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FOUNDATION YEARS JOURNAL 2018

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Foundation years journal

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THE PSYCHOSOCIAL HISTORY & SAFEGUARDING ADOLESCENTS: WHAT ALL FOUNDATION DOCTORS SHOULD KNOW

J Salkind, R Viner

Abstract

All foundation doctors will be involved in the initial assessment and ongoing care of adolescents, in paediatric rotations but also within General Practice, the Emergency Department and on surgical rotations. It is therefore important that all foundation doctors feel comfortable communicating with this important patient group, whose needs are often not met by either paediatric or adult services. There is an evolving understanding of the complexities of adolescent safeguarding, and the importance of a comprehensive psychosocial history to explore potential risks and take appropriate action.

Here, we give advice on building rapport, clarity around confidentiality and taking a thorough history via the HEEADSSS framework: Home, Education/Employment, Eating, Activities, Drugs, Sexuality, Suicidal ideation and Safety, which can be easily used by any foundation doctor within a hospital or primary care setting (1). We also offer practical advice on how, as a junior doctor, you can escalate potential safeguarding concerns through the appropriate channels.

Introduction

We spend a lot of time in clinical practice ensuring that we don't treat children as merely small adults, yet we tend to fall into this trap with adolescents, treating them as either big children or small adults. Adolescence is defined by the World Health Organisation as the age bracket from 10 to 19 years, and is a time of significant biological, psychological and social change. This important developmental period can be disrupted by illness or psychosocial stressors, including neglect and physical, emotional or sexual abuse (2,3).

In this age bracket, there is the potential for the emotional and behavioural changes secondary to serious maltreatment to be misinterpreted by professionals as normal 'teenage angst' (4). Safeguarding adolescents is also more complex than safeguarding younger children due to the broader range of potential risks encountered by adolescents, many of which are now online (2,5) and because the views of the young person need to be taken into account as well as those of their parents and professionals.

For this reason, whenever you encounter an adolescent in clinical practice, it is important to take a broad psychosocial history. One way of systematically inquiring about the multiple domains across which risks may occur, is to use the mnemonic HEEADSSS: Home, Education/Employment, Eating, Activities, Drugs, Sexuality, Suicidal ideation and Safety, (1).

The HEEADSSS framework proceeds from theoretically more neutral areas (home and education) to those that are more sensitive and difficult to talk about (substance use and sex). Junior doctors are well placed to take this history, and may uncover risks not uncovered previously, as shown in one Australian study which used an adaptation of the HEEADSSS framework to opportunistically screen adolescent surgical inpatients (6).

While the psychosocial history is of the utmost importance in a young person presenting with psychological distress or self harm, this study highlighted the benefit of making time to discuss these issues with all young people, even when they present with a wholly physical complaint such as appendicitis. The structure should be used flexibly, allowing for discussion to flow naturally, directed by the young person. This will help to build rapport and avoid the interview feeling like an interrogation.

The adolescent is your patient; speak to them alone as well as with their parents

For younger children, we naturally communicate and make treatment decisions with the parents, although we increasingly recognise the importance of hearing the child's voice in treatment decisions. For adolescents, it is important to remember that the young person is your patient and that they are somewhere on a journey between child-like dependence and full adult autonomy. Neither treating them like a child nor like an adult is providing good care. Whilst this sounds complex, in practice there are some simple guidelines to follow.

When meeting young people, is important to first address them directly before moving on to introduce yourselves to their parent or caregiver. Make the young person the centre of attention in the consultation or during bedside rounds, making it clear that you understand them as the key player rather than merely as a dependent child. This said, parents are a central part of young people's lives, particularly when they are sick, and need to be a part of the history-taking and (usually) part of decision-making. Parental involvement will depend on the age of the adolescent and their capacity (see later).

Seeing adolescents by themselves, as well as with their parents is important and good practice in most (but not all) situations. This should depend on the maturity of the adolescent but is good practice to consider for all young people over 12-13 years (and some below this!). It is the psychosocial history which is particularly useful to take from the young person on their own (1).

This allows them to start to take responsibility for their own health and gain confidence in talking to professionals, but it also means that they may be able to be more open about potentially sensitive topics. This is important for all young people who may be embarrassed to talk freely in front of their parent or caregiver. In rare cases,that parent or caregiver may be the perpetrator of abuse whose presence will prevent disclosure.

A helpful phrase is "I usually speak to young people by themselves as well as seeing them together with their parents. This is a good thing because it allows young people to start being responsible for their own health and also gives them a chance to talk about anything which is private or difficult to talk about." Explain to the parent or caregiver that you will be able to have a joint consultation before and/or afterwards and escort them to a place to wait, which is sufficiently far enough away that there is no chance of them overhearing your consultation.

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Establishing confidentiality

Confidentiality is key to maintaining trust in the medical profession and the General Medical Council is clear that 'the same duties of confidentiality apply when using, sharing or disclosing information about children and young people as about adults' (7). Like adults, young people do not have an absolute right to confidentiality and you may be compelled to break confidentiality by law or have a defence in doing so where there is overriding public interest or it is in the best interests of the young person.

The best way to maintain trust is to be open and honest with your patients about who you will share information with. For example, as a foundation doctor, you are likely to discuss every adolescent from whom you take a psychosocial history, with a more senior doctor on your team. Ask your patient to tell you if there are things that they do not want their parents to know. You could say "I will not share things with your parents or anybody outside the medical team without your permission.

The exception to this would be if I thought there was a serious risk to you or to somebody else. In that scenario, I might need to speak to somebody else but I would discuss it with you before doing so and you would be able to give me your opinion." If a young person says that they will only tell you something if you promise not to tell anybody else, you should not make a promise that you may have to break.

Opening the discussion and your use of language

Communication skills are key for any consultation, but perhaps more so for consultations with adolescents than at any other time of life. Even within a short consultation, it is important to take the time to build rapport and to acknowledge that some topics might be difficult for the young person to talk about. For example, you could say "I am going to ask you about many different aspects of your life – some of these might feel quite personal so please let me know if there are things that you don't feel comfortable discussing and we can explore why that might be."

As you ask about the different domains, try to ask open questions, for example, "tell me a bit about your school", rather than "is school going okay?", the latter allowing them to give you a one word reply. It can be helpful to reflect back to them things that they have told you, giving them time to expand on things that they have said. When your time is limited, it is easy to focus only on negative factors and potential risks. It is important to actively identify positives and protective factors, for example by asking, "what is something good that your friends might say about you?" or "what things about yourself are you proud of?"

It can be helpful to mirror the language used by the young person, particularly when they are describing their own identity. While you want to be approachable, you should avoid using overly informal language to maintain a clear professional boundary. In other words, do not try to be cool or down with the kids! Young people tell us clearly that they want to value you as their doctor, not their friend.

If the young person uses a term that you do not understand, you can ask them to clarify what they meant. Non-verbal communication is also important – assess the young person's posture, eye contact and how well they engage with you, and document this in the notes.

Home

A useful place to start is establishing where the young person lives, who they live with and what the relationships are like within their family. Changes at home such as parental unemployment, divorce or bereavement may have a big impact on a young person's life. It is important not to make assumptions, as for some young people, this will be a difficult topic to talk about, especially if they have experienced abuse or neglect at home, or witnessed domestic violence.

In a UK study in 2009, 18.6% of 11-17 year olds had experienced severe maltreatment(8). As well as what the young person says (or does not say), observing their interactions with their parent or caregiver may indicate if the relationship is problematic. It can be very difficult for adolescents to reveal abuse: barriers to doing so include feelings of guilt and fear of the consequences of disclosure (9). A young person may not be living with their family: you may come across 'looked after children' who are in a foster care placement, or child refugees.

Education/employment

It is important to identify what the young person does day to day in terms of education and/or employment. For those at school and college, you could explore whether they feel able to talk to teachers, whether they have friends at school, and ask directly whether they have experienced bullying, either as the victim or the perpetrator.

Bullying has long-term effects on self-esteem and, can lead to school avoidance, self harm and even death by suicide. Remember that a lot of bullying occurs online and that minority groups are at higher risk, for example, 45% of lesbian, gay, bisexual and transgender (LGBT+) pupils in UK schools report having been bullied(10).

Eating

Adolescence is the time where patterns of eating and exercise can be set for adult life(3). Both obesity and eating disorders have extensive physical and psychological consequences. This can be a difficult topic for adolescents to talk about. One way to bring it up is to acknowledge this is a common problem, for example, "we know that during puberty, when teenagers bodies are changing, it can be a time when they think more about their weight and body shape. Is that something you tend to do?" Where a problem is identified, explore it in more detail, praising positive behaviour you identify, and being honest if you think there is a problem.

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Activities

Asking young people open questions such as "what kind of things do you do for fun?" or "how do you spend your time outside of school?", is a good way to build rapport and identify protective factors. If they are anhedonic and unable to identify anything that they enjoy, this may be an indicator of a depressive illness, although be careful not to confuse this with the typical adolescent tendency to find most things 'boring'. This point in the interview is an appropriate time to ask about smart phone and social media use – explore which platforms/applications(apps) they are using, and how much time they spend per day on electronic devices.

There are positive aspects to social media, for example, the possibility of online education, the oportunitiy to make new friends, and to find online supportive communities. For example, many LGBT+ young people seek help and find support online (11). However, the risks online are manifold and include cyberbullying, exposure to explicit sexual contact, and the possibility of grooming, child sexual exploitation and radicalisation (12).

What's new on this topic?

- Smart phones and social media have a huge implication in adolescent safeguarding due to cyberbullying, sexting. child sexual exploitation and grooming.
- Lesbian, gay, bisexual and transgender (LGBT+) young people are an emerging focus of safeguarding research due to high rates of self harm, suicide, homelessness and conversion therapy.
- With an increasing focus on the need for a supportive transition between paediatric and adult services, this will also need to incorporate safeguarding young adults over the age of 18.

Drugs

Young people may be very anxious about revealing illegal drug or alcohol use to you. A way to put them at ease is to use a third person approach, asking whether any of their friends or family use drugs or alcohol, and then opening the conversation up to find out what they have tried. If they have used any substances, you should ask about how often, how much and how it made them feel. It is important to discuss peer pressure which may be relevant and also to find out the source of the substance and how they have paid for it. There may be an underlying problem, for example, neglect and abuse cause an increase in the rates of smoking, alcohol use and drug use(3, 13).

Sexuality

Talking about sex and relationships may be the most difficult part of the interview for some adolescents (and for many doctors!) It is useful to acknowledge this, for example "I'm going to ask you some more personal questions now. Some young people find these questions difficult or embarrassing to talk about but it's important to do so to make sure you have all the information that you need".

It is best to ask open questions, which do not make assumptions about the young person's sexual orientation, for example asking "have you been in a relationship with anyone" or "have you had sex before" rather than automatically asking a girl "do you have a boyfriend?" Ask directly what they know about safer sex, exploring risks of sexually transmitted infections and unplanned pregnancy as appropriate (14). Over a third of girls in UK secondary schools have experienced sexual harassment (15) therefore it is key to discuss the concept of valid consent and whether the young person has felt pressured or forced into sexual activity.

Here is a good place to ask about 'sexting' (the act of sending photos or videos with explicit content via text message or social media) - this is illegal in the UK for under 18s and has many potential repercussions of the young person(2). Be wary of relationships where one partner is older, in a position of power, or there is any indication of coercion.

In the course of the interview, a young person may reveal to you that they are LGBT+. For many young people, their sexual orientation and/or gender identity will be an aspect of their identity that they are proud of, however 'coming out' as LGB or as transgender, can be a dangerous process with risks of family rejection, homelessness, bullying and exposure to the dangerous practice of 'conversion therapy' (10,11,16).

Suicidal ideation

During the course of your interview, you will be able to make an assessment about the young person's mental state, based on what they say and whether their affect is reactive or, for example, flat, indicating depression. It is important to ask them to describe how their mood has been, and to specifically ask about self harm and suicide. It can be helpful to generalise, for example "sometimes when people are having a difficult time or feeling very low, they might have thoughts of hurting themselves or of killing themselves – have you ever had thoughts like that?"

Many clinicians are worried about asking about suicide, but but there is very good evidence that asking about self-harm does not increase the risk but can be preventive. A particularly high risk group is the LGBT+ group: in a survey of over 3000 young people in the UK, of the LGB cohort, 61% reported self-harm and 22% had made a suicide attempt. Of the transgender cohort, 84% reported self-harm and 45% had made a suicide attempt(10).

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Safety

By the end of the interview, you will have covered many of the potential threats to a young person's safety but it is important to directly ask them whether there are places or people with whom they do not feel safe. Specific threats to safety include neglect and abuse, gang violence, grooming and child sexual exploitation, forced marriage, 'honour' based violence, female genital mutilation, human trafficking and modern slavery, and radicalisation (2,5). Many of these are crimes and will require escalation to the police.

Finishing the interview and your next steps

You should conclude your interview by summarising what has been discussed and giving the young person a chance to add anything and ask questions. Discuss what kind of support the young person thinks will be helpful. If you have identified a safeguarding concern, explain your next steps, in terms of who you will speak to.

As a foundation doctor, your main roles are to take a comprehensive history, to document clearly and to escalate rapidly to a senior, who should advise on the next steps which may require involvement of the local child protection team, social services or the police.

As a foundation doctor, you are often the most present doctor on the ward and will therefore get to know your patients and their families well – you may be uniquely placed to identify and escalate problems. When acting as 'scribe' for a registrar or consultant, document verbatim what is said by the patient, their family and the healthcare professional, as these notes may form the basis of later court statements.

When should I call my registrar?

- A young person who does not feel safe at home
- Severe bullying
- Concerning sexual activity e.g. with a partner who is much older/where there is a power imbalance
- Drug and alcohol use
- Mental health conditions, self harm or suicidal ideation
- Any other child protection concerns

Conclusion

Working with adolescents is often considered challenging, but they can be amongst the most rewarding patients to work with. Adolescent safeguarding is the responsibility of all professionals who work with them. Taking a comprehensive psychosocial history can help you to identify potential risks and take appropriate action, in line with the law and the General Medical Council guidance.

Best of five questions

1. A 12 year old girl tells you she has a 14 year old boyfriend who goes to her local drama club. She feels safe with him and they have had sex using condoms which was her choice. What are your next steps:

A) Do nothing – the two are close in age and she has clearly given valid consent to have sex which they are doing safely.

B) Assess her to see whether she is Gillick competent to be consenting to have sex.

C) Supply her with condoms and advise her to seek sexual health testing.

D) Try to persuade her to tell her parents as you are worried that she is too young to make this decision, but accept it is her decision if she does not want to.

E) Escalate immediately to your consultant and child protection team.

2. A sixteen year old girl tells you that since she came out as a lesbian, her parents have been taking her to therapy to 'convert' her to being straight. They have strong religious beliefs that homosexuality is wrong. Which of the following should you do?

A) Respect her parents right to religious freedom and do not pass judgement on their actions.

B) Explain to her that the law in the UK allows 16 and 17 year olds to consent to treatment but not to decline treatment which their parents have consented to, and therefore she must wait until she is 18 to stop going to conversion therapy.

C) Call her parents and explain to them that they need to respect her sexual orientation as it is a normal variant.

D) Reassure her that what she has told you is confidential and give her the details of an online support group for LGBT+ young people.

E) Escalate immediately to your consultant and child protection team.

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Answers

1. Answer - E

Sex under the age of thirteen is statutory rape and a child under this age is not legally capable of giving consent to have sex. This will need to be urgently discussed with your consultant and child protection team, and escalated to the police.

2. Answer – E.

All major psychotherapy and counselling bodies, along with NHS England and the British Medical Association have condemned conversion therapy due to its harmful effects on LGBT+ people's mental health (17).

This represents a major safeguarding concern and needs to be escalated appropriately. While part of this is likely to involve a discussion with the young person's parents, this is not something you should attempt yourself as a foundation doctor, and it would not be appropriate to do this over the phone

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CHILDHOOD OBESITY

MR Begum, AR Moodambail

Abstract

In developed countries such as the UK and USA, obesity in a paediatric population has been a public health issue since the late 1980s. (1) The incidence of this condition has further increased significantly in this last decade and it is now a public health priority. Children who suffer from obesity experience difficulties in many aspects of their lives including their physical, mental and emotional wellbeing.

Long term complications of untreated paediatric obesity include diabetes, asthma and cardiovascular disease amongst many others. (2) This has significant health implications for the patient but equally substantial financial strains on a pressurised healthcare system such as the NHS. Reports have estimated that this condition and its sequalae could cost the NHS £4.2 billion per year. (3)

For these reasons, the assessment of childhood obesity needs to be effective at correctly identifying the condition, requesting appropriate investigations and referrals and delivering evidence-based holistic case. This article focuses on a local audit which investigates the assessment of childhood obesity in a paediatric hospital. The aim was to identify areas for improvement in assessment in a bid to improve clinical practice and child health outcomes.

Background

Between 2016-17, 10% of boys and 9.2% of girls in Reception (aged 4-5 years) and 21.8% of boys and 18.1% of girls in Year 6 (aged 10-11 years) were classified as obese in England according to the National Child Measurement Programme. (4) In London, 10.3% of boys and girls (aged 4-5 years) and 23.6% of boys and girls (aged 10-11 years) were classified as obese. (4) London, in comparison to the rest of England, had the highest rates of obesity in children in Year 6 (aged 10-11 years). (4)

This exhibits the relevance of appropriate assessment of paediatric patients to allow for timely management prior to the development of further complications. Based upon OSCA (Obesity Services for Children and Adolescence) guidelines, an audit was carried out in a General Paediatric Clinic at a District General Hospital in the UK to evaluate the effectiveness of childhood obesity assessment.

Method

The records of 72 children aged one to 16 years between the dates of February 2010 and October 2014 were retrospectively analysed according to a pro forma created using the OSCA guidelines with a scoring system formed to observe effectiveness. The total possible score that could be obtained was 30, with the essential score, deemed as effective assessment, as 23. The scoring category used have been summarised in Table 1 below.

Scoring Category	Scores
Total possible score	30
Essential score (Effective assessment)	23
Good assessment	≥15
Ineffective assessment	≤14

Table 1: Showing the scoring categories used assist defining results.

The pro forma included aspects assessing clinical history, clinical examination, basic investigations and any special investigations carried out. Furthermore, co-morbidities obtained were noted throughout in addition to assessment by a dietician, paediatrician and physical activity programme referral.

Results

39 patients (54% of total patients) obtained a score of ≥15, deemed as good assessment, with the remaining 33 patients (46% of total patients) scoring ≤14, deemed as ineffective assessment. The highest score obtained was 20 out of 30, achieved by two patients. The distribution of data is shown below in Figure 1.

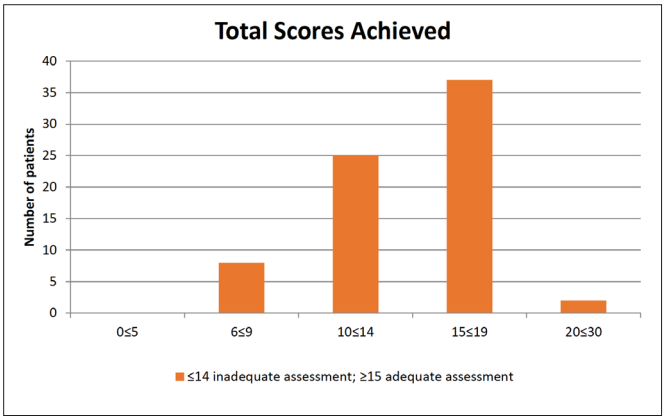


Figure 1: Depicting the trend of the data collection quality.

Common co-morbidities identified include Vitamin D deficiency (40% of patients), polycystic ovarian syndrome ((PCOS) 36% of female patients aged between 11 and 16 years) and Type 2 diabetes or impaired glucose tolerance (7% of patients).

Other co-morbidities were also identified; neurodevelopmental/learning/behavioural difficulties (23.6%), hypertensive or obtained abnormal blood pressure readings (5.6%), psychologically distressed due to weight-related bullying at school (5.6%) and dyslipidaemia (2.7%). Figure 2 summarises these findings on a graph.

CHILDHOOD OBESITY

MR Begum, AR Moodambail

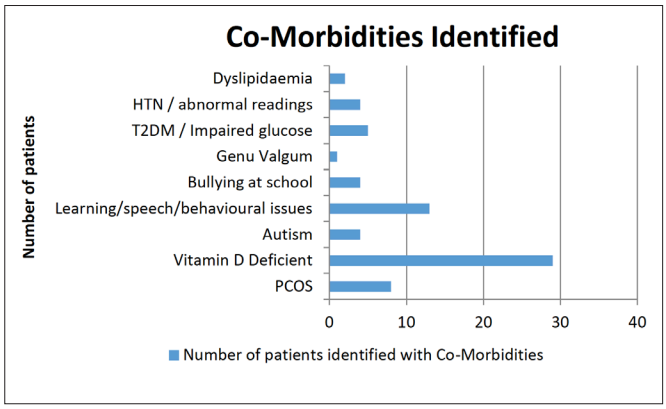


Figure 2: Showing the number of patients identified with co-morbidities.

Overall, 46% of patients were inadequately assessed, leading to possible non-identification of early stages of co-morbidities and a further delay in intervention. It also suggests the importance of checking Vitamin D levels in all overweight children, with possible consideration of sex hormone profile testing to exclude PCOS in overweight pre-pubertal girls.

This audit, although based on one hospital in the UK, highlights the need for adequate assessment of childhood obesity. To further analyse this on a local basis, two focus groups were established to obtain the views of community and hospital clinicians regarding adequate childhood obesity assessment. One focus group included clinicians from Newham Clinical Commissioning Group (CCG) whilst the second focus group included paediatricians, dieticians and physical activity specialists from Newham University Hospital.

Outcomes of both focus groups identified that:

1. Current obesity assessment were inadequate in all types of care for children and young people.

2. Creation of a practical pro forma for assessment of obesity in primary and secondary care.

3. The pro forma should reflect relevant national guidelines.

As a result, a set of evidence-based clinical orientated pro formas were created to better assess obesity in both primary and secondary care, shown in Appendix 1 and 2. The guidelines used to develop the pro forma comprised of the following:

1. NICE guideline CG43 aims to increase the effectiveness of interventions to prevent obesity whilst improving the care provided to patients at risk of being overweight and obese. (5)

2. NICE guideline PH47 aims to provide recommendations for local authorities in the community on lifestyle weight management for those under 18 years of age who are identified as overweight or obese. (6)

3. NICE guideline CG189 provides evidence-based advice on the identification, assessment and management of obesity. (7)

4. OSCA guidelines provide a guidance on assessment for childhood obesity in secondary and tertiary settings, including the referral criteria, examinations and investigations to consider in secondary care and management. (8)

5. Map of Medicine Pathway provides steps for initial assessment in primary and secondary care. (9)

6. Chervin Pediatric sleep questionnaire provides more in-depth history taking for symptoms of obstructive sleep apnoea, a known association of obesity. (10)

Both primary and secondary care pro formas include some overlapping in history taking areas, with the secondary care pro forma offering a more thorough assessment of obesity in children and young people and the inclusion of all possible differential causes of obesity. Additionally, the inclusion of a sleep questionnaire in the secondary care pro forma allows a more effective assessment of possible symptoms of obstructive sleep apnoea, which was found to be ineffective in the audit.

Provisional pathways have also been created to allow clinicians and health professionals to be aware of the next steps in patient management. A separate pathway has been created for primary and secondary care shown in Appendix 3 and 4.

The pro forma in both care settings can be adjusted to suit the needs of different areas in the UK to tackle the issue of obesity. To improve the assessment of obesity, our recommendation is to use the pro formas provided in the Appendix as the next steps forward for both primary and secondary care followed by assessment of whether obesity assessment is more effective in the UK.

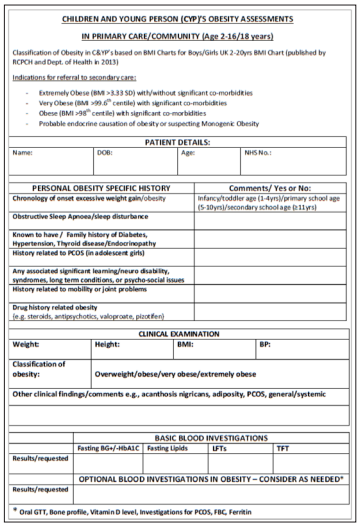
Conclusion

We suggest use of an obesity assessment pro forma in clinics to improve assessments in primary and secondary care. The audit study and the focus group suggests a pragmatic approach to check Vitamin D levels in all overweight children, and sex hormone profile to exclude PCOS in obese pubertal girls.

The evaluation highlights the lack of customised physical activity programmes; some children will benefit from psychological support while dealing with their weight management. These newly-created pro formas have been implemented in the Newham area in London, but can easily be manipulated and tailored to suit other units in the UK. Additional re-auditing will be required in 2019 to assess the outcome of the use of these pro formas to continue to assess and improve clinical care.

CHILDHOOD OBESITY

MR Begum, AR Moodambail



CHILDREN AND YOUNG PERSON (CYP)'S OBESITY ASSESSMENTS

IN PRIMARY CARE/COMMUNITY (Age 2-16/18 years)

Classification of Obesity in C&YP's based on BMI Charts for Boys/Girls UK 2-20yrs BMI Chart (published by RCPCH and Dept. of Health in 2013)

Indications for referral to secondary care:

- Extremely Obese (BMI >3.33 SD) with/without significant co-morbidities
- Very Obese (BMI >99.6th centile) with significant co-morbidities
- Obese (BMI >98th centile) with significant co-morbidities
- Probable endocrine causation of obesity or suspecting Monogenic Obesity

PATIENT DETAILS:

Name:	DOB:	Age:	NHS No.:

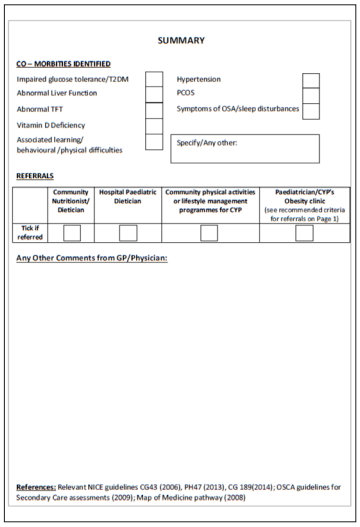
PERSONAL OBESITY SPECIFIC HISTORY	Comments/ Yes or No:
Chronology of onset excessive weight gain/obesity	Infancy/toddler age (1-4yrs)/primary school age (5-10yrs)/secondary school age (≥11yrs)
Obstructive Sleep Apnoea/sleep disturbance	
Known to have / Family history of Diabetes, Hypertension, Thyroid disease/Endocrinopathy History related to PCOS (in adolescent girls)	
Any associated significant learning/neuro disability, syndromes, long term conditions, or psycho-social issues	
History related to mobility or joint problems	
Drug history related obesity (e.g. steroids, antipsychotics, valoproate, pizotifen)	

CLINICAL EXAMINATION

Weight:	Height:	BMI:	BP:
Classification of obesity:	**Overweight/obese/very obese/extremely obese**		
Other clinical findings/comments e.g., acanthosis nigricans, adiposity, PCOS, general/systemic			

	BASIC BLOOD INVESTIGATIONS			
	Fasting BG+/-HbA1C	**Fasting Lipids**	**LFTs**	**TFT**
Results/requested				
	OPTIONAL BLOOD INVESTIGATIONS IN OBESITY – CONSIDER AS NEEDED*			
Results/requested				

* **Oral GTT, Bone profile, Vitamin D level, Investigations for PCOS, FBC, Ferritin**



SUMMARY

CO – MORBITIES IDENTIFIED

Impaired glucose tolerance/T2DM	☐	Hypertension	☐
Abnormal Liver Function	☐	PCOS	☐
Abnormal TFT	☐	Symptoms of OSA/sleep disturbances	☐
Vitamin D Deficiency	☐		
Associated learning/ behavioural /physical difficulties	☐	Specify/Any other:	

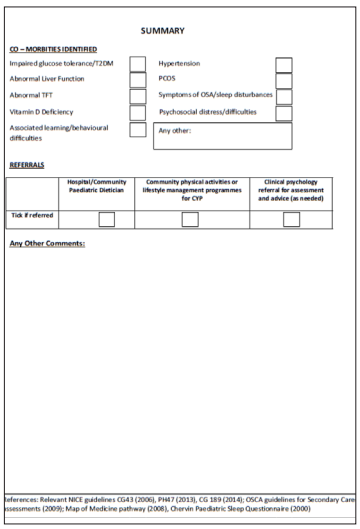
REFERRALS

	Community Nutritionist/ Dietician	Hospital Paediatric Dietician	Community physical activities or lifestyle management programmes for CYP	Paediatrician/CYP's Obesity clinic (see recommended criteria for referrals on Page 1)
Tick if referred	☐	☐	☐	☐

Any Other Comments from GP/Physician:

References: Relevant NICE guidelines CG43 (2006), PH47 (2013), CG 189(2014); OSCA guidelines for Secondary Care assessments (2009); Map of Medicine pathway (2008)

Appendix 1: Primary Care Pro-forma.



SUMMARY

CO – MORBITIES IDENTIFIED

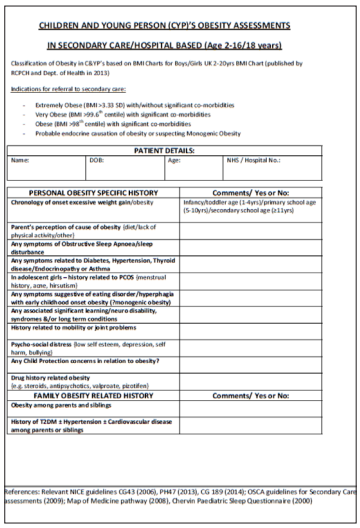
Impaired glucose tolerance/T2DM	☐	Hypertension	☐
Abnormal Liver Function	☐	PCOS	☐
Abnormal TFT	☐	Symptoms of OSA/sleep disturbances	☐
Vitamin D Deficiency	☐	Psychosocial distress/difficulties	☐
Associated learning/behavioural difficulties	☐	Any other:	

REFERRALS

	Hospital/Community Paediatric Dietician	Community physical activities or lifestyle management programmes for CYP	Clinical psychology referral for assessment and advice (as needed)
Tick if referred	☐	☐	☐

Any Other Comments:

References: Relevant NICE guidelines CG43 (2006), PH47 (2013), CG 189 (2014); OSCA guidelines for Secondary Care assessments (2009); Map of Medicine pathway (2008), Chervin Paediatric Sleep Questionnaire (2000)



CHILDREN AND YOUNG PERSON (CYP)'S OBESITY ASSESSMENTS

IN SECONDARY CARE/HOSPITAL BASED (Age 2-16/18 years)

Classification of Obesity in C&YP's based on BMI Charts for Boys/Girls UK 2-20yrs BMI Chart (published by RCPCH and Dept. of Health in 2013)

Indications for referral to secondary care:

- Extremely Obese (BMI >3.33 SD) with/without significant co-morbidities
- Very Obese (BMI >99.6th centile) with significant co-morbidities
- Obese (BMI >98th centile) with significant co-morbidities
- Probable endocrine causation of obesity or suspecting Monogenic Obesity

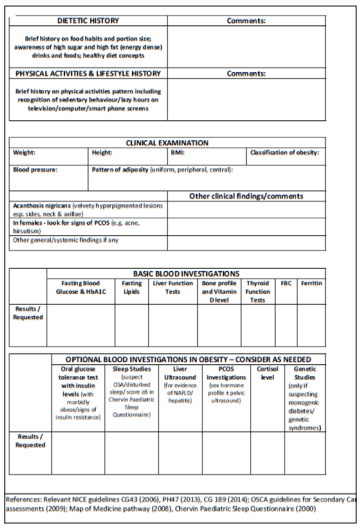
PATIENT DETAILS:

Name:	DOB:	Age:	NHS / Hospital No.:

PERSONAL OBESITY SPECIFIC HISTORY	Comments/ Yes or No:
Chronology of onset excessive weight gain/obesity	Infancy/toddler age (1-4yrs)/primary school age (5-10yrs)/secondary school age (≥11yrs)
Parent's perception of cause of obesity (diet/lack of physical activity/other)	
Any symptoms of Obstructive Sleep Apnoea/sleep disturbance	
Any symptoms related to Diabetes, Hypertension, Thyroid disease/Endocrinopathy or Asthma	
In adolescent girls – history related to PCOS (menstrual history, acne, hirsutism)	
Any symptoms suggestive of eating disorder/hyperphagia with early childhood onset obesity (?monogenic obesity)	
Any associated significant learning/neuro disability, syndromes &/or long term conditions	
History related to mobility or joint problems	
Psycho-social distress (low self esteem, depression, self harm, bullying)	
Any Child Protection concerns in relation to obesity?	
Drug history related obesity (e.g. steroids, antipsychotics, valproate, pizotifen)	
FAMILY OBESITY RELATED HISTORY	**Comments/ Yes or No:**
Obesity among parents and siblings	
History of T2DM ± Hypertension ± Cardiovascular disease among parents or siblings	

References: Relevant NICE guidelines CG43 (2006), PH47 (2013), CG 189 (2014); OSCA guidelines for Secondary Care assessments (2009); Map of Medicine pathway (2008), Chervin Paediatric Sleep Questionnaire (2000)

Appendix 2: Secondary Care Pro-forma.



DIETETIC HISTORY	Comments:
Brief history on food habits and portion size; awareness of high sugar and high fat (energy dense) drinks and food; healthy diet concepts	
PHYSICAL ACTIVITIES & LIFESTYLE HISTORY	**Comments:**
Brief history on physical activities pattern including recognition of sedentary behaviour/lazy hours on television/computer/smart phone screens	

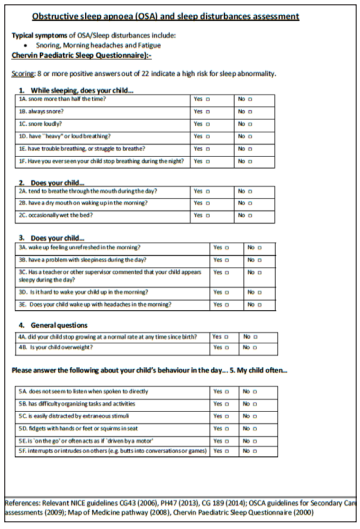
CLINICAL EXAMINATION

Weight:	Height:	BMI:	Classification of obesity:
Blood pressure:	**Pattern of adiposity** (uniform, peripheral, central):		
		Other clinical findings/comments	
Acanthosis nigricans (velvety hyperpigmented lesions esp. sides, neck & axillae)			
In females - look for signs of PCOS (e.g. acne, hirsutism)			
Other general/systemic findings if any			

	BASIC BLOOD INVESTIGATIONS						
	Fasting Blood Glucose & HbA1C	**Fasting Lipids**	**Liver Function Tests**	**Bone profile and Vitamin D level**	**Thyroid Function Tests**	**FBC**	**Ferritin**
Results / Requested							

	OPTIONAL BLOOD INVESTIGATIONS IN OBESITY – CONSIDER AS NEEDED					
	Oral glucose tolerance test with insulin levels (with morbidly obese/signs of insulin resistance)	**Sleep Studies** (suspect OSA/disturbed sleep/ score ≥8 in Chervin Paediatric Sleep Questionnaire)	**Liver Ultrasound** (for evidence of NAFLD/ hepatitis)	**PCOS Investigations** (sex hormone profile ± pelvic ultrasound)	**Cortisol level**	**Genetic Studies** (only if suspecting monogenic diabetes/ genetic syndromes)
Results / Requested						

References: Relevant NICE guidelines CG43 (2006), PH47 (2013), CG 189 (2014); OSCA guidelines for Secondary Care assessments (2009); Map of Medicine pathway (2008), Chervin Paediatric Sleep Questionnaire (2000)



Obstructive sleep apnoea (OSA) and sleep disturbances assessment

Typical symptoms of OSA/Sleep disturbances include:

- Snoring, Morning headaches and Fatigue

Chervin Paediatric Sleep Questionnaire):-

Scoring: 8 or more positive answers out of 22 indicate a high risk for sleep abnormality.

1. While sleeping, does your child...		
1A. snore more than half the time?	Yes ☐	No ☐
1B. always snore?	Yes ☐	No ☐
1C. snore loudly?	Yes ☐	No ☐
1D. have "heavy" or loud breathing?	Yes ☐	No ☐
1E. have trouble breathing, or struggle to breathe?	Yes ☐	No ☐
1F. Have you ever seen your child stop breathing during the night?	Yes ☐	No ☐

2. Does your child...		
2A. tend to breathe through the mouth during the day?	Yes ☐	No ☐
2B. have a dry mouth on waking up in the morning?	Yes ☐	No ☐
2C. occasionally wet the bed?	Yes ☐	No ☐

3. Does your child...		
3A. wake up feeling unrefreshed in the morning?	Yes ☐	No ☐
3B. have a problem with sleepiness during the day?	Yes ☐	No ☐
3C. Has a teacher or other supervisor commented that your child appears sleepy during the day?	Yes ☐	No ☐
3D. Is it hard to wake your child up in the morning?	Yes ☐	No ☐
3E. Does your child wake up with headaches in the morning?	Yes ☐	No ☐

4. General questions		
4A. did your child stop growing at a normal rate at any time since birth?	Yes ☐	No ☐
4B. Is your child overweight?	Yes ☐	No ☐

Please answer the following about your child's behaviour in the day... 5. My child often...

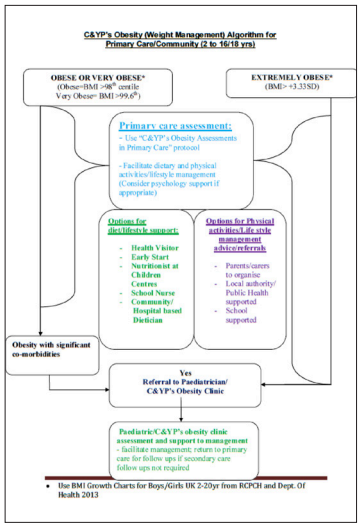
5A. does not seem to listen when spoken to directly	Yes ☐	No ☐
5B. has difficulty organizing tasks and activities	Yes ☐	No ☐
5C. is easily distracted by extraneous stimuli	Yes ☐	No ☐
5D. fidgets with hands or feet or squirms in seat	Yes ☐	No ☐
5E. is 'on the go' or often acts as if 'driven by a motor'	Yes ☐	No ☐
5F. interrupts or intrudes on others (e.g. butts into conversations or games)	Yes ☐	No ☐

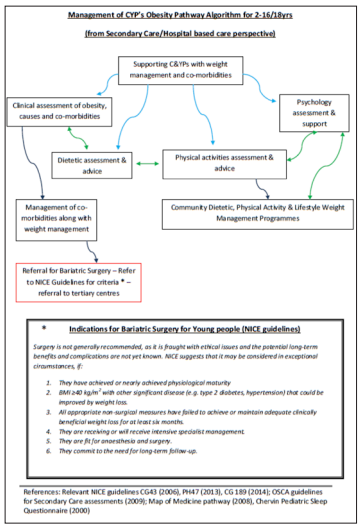
References: Relevant NICE guidelines CG43 (2006), PH47 (2013), CG 189 (2014); OSCA guidelines for Secondary Care assessments (2009); Map of Medicine pathway (2008), Chervin Paediatric Sleep Questionnaire (2000)

Appendix 3: Primary Care Pathway.

CHILDHOOD OBESITY

MR Begum, AR Moodambail





Appendix 4: Secondary Care Pathway.

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DON'T MISS A CLEFT PALATE!

RS Pryce, H McElroy

Abstract

A cleft of the palate and/or lip is one of the commonest birth defects observed in the UK. Whilst recognising a cleft lip is relatively straightforward, detecting a cleft palate requires a thorough visual examination of the palate. Poor practice can result in a cleft palate being missed with significant impact on baby and family. Prompt detection is essential to ensure appropriate management and subsequent treatment.

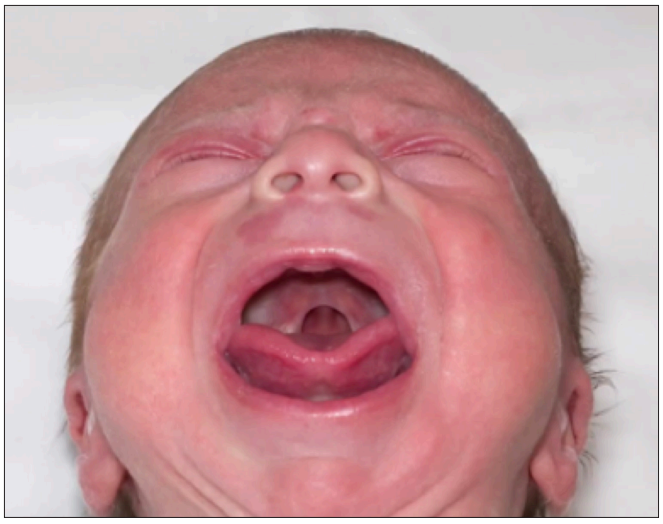

Figure 1: Baby with cleft palate.

Case study 1

Billy Jones was born at 38 weeks by normal vaginal delivery. Antenatal scans were normal. He was on the routine baby check list for the paediatric SHO who had just rotated into the speciality. She had been shown how to do a baby check as part of the induction. His mother was concerned that he seemed to feed for over an hour and made a clicking noise whilst trying to suck. During the examination, she assessed Billy's suck and felt the palate, which she thought was normal.

He gave a cry during assessment of his moro reflex and she saw his open mouth and tongue. She was unable to see the back of the throat but because it felt normal, she reassured parents and discharged Billy home. Billy was referred to the paediatric assessment unit 3 days later due to poor feeding and 12% weight loss.

During the examination, he cried and the on call SHO thought his palate looked abnormal. She examined his mouth using a tongue depressor and pen torch and discovered a cleft palate. She contacted the local cleft team and Billy was seen by the clinical nurse specialist later that day.

Incidence of cleft palate

A cleft palate is a birth defect affecting the roof of the mouth. Cleft of the palate and/or lip is common, occurring in approximately 1 in 700 births; over 40% of these comprise an isolated cleft palate. A cleft palate results from failure of fusion of the segments of the palate, a complex process that occurs between 8 and 12 weeks of fetal life.

The aetiology behind dysregulation of the formation of palate is uncertain for the majority of cases but it is thought that both genetic and environmental factors play a role. (1,2) A cleft can involve the soft palate only, the posterior part of the hard palate or can be complete.

Approximately 15% of affected babies will have additional abnormalities; detecting a cleft palate should lead to a careful examination for other subtle clinical features that might suggest an underlying syndrome. (2)

Signs of a cleft palate

National standards of care issued by cleft services advocate assessment of the palate within the first 24 hours of life as these babies present with early feeding difficulties (Box 1). (3) Affected babies are at risk of choking and aspiration.

- **Nasal regurgitation of milk during feeding**
- **Poor latching with prolonged feeding**
- **Clicking sound during feeding**
- **Choking, gagging, coughing during/after feeding**
- **Frequent burping due to excessive intake of air**
- **Disorganised sucking pattern**

Box 1: Signs of a cleft palate in the newborn.

How often is a cleft palate missed?

Approximately 30% of cleft palates are missed in the first 24 hrs of life; this figure has changed little over the past 30 years. (4,5) This is despite affected babies having the newborn examination performed by a doctor or midwife, and in most cases exhibiting classic signs of a defective palate.

DON'T MISS A CLEFT PALATE!

RS Pryce, H McElroy

Why is a cleft palate missed at the newborn examination?

The delay in detection of a cleft palate is a consequence of inadequate examination. Historically it was thought that either observing the palate through elicitation of the gag reflex, or when baby cries or yawns were sufficient. If this didn't occur, direct palpation of the palate with the little finger was undertaken.

A study of this approach in 125 babies found that an incomplete visualisation of the whole palate occurred in 45%, with potential to miss a cleft.6 A wider review of clinical practice also revealed a delay in detection that was felt primarily due to the omission of visual inspection. (5,7)

In response to the persisting problem of missed diagnosis, the RCPCH (Royal College of Paediatrics and Child Health) produced a best practice guide in 2014 advising that the palate should always be visually inspected using a torch and tongue depressor. (8) This is emphasised in an e-Learning module for trainees to highlight this condition and improve early detection - test your knowledge at http://rcpch.learningpool.com. (9)

What is the impact of delayed detection?

Newborns with a cleft palate have difficulty establishing feeding with poor latching, prolonged feeding times and consequent faltering weight. This can result in unnecessary admission to hospital with the need for nasogastric feeding. The lack of a protective gag reflex poses a risk of aspiration and choking (Box 2). The incomplete fusion of the palate can disrupt Eustachian tube ventilation causing recurrent bouts of otitis media.

This can lead to persistent effusions resulting in conductive hearing loss with subsequent delay to speech and language. Hearing loss can be found in over 30% of children with cleft palates. (1) Delayed detection can lead to delays in surgery which is usually performed between 9 months to 1 year of age.

Optimal hearing and speech development is achieved when palatal integrity is restored before the 2nd year of life.10 Underlying this is the distress to parents caused by a missed diagnosis and the disruption to the family- doctor relationship.

- **Poor weight gain and failure to thrive**
- **Recurrent chest infections as a result of aspiration**
- **Recurrent otitis media and hearing loss**
- **Delayed speech and language**
- **Emotional impact to family from delayed detection**
- **Delayed surgery**
- **Litigation claims to NHS Trusts**

Box 2: Consequences of a missed cleft palate.

Visual inspection of the palate - the gold standard

The RCPCH Best Practice Guide to examination of the palate advocates a thorough visual inspection of the mouth using tongue depressor and torch to inspect the whole palate from gums to uvula. This part of the examination is best left till the end of the newborn and infant physical examination (NIPE) and it's important to have either parent or midwife to help keep baby still.

Technique

- *Always visualise the palate with a good light source (pen torch) and a tongue depressor (e.g. a wooden spatula).*

- *Explain clearly to parents that you are going to check that the roof of the mouth is normal.*

- *Lie baby flat in the cot. Ask parent or midwife to hold baby's hands crossed on the chest with one hand and the head gently with the other hand (Figure 2).*

- *Gently press the depressor down onto the tongue with one hand and use the pen torch to visualise the palate. Avoid going in too deep with the depressor as this can cause trauma to the back of the mouth and you might elicit a gag reflex.*

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Figure 2: Demonstrating the ideal holding position to inspect the palate.

· You must visualise the entire length of the palate from gums to uvula (Figure 3).

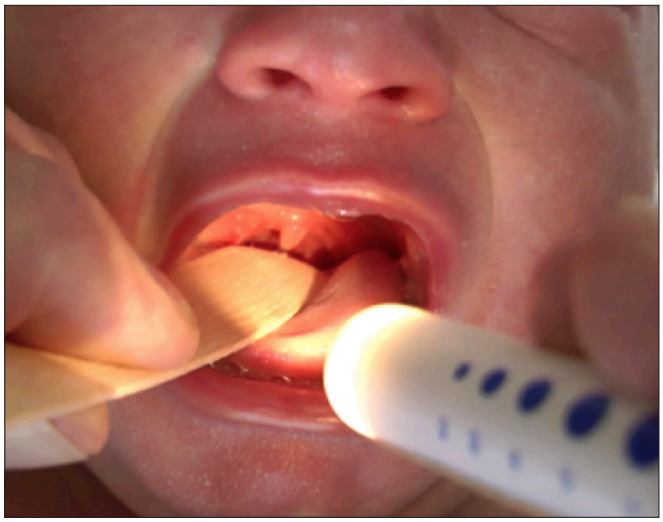

Figure 3: Correct use of tongue depressor and torch to visualise the palate (the uvula is clearly visible at the tip of the wooden spatula).

Types of cleft palate

Clefts can vary in length and width, either involving the soft palate only or extending into the hard palate. Broad clefts are easier to detect than narrow clefts and it is more difficult to view posterior clefts involving only the soft palate (Figure 4).

A bifid (split) uvula can be associated with a submucous soft palate. This is a subtype of cleft palate that is more difficult to diagnose, as there is a palpable notch at the back of the hard palate rather than a visible cleft. Refer babies with a bifid uvula to the clinical nurse specialists who will assess for the presence of a submucous cleft.

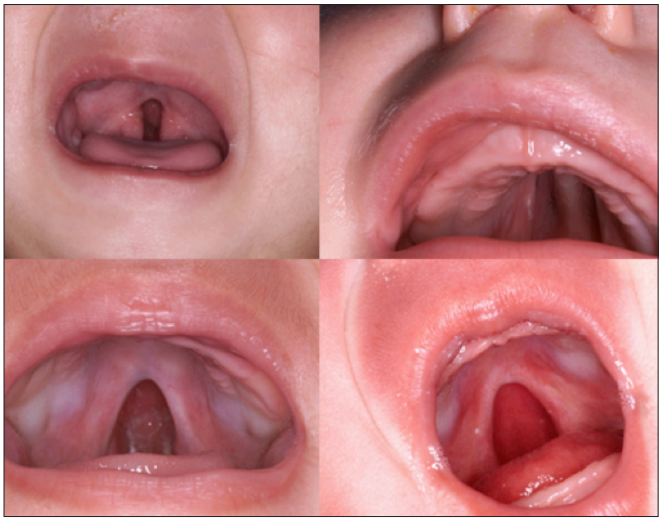

Figure 4: Types of cleft palate (clockwise from top left): A: cleft soft palate. B: wide cleft of hard palate – bony plates of the nasal septum are visible. C: narrow cleft hard palate. D: wide cleft hard palate. (Photographs courtesy of Alex Habel)

Documentation

Clearly document the findings of the NIPE in both the Child Health Record (the 'red book'), which is kept by parents, and the clinical notes as part of good medical practice. If the whole palate cannot be visually inspected at first attempt, document 'palate not seen completely' and ensure handover to your team so that a further attempt is made within 24 hours.

Referral

All babies with a cleft palate and/or lip should be referred as soon as possible to the local clinical nurse specialists for cleft services; they will assess each baby and provide specialist support to the family alongside a comprehensive cleft team that includes surgeons, orthodontists, speech and language therapists and other key health professionals. (11)

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Case study 2

The paediatric trainee is asked to perform a routine baby check on Destiny Evans, a term baby born by spontaneous vaginal delivery, currently on the postnatal ward. Destiny's antenatal scans were normal. She is now 10 hours old and her mother is keen to breastfeed but is worried because she doesn't latch on very well and seems to choke during a feed.

During the examination, Destiny is found to have a small jaw and what appears to be a large tongue. The paediatrician is unable to visualise Destiny's palate as she is very active and becomes unsettled. Her mother tries to put Destiny to the breast to calm her but she suddenly chokes and struggles to breath. This resolves after she is turned prone and patted on the back.

She is admitted to the neonatal unit for further assessment where she is found to have a cleft palate. A diagnosis of Pierre Robin sequence is made. A nasogastric tube is inserted so that Destiny can be fed whilst a referral to the local cleft nurse specialist is made.

Syndromes associated with cleft palate

Cleft lip and palate (CLP) can be considered as one of three groups: syndromic, familial or sporadic. (12) Whilst there are over 500 syndromes associated with cleft lip and palates, only 30% of cases of CLP are considered syndromic. (13) The most common anomalies associated with a cleft palate are a result of either chromosomal abnormalities (including Trisomy 13, 18 or 21) or specific gene mutations (Stickler syndrome, Van der Woude and 22 q11).

Autosomal dominant patterns of inheritance can be seen in Stickler syndrome and Van der Woude syndrome, highlighting the importance of taking a detailed family history (see Table 1). Other factors that increase the chance of having a baby with an orofacial cleft include maternal diabetes, alcohol, smoking and antiepileptic drugs (sodium valproate and topiramate). (14-17)

Pierre Robin sequence describes the clinical features of a small lower jaw (micrognathia) and a posteriorly placed tongue (glossoptosis), with potentially life threatening upper airway obstruction, and variable association with a cleft palate. (18)

It occurs in approximately 1 in 8500 births and may present with breathing difficulties before the facial features and cleft are identified. Airway management in Pierre Robin sequence can be challenging. Airway obstruction may be relieved by positioning prone or lateral, allowing the tongue to fall forward. (19) If these fail, insertion of a nasopharyngeal airway is recommended.

	Features	Aetiology	Inheritance pattern
Pierre Robin sequence	Micrognathia, glossoptosis, upper airway obstruction, cleft palate	Monogenic, chromosomal or teratogenic.	Variable
Stickler's syndrome	Pierre Robin sequence, flat facies, sensorineural deafness, myopia, glaucoma, cataracts and retinal detachment.	Mutation in collagen genes COL11A1, COL11A2 or COL2A1, COL9A1	Autosomal Dominant
22q11 (Di George)	Cleft palate, congenital heart disease, thymic hypoplasia, hypoparathyroidism and immune deficiency	Microdeletion of chromosome 22q11.2	Autosomal dominant
Van der Woude syndrome	Lower lip pits, cleft lip and/or palate, hypodontia	Mutation in 1q32	Autosomal Dominant

Table 1: Conditions commonly associated with a cleft palate.

Initial investigations

All infants with a cleft palate should have a full examination to exclude associated anomalies and distinctive features suggestive of a syndrome. The presence of a heart murmur should prompt evaluation with echocardiography. Infants with distinctive features or confirmed cardiac defects should have genetic testing with array CGH (comparative genomic hybridization). Those with Pierre Robin sequence should also be referred for ophthalmology assessment to consider Stickler's syndrome.

Conclusion

Examination of the palate of a newborn should always be performed using a tongue depressor and torch. All doctors should be able to recognise a normal palate and a cleft defect. Prompt detection with rapid referral allows appropriate support to be put in place for infant and parents. The RCPCH Compass E-learning provides an excellent online module to enforce understanding of cleft palate for health professionals.

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Multiple Choice Questions

1. You are asked to review a 2 day old baby on the postnatal ward who is struggling to breastfeed. His mother has noticed milk coming out of his nose when feeding. Which of the following would most help in making an immediate diagnosis?

a. Direct visualisation using a pen torch and tongue depressor.

b. Giving the baby some sucrose solution and allowing him to suck on your finger to assess the quality of his suck.

c. Ask the midwife to observe him feeding to assess technique

d. Ask the midwife to weigh the baby so you can see how much he has lost.

e. Apply pulse oximetry to check oxygen saturations during his next feed.

2. Which of the following signs are suggestive of a baby having a cleft palate?

a. Regurgitation of milk after feeding.

b. Gagging during feeding.

c. Difficulty winding the baby.

d. Clicking sound during feeding.

e. Disorganised sucking pattern.

3. What is the incidence of cleft palate in babies?

a. 1 in 50

b. 1 in 200

c. 1 in 400

d. 1 in 700

e. 1 in 1000

4. During 2016 in the UK and Ireland, how many babies with cleft palate had a delayed diagnosis i.e. after 24hrs age?

a. 15%

b. 25%

c. 32%

d. 50%

e. 65%

5. What is the cost to the NHS in negligence litigation claims for missed cleft palate in the last 15 yrs?

a. £90,000

b. £250,000

c. £1 million

d. £3 million

e. £15 million

Answers

1. a.

Nasal regurgitation of milk during feeding is characteristic of a cleft palate. Only direct visualisation of the palate using the correct method will give you an immediate answer.

2. b.

Gagging during feeding, d. Clicking sound during feeding and e. Disorganised sucking pattern of signs suggestive of a cleft palate. Regurgitation of milk after feeding (posseting) is common in babies.

3. d.

1 in 700. Cleft palate is one of the most common congenital craniofacial anomalies.

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4. c.

32% of babies had a missed cleft palate within the first 24 hours of life. This statistic highlights the need for ongoing education and training of all health professionals to correctly examine the palate with a tongue depressor and torch to properly visualise the whole palate from gums to uvula.

5. c.

The NHS has paid out over £1 million in claims due to missed diagnosis of cleft palate.

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HAVE THE GOALPOSTS CHANGED WITH RESPIRATORY SYNCYTIAL VIRUS?

G Wilson, P Desai

Abstract

Respiratory syncytial virus (RSV) is recognised as a common cause of bronchiolitis in children < 2 years which can be of variable severity. It accounts for 4.4 per 100 admissions of children < 6 months. In children over 2 years it usually causes a mild lower respiratory tract infection and is therefore not commonly tested for in this age group.

We present the cases of 3 children over the age of 2 years presenting to a hospital within 1 week, all requiring prolonged stays and significant respiratory support, found to be positive for RSV. These cases have led to the question of whether the virulence of RSV is changing, or whether it is the culprit in many cases of severe viral lower respiratory tract infections in older children.

Background

Respiratory syncytial virus (RSV) is an enveloped RNA virus that usually causes a mild lower respiratory tract infection in children and adults, but is typically known for causing bronchiolitis in infants which can be of variable severity. It is highly communicable and is spread by respiratory secretions through close contact with infected individuals (1).

Risk factors for more severe infection include prematurity, chronic lung disease, congenital heart disease and immunodeficiency (2). The mean incidence of hospital admissions is 4.4 per 100 admissions of children <6months old, and 0.5 per 100 of children 6 months to 5 years (using multivariate regression analysis) (3).

The National Institute for Health and Care Excellence (NICE) defines bronchiolitis as being an illness in children under 2 years, with a peak at 3-6 months (4). Given this evidence of more severe infections in children under 2 years with bronchiolitis compared with older children, RSV is rarely tested for in older children.

We present the cases of 3 children over the age of 2 years admitted to hospital within 1 week of each other in the winter of 2017, all RSV positive and requiring significant respiratory support. This evidence could suggest that the general consensus of RSV causing a mild illness in older children may be changing.

Case 1

A 5 year old girl, previously fit and well, presented to A&E after a possible febrile convulsion following a 24 hour history of fever with productive cough and increased work of breathing. Her oxygen saturations on arrival were 88% in air so she was commenced on oxygen and IV amoxicillin, prednisolone and salbutamol.

Her initial blood tests showed a CRP of 42 but were otherwise unremarkable; chest X-ray (CXR) showed basal atelectasis and right midzone patchy consolidation.

She required high flow oxygen for 8 days which was difficult to wean off despite otherwise being clinically well. Repeat CXR showed similar findings so she was switched to oral amoxicillin and oral azithromycin was added.

On day 4 of her admission, her oxygen requirement increased to 11 litres so a nasopharyngeal aspirate (NPA) was performed. This was positive for RSV and so she was moved to be isolated in the RSV positive bay. On day 9 she was weaned to nasal cannulae and then off oxygen the following day, having completed 7 days of amoxicillin and 3 days of azithromycin for possible superimposed bacterial infection.

Case 2

A 3 year old girl with VACTREL association, including tracheo-esophageal repair, complex congenital heart disease, right renal agenesis, and vertebral and rib abnormalities, was brought to A&E just a few days after the first patient. She presented with fever, increased work of breathing and a barking cough.

She also required high flow oxygen up to 18 litres/min in FiO2 85% during her admission and received IV co-amoxiclav. Her initial CRP was 106 but subsequently rose to 244 and she had increasing oxygen requirements; she was therefore switched to IV ceftriaxone and azithromycin but made little improvement.

CXR showed multiple long-standing changes, but with some right basal atelectasis. An NPA was performed on day 4 of her admission after poor response to antibiotics which also showed RSV. After 10 days, her high flow oxygen was gradually weaned off and her CRP came down to 9 so she was discharged home.

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Case 3

A 2 year old girl with congenital hypothyroidism, eczema and previous admission with viral induced wheeze presented with a 3 day history of coryza, wheeze and increased work of breathing. CXR showed hyperinflated lungs and CRP of 25.

She required 15 litres of high flow oxygen to maintain her oxygen saturations and hourly nebulisers. She also received ipratropium and IV aminophylline however she continued to have high oxygen requirements up to 25 litres.

She began to tire the following day so had a semi-elective intubation, was commenced on IV ceftriaxone and was transferred to a Paediatric Intensive Care Unit (PICU). She was also found to be positive for RSV in addition to enterovirus.

She was mechanically ventilated for 5 days and received an IV salbutamol infusion in addition to previous measures. She was discharged home self-ventilating in air 6 days following admission.

Discussion

As RSV is typically known for causing bronchiolitis in children under 2 years old, these children were not tested for RSV on presentation to hospital. They were investigated and treated as per the guidelines for lower respiratory tract infections with blood tests, blood cultures, chest X-ray, antibiotics, fluids and oxygen. This contrasts with the investigation and treatment of bronchiolitis in which none of the stated investigations are performed - a blood gas is recommended if oxygen concentration requirement exceeds 50%.

Treatment only involves oxygen support, nasogastric feeding if inadequate oral intake (50-75% of usual volume), and occasionally intravenous fluids if nasogastric feeds are not tolerated or there is evidence of impending respiratory failure (4).

It is appropriate and correct to treat a child with antibiotics presenting to hospital with the symptoms of the cases described. This is because a severe bacterial infection is not only treatable but can cause significant morbidity and even mortality if not treated appropriately and in a timely manner.

The decision to test for RSV in each case was due to failure of clinical improvement with antibiotics, which would be suggestive of a viral rather than a bacterial cause. Despite having a positive RSV NPA, each child completed the course of antibiotics as it is possible that they had a concomitant bacterial infection. Additionally, it is important to complete a course of antibiotics for prevention of drug resistance.

These cases have highlighted the ability of RSV to cause severe illness in children over the age of 2 years. This may not be considered initially when a child presents to hospital unwell. Possible explanations for this apparent epidemiological change observed in our hospital may be that, as we generally do not test older children for RSV with NPAs, we are under diagnosing cases.

The children improve with time, as most viral respiratory tract infections do with supportive measures, and improvement is incorrectly attributed to antibiotics. Alternatively, the strain of RSV during the winter observed was an especially virulent one, causing unusually severe infections in older children.

It is difficult to discriminate between these two explanations due to the rarity of testing for RSV in the over 2 year olds; the first step in trying to distinguish between them would be to test all children over the age of 2 with an NPA, in addition to blood tests, cultures and chest X-rays. This however would not necessarily be cost-effective as the tests are expensive and, as we can see from the cases, would not change our management of these children.

Of note, although RSV accounts for fewer hospital admissions in adults compared with children, inpatients with confirmed RSV are more likely to have severe illness with respiratory insufficiency with up to 16% requiring assisted ventilation. (6)

Therefore, learning points should include the consideration of testing for RSV and other respiratory viruses in critically ill patients, those requiring significant respiratory support, and those with prolonged hospital stays. Additionally, to be aware that RSV may be responsible for common presentations of lower respiratory tract infections such as viral induced wheeze in pre-school children.

Test Yourself – Best of 5

1. What investigations should be considered if a child presents with bronchiolitis and a high oxygen requirement?

a) Full blood count

b) Blood Culture

c) Blood gas

d) CXR

e) CRP

HAVE THE GOALPOSTS CHANGED WITH RESPIRATORY SYNCYTIAL VIRUS?

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2. Which of the following is NOT a risk factor for severe infection with RSV?

a) Congenital Heart Disease

b) Prematurity

c) Chronic Lung Disease

d) Immunodeficiency

e) Family history of asthma

Answers

1. Answer: C

2. Answer: E

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NEONATAL JAUNDICE: AN APPROACH FOR JUNIOR DOCTORS

T Conway, P Mallett, A Thompson

Abstract

Neonatal jaundice encompasses a spectrum of conditions characterised by the accumulation of bilirubin causing yellow discoloration to the skin, gums and sclera in a baby less than 28 days old. We present a case of a 10 day old baby with significant weight loss, vomiting and jaundice.

Jaundice is the most common condition which requires medical attention in the neonatal population [1]. We discuss the common causes of jaundice in the neonate, highlight the importance of obtaining a detailed clinical history, performing a thorough examination and provide a framework for foundation doctors to compile an appropriate list of differentials, investigations and provisional management plan. This is an important topic for all junior doctors as it is commonly encountered in both primary, secondary and tertiary level care.

Case Report

A 10-day-old girl, born at full term, presented with jaundice and 17% weight loss to her local Children's Hospital, a tertiary-level regional unit. She was brought to the Emergency Department (ED) by her mother, who reported a three-day history of vomiting after feeds.

The mother described projectile vomiting of milky fluid, with no reported bile-stained vomit. She was exclusively formula-fed, had been feeding 30-45 mls every two hours, and had been waking appropriately for feeds. She was passing stool regularly and these were described as yellow/green in appearance. Urine was yellow in colour.

Birth history revealed an uneventful pregnancy. She was born at 40 +2 weeks weighing 3100 grams. There were no risk factors for sepsis. On presentation to hospital she weighed 2569 grams, highlighting a 17% loss from her birth weight as calculated below (Figure 1).

(Current weight (g) ÷ Birth weight (g)) x 100 = (2569 ÷ 3100) x 100 = 82.8

100 – 82.8 = 17.2 % weight loss

Figure 1: Calculating degree of weight loss (%)

On initial examination, she appeared unwell, lethargic and her skin was noted to be a green complexion. She was visibly jaundiced with icteric sclera. Her liver edge was 2cm palpable below her costal margin. Admission observations were all within normal limits.

Temperature was within normal range. As she appeared clinically unwell, she received a 20ml/kg fluid bolus of crystalloid. She was commenced on broad spectrum antibiotics of cefotaxime and amoxicillin to cover for late-onset neonatal sepsis.

Initial blood tests taken included full blood count (FBC), renal function (U&E), liver function (LFTs), C - reactive protein (CRP) and venous blood gas. Summary of these results are included below (Figure 2).

Blood test	Result	Reference range [2]	Unit
Full Blood Count	Haemoglobin- 224	140-186	g/L
	Platelets- 11	150-400	x 10^9/L
	White cell count- 8.2	5-21	x 10^9/L
C reactive protein	CRP- 7	0.6-5	mg/l
Liver Function tests	Total Bilirubin- 280	<17	µmol/L
	Direct-201	0-5	µmol/L
Ammonia	Ammonia- <100	<100 in neonates	µmol/L
Ferritin	2500	12-200	µg/L
Venous Blood gas	ph- 7.17	7.35-7.45	
	Bicarbonate- 13	22-26	mmol/L
	Lactate- 4.2	<1	mmol/l

Figure 2: Admission Investigation results with age-related neonatal reference ranges [2]

She was reviewed by the paediatric consultant on-call, who recommended a metabolic workup, including a blood test for galactose-1-phosphate uridyltransferase (Gal-1-PUT), and suggested liaison with paediatric hepatology for further advice.

Further blood tests revealed a coagulopathy, with PT 15.50 (8.5-14.1 seconds), for which she received several doses of intravenous vitamin K. She was noted to have a conjugated hyperbilirubinaemia, with direct bilirubin 71.7 % of total bilirubin.

As a result, an abdominal ultrasound (USS) was performed, which was unable to definitively identify the biliary tree. Concurrently, her Gal-1-PUT test revealed absence of the enzyme and a diagnosis of galactosaemia was made. Following this a repeat interval ultrasound was planned.

She was reviewed by the metabolic team who commenced her on a lactose free diet for life and long-term vitamin replacement. She was reviewed by the Ophthalmology team who diagnosed bilateral 'oil-drop' cataracts. She clinically improved and was fit for discharge home with close metabolic follow-up.

She re-presented several days later post-discharge with symptoms of an upper respiratory tract infection. At this time, deterioration in her LFTs was noted (Total bilirubin -119mol/L, Direct bilirubin - 107mol/L, 90% conjugated). A repeat USS abdomen failed to show a definite common bile duct.

Her case was discussed with the hepatobiliary team in Birmingham Children's Hospital who advised that a Hepatobiliary Iminodacetic Acid (HIDA) scan should be performed. The HIDA scan showed a normal biliary tree.

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The hepatobiliary team suggested that her persistent moderately deranged liver function tests (GGT of 840U/L, AST of 60U/L and ALT of 43U/L) were thought to be secondary to ballooning of hepatocytes due to galactosaemia. She is now clinically stable. Weekly liver function tests indicate gradual improvement, her weight is increasing, and she is at home.

Red flag markers in this case, suggesting potentially serious underlying pathology, include significant weight loss (>10% of birth weight), green skin complexion and initial high conjugated hyperbilirubinaemia.

Discussion

Jaundice refers to the yellow discolouration of the skin, gums and the sclerae caused by the accumulation of bilirubin in the skin and mucous membranes [1], as demonstrated below (Image 1).

Image 1: Neonatal Jaundice: note yellow discolouration to skin and sclerae.

Neonates (a baby aged 28 days or less) generally appear clinically jaundiced when the serum bilirubin level is greater than 80mol/L. Approximately 60% of term and 80% of preterm babies develop jaundice in the first week of life, and about 10% of breast-fed babies are still jaundiced at 1 month [1, 3]. Neonatal jaundice can be physiological or pathological.

Causes of Neonatal Jaundice

A useful way of categorising the different causes of jaundice in the neonate, is to first differentiate whether the jaundice first appeared in the first 24 hours of life of thereafter (Figure 3).

Jaundice within 24 hours of birth is exclusively pathological in nature. It suggests an antenatal disease process. The most common causes are due to haemolysis such as cases of Rhesus iso-immunisation or ABO incompatibility.

In these instances, it is important to check maternal and baby blood group, rhesus status and direct Coombs test. Congenital infection is also an important cause which must be investigated. [3, 4].

TORCH screening should be carried out including:

- *Toxoplasmosis*
- *Other*
- *Rubella*
- *Cytomegalovirus*
- *Hepatitis or Herpes*

Most cases of jaundice between days 2-14 are physiological or related to breastfeeding, but include other important causes such as infection, bruising, and conditions such as hypothyroidism or metabolic disorders [5].

Physiological jaundice is usually present within the first week of life, generally within Day 2-4 of life and often regresses by around Day 10 of life. It is felt likely due to several contributing factors including the immature neonatal liver leading to bilirubin accumulation, and excessive bilirubin formation following degradation of persistent foetal haemoglobin.

Breastmilk jaundice is often seen in otherwise healthy breast-fed term babies. The exact clear is unknown but has been linked to a substance in breast milk which slows the breakdown of bilirubin. This can persist for the first few weeks of life and so is a cause of prolonged jaundice also.

Breastfeeding jaundice is a different entity and it is important to separate these two conditions. This is related to an insufficient volume of milk received by the baby and can often be aggravated by situations such as an inadequate latch, maternal anxiety, or lack of support during the initial stages of establishing a regular feeding routine. Careful monitoring of weight and feed volume is required in these circumstances. This can often be aided by input from breast-feeding support coordinator and community midwives.

Jaundice after day 14 in term neonates is known as prolonged jaundice. In preterm infants (babies born < 37 weeks gestation), this as defined as jaundice persisting after day 21.

Another useful tool to distinguish between different causes of neonatal jaundice, is to ascertain whether it is an unconjugated or conjugated hyperbilirubinaemia. Any cause of jaundice that leads to excess formation of unconjugated bilirubin such as haemolysis, iso-immunisation, physiological or breastmilk jaundice leads to an unconjugated hyperbilirubinaemia.

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Conjugated hyperbilirubinaemia is generally regarded when levels are >25% of total bilirubin [1, 3, 4, 6]. Patients can present as green in appearance (as opposed to traditional yellow jaundice appearance) which may suggest an underlying cholestatic problem. The urine may be dark and stool pale or clay-coloured (Image 2) or acolic due to absence of bile pigment [6].

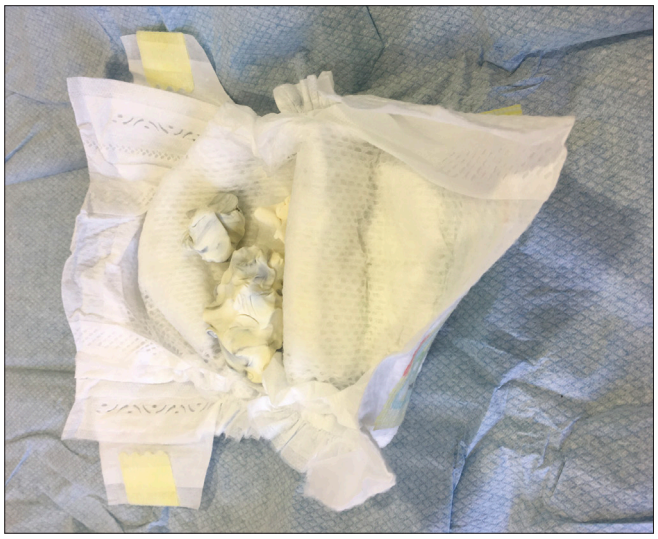

Image 2: Soiled nappy with clay-coloured/pale stool.

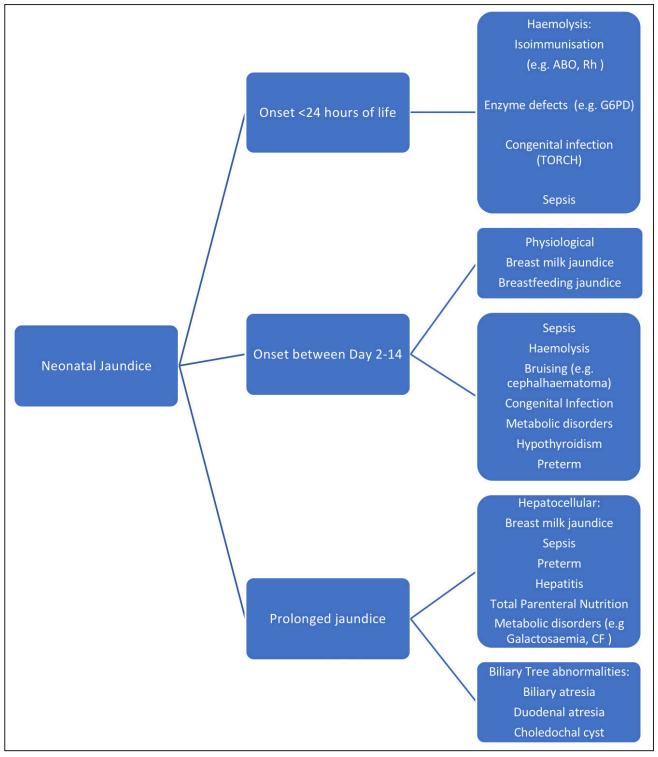

Figure 3: Causes of Neonatal Jaundice [3, 5]

Complications of Jaundice in the Neonate

Jaundice is important to recognise and treat promptly, failure to do so can lead to serious complications of cholestasis such as [7]:

- *Intracranial bleeding from malabsorption of vitamin K*
- *Hypoglycaemia*
- *Kernicterus*

Kernicterus is a life-threatening condition which occurs when the bilirubin levels are >360µmol/L. Unconjugated bilirubin can then cross the blood brain barrier, causing encephalopathy by forming deposits in the basal ganglia and the brainstem nuclei.

The neurotoxic effects can vary in severity from transient disturbance to severe damage. This can lead to lethargy, poor feeding, seizures, coma and death. Infants who survive may develop choreoathetoid cerebral palsy, learning difficulties and sensorineural deafness [6, 8].

Top Tips For Foundation Doctor Assessing A Neonate With Jaundice

History

Detailed History including:

- *Source of referral – Parental concern? Community midwife? Health-visitor? GP? ED?*
- *At what day of life was the jaundice first noticed and by whom?*
- *Is the baby breast or bottle fed?*
- *How often do they feed? Duration and volume?*
- *What feeding support does mum have?*
- *Calculate fluid volume requirements for day of life-*
- *Term infant requirements:*
- *Day 1- 60ml/kg/day*
- *Day 2- 80ml/kg/day*
- *Day 3-100ml/kg/day*
- *Day 4-120 ml/kg/day*
- *Day 5 onwards- 150ml/kg/day*

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- *Is there weight loss and calculate % loss from birth weight.*

- *Ask about vomiting. Is it milky content? Is it always after feeding? Is it projectile? Is it bile-stained (bright green)?*

- *Ask about colour of stools and urine-is there pale chalky stools and/or dark urine that stains the nappy.*

- *Any temperatures or feeling cold? Irritability, poor handling or drowsiness?*

- *Birth History- Any antenatal, perinatal or postnatal complications? Maternal Blood group and Rhesus status? Risk factors for sepsis? Birth weight? Condition at birth? Date of discharge home? Postnatal Baby check examination abnormalities? Has the baby had their heel-prick blood test (~Day 5 of life)? Are family aware of results?*

- *Family history-of jaundice? Any relatives affected with blood disorders or other conditions?*

- *Drug history- On any regular medication? Over the counter or herbal remedies?*

- *Social history- Ascertain support status in the home. Regular midwife/ health visitor attendance?*

Examination

Examine the exposed infant in bright and preferably natural light. Remember, with babies and children, to try examine in an opportunistic manner:

- *Comment on the general appearance of the infant- do they look well, unwell, lethargic, drowsy, dehydrated, active and alert? Remember to relate it to their developmental stage. If it any time you are concerned about the appearance of child, contact for senior review early.*

- *Examine the sclerae, under tongue and gums for yellow discolouration, and press lightly on the skin to check for signs of jaundice in 'blanched' skin, check if it appears yellow/green.*

Note it is easy to underestimate jaundice in Afro-Caribbean, Asian and pre-term infants or those with anaemia.

- *Check naked weight, birth weight and ascertain if any weight loss or gain.*

- *Inspect the nappy area and comment on urine and stool colour if present.*

- *Assess hydration status- fontanelle, mucous membranes, skin turgor, capillary refill time and skin perfusion.*

- *Perform baseline observations*

- *Perform cardiovascular, respiratory and abdominal and neurological examination. Assess for organomegaly or abdominal distension. Assess for a rash.*

Investigations

First line investigations may include [4,9]:

- *Full blood count, Blood Film, Coomb's test, Blood Group- (mother and baby)*

- *U&E, Bone Profile*

- *LFT's & Serum Bilirubin -total and direct*

- *TFTs*

- *Blood sugar*

- *Blood cultures*

- *TORCH Screen*

- *Guthrie Card*

- *Urine for protein, bilirubin, nitrites, leucocytes and culture [6]*

Further blood/urine tests if appropriate may include:

- *Cholesterol/Triglycerides*

- *Hepatitis A, B, C*

- *G6PD*

- *Metabolic/Endocrine Investigations*

- *IRT/ Sweat test*

- *GAL-1-PUT*

- *Alpha 1 antitrypsin*

- *Plasma amino acids, Urine amino/organic acids*

- *Cortisol*

Additional Investigations may include:

- *USS Abdomen*

- *Liver Biopsy*

- *Specialist review e.g. GI, Metabolic, Hepatobiliary, Ophthalmology, Genetics*

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Management

At all times, seek senior advice and support when managing a neonate with significant jaundice. For the vast majority of cases of neonatal jaundice, these are managed through strict fluid monitoring, repeated clinical assessment and treatment with phototherapy. For a small number of cases of patients with extremely high levels of bilirubin, exchange transfusion may be required.

Phototherapy

The baby is subjected to light (450nm wavelength) from the blue-green band of the visible spectrum which coverts unconjugated bilirubin into a harmless water-soluble pigment excreted predominately in the urine [1, 4, 10]. A separate graph is required depending on what gestation the baby was born.

Depending on the level of bilirubin, this treatment can range from single to quadruple light phototherapy. This can be discontinued once serum bilirubin has fallen to a level at least 50 micromol/litre below the phototherapy threshold [1].

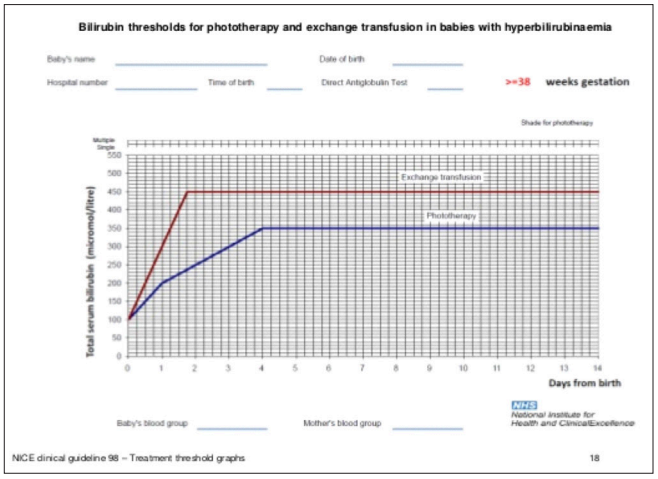

Image 3: Phototherapy treatment graph for baby born >38 weeks gestation. [1]

Exchange transfusion

In cases of severe unconjugated hyperbilirubinaemia, a baby's blood is removed in small aliquots and replaced with donor blood that has been carefully matched and screened. Twice the infant's blood volume is exchanged and intensified phototherapy should continue during exchange transfusions [11]. This procedure is high-risk and is often performed by senior paediatricians or neonatologists in a neonatal unit.

Intravenous Immunoglobulins

This form of treatment may be used as an adjunct to continuous intensified phototherapy in cases of rhesus haemolytic disease or ABO haemolytic disease when the serum bilirubin continues to rise by more than 8.5 micromol/litre per hour [8]. Vitamin K may be given if patient is coagulopathic, this can be repeated after 12 hours if patient is resistant to treatment [8].

Acknowledgements

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- *The Department of Play Therapy in Royal Belfast Hospital for Sick Children for assistance in creating our simulated nappy.*

Test Yourself: Neonatal Jaundice

1. You are asked to review a 3 day old term baby boy on the postnatal ward. The midwives are concerned about increasing signs of jaundice. This is mum's first child.

Antenatal history was unremarkable. Delivery was following face presentation and moderate facial trauma was noted. In general, baby was born in good condition and suitable for admission to postnatal ward for close observation.

Birth weight was 4.2 kg. There were no risks factors for infection. Baby has been breastfeeding well. Which ONE of the following is the most likely cause of jaundice?

a) Breast milk jaundice

b) Sepsis.

c) ABO incompatibility.

d) Bruising.

e) Physiological jaundice.

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2. A term 4 day old baby has been admitted to the paediatric ward with jaundice. She is bottle fed, and has had some milky vomits after most feeds. Bowels have opened. She has 8% weight loss, and appears dehydrated.

Bilirubin is 4 squares into the single phototherapy zone, with Direct bilirubin <10% of total. Direct Coombs is negative and Haemoglobin, White cell count, U&E and CRP are in normal range. Current management of this baby includes all EXCEPT which option:

a) Frequent weights.

b) Strict fluid balance and monitoring.

c) Exchange transfusion.

d) Single Phototherapy.

e) Feeding support and supervision.

3. A 6 day old caucasian baby girl is referred to the Paediatric ward from the community midwife with jaundice and 12% weight loss. Her jaundice was first noticed on Day 4 and has become more apparent since then. She was born at full term via normal delivery.

Mother and baby were discharged on Day 2. This is mum's first baby and she is breastfeeding. She is afebrile, has passed urine and opened bowels, and appears hungry. She has had a few small milky vomits. The most likely cause of this baby's jaundice is:

a) ABO incompatibility.

b) Breast milk jaundice.

c) Infection.

d) Pyloric Stenosis.

e) Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency.

4. A 16 day old term baby girl is an inpatient on the paediatric ward having been referred by the community midwife with jaundice. On reviewing her latest blood results, you see her total bilirubin is 300 and her direct bilirubin is 85 (28% of total).

You review the blood results and the patient with the registrar and make an appropriate management plan. Causes of conjugated hyperbilirubinaemia include all of the conditions EXCEPT which one:

a) Biliary Atresia.

b) ABO incompatibility.

c) Galactosaemia.

d) Congenital TORCH Infections.

e) Neonatal Hepatitis syndrome.

5. A baby born at 36 weeks has been noticed to become jaundiced in the first day of life on the postnatal ward. You are asked to review the patient. Reviewing maternal history, you note that this is mother's second pregnancy. Her first baby was born in good condition with no issues.

Mother's serology at that time was negative. This pregnancy was an unscheduled and she presented for first antenatal appointment at 35 weeks. Mother's blood group is A Rh Negative. There are no known risk factors for sepsis. You examine baby and take some blood tests including Full Blood Count, Bilirubin, Blood Cultures, and Group and Direct Coombs Test. Which ONE of the following is the most likely cause of jaundice?

a) Hypothyroidism.

b) Cystic Fibrosis.

c) Polycythaemia.

d) Rhesus disease of the newborn.

e) Physiological jaundice.

Answers

1. Answer:

The history of traumatic delivery offers a clue to the likely diagnosis in this instance. Most commonly, babies are born head-first (cephalic) where the occiput is the leading part and the neck is flexed and chin is tucked in towards the chest (cephalic vertex).

A face presentation, which is one type of malpresentation, means that the neck is extended. This inhibits head engagement and descent of the baby through the birth canal. A large baby (macrosomia) is defined as a baby weighing over 4kg at birth. This is one of the causative factors associated with face presentation.

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Complications of this form of presentation include prolonged labour, facial trauma, bruising, and respiratory distress. Extensive bruising predisposes babies to develop signs of jaundice. A bruise forms when blood leaks from the blood vessels. In repair, haemolysis occurs as these red cells are broken down, causing release of bilirubin.

2. Answer:

Difficulties establishing feeding in the first few weeks of life are extremely common. Mothers and babies need support and guidance during this period. It is important to take a thorough history to ascertain the degree of vomiting, the colour of the vomitus (to ensure not bilious suggesting a possible obstructive cause). Other causes include reflux, milk intolerance and often simple posseting until feeding is established.

In this case, the baby has lost some of her birthweight, appears dehydrated and is likely not receiving enough volume. It is important to closely supervise feeding technique, provide appropriate support and monitor fluid balance and weight. With reference to the NICE Phototherapy graphs, you have plotted that the Bilirubin falls in the single phototherapy zone, and so it is appropriate to commence this, following discussion with parents.

Regular monitoring of clinical status and serum bilirubin's are important to ensure resolution. In cases of extremely high hyperbilirubinemia, exchange transfusion is required and requires transfer to the neonatal unit.

3. Answer:

The most likely cause in this instance is breast milk jaundice. Breast milk may exacerbate jaundice in healthy infants. The exact mechanism is unclear. Increased enterohepatic reabsorption of unconjugated bilirubin is suggested as one possible reason. In some infants, this may be exacerbated if milk intake is poor from delay in establishing breastfeeding and dehydration occurs.

Electrolyte disturbances, worsening dehydration and hypovolaemic shock may occur if not corrected. Prompt discharge from hospital, inexperienced mothers and inadequate milk supply may all be contributing to this situation. Extensive support to the mother should be provided to encourage breastfeeding throughout, but alternative methods such as strict monitoring of intake using expressed breast milk or in some cases supplementation with IV Fluids may be required.

ABO incompatibility often presents in the first 24 hours of life. In this case, there were no risk factors for infection. Pyloric Stenosis presents in babies usually between 2 and 7 weeks of age, often with projectile vomiting, and is more common in boys. In cases of neonatal jaundice due to G6PD deficiency, onset is usually within the first 3 days of life. The gene for G6PD is located on the X chromosome and so the deficiency mainly affects males. It is more common in those of mediterranean, asian and afro-carribean descent.

4. Answer:

Conjugated hyperbilirubinaemia results from a variety of different mechanisms. These include reduced secretion of conjugated bilirubin into the bile such as in hepatitis and in cases where there is impaired flow of bile into the intestines such as in biliary atresia. Bile formation is sensitive to inflammatory cytokine release, and this may occur in congenital infections. Galactosaemia can lead to either forms of hyperbilirubinaemia.

If direct bilirubin is measured, it is considered elevated if it is greater than 25 percent of the total serum bilirubin, and may suggest an underlying hepatic or extrahepatic (cholestatic) cause of jaundice. Patients often can appear green in appearance as opposed to the traditional yellow appearance in unconjugated cases of jaundice.

In these cases, urine is dark and stool as pale or acolic (due to absence of bile pigment). Breakdown of red blood cells, caused by haemolysis, such as in cases of ABO incompatibility contributes to release of indirect bilirubin and is a well-recognised cause of unconjugated hyperbilirubinaemia.

5. Answer:

Jaundice appearing in the first 24 hours of life is pathological. It usually results from cases of haemolysis or congenital infection. It is particularly important to identify as bilirubin is unconjugated and can rise very rapidly reaching dangerous levels.

Rhesus (Rh) disease occurs during pregnancy when there is an incompatibility between the blood types of the mother and baby. When a Rh negative mother has a baby that is Rh positive, problems can develop if the baby's red blood cells cross to the mother.

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This often occurs at delivery following detachment of the placenta, but may occur at any stage. The mother's immune system identifies the baby's Rh positive blood as foreign, and develops antibodies against these. The mother's immune system retains these antibodies in case of future pregnancies. The mother is now Rhesus sensitised.

In a first pregnancy, Rhesus sensitisation is not likely. It usually only becomes a problem in future pregnancies with another Rh positive baby. During this pregnancy, the mothers' antibodies can cross the placenta to attack the baby's Rh positive blood cells. Complications include anaemia, jaundice and a severe condition known as hydrops fetalis.

In the newborn baby, this condition is known as Haemolytic disease of the newborn. Usually, in the antenatal periods, Rhesus negative mothers receive a drug called Rhesus immunoglobulin (RhIg), at around the 28th week of pregnancy, and a second dose several days after delivery. This destroys any Anti-Rh antibodies which have been created in the mothers' circulation, and helps protect future Rh positive baby.

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VIRAL-INDUCED RASH IN A PATIENT WITH CUTANEOUS GRAFT V HOST SKIN DISEASE

Z Zair, E Simmonds

Abstract

Identifying the cause of a rash in a child can present a challenge for many clinicians. Here we discuss the case of a 12 year old boy presenting with features consistent with a viral induced rash, complicated by a pre-existing cutaneous graft versus host disease post bone marrow transplant for acute myeloid leukaemia treatment.

We utilise this case to enhance our understanding on how best to identify and distinguish between the different types of common viral induced rashes. We discuss the characteristics associated with cutaneous graft versus host disorder and appropriately differentiate this patient's presentation from that of acute myeloid leukaemia relapse.

Case Presentation

A 12 year old presented to Children's Emergency Department with pyrexia, lethargy, headache and widespread maculopapular rash. The rash presented initially, on the chest and then spread peripherally to include the face and buttocks. The rash was preceded by pruritus although at presentation the patient no longer complained of being itchy. The rash was non-blanching, erythematous and macular with flattened papules and plaques [Figure 1].

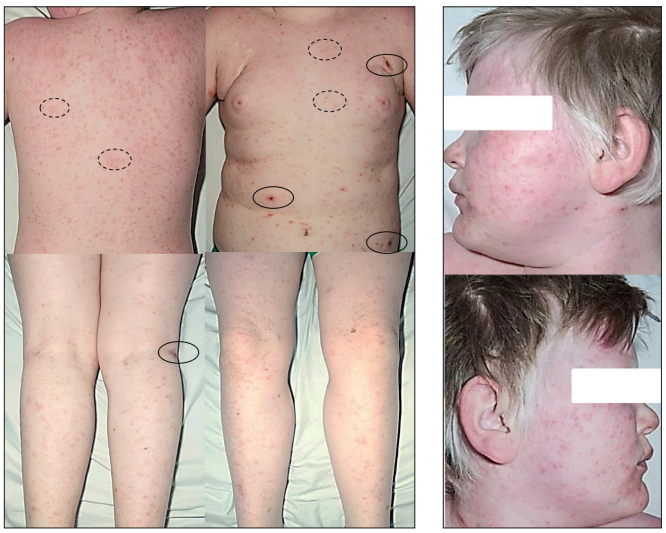

Figure 1: Photograph of patient's skin taken one day post hospital admission. The pictures show widespread maculopapular rash including the face, arms and buttocks. These pictures are representative of the patterns shown on the arms and buttocks, which are not shown here. Areas of erosion are demarcated by a black circle.

Eroded areas were noted at the right axilla, abdomen and right posterior knee. Headache, lethargy and pyrexia developed three days after the rash. The headache was intermittent in nature with no associated audio-visual changes. There was no history of recent travel, contact with animals or change in medication.

Identifying the cause of the rash on clinical presentation alone was complicated by a pre-existing cutaneous graft versus host disease (GvHD) which in this case comprised of areas of dyspigmentation with xerosis and hyperkeratotic acral palmar erythema [Figure 1, 2].

The patient was admitted to hospital and treated with a course of flucloxacillin for probable staphylococcus infection secondary to epithelial breaks caused by persistent scratching of the viral-induced rash. He was also given intravenous paracetamol as an anti-pyretic and analgesic. A dermatological review was also arranged.

Patient Background

The patient had previously been diagnosed and treated for acute myeloid leukaemia (AML) at the age of four years. Late onset acute GvHD was a complication of the allogenic haematopoietic bone transplant received as part of his AML treatment. In this case the GvHD manifested with cutaneous [Figure 2] and oesophageal complications. The patient has been in remission since 2013.

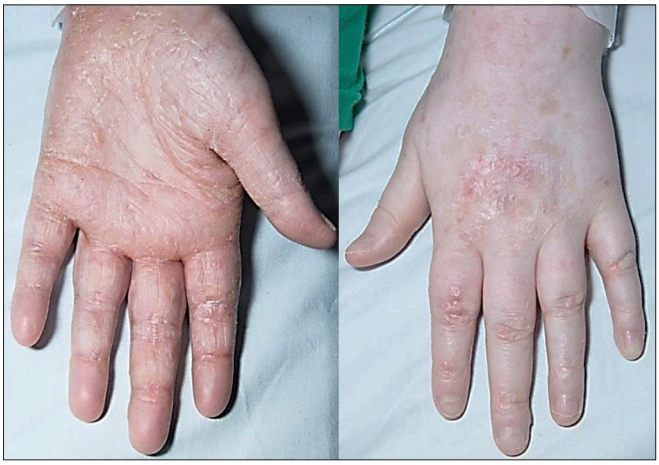

Figure 2: Photograph of patient's hand taken one day post hospital admission. This figure shows hyperkeratotic acral palmar erythema on the left hand and is representative of the skin pattern noted in both hands and both feet.

VIRAL-INDUCED RASH IN A PATIENT WITH CUTANEOUS GRAFT V HOST SKIN DISEASE

Z Zair, E Simmonds

Investigation

A viral screen was performed and did not positively identify a causative strain [Table 1].

Virology/Serology Tests:			
Influenza A RNA	NOT DETECTED	Varicella zoster Virus DNA	NOT DETECTED
Influenza A RNA	NOT DETECTED	Herpes Simplex Virus 1	NOT DETECTED
Respiratory Syncytial Virus RNA	NOT DETECTED	Herpes Simplex Virus 2	NOT DETECTED
Human metapneumovirus	NOT DETECTED		
Rhinovirus RNA	NOT DETECTED		
Enterovirus RNA	NOT DETECTED	Cytomegalovirus IgM Antibody	NOT DETECTED
Adenovirus DNA	NOT DETECTED	Cytomegalovirus IgG Antibody	NOT DETECTED
Bocavirus DNA	NOT DETECTED	Parovirus B19 IgM Antibody	NOT DETECTED
Coronavirus RNA	NOT DETECTED	Parovirus B19 IgG Antibody	DETECTED
Parainfluenza virus RNA	NOT DETECTED	EBV VCA IgM Antibody	NOT DETECTED
Parechovirus RNA	NOT DETECTED	EBV VCA IgG Antibody	DETECTED

Table 1: Virology Screen. Blood, sputum, throat and nasal cultures were taken and a virology/serology screen performed. None of the major viruses were detectable on PCR. A previous infection with Parovirus B19 and EBV was noted.

Bloods were taken to monitor renal and liver function, which were both normal. A full blood count (FBC) revealed mild pancytopenia and moderate thrombocytopenia [Table 2]. Parvovirus B19 and EBV antibodies were IgG positive but IgM negative [Table 1] suggesting a previous rather than a current infection and less likely to account for the observed pancytopenia [Table 2].

Day	Hb	MCHC	Plts	RBC	WCC	Neutr	Eosin	Basoph	Lymph
1	144 (N)	364 (N)	79 (L)	4.38 (N)	3.31 (L)	1.25 (L)	0.33 (L)	0.02(N)	1.41 (N)
2	128 (L)	357 (H)	122 (L)	3.78 (L)	5.30 (N)	2.99 (N)	0.54 (N)	0.05 (N)	1.36 (N)
4	124 (L)	363 (H)	U	3.77 (L)	5.45 (N)	2.95 (N)	0.52 (N)	0.03 (N)	1.55 (N)

Day	Blood Film
1	Platelet anisocytosis Reactive lymphocytes
4	Spurious thrombocytopenia Polychromasia, mature neutrophils with toxic granulation No blasts noted

Laboratory Normal Ranges:	
Hb	130 - 160
MCHC	315 - 345
Platelets	140 - 400
RBC	4.10 – 5.10
WCC	4.5 – 13.00
Neutrophils	1.8 – 8.0
Eosinophils	0.5 – 1.0
Basophils	0.01 – 0.10
Lymphocytes	1.0 – 4.0

Table 2: FBC and blood film results. Bloods and blood films were taken over the course of four days and a notable change in pattern can be seen. Initially the patient presented with mild thrombocytopenia, however, this later changed to a mild macrocytic anaemia.

The blood films did not contain any blastocyst but instead contained reactive lymphocytes, confirming a current infection. Plts = platelets, Neutr = Neutrophils, Eosin = Eosinophils, Basop = Basophils, Lymph = Lymphocytes, N = normal value, H = high value, L = low value U = unavailable.

Skin cultures were taken and positive for staphylococcus aureus. The course of flucloxacillin, commenced on admission, was continued in line with our hospital guidelines. Blood and urine cultures were negative.

A blood film performed to rule out AML relapse was further performed. No immature blasts were detected on the film, however, reactive lymphocytes were present, consistent with an infective cause for this patient's presentation [Table 2].

During the admission the patient had complained of chest pain and so a chest x-ray (CXR) was performed [Figure 3]. This showed an ill-defined small opacity at the left base close to the left heart border. Given that the patient was not exhibiting any respiratory symptoms/signs, had no evidence of increased work of breathing and normal oxygen saturations in air, it was decided that no treatment was required. A repeat CXR would be performed in 4 – 6 weeks' time only if the patient complained of respiratory problems.

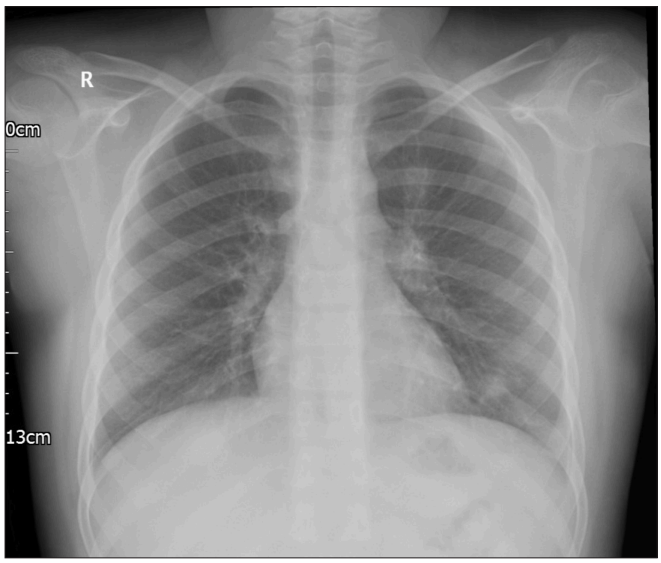

Figure 3: Anterior-posterior chest x-ray of a 12 year old male patient complaining of chest pain. A chest x-ray performed whilst in hospital shows an ill-defined small opacity at the left lung base suggestive of mild infection.

A repeat FBC test performed on day 2 and day 4 of admission showed a mild hyperchromic macrocytic anaemia [Table 2].

Patient Outcome

Despite the deranged FBC, the patient had much improved and was no longer pyrexic or exhibiting any systemic symptoms of illness. The haematology team were consulted when the patient's blood film results were initially produced and there were no plans for a haematology follow up. The rash had improved slightly, albeit still visible on discharge.

VIRAL-INDUCED RASH IN A PATIENT WITH CUTANEOUS GRAFT V HOST SKIN DISEASE

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An outpatient follow up appointment was arranged with the dermatology team who deemed the rash to be a probable reactive exanthema. The patient was offered a skin biopsy if the rash did not improve with time. At the time of publication the patient's skin condition had improved and the patient did not exhibit any recurrence in their symptoms.

Dermatological Features

Pyrexic children with a rash can present in a myriad of ways including maculopapular rash, generalised diffuse erythema, vesicular, pustular, nodular or petechial/purpuric. Viral infection is the most common cause. In this case the patient exhibited a maculopapular exanthema of unknown viral strain.

In the majority of cases early diagnostic testing for a febrile illness with a rash is inefficient. Tests may prove of superior diagnostic use if repeated later on and the clinical course monitored. Whilst of academic or educational interest, the repetition of such tests needs to be weighed against the inconvenience and potential unnecessary distress to the child having to undergo multiple blood tests and could be reserved for patients whose symptoms have not improved.

It is acceptable to diagnose a patient as having an unknown causative viral strain provided that a) the overall clinical picture is in keeping with a viral infection, b) we have ruled out other causes such as bacterial, fungal and c) the patient is responding to treatment. Indeed the management for viral induced rash with systemic features is conservative; treatment comprising of hydration, analgesia and if required skin emollients.

When approaching a patient with a viral induced rash, we recommend the following approach to help in guiding the diagnosis [Figure 4]:

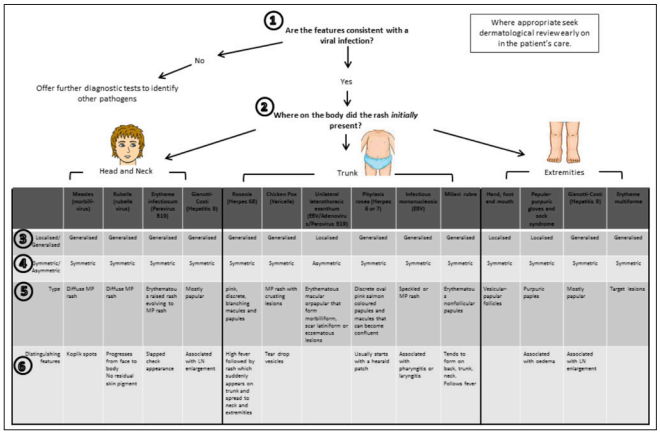

Figure 4: Overview of the most common viral induced maculopapular rashes in children according to location, distribution and pattern. Body part figures taken from http://mzayat.com and https://openclipart.org. This table includes only the most common viral causes and is not exhaustive.

1) Are the systemic features consistent with a viral infection?

2) Where on the body did the rash initially present?

3) Is the rash localised or generalised?

4) Is the rash symmetric or asymmetric

5) What type of rash is it i.e. petechial or nodular etc.?

6) Are there any additional features that may help distinguish the rash?

It is worth noting that rash severity is not related to the severity of the underling disease, however, it is always best to obtain dermatological input from an early stage of presentation.

Patients with cutaneous GvHD can initially present with a maculopapular rash on the palms and soles however the rash can begin anywhere in the body. Early lesions are usually entered around hair follicles which is pathegnomic of cutaneous GvHD (Friedman et al., 1988). Later on patients exhibit a lichenoid appearance evolving to form a more sclerodermatous appearance (Peñas et al., 2004).

Scleroderma GvHD is preceded by lichenoid lesion in only 40% of patients. Moreover not all patients with lichenoid GvHD progress to a sclerodermatous appearance. It is important to recognise cutaneous GvHD early on in order that appropriate treatment is given to these patients. It is also important to identify the lichenoid or scleroderma appearance in patients with established GvHD so that when they present with new onset skin dysfunction, it can be correctly characterised.

Our patient had a staphylococcus aureus positive skin swab. It was thought that this was a complication of the pre-existing viral rash where excoriations and resulting epithelial breaks were infected with the natural skin flora rather than a primary infection. Children with primary staphylococcus infections tend to look more 'toxic' in appearance, sometimes presenting with hypotension, severe muscle and bone pain as well as fever.

Haematological Features

In light of the patient's medical background, coupled with the constellation of symptoms/signs that he presented with, it would be of relevance to simultaneously investigate for AML relapse as was done in this case. This is always good clinical practice, nevertheless, we would not expect a patient with AML relapse to present in this way.

There is no clearly defined guideline regarding signs/symptoms to look for in patients querying AML relapse, however, patients typically present with abdominal pain, petechiae and lethargy rather than fever, rash, lethargy which although can be indicative are less likely associated with AML relapse and more in keeping with a common viral infection.

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Interestingly current NICE guidelines do not include abdominal symptoms or weight loss in as a characteristic feature of AML despite a recent systematic review and meta-analysis showing these symptoms to occur in over one third of patients with leukaemia [Clarke et al., 2016; NICE CKS 2015].

Other factors that would detract away from a diagnosis of AML relapse include the timeframe in presentation and the development of GvHD. In general, AML relapse tends to occur after the first cycle of treatment and/or within the first year of remittance rather than several years after remittance. Moreover, patients with cutaneous GvHD are associated with fewer instance of AML relapse thought perhaps due to the high antileukemic effect of the initial treatment regimen (Enright et al., 1996; van Rhee et al., 1997; Lee et al., 2002).

Conclusion

A patient unwell enough to require hospital admission with a background of AML should be checked for relapse regardless of presentation. Nevertheless, common things are common and so a child presenting with fever, lethargy, fatigue and widespread maculopapular rash is more likely to have a viral infection and should be treated accordingly.

Adopting a systematic approach can be of benefit when trying to identify the type of viral-induced rash. We have provided a flow chart which can be used and is centred on the primary location of the rash. Lastly, interpretation of blood results should be done in conjunction with and not in spite of the patient's presentation. In this case the patient initially presented with a pancytopenia/thrombocytopenic picture that later changed to a macrocytic anaemia, however, the patient's symptoms were resolving. This transient change in blood component levels, whilst unusual, may be characteristic of the type of viral infection that the patient had.

Multiple Choice Questions

1) According to the World Health Organization (WHO) diagnostic criteria for AML, what is the minimum number of blasts that should be present within the bone marrow?

a. 5%

b. 10%

c. 15%

d. 20%

e. 25%

2) Graft versus host disease is an immunological process initiated and mediated by which type of cell?

a. Plasma cells

b. T cells

c. B cells

d. Natural killer cells

e. Mast cells

3) Which viral rash is caused by morbillivirus?

a. Chickenpox

b. Measles

c. Mumps

d. Rubella

e. Erythema multiforme

4) Aciclovir may be used in the treatment of which condition?

a. In adults with chickenpox

b. In children with chickenpox

c. In immunocompromised children with measles

d. In pregnant women with chickenpox

e. In children with respiratory syncytial virus infection

5) Which viral infection is most typically associated with leukopenia?

a. Chickenpox

b. Erythema infectiosum

c. Mumps

d. Rubella

e. Measles

VIRAL-INDUCED RASH IN A PATIENT WITH CUTANEOUS GRAFT V HOST SKIN DISEASE

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Answers

1) Answer: d

The WHO diagnostic criteria for AML has recently been updated and states that a patient should have greater or equal to 20% blasts within the bone marrow or peripheral blood with molecular genetics as well as immunohistochemistry and/or cytochemistry data for disease classification [Arber et al., 2016; Döhner et al., 2018].

2) Answer: b

It is initiated by the activation of donor T cells after adoptive transfer into an allogeneic recipient. T cells then mediate the induction of graft versus host response.

3) Answer: b

The morbillivirus causes measles and is an enveloped single stranded RNA virus.

4) Answer: a

The Varicella Zoster virus cause chicken pox and can be effectively treated with aciclovir, but it is only usually given in adults where the disease tends to be more severe, or in severe childhood infections.

5) Answer: e

Measles is associated with a transient immune suppression, the mechanism of which is not fully understood. Patients are monitored and where appropriate antibiotics are sometimes given to prevent infection during the course of the disease.

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PRESENTATIONS OF INHALED FOREIGN BODIES IN CHILDREN

R Devaney, M Kurc, M Daniel, M Hurley, S Rathi, M Yanney, JM Bhatt

Abstract

Foreign body inhalation is recognised as a significant cause of morbidity and mortality in children. [1] Whilst organic foreign bodies are the most commonly inhaled [2], it is important to also consider inorganic foreign bodies and how these may present. Obtaining the relevant history is key to making a prompt diagnosis and to avoid complications associated with delay.

Background

Foreign body inhalation is a significant cause of morbidity and mortality in children [1]. It is most common in children aged 1-3 years, who are mobile and exploring their environment. Organic foreign bodies are most commonly inhaled [2], however, there may be differences in how organic and inorganic foreign bodies present, possibly related to the different nature of inflammatory response that these might incite in the lungs. Delayed diagnosis is not unusual and may result in potentially avoidable additional complications [3,4].

Diagnosis can be a significant challenge, especially if unwitnessed and the child is unable to provide a history. A suspicion of foreign body inhalation may come from clinical history, symptoms (especially a cough, choking episode or sudden onset dyspnoea), and findings on chest examination. A history of a witnessed choking event should always be taken seriously.

Findings on clinical and radiological examination associated with foreign body aspiration, can be subtle and non-specific and foreign bodies are rarely radio opaque. A normal CXR, even if a child is old enough and able to breath hold and it is possible to obtain an expiratory film, is not sufficient to exclude foreign body inhalation; bronchoscopy is the gold standard [4, 5]

The impact on the nature of the foreign body on the inflammatory response has been reported in other areas e.g. intraocular foreign bodies [6] and may result in a difference in their presentation.

Whilst there are reports in the literature of organic foreign body inhalation e.g. jelly sweets causing significant lung collapse and a requirement for emergency bronchoscopy and intensive care [7], there are also reports of intervals of several days or even longer in the diagnosis of non-organic foreign bodies e.g. metal balls [8]. It is important to consider foreign body aspiration in children with persistent respiratory symptoms that fail to improve.

Case 1

A 2yr old boy attended the emergency department (ED) having choked on an orange pip, 2 hours previously. He briefly appeared blue after choking and then had some difficulty breathing. On arrival in ED he had soft stridor which became louder when upset. Oxygen saturations were 88% in air. He had mild recession and tracheal tug when crying.

He was reviewed by the ENT team who arranged a prompt bronchoscopy about 3 hours after the suspected inhalation. A hyperaemic airway was found, with copious secretions. No foreign body was identified. Twenty-four hours later he became unwell with an increased oxygen requirement. He had decreased air entry in the right lower zone with some inspiratory squeaks and he had to be transferred to PICU where he was ventilated.

A CXR showed a near total collapse of the right lung (figure 1) with mediastinal shift consistent with a mucus plug or dislodged foreign body occluding the right main bronchus. He had a repeat bronchoscopy where an orange pip was retrieved from the right main bronchus. He was extubated the following day. A repeat CXR was clear. The patient completed a 5 day course of antibiotics and was discharged home.

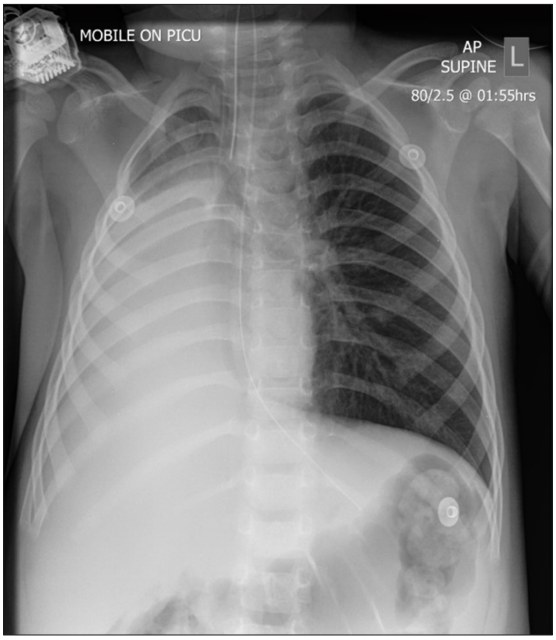

Figure 1: CXR showing near total collapse of the right lung

Case 2

A 2-year-old boy presented with a 2 week history of fever, cough and reduced appetite. Examination findings and CXR indicated left lower lobe pneumonia. He was treated with a course of Amoxicillin but made a partial recovery. In view of persistent symptoms he received two further courses of antibiotics but was referred to paediatrics after failing to improve.

He had persistent cough and wheeze at outpatient review 8 weeks later and received a further course of antibiotics. He was admitted soon after with acute wheeze requiring oxygen, bronchodilators and prednisolone.

A CXR done at the clinic review was later reported to show a foreign body in the left main bronchus with distal consolidation. A retrospective review of the CXR performed 2 months earlier (figure 2) also showed the likely foreign body, which was not reported at the time.

A history of sudden onset choking and coughing a few days prior to the onset of cough and fever was obtained retrospectively. He had been reviewed at his local hospital but reassured and discharged.

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A bronchoscopy was performed and a pebble removed from the right main bronchus. The foreign body most probably moved from the left to the right main bronchus after a significant bout of coughing and vomiting a few days earlier. A further 2 week course of antibiotics was given. He has subsequently remained well.

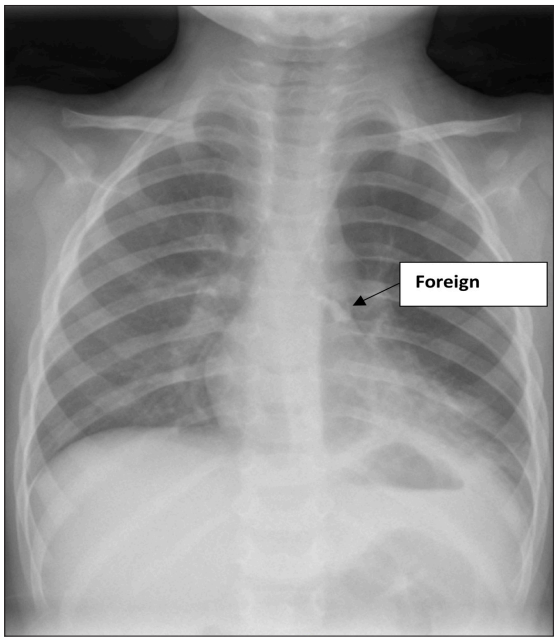

Figure 2: CXR showing foreign body in left main bronchus and consolidation of left lower lobe

Case 3

A 4 year-old girl presented with a history of choking on a sweet wrapper. She initially had some noisy breathing associated with cough and choking but this quickly settled. Her parents were reassured and she was sent to bed. Overnight she woke with noisy breathing and her parents took her to ED.

She was treated with antibiotics and an inhaler and discharged. Over the next few months, she had problems with halitosis and a chronic wet cough, productive of green/ brown coloured sputum and her parents attended primary care and hospital multiple times. A fluoroscopy showed some slight mediastinal shift to the left on expiration, a HRCT scan of her chest was reported as normal and she had multiple courses of antibiotics. .

In view of the initial history and ongoing symptoms she was referred for a paediatric respiratory opinion. A bronchoscopy (figure 3) was promptly arranged. A sweet wrapper (figure 4) was successfully removed from the right main bronchus 10 months after her initial presentation. Her halitosis immediately improved and she has remained well since.

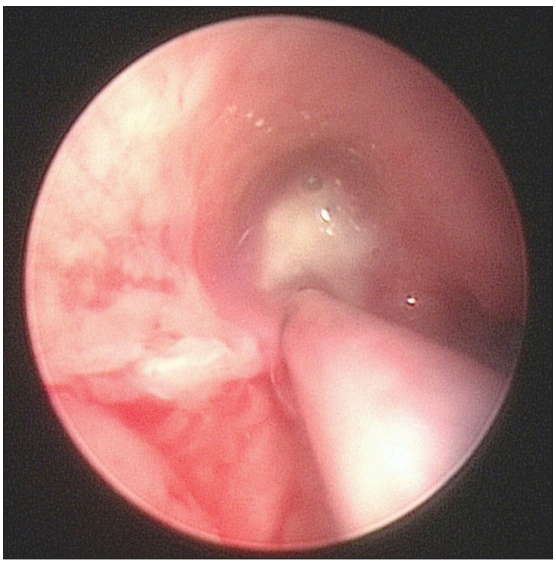

Figure 3: Bronchoscopy image showing secretions surrounding the foreign body

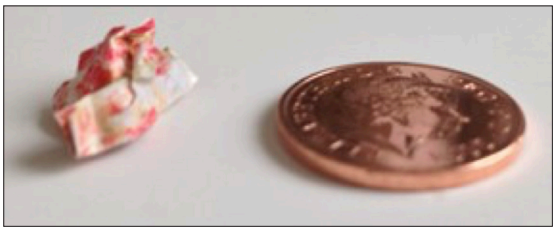

Figure 4: Image of sweet wrapper removed from right main bronchus (coin for comparative purposes)

Discussion

These cases demonstrate how the nature of an inhaled foreign body (organic vs non-organic) may have a significant impact on clinical presentation. A study from Great Ormond Street looking at timing of removal of aspirated foreign bodies showed that of 94 patients analysed, 7 required an emergency procedure out of hours due to signs of severe respiratory distress, and in all 7 of these cases the foreign body was organic [9]

It is likely however, that other features such as the age of the child, size, shape, site of impaction and degree of airway obstruction of the foreign body may also influence the clinical manifestation.

Shubha et al, describe a series of 102 infants who underwent bronchoscopy for suspected foreign body aspiration between 1997 and 2007 in India. There was a wide range of delay in presentation (1 day- 3 months) and symptoms, yet most of the foreign bodies in this study were organic e.g. mostly nuts and seeds [10].

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A prolonged history and normal radiographic findings should not confer inappropriate reassurance with an accompanying history of inhalation/choking. The classical triad of coughing, wheezing and diminished unilateral breathing sounds are present in less than 50% of clinical cases [11].

It is important to have a high level of suspicion with these patients and to consider further investigations. These include radiological (CXR) and endoscopic which can be selected according to patient's response to treatment or observations.

Learning Points

• Eliciting the relevant history is crucial to making a prompt diagnosis of foreign body inhalation.

• Foreign bodies can present with a range of symptoms including wheeze, stridor, an oxygen requirement and reduced air entry

• Symptoms of foreign body inhalation may be confused with other conditions e.g. croup, viral wheeze or a lower respiratory tract infection

• The nature of an inhaled foreign body (organic vs non-organic) may have a significant impact on the clinical presentation.

• Delayed diagnosis may result in potentially avoidable complications.

• A prolonged history and normal radiographic findings may not be reassuring in foreign body aspiration.

• The gold standard for respiratory-tract foreign-body retrieval is a rigid bronchoscopy [11]

Test Yourself

Test Yourself 1

WHAT IS THE MOST RELIABLE METHOD OF FOREIGN BODY REMOVAL FROM THE LOWER RESPIRATORY TRACT?

A) HEIMLICH MANOUVRE
B) RIGID BRONCHOSCOPY
C) FLEXIBLE BRONCHSCOPY
D) FORCEPS RETRIEVAL
E) "PATTING" OF THE BACK

Test Yourself 2

WHAT ARE THE CLINICAL MANIFESTATIONS OF FOREIGN BODY INHALATION?

A) DESATURATION
B) WHEEZING
C) STRIDOR
D) UNILATERAL DIMINISHED BREATHING SOUNDS
E) ALL OF THE ABOVE

Test Yourself 3

WHICH OF THE FOLLOWING CHEST XRAY FINDINGS ARE NOT SUGGESTIVE OF FOREIGN BODY INHALATION?

A) LOBAR HYPERINFLATION
B) SEGMENTAL COLLAPSE OR CONSOLIDATION
C) BILATERAL DIFFUSE GROUND GLASS APPEARANCE
D) OPACITY WITHIN BRONCHIAL TREE
E) NORMAL APPEARANCE

Test Yourself 4

WHICH OF THE FOLLOWING STATEMENTS ABOUT FOREIGN BODY INHALATION IS NOT TRUE?

A) OVER 300 CHILDREN ARE ADMITTED ANNUALLY IN ENGLAND
B) THE PEAK INCIDENCE IS IN CHILDREN AGED BETWEEN 1-3 YEARS
C) DIAGNOSIS IS DELAYED BY MORE THAN A WEEK IN ABOUT 30% OF CASES
D) ACCIDENTAL DEATH HAS BEEN REPORTED IN 7% OF CHILDREN UNDER 4 YEARS OF AGE
E) MOST FOREIGN BODIES ARE RADIO OPAQUE AND VISIBLE ON A CHEST XRAY

Test Yourself 5

WHICH OF THE FOLLOWING IS NOT A RECOGNISED COMPLICATION OF FOREIGN BODY INHALATION?

A) RECURRENT PNEUMONIA
B) PNEUMOTHORAX
C) LUNG ABSCESSES
D) TUBERCULOSIS
E) BRONCHIECTASIS

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Answer Key

Question 1: B

Rigid Bronchoscopy is the most reliable tool for removal of foreign bodies located in airway.

Question 2: E

Depending on location of the foreign body, all of these symptoms can manifest in a patient.

Question 3: C

Diffuse ground glass (reticular) shadowing is suggestive of an interstitial lung disease.

Question 4: E

Three quarters of inhaled foreign bodies are radiolucent and therefore not visible on a chest xray.

Question 5: D

Recurrent pneumonia, lung abscesses, pneumothorax and bronchiectasis are all recognised complications of FB inhalation but not tuberculosis.

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A CASE OF BRONCHIOLITIS WITH MNEMONICS TO GUIDE MANAGEMENT DECISIONS

SJ Farrelly, DK Luyt

Abstract

We present a case of acute bronchiolitis in a child who required admission to our high dependency unit for treatment with heated humidified high-flow nasal therapy. We discuss the spectrum of the presentation of bronchiolitis with emphasis on guiding an inexperienced paediatric junior doctor in decisions on whether to admit to hospital and when to escalate the supportive management in hospital.

Case presentation

A 5 month old boy presented to his local accident and emergency department with increasing breathlessness. He had developed coryzal symptoms with a cough for four days previously. His parents reported that over the 24 hours preceding presentation, his cough had become persistent and they noted his abdomen was 'sucking in' when he was breathing. These together with diminished feeding had triggered the visit to A&E. This was his first episode of illness. He was born at term following an uneventful pregnancy. There were no concerns with feeding or development and his immunisation were up to date. There is no smoking in the home and his father has asthma.

Observations at presentation were a heart rate of 171 beats/minute, respiratory rate of 52 breaths/minute, oxygen saturations of 88% in air, and a temperature of 36.8°C. He was alert and active yet agitated. His chest inspection revealed moderate to severe subcostal and intercostal recessions, sternal recession and a tracheal tug. Chest auscultation revealed reduced air entry bilaterally but no crackles or wheeze.

Cardiovascular system examination was normal with normal heart sounds and palpable good volume femoral pulses. His abdomen was soft on palpation with no evidence of an enlarged liver. A capillary blood gas showed respiratory acidosis with pH 7.290 (7.350 – 7.450), PCO_2 7.37 kPa (4.70-6.40) and lactate 2.4 mmol/L (0.6-1.4).

Initial management involved administering face mask oxygen at 1 litre/minute and commencing intra-venous fluids as he was too breathless to feed. His condition did not improve with these measures. He remained tachypnoeic with a respiratory rate persistently above 50 breaths/minute, his oxygen saturations remained around 85% and he continued to have to work very hard to breathe. At this point the decision was made that the child required further respiratory support and would need to be transferred to the high dependency unit (HDU).

A chest radiograph was performed to exclude complications, particularly atelectasis, that could contribute to the severity of his clinical presentation. His image (figure 1) was however characteristic of uncomplicated bronchiolitis with hyperinflated lungs with bilateral perihilar inflammatory change. A nasopharyngeal aspirate confirmed bronchiolitis as it tested positive for respiratory syncytial virus type A.

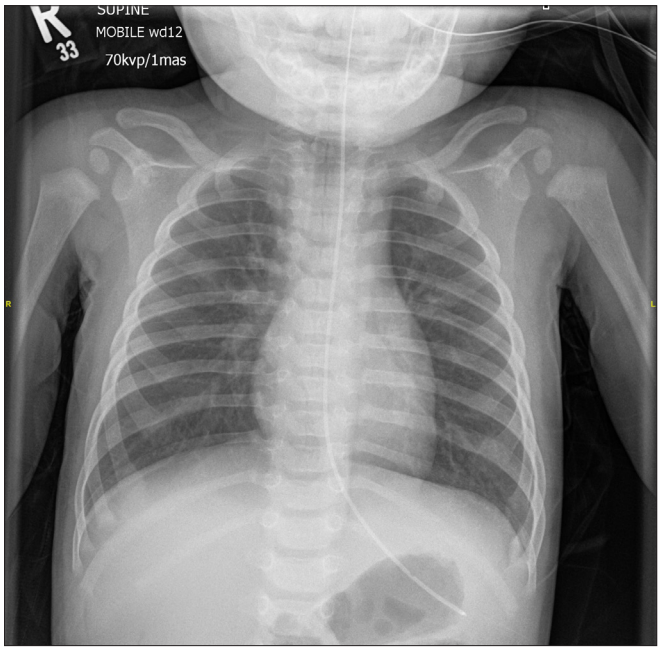

Figure 1: Chest radiograph from the infant.

In HDU, treatment was initiated with heated humidified high-flow nasal therapy at a flow of 8 litres/minute and FiO_2 of 48%, a nasogastric tube was passed and 0.9% saline nebulisations were prescribed for as needed use. His respiratory status improved as he appeared less distressed and his oxygen saturations were maintained at 92%.

Over the next 24 hours with further improvement as evidenced by improved oxygenation on a lower FiO2 and decrease in respiratory rate and work of breathing, intravenous fluids were discontinued and naso-gastric feeds started. Over a further 24 hours he was weaned from the high-flow nasal therapy and oral feeds were re-established. He spent 4 days in hospital.

Discussion

Bronchiolitis is a viral lower respiratory tract infection which predominantly affects children between three and six months of age. As with most viral respiratory infections, symptoms (and therefore potential severity) peak between day 3 and 5 of the illness.

This poses an unusual challenge to clinicians assessing affected infants as the severity at presentation may vary not only because of patient factors and intensity of infection but also because of timing in illness evolution. Indeed, on the very same evening that this child was admitted, two other children were seen also with respiratory syncytial virus confirmed bronchiolitis.

A CASE OF BRONCHIOLITIS WITH MNEMONICS TO GUIDE MANAGEMENT DECISIONS

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A 6 month old girl was discharged after assessment and a 6 month old boy who was observed for a few hours to ensure oral intake was adequate before being discharged home. This is not an uncommon scenario facing junior doctors all over the UK in the winter. Hospital admissions for infants with bronchiolitis have been increasing, an estimate in 2011 stated that an annual rate of 46.1 per 1000 infants less than 1 year were admitted to hospital with bronchiolitis. (1)

There are therefore some children, like this case, who will not only require admission but also escalation in treatment to an HDU, whilst by contrast some do not even need hospitalisation. Making that correct decision can be difficult for an inexperienced doctor who may be facing bronchiolitis for their first season of it. There are no reliable investigations that aid that assessment, which is only likely to increase the doctor's anxiety levels.

The UK National Institute for Health and Care Excellence (NICE) and American Academy of Paediatrics recommend against routinely performing blood tests or chest radiographs in the assessment of bronchiolitis. (2-3) NICE do however provide good guidance of factors to consider when deciding on who to admit. (3) We propose a 'SAFER' mnemonic based on the NICE guidance to aid the decision-making process. Indications for admission are as follows:

S – Saturations:

Oxygen saturations <92% when breathing air.

A – Apnoea:

Both reported and observed apnoeic episodes.

F – Feeding:

Poor oral intake, defined by NICE as 50-75% of usual volume.

E – Extra factors:

These can broadly be split into two categories:

- Patient factors - *<3 months old, were born prematurely, have existing chronic lung disease, have a diagnosis of congenital heart disease, suffer from neuromuscular disorders or have an immunodeficiency.*

- Carer factors - *consider social circumstances, competence in being able to look after a child with bronchiolitis, the ability to spot red flag symptoms that their child is deteriorating to the point of requiring medical attention, how far away they are from access to healthcare in the event of deterioration.*

R - Respiratory distress:

On examination of the child always look out for grunting, moderate to severe chest recession or a respiratory rate of greater than 70.

As the hospital admission continues it is likely the clinical picture will change as the condition runs its course. This means that as well as the child recovering there is also the possibility of deterioration. This deterioration can be a secondary cause of anxiety to the inexperienced junior doctor called to review the child with bronchiolitis.

The decision of when to escalate care is again a clinical one. We have developed a second mnemonic 'SAD', based on indications for intensive care referral, to help with the clinical decision of when to escalate care to HDU and above. (4) These are:

S – Saturations:

failing to maintain > 92% with increasing oxygen support.

A – Apnoea's:

worsening episodes in either frequency or duration.

D – Deteriorating respiratory status and impending exhaustion:

A child who is persistently tachypnoeic and continually showing signs of marked respiratory distress is further indicator of the prompt need to escalate care.

Conclusion

We have described a case of bronchiolitis that needed escalation of treatment. We propose some memory aids that can guide trainee doctors to guide decision making in acute bronchiolitis. These will hopefully improve confidence in assessment and management and alleviate anxiety.

MCQ

1. Which virus is the most common cause of bronchiolitis?

A) Metapneumovirus

B) Rhinovirus

C) Respiratory Syncytial Virus

D) Coronavirus

E) Enterovirus

A CASE OF BRONCHIOLITIS WITH MNEMONICS TO GUIDE MANAGEMENT DECISIONS

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2. If it is indicated that an infant should receive palivizumab prophylaxis, what is the usual regimen for the prophylaxis?

A) 5 doses, one dose a month

B) 10 doses, one dose a month

C) 5 doses, one dose every 2 weeks

D) 10 doses, one dose every 2 weeks

E) 10 doses, one every 1 week

3. A 4 month old boy is seen on the admissions unit with a 2 day history of cough and coryzal symptoms. Parents report he isn't feeding as well as he normally does, on further discussion this is difficult to quantify. His oxygen saturations are 96% in air and on-examination there is no respiratory distress and the chest sounds clear. The rest of the examination is otherwise normal. The boy was born at term and is otherwise fit and well. What would be the most appropriate step in his management from the options below?

A) Contact the intensive care registrar to discuss about a potential admission

B) Discharge home immediately

C) Admit to the unit to monitor the infants feeding, perform some routine blood tests and a chest x-ray before deciding on discharging home

D) Admit to the unit to monitor the infants feeding before deciding on discharging home

E) Admit to the ward for an overnight stay regardless of how well the infant feeds over the next few hours

Answers

1. C: *Respiratory syncytial virus is the most frequent cause of bronchiolitis with the rest being due to other viruses such as; human metapneumovirus, rhinovirus, adenovirus, enterovirus. (2)*

2. A: *Infants who qualify for palivizumab should receive a maximum of 5 monthly doses or in some cases less doses if the end of the RSV season comes before the 5 monthly doses are administered. (2)*

3. D: *From the above information the infant does not seem to be in respiratory distress, he seems to be able to saturate well in air and there is no mention of any apnoea in the history. There does not seem to be any co-morbidities in his history. There is a lack of clarity on how well the infant is feeding and for that reason it would be wise to admit for a period of observation to ensure there is adequate oral intake. Further investigations such as blood tests and chest radiographs are not indicated at this point. If after observation the infant is feeding well and the clinical picture does not change then he could potentially go home, so he does not need overnight admission straightaway and there is no indication to involve the intensive care team at present.*

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A REVIEW OF COMMUNITY ACQUIRED PNEUMONIA & THE MANAGEMENT OF ITS COMPLICATIONS

O Baker, M Avari, K Pannu, DK Mukherjee

Abstract

Respiratory symptoms are a common reason for patients to present to the emergency department. Acute symptoms of cough, purulent sputum, shortness of breath and fever combined with new focal chest signs on examination and no other explanation for symptoms define pneumonia (1).

Pneumonia is a common presentation: as a junior doctor it is useful to know the management of this condition and be able to identify and manage the complications.

Case Presentation

A fit and well 37-year-old gentleman, a window-cleaner by trade, was admitted feeling generally unwell for one week with green productive cough. He complained of left sided pleuritic chest pain but no haemoptysis. Of note, his mother had flu-like symptoms the week previously. He is a non-smoker with no previous respiratory disease.

On admission, his vitals showed a respiratory rate of 26 breaths/minute, oxygen saturations of 86% on room air, pulse rate of 125 beats/minute, blood pressure 111/70 and a temperature of 37.9˚C. Electrocardiogram showed sinus tachycardia and his blood tests showed raised inflammatory markers with a C-reactive protein of 451 mg/L and urea 8.8. Sepsis protocol was initiated.

As viral pneumonia could not be excluded anti-viral treatment (Tamiflu®) was started alongside antibiotics. Arterial blood gas (ABG) on 15 litres of oxygen showed a significantly increased Alveolar-arterial (A-a) gradient along with a raised lactate (pH 7.44, $PaCO_2$ 4.49, PaOÐ 10.28, HCO3- 22.5, BE -0.8, lactate 7.3). His chest x-ray (CXR) showed left sided pleural effusion and right sided consolidation (figure 1).

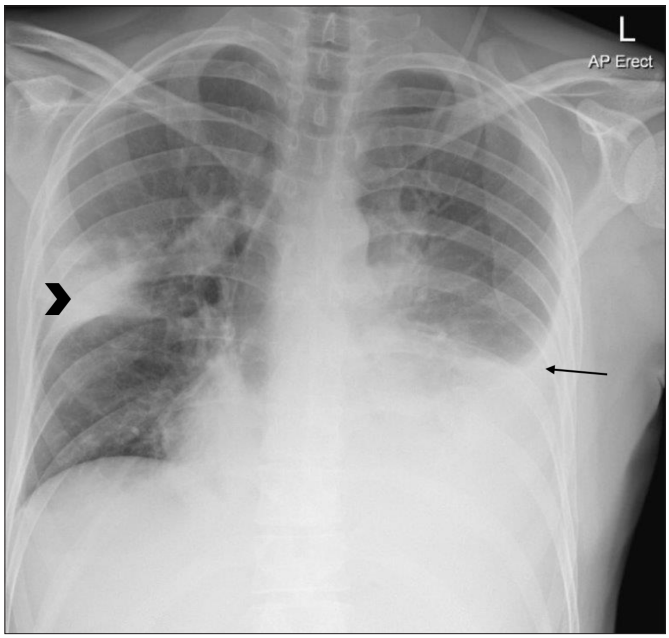

Figure 1: Anteroposterior CXR showed left sided effusion (black arrow pointing to meniscus) and right sided consolidation (black chevron)

Within 24 hours, the patient's oxygen requirements continued to increase and optiflow was required as the patient was getting more hypoxic. CXR was repeated (figure 2).

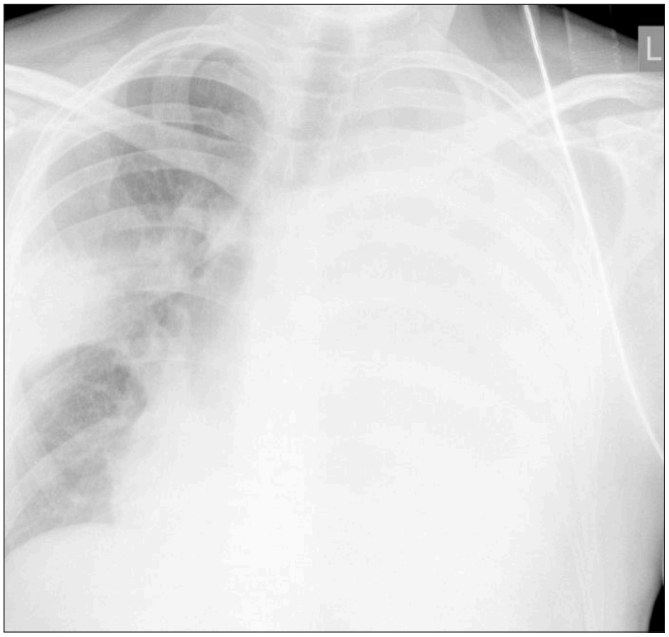

Figure 2: CXR shows progression of left sided effusion within 24-hour period.

Diagnostic pleural fluid was sent for analysis; pH was 6.85 and turbid fluid present indicating an empyema hence a 12F chest drain was inserted. Further analysis showed that pleural fluid was exudative using Light's Criteria and acid-fast bacilli was negative. Blood cultures taken on admission and pleural fluid grew beta-haemolytic streptococcus group A. Therefore, antibiotics were changed to benzylpenicillin and clindamycin on microbiology advice. Viral swab and high-risk screening bloods returned negative.

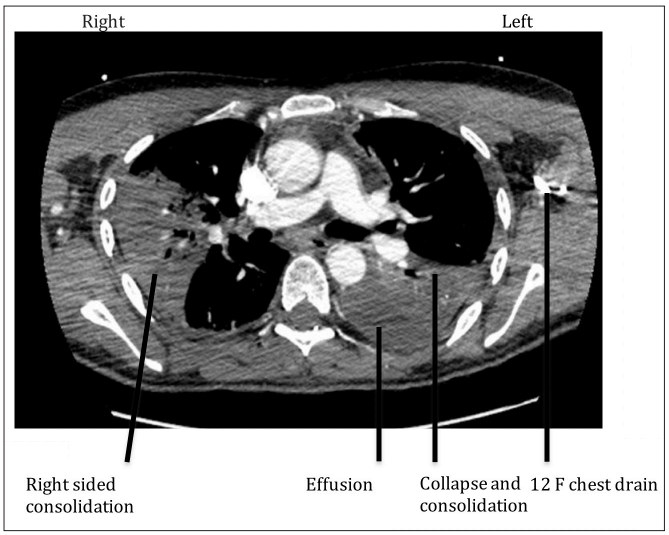

Figure 3: Chest CT scan post drain insertion, which shows left effusion, collapse and consolidation and right sided consolidation

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A bedside ultrasound scan of his chest was done which showed loculated pleural fluid. In light of this multiloculated effusion, a discussion was held with cardiothoracic surgeons and a repeat CT ordered. The new CT showed resolution of the right upper lobe consolidation but a new right sided pleural effusion. It also showed increased left basal empyema (figure 4).

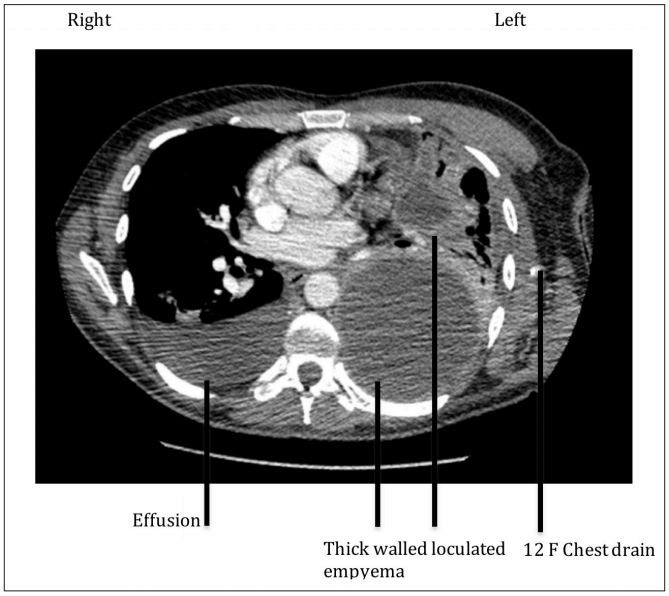

Figure 4: Second CT chest showing right sided effusion and loculated empyema

Another chest drain was inserted which drained the remaining loculated fluid. The patient was discharged with a total 6 weeks of antibiotics and follow-up in clinic with repeat CXR.

Discussion

There are many different types of pneumonia such as community acquired (as in this patient), hospital acquired, atypical (Legionella, mycoplasma and pneumococcal), ventilator associated, viral and fungal.

1. CURB65

Once a diagnosis of CAP is made the CURB65 score can be used to assess mortality and subsequently guide treatment (1, 2) (Table 1). Each criterion is one point - confusion (abbreviated Mental Test score 8 or less,) raised blood urea nitrogen greater than 7mmol/L, respiratory rate >30bpm, low blood pressure <90/60 and age 65 years or more.

CURB65 score	Mortality	Treatment
0 to 1	Low: <3%	Consider home treatment, 5 days PO antibiotics
		Amoxicillin Penicillin allergy = macrolide or tetracycline
2	Moderate: 9%	Hospital antibiotics oral or I.V. 7-10 days
		Amoxicillin + macrolide Penicillin allergy = doxycycline + macrolide
3-4	High: 15-40%	Hospital antibiotics I.V. 7-10days
		Co-amoxiclav + macrolide Penicillin allergy = glycopeptide + macrolide

Table 1: Treatment of CAP using CURB65 score.

In our case, even though the patient was severely septic, the calculated CURB65 was only 1. It is important to remember clinical judgment must be used on an individual basis when deciding best treatment.

If, after suitable treatment, persistent pneumonic consolidation remains other differential diagnoses and biopsy should be considered. Underlying conditions such as lung cancer (adenocarinoma in figure 5) or eosinphilic pneumonia (figure 6) may be the cause.

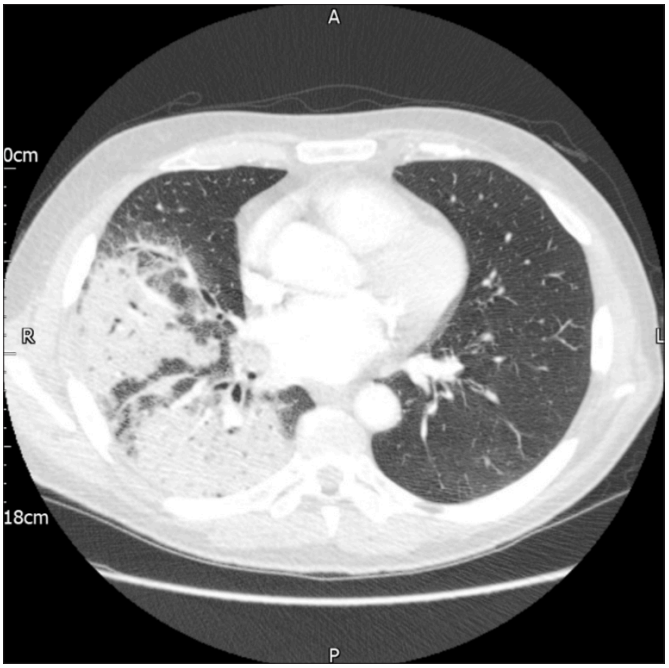

Figure 5: CT scan showing left Upper Lobe adenocarcinoma.

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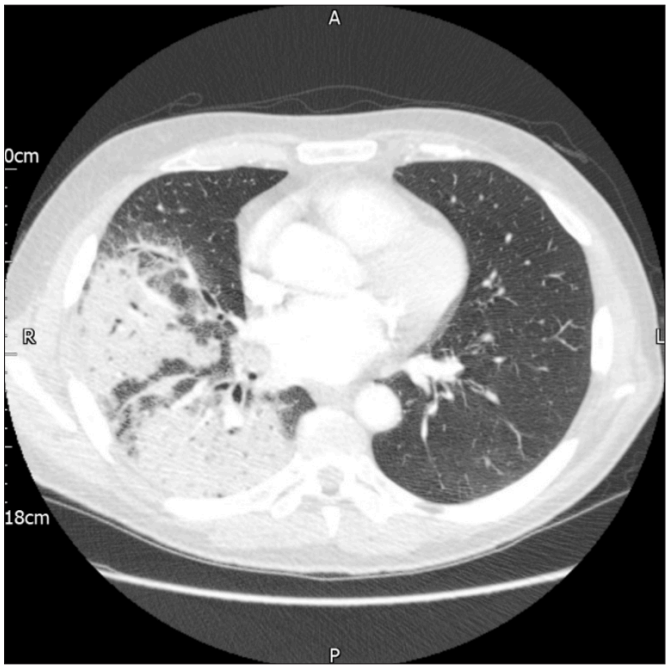

Figure 6: CT scan showing eosinophilic pneumonia.

2. Respiratory failure

One of the most common complications of pneumonia is type I respiratory failure. Type I respiratory failure is hypoxia (PaO_2 <8kPa) with low or normal PCO_2, whereas type II respiratory failure is both hypoxia and hypercapnia ($PaCO_2$ >6.5) (3). The hypoxaemia in type I results most commonly from ventilation/perfusion (V/Q) mismatch and also right-to-left shunts: both of which show an increased A-a gradient. (Table 2) (4).

V/Q mismatch	Right-to-left shunt
Pneumonia	Lobar Pneumonia
Pulmonary Embolism	Adult Respiratory Distress Syndrome (ARDS)
Obstructive lung disease	Congestive cardiac failure
Atelectasis	
Pneumothorax	

Table 2: Causes of type1 respiratory failure.

Type 2 respiratory failure can be due to obstructive lung diseases (COPD, life threatening asthma,) obstructive sleep apnoea, obesity hypoventilation syndrome, neuromuscular problems, chest wall deformities, reduced breathing effort (opioid toxicity)

3. Pleural effusion

Fluid in the pleural space occurs in over forty percent of patients diagnosed with a bacterial pneumonia (5). It can be detected on examination by decreased chest expansion and breath sounds with a stony dull percussion note. Radiologically a meniscus will be seen on CXR (figure 1) but ultrasound has the highest sensitivity.

Unilateral pleural effusions, as is often the case in parapneumonic effusions, need an early diagnostic pleural tap and if turbid in consistency or a pH <7.2 a chest drain should be inserted. The pleural aspiration should be sent to biochemistry for pH, glucose, LDH and protein levels.

It should also be sent to cytology, microbiology and for acid fast bacilli culture. Light's Criteria is used to indicate the cause of the effusion – in pneumonia the fluid is exudative and results from increased pleural capillary leak (4). Analysis of the pleural effusion fluid will allow treatment of the underlying condition. Junior doctors should remember to take venous blood cultures for microbiological analysis as this can increase diagnostic yield.

4. Empyema

The presence of frank pus in the pleural space and, as seen in our patient, a pleural fluid pH of <7.2 with positive microbiology confirms empyema (6). A key differential to rule out is lung abscess.

Since the 1850s serial therapeutic thoracocentesis has been used to treat pleural effusions and empyema however today the main stay of treatment is chest drain insertion and antibiotics (7). Treatment today should involve quick, effective drainage with a chest drain to improve outcome and need for invasive procedures (8).

Our patient had a 12 F chest drain inserted using Seldinger technique under ultrasound guidance – this smaller size has been shown to be just as effective as larger bore drains even in the presence of pus (9, 10, 11). As a junior it is important to remember to review and flush these smaller drains with normal saline four times a day to avoid blockage. Empyema and other complicated pleural effusions can also be treated by intrapleural therapy with DNase and tissue plasminogen activator (12).

Referral to a thoracic surgeon should be considered for loculated empyemas but there is no objective criterion for when to refer (5).

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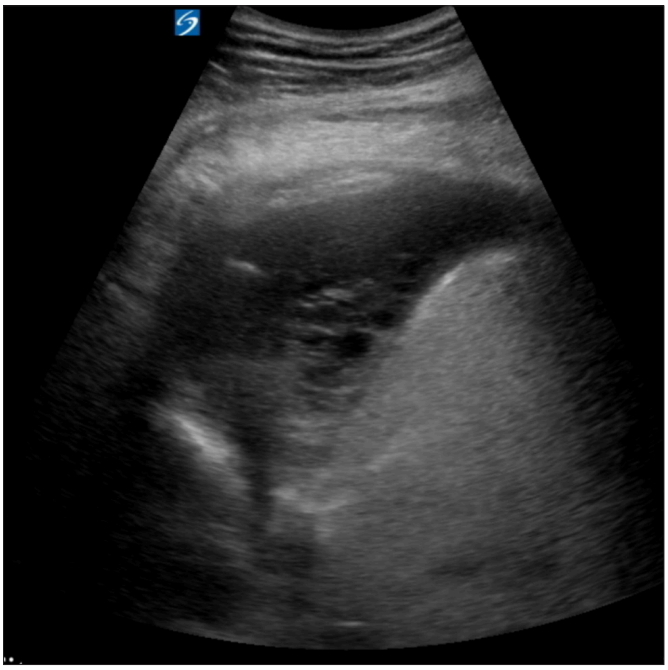

Figure. 7: Ultrasound Scan showing loculated empyema.

Multiple Choice Questions

1. What is the most common microorganism causing community acquired pneumonia?

a. Mycoplasma pneumoniae

b. Pseudomonas aeruginosa

c. Haemophilus influenza

d. Staphylococcus aureus

e. Streptococcus pneumonia

2. A 65 year old is admitted with CAP. They are not confused but have a respiratory rate of 32 bpm, urea of 7.2mmol/L and BP 95/65. No known drug allergies. Which antibiotics are most suitable?

a. Macrolide

b. Macrolide and a beta-lactamase stable beta-lactam

c. Amoxicillin and a macrolide

d. Tetracycline

e. Glycopeptide and macrolide

3. How many weeks after treatment for CAP should a repeat CXR be arranged if symptoms persist?

a. 2

b. 3

c. 5

d. 6

e. >10

4. When aspirating a pleural effusion what maximum volume does the British Thoracic society suggest aspirating in one attempt?

a. 0.5L

b. 1.0L

c. 1.5L

d. 2.0L

e. 3.0L

5. Hospital acquired pneumonia (HAP) can be diagnosed after how many hours stay in hospital?

a. 12

b. 24

c. 48

d. 62

e. 72

Answers:

1. E

Streptococcus pneumonia can be diagnosed using urinary antigens. In COPD, haemophilus influenza is most common; and in Hospital Acquired Pneumonia (HAP) it is pseudomonas.

2. B

For a CURB score of 3 or above NICE recommend a beta-lactamase such as co-amoxiclav and macrolide such as clarithromycin for 7 to 10 days IV (2).

A REVIEW OF COMMUNITY ACQUIRED PNEUMONIA & THE MANAGEMENT OF ITS COMPLICATIONS

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3. D

BTS guidelines suggest 6 weeks if symptoms persist or there is a high risk of underlying malignancy (1).

4. C

1.5L. This is to prevent rapid lung re-expansion – causing the patient to experience sudden shortness of breath. One of the most serious side effects is re-expansion pulmonary oedema and can occur if >3L is drained quickly (13).

5. C

HAP can be diagnosed after a patient has been in hospital for more than 48 hours. Antibiotics should cover gram-negative organisms and there should be high suspicion of pseudomonas.

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AMNIOTIC FLUID EMBOLISM

J Olanrewaju, H Moudgil

Abstract

Amniotic fluid embolism (AFE) is a very rare but potentially fatal obstetric emergency recognised as the fifth most frequent cause of direct attributed maternal death. Although most often diagnosed at the exclusion of more common causes of acute lung injury, it has significant pulmonary and systemic complications and should remain among differentials of a sudden respiratory complication particularly during late obstetric care. This case illustrates risks as well as clinical and radiological evidence supporting this important diagnosis.

Case Illustration

A 32 year old UK born Pakistani lady, para 4+3, not currently employed and continuing to smoke 4 cigarettes/day over a 12 year history presented to the labour ward after a Spontaneous Rupture of Membrane (SROM) (at 37 +2 weeks) and was admitted for a category 3 (earlier than planned delivery but without current evident maternal or foetal compromise) caesarean section (C-section) under spinal anaesthesia.

She had a past medical history of stable asthma controlled with as required salbutamol inhalers and further admitted her occasional use of salbutamol included for anxiety and panic attacks. There had been no prior identified allergies and she didn't know her previous best or predicted peak flow readings.

She was otherwise fit and well and had experienced no major complications during her current pregnancy but had attended her General Practitioner two weeks earlier and prescribed a course of antibiotics (Amoxycillin) for a clinically diagnosed "chest infection". Her previous successful pregnancies had been delivered by caesarean section and on this occasion there were adhesions in the lower uterine segment between the bladder and anterior abdominal wall.

The C-section went well with no record of foetal distress but with an estimated maternal blood loss of 1000mls. Approximately three hours post-operatively the patient abruptly started complaining about wheeze which was not relieved with salbutamol nebulizers. Her Early Warning Score (EWS) was 0, with Heart Rate 89 beats/minute, respiratory rate 20/minute, Blood Pressure 96/54 and oxygen saturation at 96% on room air. She was reviewed by the junior doctor on call, and apart from the expected mild hypotension did not appear to be in respiratory distress.

The patient was subsequently reviewed by the on-call medical registrar who documented normal heart sounds but diffuse bilateral wheeze and basal crackles. She was managed as exacerbation of asthma, and a bed-side chest radiograph (CXR) was requested. Target oxygen saturations were being met and EWS remained at 0. She was started on oral steroids and nebulized salbutamol bronchodilators along with intravenous (IV) antibiotic (amoxicillin).

Allowing for the AP projection, figure 1 illustrates her CXR taken 10 hours post-operatively and reported as normal. Her post operative blood counts showed the expected fall in haemoglobin to 71 grams/litre (compared to 115 g/L pre-operative) with haematocrit 21.4 (compared to 35.2) suggesting some additional haemodilution on replacement intravenous fluids. Platlet count was 147 x 10^9, white cells 14.1 x10^9/L (neutrophils 13.3), and clotting profile satisfactory with international normalised ratio (INR) 1.1 with prothrombin (PT) 12 seconds and partial thromboplastin time (PTT) 31 seconds. She was transfused 3 units of packed red blood cells.

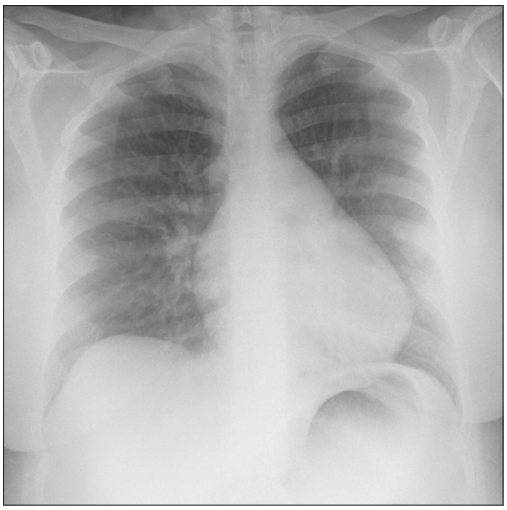

Figure 1: Initial bed-side CXR. Uncertain heart size with AP film but nil focal to lungs.

There was a progressive decline and by the following day general health had significantly worsened; her EWS was recored at 6 with tachycardia at 130 beats/minute, tachypneic at 30/min, temperature 38°C and oxygen saturation of 92% on air. Although still able to speak in full sentences, she remained wheezy, and she was continued on the earlier medications.

The patient's oxygen saturations continued to drop and she became more short of breath, pale and sweaty. With increasing oxygen demand up to 15 litres/minute, continued desaturations down to 65%, senior review was sought with the anaesthestist on call. Blood gases confirmed a type 1 respiratory failure with pH 7.36 pCO_2 5.32 pO_2 8.55 lactate 1.66 Bicarb 22.4 Base Excess -2.6. Inflammatory markers were raised with C-reactive protein (CRP) 297 U/ml, total white cell count 26.7.

Most recent sputums had been muco-purulent but had only identified a moderate growth of candida albicans. Updated CXR (figure 2) whilst still in the delivery suite showed the rapid change from earlier not thought related to intravenous fluid overload with extensive bilateral airspace and possibly interstitial shadowing. Antibiotics were changed to intravenous Cefuroxime and patient transferred to the High Dependency / Intensive Care Unit (HDU/ITU) for consideration of further ventilatory support.

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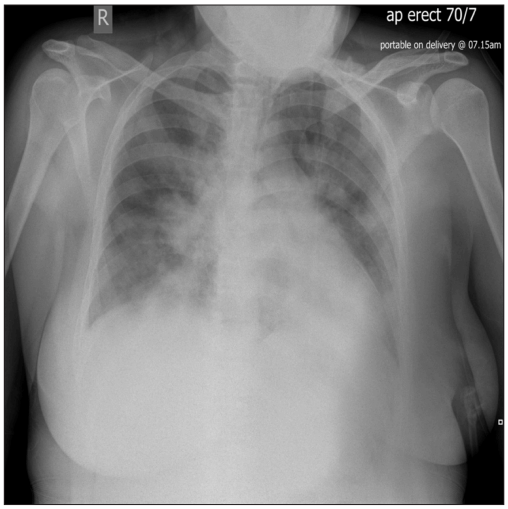

Figure 2: Extensive bilateral airspace and possibly interstital shadowing. Heart size and mediastinal assessment is less dependable with AP film.

She remained in HDU/ITU for the subsequent eight days. A chest CT pulmonary angiogram (CTPA) (figure 3) showed no acute pulmonary embolism but a mosacic pattern throughout with acute lung injury consistent with the clinical picture of an adult respiratory distress syndrome (ARDS) with some CT cuts showing an almost snow storm type appearance.

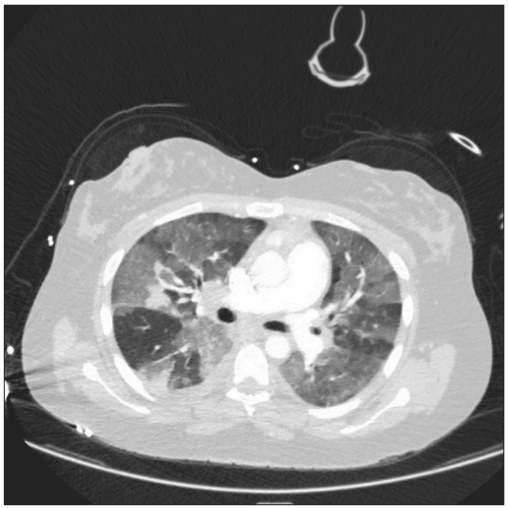

Figure 3: Chest CT scan, suggestive of Acute lung injury with mosaic pattern.

She was not intubated but remained on high concentrations of oxygen and for prolonged periods on non-invasive ventilation supports with trials on continuous positive airway pressure (CPAP) and Bi-Level positive airway pressure (Bi-PAP) and high flow nasal (NHF) oxygen eventually weaning started with FiO2 at 50%.

Prior to discharge back to a respiratory ward the oxygen dependency with targets between 94 to 98% had reduced to 1 litre/minute by nasal specks and inflammatory markers with CRP 65 and normal white count had improved. Only other positive microbiology throughout was Polymerase Chain Reaction (PCR) evidence for parainfluenza RNA positive but clinical scenario considered less likely to have contributed.

Subsequent CXR continued to improve (figure 4) but with some residual initial changes bilaterally. Clinical reviews three and then six months after that admission have continued to show improvement in general physical health but with continued patient concerns and anxiety related to her acute illness and for which she presently seeks conselling.

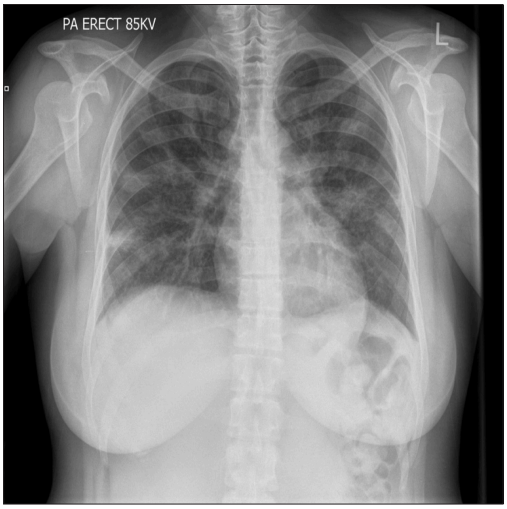

Figure 4: Improved films but with some patchy inflammatory change within both mid and some right lower zones.

Discussion

Amniotic fluid embolism occurs when foetal/placental materials and amniotic fluid surrounding the foetus in the uterus enter the mother's bloodstream [1]. Although rare, medical students and junior doctors are reminded of this important differential when mothers present unwell with respiratory complications during or shortly after pregnancy.

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Previous work through the Mothers and Babies: Reducing Risk through Audit and Confidential Enquires across the UK (MBRRACE-UK) showed a mortality rate of 0.33 per 100,000 pregnancies [2] with 11 women reported to have died because of AFE in the period 2009-2012 [2].

Most (70%) cases of AFE occur during labour, 19% during C-sections and 11% following vaginal delivery [3]; it has also been reported during early gestation, second trimester abortions, during amniocentesis, or following closed abdominal injury [1,2,4]. Other direct and indirect causes of maternal death are also listed in Box 1 and categories by risk assessment for C-sections to be performed listed in Box 2.

DIRECT	INDIRECT
ECLAMPSIA	ANAEMIA
PULMONARY EMBOLISM	HEART DISEASE
SEVERE SEPSIS	ANAESTHETIC COMPLICATION
AMNIOTIC FLUID EMBOLISM	HEPATITIS
PERIPARTUM HAEMORRHAGE	UNIDENTIFIED

Box 1: Most common direct and indirect causes of pregnancy related maternal deaths.

	RISK PROFILE
CATEGORY 1	Urgent threat to the life or the health of a woman or foetus
CATEGORY 2	Maternal or foetal compromise but not immediately life threatening
CATEGORY 3	Needing earlier than planned delivery but without currently evident maternal or foetal compromise
CATEGORY 4	At a time acceptable to both the woman and the caesarean section team, understanding that this can be affected by a number of factors

Box 2: Categories for C-section by risk profile dictating urgency.

Although not well understood, common pathology is from a breakdown in the placental barrier as from trauma and the foreign materials trigger the mother's immune system creating an anaphylaxis cascade to release products to cause an inflammatory reaction including disseminated intravascular coagulation or other coagulopathies alongside any respiratory distress with hypoxaemia and haemodynamic collapse [1,4].

Difficulty in diagnosis often arises out of non-specific features or those common to alternate presentations including acute breathlessness with pulmonary oedema without increased left heart pressures as with the adult respiratory distress syndrome (ARDS), hypotension, tachycardia, seizures, fever, altered mental state with confusion or anxiety, etc [1,2,4]. This then usually widens the initial diagnosis to include thrombotic pulmonary embolism, air embolism, fat embolism, aspiration of gastric contents with chemical pneumonitis, infection, and so on.

Among several risk factors implicated are an advanced maternal age over 35 years, Asian ethnic origin, multiparity, polyhydramnios, amniocentesis, as well as cervical laceration or uterine rupture alongside placenta previa or abruption as these can disrupt the physical barriers between mother and foetus, pre-eclampsia, operative delivery with C-sections, a forceps delivery or a vacuum extraction, but evidence on medically induced labour is conflicting [1,5,6].

Complications arise not only out of maternal (20% in developed counties) and infant deaths, but also through maternal brain injury secondary to low oxygen saturations and consequent lengthy hospital stay [1,3,5,6].

In comparison to those patients with pulmonary embolism after a C-section, the majority die within the first hour of onset of symptoms and about 85% of those who survive have permanent neurological impairment [1,3,5].

In this patient's case, the rapid change in clinical findings shortly after an otherwise unremarkable C-section provided the clinical diagnosis; the CTPA excluded acute thrombotic pulmonary embolism and although PCR was positive for parainfluenza RNA, the clinical presentation or rapid progression of disease did not support that diagnosis.

Any blood and intravenous fluid resuscitation was not excessive and not thought contributing. Specific risks with this patient apart from the C-section included her slightly increased age (usually more significant >35 years) and ethnic origins. The patient had been confused throughout most of her HDU/ITU stay but the residual anxiety presently being experienced at follow up is thought related to the stay rather than from any sustained neurological deficit.

In summary, this case illustration is of an important differential to consider particularly with respiratory problems with acute lung injury including ARDS in obstetric care. The diagnosis is one after exclusion of other pathologies but its natural history does follow a slightly different time course. Undetected, prognosis may be poor and most measures at treatment are largely supportive with aggressive oxygen and fluid balance management potentially through HDU/ITU units.

MCQ Self Assessment

1. What is the least likely risk factor for AFE?

(a) maternal age greater than 35 years
(b) increased amniotic fluid
(c) male foetus
(d) intravenous drug users
(e) uterine atony

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2. Which of the following is NOT a direct trigger for Acute Lung Injury?

(a) pneumonia, high altitude, pulmonary contusion
(b) pneumonia, reperfusion injury, drowning
(c) aspiration of gastric content, fat/amniotic embolism, pneumonia
(d) pneumonia, pancreatitis, sepsis
(e) All above are correct

3. What potential causes post-partum collapse ?

(a) Ergometrine
(b) amniotic fluid embolism
(c) uterine rupture
(d) lack of steroids
(e) intracranial haemorrhage

4. What is the most useful investigation in confirming a diagnosis of AFE?

(a) Arterial blood gases
(b) Chest radiograph (CXR)
(c) CT Thorax
(d) Pulmonary Blood sampling
(e) D-Dimer

Answers

1. (d)

No documentation of IVDU causing AFE; other answers all correct.

2. (d)

Pancreatitis and sepsis are less common indirect causes of acute lung injury.

3. (a)

Amniotic fluid embolism may cause collapse in labour or after delivery. Lack of steroid cover may occur in patients who have had their adrenals suppressed through steroid use. Ergometrine may cause the blood pressure to rise, but does not cause collapse.

4. (d)

All answers correct but single best is that pulmonary blood samples may show the presence of squamous cells coated with neutrophils and presence of foetal debris. CT scans indicate a picture of ARDS but cannot confirm the diagnosis.

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AN APPROACH TO ACUTE HYPERCAPNIC RESPIRATORY FAILURE

S Faber, H Makker

Abstract

First presentation of acute hypercapnic respiratory failure (AHRF) in the absence of a known pre-existing respiratory disorder can be a diagnostic challenge. Although COPD is a well recognised cause, Obesity Hypoventilation Syndrome (OHS) is being increasingly recognised as a leading cause of acute hypercapnic respiratory failure. Hence clinicians need to recognise how to manage such patients acutely.

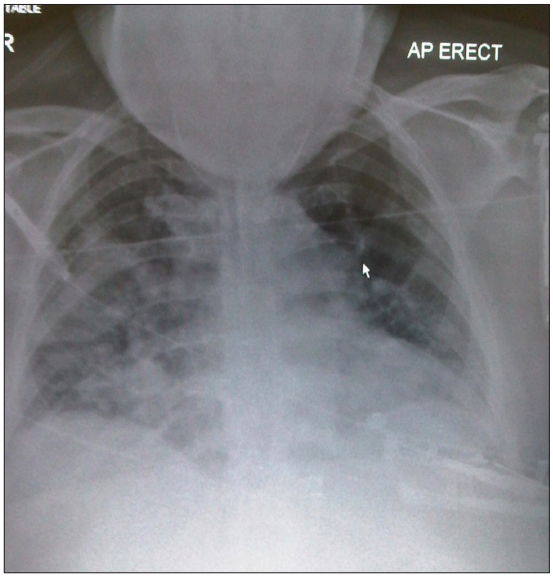
Case One

A 64 year old obese man is brought in via ambulance to accident and emergency department because of breathlessness, orthopnoea and swollen ankles. He was recently commenced on frusemide by his GP. He has diet-controlled diabetes and is a lifelong non-smoker. He is tachypnoeic RR 30/min, tachycardia HR 113/min, requiring high-flow oxygen to maintain spO_2 97% and his BP 168/88mmHg.

He is unwell, plethoric, and has bi-basal crepitations and ankle oedema. Intravenous frusemide and a GTN infusion is administered for presumed heart failure. An arterial blood gas (ABG) is taken on 8 litres of oxygen (see table).

Inspired oxygen concentration is reduced to 35% via Venturi mask.

The chest x-ray is difficult to interpret (see image). The Foundation Trainee suspects heart failure with decompensated type 2 respiratory failure, and refers to the on call medical registrar.



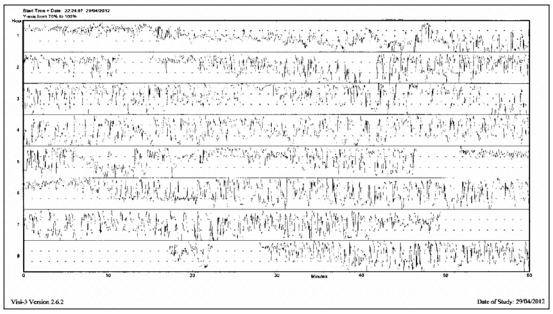
The registrar reviews the chest x-ray and notes consolidation in the right lower zone, but no pulmonary oedema. Blood tests reveal a raised white cell count of 18 and CRP 126. A previous echocardiogram performed four months ago shows normal left ventricular function but a raised pulmonary arterial pressure of 62mmHg (normal 15-30).

Further questioning reveals a long history of weight gain, recurrent headaches and daytime somnolence. Decompensated type 2 respiratory failure secondary to OHS and OSA, precipitated by pneumonia is diagnosed. The patient continues to look unwell and is desaturating despite controlled oxygen. The patient is commenced on bilevel positive airway pressure ventilation (BiPAP) and administered intravenous antibiotics.

One hour later the patients reports feeling much better and a repeat ABG shows a resolving respiratory acidosis.

Arterial Blood Gas	Initial reading	1 hour into BiPAP	4 days later
pH	**7.19**	**7.33**	**7.45**
pCO2	**13.4**	**9.9**	**7.2**
pO2	**14.2**	**7.7**	**7.7**
HCO3	**37.7**	**38.2**	**37.5**
BE	**6.9**	**11.1**	**11.0**

After four days the acidosis has resolved, although he remains hypercapnic. BiPAP is gradually discontinued. One week later, a sleep study confirms severe OSA with OHS. The patient is commenced on domiciliary CPAP. Repeat echocardiogram confirms pulmonary hypertension.



Overnight oximetry from patient's Sleep Study showing frequent desaturations during sleep.

Discussion

How can this ABG be interpreted?

This initial ABG indicates an acute on chronic hypercapnic respiratory failure because the bicarbonate levels are elevated suggesting longstanding pre-existing hypercapnia. This should make clinicians consider underlying causes such as COPD, OHS, neuromuscular and chest wall disorders. The acidosis implies decompensation from an acute insult.

What evidence is there of an underlying cause?

AHRF is commonly associated with acute exacerbations of COPD. Such patients will usually have a history of chronic progressive respiratory illness and previous exacerbations. This patient has never smoked and appears to have no history of previous respiratory illness, making COPD unlikely.

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Neuromuscular disorders will commonly present with other neurological features, particularly bulbar weakness. These are not mentioned here but should form part of the assessment. Given that the patient is obese and and gives a history of OSA symptoms, OHS should be strongly considered. There is evidence of pulmonary hypertension on a previous echocardiogram which is suggestive for an underling respiratory or chest wall disorder, including OHS.

What has precipitated this presentation?

Initially the A&E doctor suspects heart failure. Certainly the presenting symptoms are suggestive, however a recent echocardiogram showed good left ventricular function and the ECG shows only sinus tachycardia. Some fluid overload would not be surprising in OHS, however there is consolidation in the right lower zone, and together with a leukocytosis and raised CRP, pneumonia is probably the predominant feature.

How should the Acute Hypercapnic Respiratory failure be initially managed?

The respiratory acidosis is initially severe (pH 7.19) and the A&E doctor appropriately reduces the inspired oxygen concentration using a Venturi system. Chronic hypercapnia indicates the patient probably relies on lower oxygen levels to maintain respiratory drive and hence over-oxygenating could worsen the acidosis.

Oxygen should be prescribed to aim for lower target saturations (88-92%) whilst antibiotics are hastily initiated. The patient's response to treatment needs to be assessed very soon after. This implies reviewing any changes in clinical signs and symptoms, and ideally repeating an ABG. In this case the patient remains unwell and is desaturating on controlled oxygen, therefore a repeat ABG may not have changed management.

Is Non-invasive Ventilation (NIV) indicated?

The patient has a severe acidosis and clinically has not improved despite controlled oxygen and antibiotics, therefore he needs assisted ventilation. The guidance on whether to use non-invasive ventilation versus mechanical ventilation in this type of scenario is not totally clear.

Pneumonia is not in itself considered an indication for NIV because of the risks of treatment failure, however it is advised in patients with AHRF with OHS, the same way as it is in COPD exacerbations1. In this case, particularly as the pH is less than 7.25, the patient should have an urgent review by Intensive Care, but NIV should certainly be started in the meantime.

Subsequent Management

Repeat ABG after one hour shows a resolving acidosis. NIV should be continued until pH has normalised and ideally the pCO_2 is less than 6.5, however in some patients with pre-existing chronic hypercapnia this might not be possible. Diagnosis of the underlying cause of chronic hypercapnic respiratory failure will focus on excluding chronic lung disorders, neuromuscular and other chest wall disorders. In this case, a subsequent sleep study is helpful in confirming OSA and OHS. This patient must be discussed with the local home ventilation service before discharge as he is likely to require domiciliary NIV or CPAP.

Acute Hypercapnic Respiratory Failure in Obesity Hypoventilation Syndrome

Obesity Hypoventilation Syndrome is the combination of obesity and chronic daytime hypercapnia, in the absence of other causes of hypoventilation. The majority of these patients (approximately 90%) will also have OSA, which is the principal cause of chronic daytime hypercapnia, in addition to the mechanical effects of obesity (2). With the obesity pandemic continuing to grow, the acute complications of OSA and OHS are becoming increasingly recognised in medical settings.

For various reasons, OHS is often diagnosed late in its course, when patients have developed end-stage complications such as right heart failure (3). It is becoming increasingly common to diagnose OHS following a patient's first presentation with AHRF. Such patients will often reveal a more indolent history of OSA symptoms (poor quality sleep, snoring, witnessed apnoeas, daytime somnolence, morning headaches). In COPD, an acute decompensation is usually secondary to an infective precipitant, however when OHS patients present with AHRF the precipitant is not always clear. Infection and fluid overload should always be considered as potential precipitants.

It is always important to consider OHS in an obese patient with their first presentation of AHRF who has never smoked. OHS is sometimes misdiagnosed as COPD or CCF4. One should be mindful however that COPD and OSA/OHS commonly co-exist.

Arterial blood gas analysis will typically show an acute on chronic hypercapnic respiratory failure with a raised bicarbonate. Other blood tests and chest radiography may not be that helpful unless there is an obvious acute precipitant such as infection or fluid overload.

The initial management of AHRF in OHS should follow that of AECOPD with controlled oxygen therapy and treatment of any obvious precipitant. This may include antibiotics, diuretics, and steroids/bronchodilators if concomitant COPD. All patients with AHRF should be promptly referred to the on-call medical registrar as they will require regular clinical review. Non-invasive ventilation is indicated as it is in acute exacerbations of COPD (AECOPD) i.e pH < 7.35 and pCO_2 > 6.5 despite controlled oxygen therapy and treating reversible causes (1).

However the threshold for starting NIV may be lower if patients are particularly somnolent with purely a raised pCO_2 and normal pH. In OHS patients, it is not uncommon that they will require relatively high inspiratory and expiratory pressures to improve the hypercapnia, however achieving such high pressures should only be managed by someone highly trained in NIV. Contraindications to NIV apply as they do in COPD1. Similarly, urgent referral to ITU should be made in peri-arrest situations, severe acidosis (pH < 7.15), or if NIV is failing.

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NIV should be gradually discontinued as it is in AECOPD and ongoing management should then focus on excluding other causes of AHRF. If a sleep study is available this can help identify OSA and hypoventilation. All such patients should be discussed with the local home ventilation service as they are likely to require early setup on domiciliary NIV. They should also be offered support and advice on weight-loss.

Indications for Non-invasive Ventilation in Obesity

pH < 7.35, pCO_2 > 6.5, RR > 23

OR

daytime pCO_2 > 6.0 and somnolent

Contraindications to NIV

ABSOLUTE	*Severe facial deformity*
	Facial burns
	Fixed upper airway obstruction
RELATIVE	*pH < 7.15*
	pH < 7.25 and additional adverse feature
	GCS < 8
	Confusion/agitation
	Cognitive impairment

Assessment Questions

1. A morbidly obese patient is being investigated for suspected OSA and OHS. Aside from demonstrating that the patient has chronic daytime hypercapnia what other information is essential in reaching a diagnosis of OHS?

a) A long history of OSA symptoms
b) An echocardiogram confirming pulmonary hypertension
c) The absence of other causes of hypoventilation
d) Spirometry confirming a restrictive airflow pattern
e) A recent hospital admission with acute hypercapnic respiratory failure

2. A 67 year old obese male is brought into A&E via ambulance having been found collapsed and short-of-breath at home. GCS is 14/15. He is drowsy but rousable. His daughter reports he normally uses a CPAP mask at night.

Respiratory rate is 30/min, oxygen saturations 98% on 15 litres oxygen via non-rebreathe mask. Chest x-ray shows clear lung fields. An ABG is taken and shows pH 7.27 pO2 16.4 pCO2 10.2 bicarb 38.3 BE 6.3. What should be the immediate management to improve this patient's respiratory failure?

a) Turn the oxygen off completely
b) Commence Non-invasive ventilation
c) Call the ITU registrar to intubate the patient
d) Adjust the oxygen to deliver 28% oxygen via a Venturi mask
e) Continue high flow oxygen and repeat the ABG in 1 hour

3. A 59 year old man is bought in via ambulance with a history of breathlessness and reduced conscious level. Clinic letters reveal he was previously diagnosed with OSA and OHS and he uses a CPAP mask at home.

On arrival GCS 8/15, RR 30 spO_2 96% on 10 litres oxygen, HR 115/min BP 100/42. Chest x-ray shows dense consolidation in the right lung. ABG on 10 litres oxygen shows pH 7.11 pO_2 7.3 pCO_2 13 bicarb 33.3 BE 3.2. What is the correct oxygen delivery system in this acute situation?

a) Oxygen via a Bilevel positive airway pressure mask
b) Oxygen via a venturi mask
c) High flow oxygen via a non-rebreathe mask
d) Oxygen via a Continuous positive airway pressure
e) Oxygen via an endotracheal tube and mechanical ventilation

4. Which of these treatment options will not usually form part of the initial management of a patient with OHS presenting with acute on chronic hypercapnic respiratory failure?

a) Intravenous diuretics
b) Intravenous antibiotics
c) Controlled oxygen therapy
d) Continuous positive airway pressure
e) Bilevel positive airway pressure

5. A patient has been successfully weaned off Bipap following their first presentation with acute hypercapnic respiratory failure and has been diagnosed with severe OSA with OHS. Which of these is the most important intervention before organising their discharge?

a) Referral for bariatric surgery
b) Referral for rehabilitation and exercise programme
c) Referral to local Home Ventilation service
d) Referral to Smoking cessation
e) Referral for lung function testing

AN APPROACH TO ACUTE HYPERCAPNIC RESPIRATORY FAILURE

S Faber, H Makker

Assessment Answers

Question 1, answer c.

OHS is a diagnosis of exclusion. It can therefore only be diagnosed if there is chronic daytime hypercapnia without evidence of other underlying causes of hypoventilation such as obstructive airway disorders, neuromuscular disorders, and chest wall disorders. The other options are supportive of OHS but are by no means diagnostic.

Question 2, answer d.

The ABG confirms an acute on chronic hypercapnic respiratory failure which suggests the patient is oxygen sensitive, perhaps from underlying OSA and OHS. This patient requires controlled oxygen therapy at a reduced concentration via a Venturi mask. The ABG should be repeated within the next hour to see if the hypercapnia and acidosis has improved.

NIV may still be required but only after a trial of controlled oxygen and treatment of any acute precipitating cause of ventilatory failure. Intubation and mechanical ventilation is too invasive at this stage but would be considered if NIV failed or was contra-indicated. Removal of oxygen completely would risk catastrophic hypoxia.

Question 3, answer e.

This patient has a severe pneumonia with an acute on chronic hypercapnic respiratory failure on a background of OHS. His GCS is low and he is at high risk of further deteriorating on NIV. He requires immediate ventilatory support and transfer to Intensive care where he can be closely monitored. The other options are unlikely to correct the ventilatory failure and would be unsafe.

Question 4, answer d.

Fluid overload is often underestimated in patients with OHS presenting with AHRF and high doses of diuretics will sometimes be required. Infection can also precipitate acute decompensation and hence antibiotics should be given if there is evidence of underlying infection.

Controlled oxygen therapy aiming for lower oxygen target saturations should always be a part of initial management if there is evidence of chronic hypercapnic respiratory failure. In the acute setting CPAP will not provide the pressure support that BiPAP does to improve hypoventilation and is therefore not indicated.

Question 5, answer c.

Whilst all these options are likely to play a part in the longterm management of patients with OSA and OHS, it is strongly recommended that any patient presenting with AHRF secondary to OHS are discussed with the local Home ventilation service. Many of them will require longterm domiciliary NIV or CPAP. The decision about whether to implement this as an inpatient or outpatient should be based on individual patient characteristics and be made by a Ventilation Specialist.

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BRONCHIECTASIS: A CASE STUDY

D Cheng, J Brown

Abstract

We discuss a case of a 44 year old man with known asthma who presented with a history highly suggestive of the undiagnosed additional clinical problem of severe bronchiectasis. Treatment required a combination of systemic corticosteroids, prolonged intravenous antibiotics, nebulised bronchodilators, and non-invasive ventilation. Despite intense therapy, he remained in respiratory failure that earlier diagnosis of the bronchiectasis could have delayed. We discuss how to identify and investigate patients with suspected bronchiectasis and outline the principles of management and prevention of progressive lung function decline.

Case History

A 44 year old gentleman presented on the acute medical take with a six week history of increasing shortness of breath and a productive cough of between 1 to 2 egg cupfuls of thick yellow-green sputum per day. He had had multiple previous admissions to other hospitals with similar symptoms, with 6 admissions in the previous year, but had been subsequently lost to respiratory follow up.

This included 3 previous ITU admissions, but he had never been intubated. His exercise tolerance had been slowly deteriorating over the course of several years, and even when stable he was only able to walk 15 minutes without having to stop (MRC dyspnoea score 3) and had a cough productive of 150 mls of phlegm per day. He had a history of regular intravenous recreational drug use and crack cocaine smoking.

How is the patient clinically?

The initial clinical assessment is invaluable. Specifically, the respiratory rate and examination findings will help guide the urgency of review and treatment. If possible, an ABG can be very useful to determine how hypoxic and how hypercapnic is and whether non-invasive ventilation may be required.

What previous microbiology results are known?

Previous sputum results are crucial. Depending on which organisms have been isolated previously, and which resistances these have shown, antimicrobial therapy can differ greatly. Patients with bronchiectasis may often have resistance patterns which mean guidelines with empirical therapy are less helpful.

Does the patient have home nebulisers, home oxygen or domiciliary NIV?

This is a very helpful piece of information: it helps determine, along with spirometry and imaging results, exactly how severe the bronchiectasis is. Not only does this help guide how much the patient has deteriorated during an acute exacerbation, it also helps guide what further treatments may be appropriate. In particular, a treatment escalation plan should be considered as to whether NIV, critical care, or intubation and ventilation is appropriate.

Box 1: What will my registrar want to know?

On initial assessment, he had a temperature of 38°C, a respiratory rate of 25 breaths per minute, and he was only able to talk in short sentences. There was no evidence of finger clubbing. Auscultation revealed widespread inspiratory coarse crackles and loud expiratory wheeze throughout both lungs. Oxygen saturations were 93% on 4L / minute via nasal cannulae.

Blood tests revealed a C reactive protein level of 21.6 mg / L, and a white cell count of 13.9 10^9/L (96% neutrophils). The chest radiograph showed markedly increased bronchovascular markings with a visible bronchial fluid level in the right midzone, and multiple ring shadows throughout both lungs (Figure 1).

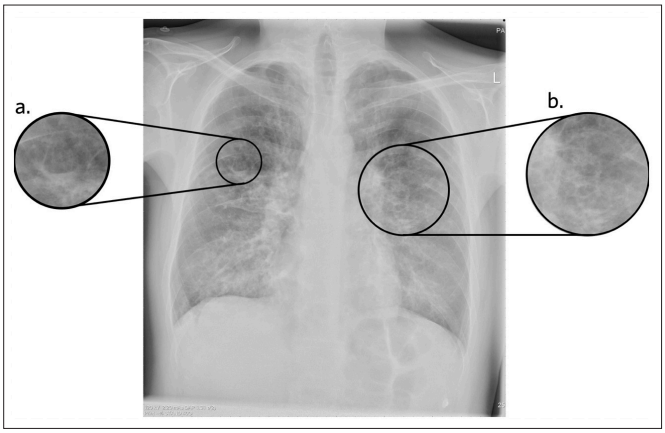

Figure 1: Chest radiograph showing increasing bronchovascular markings. Bronchial fluid level (a), and ring shadows (b) outlined and magnified.

Peak flow on admission was 220l/min; his best peak flow when he was well was 450l/min. He was unable to perform spirometry. Results of the admission arterial blood gas on an FiO_2 28% were pH 7.33, $PaCO_2$ 8.06, PaO_2 7.54, and bicarbonate 28 mmol/L, indicating compensated type 2 respiratory failure. Sputum culture grew a fully sensitive Pseudomonas aeruginosa. High resolution CT scan showed widespread cystic bronchiectatic changes involving all lobes, particularly both upper lobes, the middle lobe and the superior segments of the lower lobes (Figure 2).

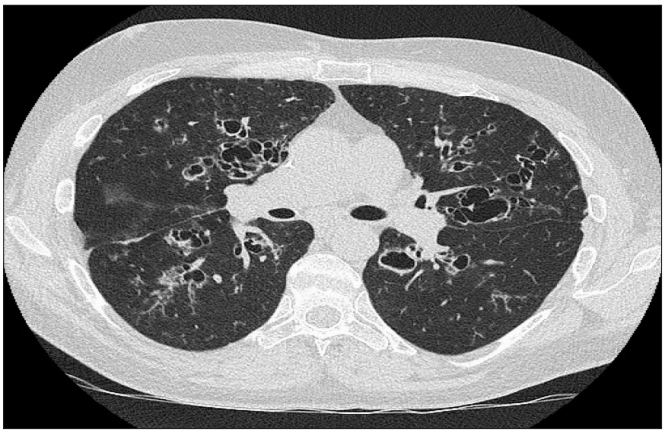

Figure 2: High resolution CT scan showing cystic bronchiectasis in all visible lobes.

BRONCHIECTASIS: A CASE STUDY

D Cheng, J Brown

There was no evidence of lobar consolidation or collapse. HIV serology was negative, Aspergillus specific IgG levels were within the normal range at 22.7 mg/L, and total IgE and IgG levels were both normal (81 and 12.1 g/L respectively).

He was started on a 14 day course of intravenous ceftazidime, 4 hourly salbutamol and 6 hourly ipratropium nebulisers, prednisolone 40 mg for 5 days then reducing to 0mg over 35 days, and regular chest clearance physiotherapy. Despite this treatment, his condition initially deteriorated and he developed a respiratory acidosis with arterial blood gases 72 hours after admission on FiO_2 of 28% showing a pH of 7.29, $PaCO_2$ 9.2 kPa, PaO_2 8.4 kPa, and a bicarbonate of 37 mmol / L.

He was started on non-invasive ventilator support (NIV) with inspiratory positive airway pressure (IPAP) of 22cm H_2O and expiratory pressure (EPAP) of 4 cm H_2O. On NIV his arterial blood gases improved with a pH of 7.33, $PaCO_2$8.3 kPa, PaO_2 8.9 kPa with an FiO_2 of 24%. His clinical condition improved, and NIV was weaned after 3 days.

Over the course of his admission the quantity of his daily phlegm production reduced to half a sputum pot a day, becoming mucoid rather than frankly purulent. Blood gases taken on air at the end of the admission showed persisting type 2 respiratory failure with pH of 7.37, $PaCO_2$ 7.6 kPa, PaO_2 8.6 kPa, and a bicarbonate of 32 mmol/L. He was discharged with domiciliary NIV, regular salbutamol nebulisers (2.5mg four times daily), azithromycin 250mg per day long term antibiotic prophylaxis , and 0.9% saline nebulisers (5 ml four times daily).

Discussion

The above case had had multiple admissions due to asthma over several years that were at least partly related to inhalation of crack, which is known to cause marked acute bronchospasm.

However, there were important clinical fatures that demonstrated an additional respiratory problem was present including: (a) production of a large volume of purulent sputum during this exacerbation, and significant daily sputum production even outside of exacerbations; (b) widespread audible crackles on chest auscultation, which are not a clinical sign caused by airways obstruction; (c) and a chest radiograph showing ring shadows and even a bronchial fluid level.

These features all suggest a diagnosis of bronchiectasis which was confirmed by the CT scan of the thorax. In addition, the long history of chronic deteriorating dyspnea and presentation with type 2 respiratory failure indicated severe chronic airways obstruction that would be unusual with asthma alone, and suggested a diagnosis of COPD (related to cigarette and recreational drug inhalation) or progressive bronchiectasis.

An alternative diagnosis would be pneumonia associated with asthma or COPD, but this would usually cause focal crackles over an area of obvious airspace shadowing on the chest X ray, associated with a more marked inflammatory response (e.g. C reactive protein greater than 40 and often well over a 100 mg/L).

Cause	% of cases	Clinical picture	Diagnostic tests
Idiopathic	29	Usually bilateral bibasal disease	N/A – diagnosed when other causes have been excluded
Post-infective	14	Onset after severe respiratory infection eg TB, pneumonia, whooping cough, measles	History; and in some cases localised disease restricted to the site of known previous infection
COPD	15	Marked airways obstruction	Lung function tests, plus a significant smoking history
Asthma	7	A pre-existing diagnosis of asthma then developing bronchiectasis symptoms	Lung function tests to look for reversible airways obstruction
Connective tissue diseases (CTD)	9	Concurrent CTD e.g. rheumatoid arthritis	Autoantibodies, ANA
ABPA	5	Severe but usually partially reversible airways obstruction and proximal bronchiectasis. Production of bronchial casts	Total IgE almost always very high *Aspergillus* specific IgG and / or IgE, and *Aspergillus* skin test positive
Immunodeficiency	5	Recurrent infections (congenital or acquired), IV drug use, haematological malignancy	Immunoglobulins, HIV test, vaccine response to *Haemophilus influenzae* and *Streptococcus pneumoniae* vaccines
Cystic fibrosis	<5	Childhood onset of symptoms, upper lobe disease, *S. aureus* isolation	Sweat chloride levels, nasal potentials, genetics
GORD/aspiration	4	Symptoms of reflux, choking coughing attacks (especially after eating food)	24 hours oesophageal manometry Video laryngoscopy
Primary ciliary dyskinesia	<1	Recurrent sinusitis and otitis media from childhood	Low exhaled NO Cilia function assays
Alpha-1 antitrypsin deficiency	<1	Associated with COPD and evidence of basal emphysem	A1AT level

Table 1: Aetiologies of bronchiectasis, adapted from Araújo et al 2017.

BRONCHIECTASIS: A CASE STUDY

D Cheng, J Brown

Clinical recognition and investigation of bronchiectasis

Bronchiectasis is defined as abnormal and permanent dilatation of one or more central or medium-sized bronchi. Patients describe daily cough productive of phlegm, the quantity of which increases in more severe bronchiectasis.

Exacerbations are common during which the quantity and purulence of phlegm produced increases, and the patient may develop malaise, a fever, chest pains, and increased dyspnoea.

Many patients have a degree of airways obstruction due to inflammation of the small airways downstream of the bronchiectasis. As shown by this case, in some patients the airways obstruction element progresses to cause respiratory failure.

Chest X rays only show abnormalities on 50% of bronchiectasis cases, and the diagnosis usually needs a CT scan to identify dilated non-tapering bronchi, often with thickened walls. The CT scan combined with lung function tests will also evaluate the extent and severity of the disease. Further investigations are needed to identify the potential cause of the bronchiectasis.

These could include:

(a) total IgG to look for immunodeficiency

(b) total IgE and Aspergillus specific IgG and IgE levels and skin testing for allergic bronchopulmonary aspergillosis (ABPA).

(c) autoantibodies, to look for rheumatoid arthritis and other autoimmune disease

(d) tests for cystic fibrosis or cilia function assays

(e) bronchoscopy to look for localised obstruction, and to obtain cultures for mycobacteria to exclude non-tuberculous mycobacterial infection

(f) HIV test

Which tests are used depend on the clinical pattern of the disease; for example, bronchoscopy is necessary in patients with single lobes affected to exclude bronchial obstruction, and tests for cystic fibrosis are necessary in younger patients especially of they have upper lobe disease.

Sputum microbiology is essential to guide antibiotic choice. Common pathogens are Haemophilus influenzae, Moraxella catarrhalis, Streptococcus pneumoniae, and Pseudomonas aeruginosa. It is important to identify non-tuberculous mycobacterium (NTM) and P. aeruginosa as these pathogens require specific antibiotic therapy and often associated with progressive disease.

Exacerbations often do not cause new lung signs, major changes in blood test results, or new changes on the chest X ray, and therefore are frequently diagnosed using history alone (Brill et al 2015). Even in this case when the patient presented extremely unwell with a severe exacerbation, the C reactive protein level was only mildly raised.

Management

Acute exacerbations need effective airway clearance and antibiotic therapy. Airway clearance is achieved through daily chest physiotherapy techniques, supported when necessary by nebulised saline or bronchodilators, mucolytics (e.g. carbocisteine), and mechanical assistance with, for example, oscillatory positive expiratory pressure devices.

The choice of antibiotic therapy depends on the patient's previous response to antibiotics and the sputum microbiology. H. influenzae is the commonest pathogen in bronchiectasis and is usually resistant to amoxicillin and macrolides, and many patients need doxycycline or co-amoxiclav. As seen with this case, P. aeruginosa is a common pathogen in severe bronchiectasis and will often require intravenous treatment.

Patients with bronchiectasis require longer courses of antibiotics as short course often result in the rapid recurrence of the infective exacerbatio; expert consensus suggest a duration of 14 days (Chalmers et al 2014). With more severe exacerbations associated with marked airways obstruction as illustrated by this case, treatment will include nebulised bronchodilators, possibly oral prednisolone, and oxygen or ventilatory support according to the blood gas results.

The long term aims of management are to improve quality of life and minimise lung function loss by reducing exacerbation frequency and duration. This will require daily effective airway clearance by the patient, prompt treatment of exacerbations with effective antibiotics, smoking cessation, and vaccination against influenza and pneumococcus.

The British Thoracic Society guidelines advocate the use of long term antibiotic prophylaxis in patients colonised with Pseudomonas or in patients with 3 or more exacerbations a year (Pasteur et al 2010) with macrolides or inhaled antibiotics (eg colomycin, gentamicin, or tobramycin) (Fan et al 2015, Altenburg et al 2013). In this particular case, inadequate recognition of the underlying bronchiectasis in combination with the patient's failure to fully engage with medical services resulted in ineffective long term therapy and chronic type 2 respiratory failure.

BRONCHIECTASIS: A CASE STUDY

D Cheng, J Brown

Multiple choice questions

1. The diagnosis of bronchiectasis is excluded by:

a. A normal chest X ray
b. Normal lung function tests
c. Repeatedly negative sputum cultures
d. An existing history of asthma or COPD as an alternative cause of daily sputum production
e. A normal high resolution CT scan

2. The management of the majority of patients with bronchiectasis include:

a. Long term inhaled corticosteroids
b. Oral corticosteroids for infective exacerbations
c. Regular airway clearance exercises
d. Five to seven day courses of antibiotics for infective exacerbations
e. Nebulised prophylactic antibiotic

Answers

Question 1. Answer: E.

Chest X ray and lung function tests may not show any abnormalities in a significant proportion of patients with bronchiectasis. Sputum cultures are insensitive and can be negative even in patients with severe bronchiectasis. However, as bronchiectasis is an anatomical diagnosis a completely normal high resolution CT scan excludes the diagnosis.

Ithough daily sputum production can also be caused by asthma or COPD, bronchiectasis is a complication of both conditions. In addition, the clinical presentation of bronchiectasis can overlap with those for asthma or COPD, and hence can be misdiagnosed as one of those conditions.

Question 2. Answer: C.

The role of long term inhaled steroids is not clear, but they are usually reserved for patients with significant but clearly reversible airflow obstruction (Martínez-García et al 2006). Oral steroids are only appropriate for selected patients with bronchiectasis e.g. exacerbations of allergic bronchopulmonary aspergillosis or co-existent asthma (Pasteur et al 2010). Daily airway clearance by the patient is one of the main pillars of management for most patients and improves the patient's symptoms and probably also reduces the frequency of exacerbations.

Antibiotic choice for infective exacerbations needs ten to fourteen days of antibiotics, with short course resulting in rapid relapse, and should ideally be guided by previous microbiology seen on sputum results. Long term nebulised antibiotics are effective at reducing infective exacerbation frequency and improving quality of life for patients with severe bronchiectasis, but are not necessary for the majority of patients.

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CASE DISCUSSION: MANAGEMENT OF PRIMARY SPONTANEOUS PNEUMOTHORAX

V Lewis, A Ionescu

Abstract

31 year old male presented with shortness of breath and right sided chest pain. He was diagnosed with a right sided, primary spontaneous pneumothorax.

As per the British Thoracic Society guidelines, (1) he required active intervention. The patient was enrolled in the RAMPP trial and randomly allocated to the ambulatory management group. He underwent a Rocket Pleural vent insertion.

The RAMPP trial is a randomised controlled trial, focusing on the management of PSP, comparing the current practise of pleural aspiration, plus or minus chest drain insertion, versus ambulatory management with insertion of a Rocket Pleural vent.

The primary outcome of the study is total length of hospital stay for up to 30 days following presentation. (2, 3) Ambulatory devices could result in diminished length of admission, possibly even a purely outpatient management approach.

Case History

A 31 year old male presented to the acute medical take with a history of sudden onset shortness of breath and right sided chest pain. He had no past medical history of lung disease. He smoked 15 cigarettes per day and used cannabis regularly.

On assessment he was haemodynamically stable, his oxygen saturations were 94% on room air and he had a respiratory rate of 18 breaths per minute. On examination there was reduced air entry over the right hemithorax. A chest x-ray revealed a large right sided pneumothorax. He was diagnosed with a right sided, primary spontaneous pneumothorax.

As per the British Thoracic Society guidelines, (1) he required active intervention. The guidelines indicate pleural aspiration, followed by chest drain insertion if the lung does not re-expand sufficiently. (1) Alternatively patients are currently being enrolled in the Randomised Ambulatory Management of Primary Pneumothorax (RAMPP) trial (2,3).

Following full informed consent the patient was enrolled in the trial and randomly allocated to the ambulatory management group. He underwent a Rocket Pleural vent insertion. The pleural vent remained in situ until day three, when the lung was fully re-expanded; and the vent was then removed.

Discussion

The incidence of pneumothorax is between 1.2–28/100 000 cases per annum. (4,5) Pneumothoraxes occur more frequently in men (4,5) and cigarette smoking and cannabis use are established risk factors for developing a pneumothorax. (5,6)

The term primary spontaneous pneumothorax (PSP) refers to pneumothorax occurring with no history of trauma and in patients without underlying established lung disease. (2) The aim of treatment is to allow the lung to fully re-expand.

The management approach is based on clinical assessment of the patient, with consideration as to whether or not the patient is haemodynamically compromised. (5) The patients' symptoms and the size of the pneumothorax (measured from the lung margin to the chest wall, at the level of the hilum (1)) are also taken into account. (5)

It is important to note that in individuals with secondary pneumothorax, such as those with underlying lung disease, or pneumothorax secondary to trauma, the management approach is very different. (1,5)

The British Thoracic Society guidelines recommend PSP measuring less than 2cm, in asymptomatic individuals, may be discharged from hospital without intervention. An outpatient follow up appointment in 2-4 weeks should be arranged. (1)

Patients with larger PSP or symptoms of breathlessness are recommended to undergo pleural aspiration. (1) If this is successful they may be discharged home with outpatient follow up. (1) If following aspiration the patient remains breathless, or the pneumothorax measures greater than 2cm, then insertion of a chest drain is required and the patient will need admission to hospital. (1,5)

In cases where following chest drain insertion a persistent air leak occurs or the lung fails to re-expand, an early cardiothoracic surgical opinion is advised. (1,5)

Smoking cessation should be discussed with all patients diagnosed with a PSP (and indeed all patients with lung disease) and the use of cannabis strongly discouraged.

It is also important to advise patients to avoid air travel until one week after the pneumothorax has fully resolved (as confirmed by radiological imaging). (5). Lifelong avoidance of deep sea diving is recommended, unless the patient has undergone bilateral pleurectomy and subsequently been shown to have normal lung function tests and an unremarkable CT scan of the thorax. (1,5,7).

CASE DISCUSSION: MANAGEMENT OF PRIMARY SPONTANEOUS PNEUMOTHORAX

V Lewis, A Ionescu

All patients diagnosed with a pneumothorax, whether given active intervention or not, must be given clear advice to seek medical attention urgently should they develop further symptoms of breathlessness. (5)

The RAMPP trial is a currently in progress, randomised controlled trial, being conducted in multiple different centres across the UK. (2,3) The study is focusing on the management of PSP, in particular comparing the current practise of pleural aspiration, plus or minus chest drain insertion, versus ambulatory management with insertion of a Rocket Pleural vent.

Following their allocated intervention patients enrolled in the trial are reviewed daily, either on the ward or as an outpatient. The review involves daily chest x-rays, air leak measurements and assessment of symptoms. (2) The primary outcome of the study is total length of hospital stay for up to 30 days following presentation. (2,3)

At present the average length of hospital stay for patients requiring chest drain insertion is 6-8 days. (2) Ambulatory devices could result in diminished length of admission, possibly even a purely outpatient management approach. This could dramatically reduce the demand for hospital beds and the associated costs, as well as facilitate a better experience for the patient.

Management of pneumothorax best of five multiple choice questions

1. A 21 year old male presents with sudden onset right sided chest pain whilst at rest. Symptoms have now completely resolved and CXR shows a 1.5cm pneumothorax. What is the appropriate management of this patient?

a. Aspiration and observation

b. Aspiration and discharge

c. Chest drain insertion and observation

d. Admit, oxygen therapy and observe over night

e. Discharge and review in clinic in 2-4 weeks

2. Which of these is not a criteria for secondary pneumothorax?

a. Significant smoking history

b. Age over 50 years

c. Past medical history of previous pneumothorax

d. Evidence of underlying lung disease on imaging

e. Evidence of underlying lung disease on examination

3. A 25 year old male presents with severe, sudden onset shortness of breath. Examination revels a trachea deviated to the right side and reduced breath sounds throughout the left lung. His heart rate is 120 and BP is 80/55. What is the correct initial management for this patient?

a. Full set of observation, including O2 saturations and respiratory rate

b. CXR

c. Chest drain insertion

d. Large bore cannula inserted in second intercostal space left mid clavicular line

e. Large bore cannula inserted in second intercostal space right mid clavicular line

4. A patient presents with shortness of breath and is diagnosed with a primary spontaneous pneumothorax. The rim of air between the chest wall and lung margin is 2.5cm and the patient is breathless.

An aspiration is performed. Following this the patient is still mildly breathless and the CXR appearance is unchanged. What is the correct next step?

a. Repeat aspiration

b. Admit, oxygen therapy and observation over night

c. 12 French chest drain insertion

d. Large bore chest drain insertion

e. Discharge home with repeat CXR in 2 weeks

5. Which of these is not a risk factor for primary spontaneous pneumothorax?

a. Physical activity

b. Tall stature

c. Smoking cigarettes

d. Smoking cannabis

e. Previous primary spontaneous pneumothorax

CASE DISCUSSION: MANAGEMENT OF PRIMARY SPONTANEOUS PNEUMOTHORAX

V Lewis, A Ionescu

Answer

1. Answer: e

Discharge and review in clinic in 2-4 weeks. British Thoracic society guidelines state that in non-symptomatic patients with no underlying lung disease and pneumothorax <2cm no intervention is needed. However if the patient is symptomatic then aspiration would be considered. Patient should be advised to represent urgently if symptoms develop.

2. Answer: c

Past medical history of previous pneumothorax. A past history of a pneumothorax does not automatically mean further presentations need to be managed as secondary pneumothorax. However it is good practice to take into consideration previous management approaches and complications. These patients may benefit from early discussion with a cardiothoracic surgeon.

3. Answer: d

Large bore cannula inserted in second intercostal space left mid clavicular line. In this case the diagnosis is left sided tension pneumothorax and treatment should not be delayed whilst further investigations are organised. A chest drain will need to be inserted but the patient is haemodynamically unstable and needs urgent intervention.

4. Answer: c

12 French chest drain insertion. Following a failed aspiration the patient will need to be admitted and have a chest drain inserted. An 8 – 14 Fr chest drain is usually recommended. Large bore chest drains offer no additional benefit and may be associated with higher risks and greater discomfort.

5. Answer: a

Physical activity. Exercise is not actually associated with an increased rate of spontaneous primary pneumothorax, a pneumothorax is as likely to occur at rest as during exertion.

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RECENT DEVELOPMENTS IN MANAGEMENT OF PLEURAL DISORDERS

J Kastelik, J Flapan, M Loubani

Abstract

Pleural disorders are common. Therefore foundation year doctors may require understanding of how to investigate and manage patients with pleural diseases. Pleural effusion can occur due to many causes. Cardiac, liver and renal disorders are the most common causes of transudate. Malignancy, pleural infection pulmonary embolism and rheumatological disorders are common causes of exudative pleural effusion.

Imaging is an important aspect of investigating patients with pleural disorders including thoracic ultrasound, computed tomography and Positron Emission Tomography (PET) CT. Many patients with pleural effusion or pneumothorax would require having pleural procedures such as thoracentesis or chest drain insertion or image guided pleural biopsies. Moreover, some of those patients may need more invasive procedures tests such as thoracoscopy. For this reasons patients with pleural disorders are best managed through a multidisciplinary team approach.

Introduction

Patients with pleural disorders may be managed in many medical and surgical specialties. During their foundation years training doctors are likely to be involved in managing patients with these disorders and for this reason they should be familiar and have understanding of pleural disorders and their management.

The pleura is composed of a single layer of mesothelial cells and forms visceral pleura that covers the lungs and parietal pleura that covers the inner surface of the chest wall (1,2). Under normal physiological state the pleural space contains a very small amount of fluid, which lubricates the pleura allowing for the breathing movement. Pleural effusion occurs when the pleural cavity is filled with larger amounts of fluid. When the air enters the pleural space it results in a condition called a pneumothorax (3).

Pleural effusion

Pleural effusion occurs when there is an imbalance between pleural fluid formation and re-absorption through the mechanisms such as increased pulmonary capillary pressure, which occurs in cases of cardiac failure, decreased oncotic pressure in hypoalbuminaemia or increased permeability as seen in pleural infection or malignancy (4).

In the broad terms pleural fluid can be divided into transudate and exudate. Light's criteria define exudates when the ratio of pleural fluid to serum protein is greater than 0.5, the ratio of pleural fluid to serum lactate dehydrogenase (LDH) is greater than 0.6 and pleural fluid LDH is greater than two thirds of the upper limit of normal value for serum LDH5.

The most common causes of exudates include malignancy, pleural infection including tuberculosis, pulmonary embolism, rheumatological disorders, or following coronary artery bypass surgery (CABG). Conversely transudate that usually has low protein content is commonly caused by cardiac failure, renal or liver disorders. Low pleural fluid pH defined as pH less than 7.2 can be seen in pleural infection, tuberculosis, rheumatoid arthritis or occasionally in malignancy.

Investigation of pleural effusion

The most appropriate approach to investigation of patients with pleural disorders would require a multidisciplinary team composed of respiratory physicians, radiologists and pathologists as well as thoracic surgeons. In the context of malignant pleural effusion or pleural neoplasm other specialists may need to be involved including oncologists, palliative care specialists as well as specialist cancer nurses, physiotherapists and psychologists.

Investigation of patients with pleural disorders requires a systematic approach. In fact, many of those patients are assessed through designated pleural clinics. Although majority of patients with pleural disorders will be managed by the respiratory specialists, a proportion may present on acute take to medical or surgical wards. Similarly, patients with liver disease, cardiac failure or renal disorders or patients with rheumatological conditions may develop pleural effusion and may be managed by other specialists.

The initial assessment of patients with pleural disorders would require a careful history and examination. A detailed smoking history is of importance as it may predispose to malignant pleural disorders. Similarly, an occupational history is paramount with particular assessment for any asbestos exposure. Medication history is essential as medications such as methotraxate, amiodarone, phenytoin or pergolide have been shown to cause pleural diseases (6).

Clinical examination may reveal reduced expansion in cases of pleural thickening, pleural effusion or pneumothorax. The percussion is characteristically stony dull in cases of pleural effusion and hyper-resonant in pneumothorax. Clinical examination may reveal other findings that may point towards the aetiology of pleural effusion such as finger clubbing, stigmata of heart failure, rheumatological, liver or renal disorders.

Imaging

Imaging forms an important aspect of assessing patients with pleural disorders. A chest radiograph, which is widely available modality remains an initial imaging modality as it may reveal abnormalities such as pleural effusion, pleural diseases such as pleural thickening or pleural plaques as well as pneumothorax (7).

Chest radiograph may also help in determining the origin of pleural disorders. For example, cardiac failure may be suggested by bilateral pleural effusions and cardiomegaly, evidence of sternotomy wires may suggest recent CABG and presence of consolidation may imply pleural infection.

Thoracic Ultrasound

In the recent years, thoracic ultrasound has become an important imaging modality for the assessment of patients with pleural disorders (8,9). In fact, many trainees now are expected to gain competency in thoracic ultrasound, with training requirements being described in detail in the national guidelines (8,10). The use of thoracic ultrasound helps to confirm the presence of pleural effusion and to assess its size and appearances (11,12).

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In additon, thoracic ultrasound is required for image guidance of pleural procedures such as thoracocentesis, chest drain insertion or pleural biopsies (4,13). The evidence suggests that when pleural procedures are performed under ultrasound guidance the complications such as iatrogenic pneumothorax are much lower comapared to when performing those procedures without ultrasound guidance (14).

There are some characteristics of the pleural effusion appearance that may help in determining the aetiology of pleural effusion. For example, in cases of pleural infection, pleural effusion may have complex appearances with septations or loculations. Thoracic ultrasound can also be used to diagnose underling malignancy when there is pleural thickening of ≥1cm, pleural nodularity and diaphragmatic thickening of ≥7mm (15).

Computed Tomography

Computed Tomography (CT) has formed an invaluable tool in investigations of pleural disorders. It can help in differentiation between malignant and benign pleural disorders (16,17). CT scan in the context of pleural malignancy can aid in assessing the extent of pleural thickening, mediastinal lymph nodes or other organ involvement (18).

CT can also be of help in diagnosing complex pneumothorax and distinguishing between emphysematous bullae and pneumothorax. However, CT scan has its limitations, as it may not be as accurate as thoracic ultrasound in detecting pleural loculations and septation in cases of pleural infection. CT scan has higher sensitivity in diagnosing malignant pleural disorders compared to thoracic ultrasound (15).

Positron Emission Tomography

Positron Emission Tomography (PET) CT has become an important imaging investigation in managing patients with pleural disorders. PET CT allows for measuring of positron emission from a radiolabelled tracer, which in the context of pleural or neoplasm imaging is an 18F- fluorodeoxyglucose (FDG). PET CT with FDG can localise neoplasm through its abnormal glucose metabolism. Malignancy can be identified using PET CT with approximately 95% sensitivity and 78-92% specificity (19).

PET CT can detect mediastinal lymph nodes involvement as well as the presence of distal metastases. PET CT imaging can also help to assess whether pleural diseases are benign or malignant. Thus PET CT was reported to have sensitivity of 88.2%, specificity of 92.9% and accuracy of 88.9% when diagnosing malignant mesothelioma (20,21).

PET CT is of importance when planning for histological sampling as well in when assessing response to chemotherapy. However, it needs to be noted that following talc pleurodesis there may be observed prolonged increase in FDG uptake (22).

Pleural infection

Pleural infection can be seen in around 40% cases of pneumonia with reported mortality upto 15% (23). In the past tuberculosis related empyema used to be very common and although it still can be seen currently it accounts for less than a fifth of cases (24).

In the context of pleural infection on the background of community acquired pneumonia the most commonly isolated bacteria include Streptococcus milleri, Streptococcus pneumonia, Staphylococcus aureus and anaerobes and in hospital acquired pneumonia Methicillin-resistant Staphylococcus aureus (MRSA), gram negative bacteria such as Escherichia coli or Klebsiella species as well as anaerobes (23).

The pleural infection related to the background of hospital acquired pneumonia carries higher risk of mortality. Pleural effusion in the context of pleural infection may reveal an exudate with low pH and glucose, high LDH and protein and high number of neutrophils. In severe cases there may be frank pus. It is important to stress that when pleural fluid undergoes microbiological analysis by sending samples in addition to sterile bottles in aerobic and anaerobic blood culture bottle the yield of detecting bacterial pathogen can be increased by a fifth (23).

Pleural fluid drainage and the use of appropriate antibiotics remains an important aspect of managing patients with pleural effusion. Intra-pleural administration of tissue plasminogen activator (t-PA) and DNase may be required in some cases and was it may improve the drainage of infected fluid and the need for surgery as well as reducing length of hospital stay (25).

In some patients surgical intervention may be required such as thoracotomy and decortication. There is a proposed risk stratification score called a RAPID score that takes in consideration urea and albumin levels, fluid purulence and community or hospital origin of infection (26). The score of 2 or below predicts approximately 1% risk of mortality at 3 months and the score higher than 5 upto 51%.

Pleural malignancy

In the UK there are approximately 40,000 cases each year of malignant pleural effusion. Lung neoplasm is responsible for around a third of the cases with breast cancer, ovarian cancer and lymphoma being the other common causes4. From histological aspects adenocarcinoma is the most common subtype responsible for of malignant pleural effusion (27).

Mesothelioma is the most common primary pleural neoplasm causing malignant pleural effusion. Malignant pleural effusions are characterised by the presence of malignant cells and are exudates. The presence of malignant pleural effusion usually suggests advanced cancer with poor overall survival ranging around 3 to 12 months although some patients may survive longer. The investigations of malignant pleural effusion will involve imaging with chest radiograph, thoracic ultrasound, computed tomography and CT PET scanning.

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Sampling of pleural fluid may provide histological diagnosis in a proportion of cases with studies suggesting between 20% to 60% diagnostic yield (4,27). The diagnostic yield of cytological analysis may be as high as 60% especially when identifying metastatic adenocarcinoma but remains disappointingly low at around 20% in cases of malignant mesothelioma as it is very difficult to differentiate between normal, reactive and malignant mesothelial cells (4,27).

Therefore, negative cytology of pleural fluid does not exclude malignancy especially. For this reason, additional tests such as CT guided pleural biopsy or medical or surgical thoracoscopy are frequently required to confirm the diagnosis. The management of patients with malignant pleural effusion depends on the type of underlying neoplasm, patients' symptoms, performance status and overall prognosis. In a proportion of patients pleural effusion may improve in response to systemic treatment with chemotherapy.

In patient with poor prognosis a simple therapeutic thoracocentesis suffice to control their symptoms. Others may require chest drain insertion and medical pleurodesis. In those with trapped lung an indwelling pleural catheter may be considered. In patients with good performance status and good prognosis surgical or medical thoracoscopy may be more appropriate, which allows for the drainage of the pleural effusion and talc pleurodesis.

For those reasons patients with malignant pleural effusion are managed through multidisciplinary team approach including respiratory physicians, thoracic surgeons, oncologists, radiologists and palliative care specialist.

Pleural Procedures

Pleural procedures are performed for diagnostic purposes, therapeutic reasons or both. Thoracocentesis is a procedure when a small needle is inserted into the pleural cavity either to drain air or fluid. When patients present with pneumothorax, a thoracocentesis is performed to drain air in order to expand the lung. It is recommended that thoracocentesis is performed in the second intercostal space in the mid clavicular line. The initial aspiration may be successful in 30% to 80% of cases of pneumothorax (2).

Thoracocentesis can be performed in patients with pleural effusion for diagnostic or therapeutic purposes. In contrast to pneumothorax, thoracocentesis in cases of pleural effusion should be performed under the ultrasound guidance. During thoracocentesis pleural fluid should be tested for microbiological and cytological analysis as well as protein, lactate dehydrogenase (LDH), glucose and when appropriate for the pH28.

Therapeutic thoracocentesis is usually performed using a designated pleural drainage system of which there are a number available currently. These devices are inserted under the ultrasound guidance, using an aseptic technique and the local anaesthesia for purpose of draining fluid in order to improve patients' symptoms of dysponea or pain (29-31).

The time for the fluid to re-accumulate post therapeutic drainage varies depending on the underlying aetiology of pleural effusion. The usual volume drained at one setting is around 1000 ml. It is usually undertaken in patients with advanced neoplasm who have poor prognosis or as a bridging measure prior to a definitive procedure such as thoracoscopy (4,32).

The potential complications of the procedure include discomfort, introduction of infection, re-expansion pulmonary oedema. In addition damage to intercostal vessels or organs such as liver or the spleen may occur but this has been reduced by the use of thoracic ultrasound to guide pleural procedures (11,33,34).

The procedure of chest drain insertion involves introduction of specially designed flexible tube into the pleural cavity, which is usually performed using an aseptic technique under the local anaesthesia. The chest drain is connected to an underwater sealed drainage system that creates a one way valve system, which prevent the air to enter the pleural cavity (35,36).

Chest drain needs to be inserted by an appropriate trained individual. In the context, of pleural effusion it is paramount that the procedure should only be performed under ultrasound guidance. The recommended placement of a chest drain is in the triangle of safety which is an area of which anterior border is the pectoralis major and the posterior border is formed by the latissimus dorsi with the inferior border defined as a horizontal line in the fifth intercostals space inferiorly (10).

The chest drain allows for the drainage of the pleural effusion or air in cases of pneumothorax. In addition, once the fluid or air is fully drained a procedure called pleurodesis can be performed. Pleurodesis involves introduction of a slerosing agent into the pleural cavity with an aim to obliterate the pleural space in order to reduce the risk of recurrence of pleural effusion or pneumothorax (4,37-39).

Currently, talc slurry is the most commonly used agent for pleurodesis with reported success rates at 1 month for management of pleural effusion of around 60% to 71% (27,40,41). Moreover, a recent study showed that use of minocycline for pleurodesis in patients with pneumothorax reduced recurrence to 29.2% (42).

Pneumothorax

Pneumothorax occurs when the air enters pleural cavity (3). When this takes place especially when the pneumothorax is large an impairment can occur in ventilation and perfusion leading to breathlessness and in cases of tension pneumothorax to haemodynamic instability, collapse or cardiorespiratory compromise (43)

Therefore, tension pneumothorax that can occur in 5% of major trauma patients needs to be recognised urgently and treated rapidly (44,45). Its clinical signs include hyper-resonance, tracheal deviation due to mediastinal shift and absence of breath sounds. In approximately 1% of cases a bilateral pneumothorax can occur, which is a medical emergency requiring bilateral chest drain insertion and often surgical intervention (46).

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In general the most common type of pneumothorax is a spontaneous pneumothorax, which can be subdivided into primary spontaneous pneumothorax, which occurs in previously healthy individuals and secondary spontaneous, which occurs in patients with underlying lung diseases. Another type is so called non-spontaneous pneumothorax of which the most common are iatrogenic and traumatic subtypes (47).

The underlying mechanisms of pneumothorax have not been fully elucidated. In the context of traumatic or iatrogenic pneumothorax there is a connection formed into the pleural cavity through which the air enters or alveolar rupture due to barotrauma in ventilated patients on intensive care unit or resulting from Valsava manoeuvre that increases intra-thoracic pressure (48,49).

The most likely proposed mechanisms for spontaneous pneumothorax are related to the presence of pleural inflammation and the rupture of the sub-pleural blebs or bullae (50-52). The epidemiology of pneumothorax varies depending on the country and the size of population studied. In a recent study from France, the reported annual rate of pneumothorax was at 22.7 per 100,000 with male to female ratio of 3.3 to 153.

There were two peaks of occurrence of pneumothorax first around the age of 20 years and second peak at the age of about 50 years. Smokers were reported to have an increased relative risk of having primary spontaneous pneumothorax and smoking cessation was associated with reduced risk of recurrence (54,55). The recurrence rates between 42% and 54.2% and are much higher in smokers as well as related in male patients to their height (54,56).

The national British Thoracic Society guidelines define large pneumothorax as that measured on chest radiograph as inter-pleural distance more than 2 cm at the hilar level (2). The initial treatment option for a large pneumothorax was suggested as aspiration using a 16 to 18 G cannula with reported success rates of 30% and 80% (2). If the needle aspiration fails the guidelines recommend insertion of a chest drain with small bore drains defined as less that 14F Seldinger drains, which were reported to have similar success rates and outcomes when compared with larger surgical drains (2).

A recent meta-analysis showed that there was no difference in cases of primary spontaneous pneumothorax treated with chest drain or needle aspiration in early success rates and recurrence rates at 1 year (56). The current British Thoracic Society (BTS) guidelines recommend that a chest drain insertion should be considered for a large pneumothorax defined as over 2 cm in size 2. The smaller so called Seldinger drains are as effective as large so called surgical drains (2,29-31).

It is usual practice to provide patients with pneumothorax with oxygen to maintain their oxygen saturation and to speed up the rate of the air from the pleural cavity through lowering the partial pressure of nitrogen (43).

Advanced Pleural Procedures and New Pleural Devices

Indwelling Pleural Catheters (IPC) are specially designed silicone tubes that can be inserted into pleural cavity in order to drain pleural effusion. These devices are also called long term pleural catheters and have one way valve system that can be connected to a vacuum bottle allowing to drain pleural effusion intermittently.

The devices are tunnelled under the skin and the draining tube is positioned within the pleural cavity. The insertion of the IPC is done under aseptic technique, local anaesthesia and awake sedation if required. Once in place the the patients or a district nurse are trained on how to connect the vacuum bottles to the drainage system and how to drain pleural effusion.

The patients therefore have independence and do not require to be admitted to hospital for the drainage of pleural effusion. The IPCs were shown to improve symptoms of dyspnoea, patients quality of life as well as achieving spontaneous pleurodesis (37,57,58). The main indications for IPC include presence of pleural effusion on the background of trapped lung, failed previous pleurodesis with recurrence of pleural effusion.

It is of importance that doctors are aware of new devices that have been recently gained use in management of patients with pleural disorders. One such example are endobronchial valves (EBV), which can be used in patients with pneumothorax who have a prolonged air leak and who may not be fit to undergo surgical intervention (59-61).

EBV are introduced using a flexible bronchoscope and as they are one way valves, their function is to stop the air leak by stopping air ventilation to the part of the lung where they are placed (60). A recent systematic review showed that following EBV insertion the air leak resolved in majority of patients (62). Their main complications associated with EBV insertion include valve migration, infection or recurrence of air leak. Another example of new devices are one way valves or 'vents', which are small catheters with one way valve system.

These systems can be introduced into the pleural cavity usually in the second intercostal space under aseptic technique and local anaesthetic. Using those systems patients with pneumothorax can be managed in ambulatory manner as there is no need to connect them to an underwater seal device. These drainage systems have ben shown compared to chest drain insertion to be less painful during sleep, toilet visits hygiene and removal (63,64).

They allow for the patients to be managed on out patients basis. A recent systemic review showed that their success rates were over 85%65. Moreover, the complication rates were low mainly related to cellulitis, surgical emphysema or dislodgement of the system.

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Thoracoscopy, involves introduction of the scope into then pleural cavity and can be performed under general anaesthesia so called Video Assisted Thoracoscopic surgery or under local anaesthesia and sedation so called local anaesthesia medical thoracoscopy (40,66,67). During the procedure, pleural fluid is drained, pleural surface examined and biopsies taken and if appropriate talc poudrage may be performed (40,41).

In addition VATs allows for more complex surgery such as surgery for pneumothorax, which may include pleurectomy, bullectomy, diagnostic lung biopsy or surgery for lung cancer such as segmentectomy or lobectomy.

Asbestos related pleural disorders

Asbestos are fibrous silicate materials, which when inhaled may cause pulmonary or pleural disorders. The most common types of asbestos include chrysotile (white asbestos), amosite (brown asbestos), and crocidolite (blue asbestos) (68-70).

Exposure can occur through direct handling of asbestos as well, which is more common through the occupations that expose to the end user asbestos products such as construction workers, plumbers, joiners, electricians, heating engineers or dockers. Environmental exposure from living closely to asbestos factories or domestic exposure of families of those working with asbestos usually occurring through handling their asbestos contaminated clothing needs also to be noted (71).

Therefore occupational history remains of great importance when managing patients with asbestos related disorders. Similarly, it needs to be appreciated that there is a long time lag between exposure to asbestos on average around 20 to 30 years from being exposed to asbestos to developing asbestos related disorders.

Disorders related to asbestos are divided into lung parenchymal or interstitial changes due to lung inflammation and fibrosis defined as asbestosis and pleural disorders such as pleural plaques, pleural thickening, pleural effusion and pleurisy. Pleural plaques are result of asbestos exposure and are benign lesions formed of collagen deposits on the parietal pleura. Diffuse Pleural thickening occurs as a result of exposure to asbestos, which causes scaring and thickening of the pleura, which is not reversible.

Asbestos exposure is also the most important factor for developing malignant mesothelioma, which is an aggressive form of cancer with a poor prognosis and the most common primary neoplasm of the pleura (72). In addition, mesothelioma has been described to affect other serous areas such as he peritoneal cavity and the pericardium (72). Mesothelioma at the global level causes between 15,000 - 20,000 deaths each year (73).

Malignant mesothelioma can be divided as per the World Health Organization classification into epithelioid, which is the most common subtype responsible for 60 to 80% of cases, biphasic found in 10-15% of cases and the least common reported in less than 10% of cases sarcomatoid subtype (74).

The other types of pleural neoplasms include pleural metastases, fibroud tumours, pleural sarcoma or lymphoma. Solitary fibrous tumour arises in the visceral pleura and occasionally can produce an insulin-like growth factor II leading to hypoglycaemia.

Another important aspect when managing patient with malignant pleural effusion including those with malignant mesothelioma is assessment of the predicted survival. Recently, a LENT prognostic score, which takes into account parameters such pleural fluid lactate dehydrogenase, Eastern Cooperative Oncology Group (ECOG) performance score (PS), neutrophil to lymphocyte ratio and tumour type was reported to have a good prediction for calculating survival in patients with malignant pleural effusion including those with malignant mesothelioma (75).

The LENT score, therefore may be considered when estimating survival for patients with malignant pleural effusion. Nevertheless, the issue of prognostication in the context of malignant pleural effusion remains complex due to a relative heterogeneity of this group of patients.

Conclusions

In conclusion, pleural disorders are very common and may present to a variety of specialists. Their management is complex and frequently may require multidisciplinary approach. However, the initial presentation needs to be understood in order to manage patients with pleural disorders in the most appropriate manner.

Questions

1) Are the following statements on pleural effusion True or False?

a) Under normal physiological state the pleural space contains a very small amount of fluid, which lubricates the pleura allowing for the breathing movement.
b) Light's criteria define transudate when the ratio of pleural fluid to serum protein is greater than 0.5, the ratio of pleural fluid to serum lactate dehydrogenase (LDH) is greater than 0.6 and pleural fluid LDH is greater than two thirds of the upper limit of normal value for serum LDH.
c) The use of thoracic ultrasound helps to confirm the presence of pleural effusion and to assess its size and appearances.
d) Streptococcus milleri, Streptococcus pneumonia, Staphylococcus aureus are commonest microbiological agents causing pleural infection on the background of community acquired pneumonia.
e) Mesothelioma is the most common primary pleural neoplasm.

2) Can the following occur in the context of pleural diseases?

a) Pleural effusion in the context of pleural infection may reveal an exudate with low pH and glucose, high LDH and protein and high number of neutrophils.
b) The chest drain should be placed in the in the triangle of safety.
c) Secondary spontaneous pneumothorax usually occurs in the young and healthy individuals.
d) Smokers have an increased relative risk of having primary spontaneous pneumothorax and smoking cessation is associated with reduced risk of recurrence.
e) Pleural disorders related to asbestos exposure include pleural plaques, pleural thickening but not pleural effusion and pleurisy.

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Answers

Question 1

a) True

b) False

This is a definition of an exudate type of pleural effusion.

c) True

In addition thoracic ultrasound is required for image guidance of pleural procedures such as thoracocentesis, chest drain insertion or pleural biopsies as it reduces the risks of complications.

d) True

In contrast, the commonest microbiological agents for hospital acquired pneumonia complicated by pleural infection are Methicillin-resistant Staphylococcus aureus (MRSA), gram negative bacteria such as Escherichia coli or Klebsiella species as well as anaerobes.

e) True

Question 2

a) True

In addition in severe cases there may be frank pus.

b) True

Triangle of safety is an area of which the anterior border is the pectoralis major and the posterior border is formed by the latissimus dorsi with the inferior border defined as a horizontal line in the fifth intercostals space inferiorly.

c) False

Secondary spontaneous occurs in patients with underlying lung diseases and primary spontaneous pneumothorax occurs in previously healthy individuals.

d) True

e) False

Pleural disorders related to asbestos exposure include pleural plaques, pleural thickening, pleural effusion and pleurisy.

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