A PATIENT'S JOURNEY

Hyperhidrosis

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Aged 5 this patient was offered limited and unpleasant treatments for her hyperhidrosis. Now an adult, she describes how iontophoresis has greatly reduced her symptoms

Hyperhidrosis and its management have been a huge part of my life since I was 5 years old. As I nervously awaited being reprimanded for doing something typically naughty, I noticed the sweat on my hands. More than being clammy, they ran with sweat, and as I cupped them the sweat rested in pools in my palms. I remember feeling embarrassed, frightened, and fascinated by the sight. Ever since, my life has consisted of finding increasingly cunning ways of trying to hide my embarrassing secret.

Getting through the childhood and teenage years

In a world where many debilitating and life threatening conditions exist, it may seem ridiculous to consider hyperhidrosis as a condition that can affect self esteem and an individual's social development and identity. But it can and does, and it has certainly had a huge impact on my life. As a tactile, friendly, confident person, I had to change all my natural inclinations to disguise my hyperhidrosis for fear of discovery and the inevitable reactions of disgust and ridicule. Simple childish games and other day to day tasks required careful "coping" methods—for example, sports; turning brass door knobs (which were the only kind in my primary school); writing essays in summer; gesticulating when talking (which I do very naturally); wearing sandals; going barefoot; and shaking hands. In short, things that most people do without a thought all routinely caused me great anxiety, and I developed elaborate stories to excuse myself as there was often no way of participating and disguising my condition. If you have ever experienced revulsion as someone touches your hand or notices how damp they are, you can begin to understand how mortifying it can be when it occurs.

As a teenager, when we all want to fit in and we develop our sense of self, my body image and self esteem were damaged by my loathing of my condition and therefore of my own body. My love of dance had to be sidelined; I couldn't take part in classes barefoot as I would slide all over the floor and leave puddles. I carried on with my singing and acting but cultivated ways to hide my hands when on stage and be apologetic to fellow actors I had to touch. Relationships were very difficult. I couldn't hold hands, couldn't be as tactile as I naturally am, and was frightened to become close to someone for fear they would find me revolting.

How iontophoresis has changed my life

My family has always been very supportive and reassuring; my father, having had the condition as a child, grew out of it in adulthood. They encouraged me to see our general practitioner, who was mildly sympathetic and prescribed Anhydrol Forte and other aluminium chloride products to apply topically overnight. These were helpful and did bring me some normality and relief, but they were very unpleasant to use and by no means wholly effective. When I moved to London at 22 to study at university, I thought I would make one final attempt to seek medical help and saw my new general practitioner. He was extremely helpful and suggested a referral to the Dowling Day Centre at Guy's and St Thomas's Hospital for iontophoresis.

The only previous option that I had found during my internet research was sympathectomy surgery, which seemed rather horrific. Iontophoresis entails the patient's hands or feet being placed on a metal plate and gauze in two trays containing glycopyrronium bromide solution and water, or just water if preferred. The plates are connected to a machine generating varying degrees of electrical current, which is passed through the plates, effectively facilitating a circuit through the body (for example, left hand to right hand). This process is then repeated with the current flowing in the opposite direction.

Tap water does not work for me, so I need the glycopyrronium bromide solution; but I understand that tap water works for about 85% of people who use iontophoresis, and that this is the normal practice throughout the UK in many dermatology departments. If people do not get a complete cessation of sweating with tap water alone, then they are switched over to the solution.

The treatment feels different depending on the sensitivity of the individual. I am very sensitive to the

This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

RESOURCES

Globally, there are few reliable, non-commercial sources of information and support for people with hyperhidrosis. The following are among the best.

- Hyperhidrosis Support Group (www.hyperhidrosisuk.org)—A British group led by a nurse specialist and run by volunteers; it provides information and support to people with hyperhidrosis and health professionals and seeks to raise awareness of hyperhidrosis and its treatments
- International Hyperhidrosis Society (www.sweathelp.org)—A non-profit organisation that provides information for patients (in English, French, Italian, and Spanish), promotes research, educates physicians, and raises awareness of how hyperhidrosis affects quality of life
- DermNet NZ (http://dermnetnz.org/hair-nails-sweat/hyperhidrosis.html)—A company run by the New Zealand Dermatology Society that provides high quality information for patients and clinicians on a wide range of dermatological conditions, including hyperhidrosis

electric current and the solution, and I experience most of the known side effects, such as extremely dry and sore mouth and throat and dizziness, usually up to 24 hours after treatment. I also use Vaseline on my wrists and cuticles to prevent burning. The sensation is like placing your hands on an electric fence. It is often uncomfortable and sometimes painful, although I am particularly sensitive. It is not known precisely how iontophoresis works, but it seems to be extremely effective in blocking the sweating temporarily.

Although the glycopyrronium bromide solution provides total relief in localised areas as well as the entire body, it also prevents serious physical activity for 24 hours after a treatment as it blocks the body's ability to control temperature fully. Iontophoresis gives me relief from hyperhidrosis for about five days, with a steady

Treatments for hyperhidrosis

Aluminium chloride antiperspirants (in the UK, Anhydrol Forte, Driclor, Odaban) are the first line of treatment but can cause irritation and do not work for many people

Iontophoresis (for hands, feet, and axillas) works for 85% of people using ordinary tap water. Failures using glycopyrronium bromide are rare

Botulinum toxin (for axillas) is available in a few private clinics for other areas of the body but is often not available in NHS hospitals. It costs £300-£500 per treatment and needs to be repeated every three months for maximum effect

Anticholinergics such as oxybutynin hydrochloride and glycopyrronium bromide (prescription only) work for many patients with generalised hyperhidrosis or those who experience compensatory sweating after endoscopic thoracic sympathectomy. However, they are not without side effects

Endoscopic thoracic sympathectomy is useful only when all other treatments fail and then should be considered only with caution as compensatory sweating is extremely common and often worse than the original problem

Retrodermal curettage (for use on the axillas only) is not available in many clinics, but in two thirds of patients the effects are long lasting

Disposable underarm pads are helpful to protect clothes

Glycopyrronium bromide wipes (available only online) are good for the face and hands in difficult situations

THE CLINICIAN'S PERSPECTIVE

Hyperhidrosis is defined as sweating in excess of that required for normal temperature regulation. Primary or essential hyperhidrosis, which presents without an associated condition, is a common disorder. In developed societies, up to 1% of the population will be affected in a lifetime. The onset of symptoms, as in Laura's case, is usually in childhood or adolescence. Rarely, persistent secondary hyperhidrosis is due to underlying endocrine, neurological, febrile, or genetic conditions. A secondary cause is more likely if the onset of hyperhidrosis occurs in adulthood. Although any body site may be affected, hyperhidrosis most commonly affects the axillas, palms, and soles of the feet.

Hyperhidrosis causes great emotional distress. Also, it often causes problems for the patient in the workplace if left suboptimally treated into adulthood. Laura's story illustrates how it dictated her social activities from an early age.

Although I have never met Laura, her story provides a sadly illuminating account of the problems of delays in therapeutic interventions in this condition. I see many children and adolescents who have been referred to secondary care at an appropriately early stage. Unfortunately, many children and adults suffer in silence, with a condition that is easily perceived by some as a "non-disease."

Treatments are not always that effective either. Aluminium chloride is often ineffective for severe hyperhidrosis, but it should remain the first line treatment in the community. Iontophoresis is useful for treating the palms, soles, and axillas. The iontophoresis apparatus can be bought for home use, which may be convenient for some patients. It is ineffective for facial or truncal hyperhidrosis, both of which are common, especially after sympathectomy. Delayed compensatory hyperhidrosis is very common with this operation, and only an unpredictable minority of patients benefit, so I never recommend this procedure to anyone.

I use glycopyrronium bromide in generalised hyperhidrosis. It has quite a few potential side effects, but these are usually well tolerated in the age range that is being treated (older children and younger adults). Botulinum toxin is very effective in axillary hyperhidrosis, but it is rarely available on the NHS; the high cost of the product and the necessity to repeat applications approximately every three months limits its usefulness.

George Millington, consultant dermatologist

decline in effects thereafter. I usually have my treatment once a week to maintain the effects.

Through the guidance, warmth, and support of the dermatology nurses, iontophoresis changed my life. It gave me, for the first time ever, total temporary relief from my hyperhidrosis. In short, I felt normal, as though a black cloud had been lifted. For the first time, I could wear sandals, enjoy a summer's day, hold hands, touch someone on the arm, sit with my hands in my lap, enjoy formal occasions, and shake people's hands without dread. These examples may all sound inconsequential, but they revolutionised my life.

Professionally, the improvement in my confidence for auditions and performances as a singer and actress has been immeasurable. It has been difficult, given my sensitivity and the side effects, to plan my time around my iontophoresis and arrange treatment times around concerts, rehearsals, and anything involving physical activity. However, the treatment has such positive benefits, both to my life and to my self esteem, that it makes any difficulties worth the inconvenience. Thanks to it and to the support of groups such as Hyperhidrosis UK, I can begin to see my hyperhidrosis as a condition I manage, not as a condition that manages me.

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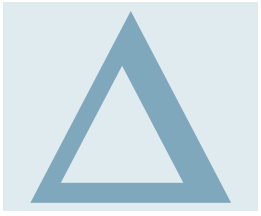

CHANGE PAGE

Prescribe systemic corticosteroids in acute asthma

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Change Page aims to alert clinicians to the immediate need for a change in practice to make it consistent with current evidence. The change must be implementable and must offer therapeutic or diagnostic advantage for a reasonably common clinical problem. Compelling and robust evidence must underpin the proposal for change. We welcome any suggestions for future articles (changepage@bmj.com).

Systemic corticosteroids reduce admission rates, relapse rates, and symptom duration and should be used for most acute exacerbations of acute asthma

The clinical problem

Globally, 300 million people are estimated to have asthma, and the prevalence in most countries is increasing.[1] Systematic reviews have found that systemic corticosteroids in acute asthma reduce admission rates, symptom duration, β agonist use, and repeat presentations for medical care.[2-5] However, evidence exists that corticosteroids are underprescribed in acute asthma, with prescribing rates ranging from <60% for mild asthma to <85% for moderate and severe asthma across multiple sites.[6] Systemic corticosteroids are recommended and should be prescribed, unless there are other contraindications, for all but the mildest of acute exacerbations of asthma.[1]

The evidence for change

Meta-analyses have reviewed the role of corticosteroids in acute asthma.[2-5] A meta-analysis of seven randomised controlled trials (426 admitted children aged 1-18 years) found that those who received systemic corticosteroids were discharged earlier (number needed to treat (NNT) 3) and were less likely to relapse within one to three months (odds ratio 0.19; 95% confidence interval 0.07 to 0.55, NNT=3).[3] A meta-analysis of six randomised controlled trials (374 discharged adults and children) showed that those receiving corticosteroids were less likely to relapse to the stage that they required additional care in the first week (0.38; 0.2 to 0.74); this effect was maintained for 21 days (NNT=13). The rate of subsequent hospital admissions in these patients was also lower (relative frequency 0.55; confidence interval 0.13 to 0.95) and the patients used β2 agonists less often.[4]

A meta-analysis of 12 randomised controlled or quasi-randomised controlled trials (863 adults and children) found that using systemic corticosteroids within one hour of arrival at hospital decreased admission rates (odds ratio 0.40; 0.21-0.78, NNT=8).[2] A meta-analysis of seven randomised controlled trials (1204 discharged adults and children) comparing rates of relapse at 7-10 or 16-21 days for high dose inhaled corticosteroids versus oral corticosteroids, found no significant difference in rates of relapse, but the heterogeneity of studies meant there was insufficient evidence to recommend inhaled corticosteroids as an alternative treatment.[5]

Despite evidence of effectiveness, studies have shown that systemic corticosteroids are underprescribed. The "snapshot of acute asthma" study was conducted in 38 Australian emergency departments and comprised 1340 acute presentations of asthma; it found prescribing rates of 59%, 86%, and 82% in mild, moderate, and severe exacerbations respectively.[6]

Barriers to change

Barriers to change may occur at the level of the patient but also at the level of the individual clinician, the

METHODS

I searched the Cochrane database of systematic reviews using the search terms "acute asthma" and "corticosteroids" in the "key words, titles and abstracts" field. From these results, I identified relevant systematic reviews relating to use of systemic corticosteroids in acute asthma.

KEY POINTS

Prescribe systemic corticosteroids for all but the mildest exacerbations of acute asthma

Systemic corticosteroids reduce admission rates, relapse rates, symptom duration, and requirement for "reliever" medications, such as short acting β2 agonists

An appropriate daily dose is 1 mg/kg a day of prednisolone (or equivalent dose of another corticosteroid) for up to seven days in adults and for three to five days in children

Insufficient evidence exists that inhaled corticosteroids are as effective as oral steroids after acute asthma attacks.

Inhaled corticosteroids have not yet been shown to be as effective as oral steroids for acute asthma attacks

healthcare team, the organisation (hospital), and the broader, national healthcare system.[7] Patient care that is based on evidence based guidelines for asthma has been shown to improve asthma outcomes,[1] and guidelines, if effectively implemented, can serve to ensure that both patients and clinicians are appropriately educated about the role of corticosteroids.[1] Yet implementation of guidelines remains an international challenge, and in emergency departments at least, barriers to change include a lack of time and resources; confidence among clinicians in what they are already doing; a chaotic, uncontrolled environment; and guidelines being available in formats that are difficult to use and access.[8]

One potential solution is for organisations to use an evidence based implementation strategy to effect change. Two studies of such a strategy for asthma guidelines found an increase in the use of systemic steroids in acute asthma.[9 10] A pre-intervention and post-intervention audit study in children presenting to emergency departments found that the use of systemic corticosteroids rose from 74% to 82%,[9] and in a controlled trial in adults presenting to emergency departments the use rose from 65% to 84%.[10] Key implementation strategies used in these studies included the use of senior medical and nursing staff as opinion leaders; reminders; audit and feedback to staff; education; and the reformatting of asthma guidelines for use in the clinical record. In these trials, of the 69 adults and children with moderate to severe presentations, 97% were prescribed systemic corticosteroids (unpublished data).

How should we change our practice?

Patients presenting with acute asthma should receive corticosteroid doses in the order of 1 mg/kg a day of prednisolone (or equivalent dose of another corticosteroid).[11] This dose is based on a double blind randomised controlled trial comparing three dosing regimens in 66 patients.[12] Systemic steroids should be prescribed for up to seven days in adults and for three to five days in children, with no need for tapering the dose.[1 11]

Although inhaled corticosteroids may yet be proved to be as effective as oral corticosteroids, most evidence to date favours systemic corticosteroids both in the acute stage and after discharge.

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The cost of carpal tunnel syndrome

As a clinical neurophysiologist, I am well acquainted with carpal tunnel syndrome and the impact that it has on affected patients that I test. Repeatedly, I hear about problems with driving, dropping objects, interrupted sleep, and the exacerbation of symptoms when performing activities of daily living such as ironing.

Last year I had the opportunity to witness the effects closely when my wife, Rebecca, developed the classic symptoms of numbness and tingling in the lateral three and a half digits of both hands. It was worst in the mornings and relieved by shaking of the fingers. The symptoms appeared from the second trimester of her pregnancy and lasted until several months after our daughter was born. During this time Rebecca became increasingly clumsy, and a heavy toll was taken of the crockery that had been our wedding presents: a dinner plate, five tea plates, nine bowls, 18 hiball glasses, and five wine glasses were destroyed. The cost of replacement will total £146.

I am yet to discover the full cost of having a daughter.

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EASILY MISSED?

Obstructive sleep apnoea in adults

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Obstructive sleep apnoea is characterised by snoring, recurrent episodes of upper airway obstruction during sleep (apnoeas and hypopnoeas), and arousals. The resulting sleep disturbance can cause excessive and disabling daytime sleepiness. The term obstructive sleep apnoea syndrome is used for people who have features of obstructive sleep apnoea on a sleep study and also have resulting daytime sleepiness.

Why is it missed?

Typically, patients with obstructive sleep apnoea have symptoms that have been present for months to years, some of which may have not been recognised by them or by healthcare professionals. Patients may complain of being "tired" rather than "sleepy," and this may be overlooked, particularly in women. A sleep history to elicit cardinal symptoms of obstructive sleep apnoea should be taken.

Why does it matter?

Driving

The possible ramifications of not diagnosing someone with symptomatic obstructive sleep apnoea and not starting treatment are enormous. They have a sevenfold increase in risk of being involved in a motor vehicle accident, resulting in a potentially fatal outcome for both the patient and others[5]; this is especially relevant in the case of the lorry driver described overleaf. Accidents caused by dangerous driving resulting from sleepiness can lead to a prison sentence,[6] and a condition such as obstructive sleep apnoea syndrome is not a mitigating circumstance.

Cardiovascular risk

As well as obesity, obstructive sleep apnoea is associated with other conditions, such as hypertension, hyperlipidaemia, insulin resistance, type 2 diabetes, and the metabolic syndrome, and therefore cardiovascular disease. Obstructive sleep apnoea worsens prognosis in chronic heart failure and is probably underdiagnosed.[7] Continuous positive airway pressure treatment significantly decreases blood pressure and sympathetic nerve activity and reduces arterial stiffness in patients with obstructive sleep apnoea syndrome; this is very likely to be beneficial to overall cardiovascular risk.[8-10] A longitudinal uncontrolled study showed that untreated severe obstructive sleep apnoea greatly increased the risk of fatal and non-fatal cardiovascular events compared with people treated with continuous positive airway pressure.[11]

How is it diagnosed?

Clinical

Consider obstructive sleep apnoea syndrome in any patient complaining of excessive sleepiness, rather than just tiredness. Ask about snoring, nocturnal choking episodes, witnessed apnoeas, and waking unrefreshed, particularly in those who are overweight and have type 2 diabetes, hypertension, or chronic heart failure.

The history, the extent of daytime sleepiness, and any abnormalities on a sleep study are necessary for making a diagnosis of obstructive sleep apnoea syndrome that merits treatment. There is a spectrum of disease, from mild snoring and occasional apnoeas at one end to regular loud snoring with recurrent apnoeas, nocturnal choking sensations, and arousals

HOW COMMON IS IT?

- Prevalence varies according to the population studied
- In the US state of Wisconsin a cohort study of 30-60 year old workers found that 24% of men and 9% of women had evidence of obstructive sleep apnoea (at least five apnoea or hypopnoea events per hour).[1] This study estimated that only 4% of men and 2% of women reported any daytime sleepiness also (that is, obstructive sleep apnoea syndrome)
- In a UK study, with lower levels of obesity, the prevalence of obstructive sleep apnoea syndrome in men was about 1%[2]
- These studies show that obstructive sleep apnoea is a common problem, which is likely to increase further as obesity levels rise
- Selected populations show a higher prevalence of obstructive sleep apnoea—for example, 23% of men with type 2 diabetes were estimated to have appreciable obstructive sleep apnoea.[3] Estimates suggest that 59 400 people receive continuous positive airway pressure treatment in England (0.2% of the population); but the National Institute for Health and Clinical Excellence (NICE) also estimates that up to 980 000 people could have undiagnosed obstructive sleep apnoea syndrome[4] (although we think it is likely that substantially fewer would want such treatment)
- From our own unpublished data, on the basis of numbers of patients being treated with continuous positive airway pressure in a selection of general practices, a primary care practice of 5000 patients can expect to have 10 people already receiving continuous positive airway pressure

This is a series of occasional articles highlighting conditions that may be commoner than many doctors realise or may be missed at first presentation. The series advisers are Anthony Harnden, university lecturer in general practice, Department of Primary Health Care, University of Oxford, and Richard Lehman, general practitioner, Banbury. If you would like to suggest a topic for this series please email us (easilymissed.bmj@bmjgroup.com)

Epworth sleepiness scale

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you. Use the following scale to choose the most appropriate number for each situation:

0 = Would never doze or sleep.
1 = Slight chance of dozing or sleeping
2 = Moderate chance of dozing or sleeping
3 = High chance of dozing or sleeping

Situation	Chance of dozing
Sitting and reading	
Watching television	
Sitting inactive in a public place (for example, a theatre or a meeting)	
Being a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
Sitting in a car while stopped for a few minutes in traffic	
Total score	

Scores of 9 or above equate with significant excess daytime sleepiness

The Epworth sleepiness scale

at the other. Patients may be reluctant to admit to sleepiness, owing to concerns about driving, but they should be reassured that if symptomatic obstructive sleep apnoea is diagnosed and effectively treated, the Driving and Vehicle Licensing Agency will allow them to drive (with annual review in the case of drivers of heavy goods or public service vehicles (www.dvla.gov.uk)).

The Epworth sleepiness scale (figure) is a simple validated tool to quantify subjective daytime sleepiness in people suspected of having obstructive sleep apnoea, although it cannot distinguish the cause.[12] In a sleep clinic, it is also used to assess response to treatment. More specific screening questionnaires, such as the Berlin questionnaire,[13] aim to identify patients at high risk of obstructive sleep apnoea, but studies have found limitations in its use, with underdiagnosis and overdiagnosis. We favour the Epworth sleepiness scale in our clinical practice. Neck size, a measure of upper body obesity, has been found to have a strong correlation with severity of obstructive sleep apnoea,[14 15] and those with a collar size of >43 cm (17 inches) and symptoms merit sleep studies.

CASE SCENARIO

A 50 year old long distance lorry driver attends for a review of his heavy goods vehicle licence. His body mass index is 30, and he says his wife complains that he snores loudly. The history of loud snoring in this overweight man should raise concern about possible obstructive sleep apnoea. As he is a lorry driver this diagnosis is particularly important.

Investigations

Many types of sleep studies are available; overnight measures of breathing and sleep disturbance (such as oximetry, pulse rate, and arousals (for example, monitored via body movement, sympathetic arousal, or electroechocardiographic activity)) are the minimum usually required to be confident of the diagnosis.

Management

If obstructive sleep apnoea seems a possible diagnosis, refer the patient to a sleep specialist, who will usually perform a sleep study. Experience and usually a face to face encounter are required to decide which patients with a positive sleep study merit treatment, particularly if they have little obvious or admitted daytime sleepiness.

Management of those with symptomatic obstructive sleep apnoea is usually with continuous positive airway pressure. This has been found to be effective in randomised controlled trials and meta-analyses.[16] A recent technology appraisal by the National Institute for Health and Clinical Excellence (NICE) has unreservedly approved continuous positive airway pressure for treating moderate and severe symptomatic obstructive sleep apnoea but for mild symptomatic cases only if other simpler treatments have not worked or are not appropriate. Other treatments include weight loss, improved sleep hygiene, and mandibular advancement devices, but so far little evidence exists to support these approaches. Ear, nose, and throat surgery has little to offer other than removal of markedly enlarged tonsils. Uvulopalatopharyngoplasty has no robust evidence to support its use in either snoring or obstructive sleep apnoea and has largely been abandoned for this reason.[17] Bariatric surgery is likely to be a more relevant operation once it becomes more widely available.

KEY POINTS

- Consider obstructive sleep apnoea syndrome in people with daytime sleepiness; snoring; and apnoeas
- Daytime sleepiness can be quantified using the Epworth sleepiness scale; scores above 9 (on a scale of 0-24) are considered abnormally high
- Refer patients for a sleep study if they have daytime sleepiness and the other suggestive symptoms
- Treatment with continuous positive airway pressure reduces sleepiness, as well as blood pressure, and thus potentially cardiovascular risk
- People without daytime sleepiness may not require treatment with continuous positive airway pressure

Patients need to inform the Driving and Vehicle Licensing Agency that they have been diagnosed with symptomatic obstructive sleep apnoea and confirm that they are receiving effective treatment with continuous positive airway pressure. For drivers of heavy goods or public service vehicles, the agency will seek medical confirmation of the treatment.

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10-MINUTE CONSULTATION

Transient ischaemic attack

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This is part of a series of occasional articles on common problems in primary care. The *BMJ* welcomes contributions from GPs

A 64 year old man comes to your surgery five days after an episode of visual loss in his left eye, followed by right sided weakness and speech disturbance lasting 10 minutes. He has made a complete recovery and has driven himself to the surgery to ask whether he can return to work.

What issues you should cover

General points

- Transient ischaemic attack (TIA) is a sudden onset focal neurological deficit that resolves completely within 24 hours.
- Amaurosis fugax, an embolic form of a TIA in the carotid territory, is painless transient monocular blindness, described as a curtain, shade, or mist descending over the eye.
- The diagnosis of a TIA is based entirely on history rather than brain imaging.
- The risk of a recurrent event after a TIA is highest in the first month (risk of 8-10% at seven days and 11-15% at 30 days).

Assessing symptoms

Onset—Symptoms are sudden in TIA, as in acute stroke. Insidious onset of symptoms is unlikely to be a TIA.

Intensity—Symptoms of a TIA are maximal at onset. Gradual progression of symptoms would point to a different pathology, such as migraine, demyelination, or a tumour. Even though recurrent events could follow a TIA, multiple and intermittent symptoms are atypical and should raise the possibility of demyelination or a tumour.

Focal symptoms—Focal neurological symptoms are the classic sign of a TIA. Carotid territory symptoms include amaurosis fugax, contralateral weakness or numbness, dysphasia, and hemianopia. Vertebrobasilar territory symptoms include ataxia, vertigo, dysarthria, diplopia, hemianopia, and bilateral visual loss. Dizziness, lightheadedness, or vertigo occurring in isolation is rare in a TIA.

Global dysfunction—Loss of consciousness (syncope) is not a TIA. Deviation of the angle of the mouth and

slumping towards one side seen during syncope could easily be mistaken for a TIA. Isolated memory loss is due to transient global amnesia, which is not a TIA.

Duration—Most TIAs last 5-15 minutes. Symptoms lasting for more than an hour are usually classed as minor strokes. Symptoms lasting less than 30 seconds are unlikely to be due to a TIA.

Recognisable pattern—Symptoms corresponding to a recognised neurological territory are supportive of a TIA. Combination of symptoms such as dysphasia, face and arm weakness (as determined by the face, arm, and speech test (FAST)) has a higher specificity in the diagnosis. Other common combinations are hemiparesis and hemisensory loss with or without hemianopia, ataxic hemiparesis and diplopia, dysarthria, vertigo, and ataxia.

Headache—Even though headache can occur during a TIA, severe headache or eye pain is not a feature of a TIA.

Excluding a diagnosis of transient ischaemic attack

Symptoms that don't suggest TIA

- Loss of consciousness
- Acute confusion
- Seizure
- Loss of memory
- Isolated dizziness, lightheadedness, or vertigo
- Gradual progression of symptoms
- Multiple recurrent symptoms
- Severe headache

Mimics of TIA

- Migraine
- Partial seizure
- Syncope
- Vestibular disorders
- Neuropathy and radiculopathy
- Ocular disorders
- Hypoglycaemia
- Tumours of the central nervous system

What you should do

Examination—Include a check of his pulse for irregular rhythm, measure his blood pressure, listen to heart sounds, and undertake a neurological examination to detect any residual signs.

Investigations—Take a full blood count and measure urea and electrolytes, fasting blood glucose, and total cholesterol. All patients with carotid circulation TIA who are fit for surgical intervention should have urgent carotid dopplers done either through primary care or secondary care, according to local guidelines.

Prognostic score—The "ABCD2" score, which is based on age (≥60 years: 1 point), blood pressure (≥140/90 mm Hg: 1 point), clinical features (unilateral weakness: 2 points; or speech disturbance: 1 point), duration (≥ 60 minutes: 2 points; 10-59 minutes: 1 point), and presence of diabetes mellitus (1 point), should be used to identify people who are at high risk of stroke (score ≥4).

Referral—All patients with a TIA should be referred to secondary care as soon as possible. The UK National Institute for Health and Clinical Excellence (NICE) recommends that patients with an ABCD2 score of ≥4 should be assessed and investigated within 24 hours.

Antithrombotic treatment—All patients with a TIA should be started on aspirin 300 mg immediately. Long term antithrombotic treatment should include combination of aspirin (75 mg once daily) and modified release dipyridamole (200 mg twice a day). Clopidogrel (75 mg once daily) should be used only if the patient is intolerant to aspirin. The combination of clopidogrel with aspirin or dipyridamole is not recommended. If he is already taking aspirin, dipyridamole should be added, and there is no reason to switch to clopidogrel routinely. In patients with atrial fibrillation, warfarin should be begun only after imaging. If a patient is already on warfarin, check his international normalised ratio and seek advice from secondary care regarding urgent brain imaging.

Other secondary preventive measures—Advise smoking cessation, regular exercise, cutting salt and alcohol intake, blood pressure control according to national guidelines, and screening for diabetes and hypercholesterolaemia (consider a statin if the total cholesterol is >3.5 mmol/l or LDL cholesterol is >2.5 mmol/l).

Driving—He should not drive for a month after a TIA.

FURTHER READING AND RESOURCES

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Stroke Association (www.stroke.org.uk) has information for patients

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LESSON OF THE WEEK

Outbreak of *Streptococcus pneumoniae* serotype 1 pneumonia in a United Kingdom school

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Healthcare workers and teachers should report suspected outbreaks of serotype 1 pneumococcal disease early, and childhood immunisation should be considered

Pneumococcal pneumonia is not generally regarded as contagious.[1] Although epidemics of pneumococcal disease have been reported (in sub-Saharan Africa[2 3] and Canada[4]), outbreaks of pneumococcal infection are uncommon and are generally restricted to high risk individuals such as alcoholics,[5] residents in shelters for the homeless,[6] and people living in close groups[7] including military camps,[8] prisons,[9] day care centres,[10] and nursing homes.[11] Recent reports have indicated that serotype 1 pneumococcus is largely responsible for the exponential increase in the incidence of empyema and complicated pneumonia seen in children in several countries over the past decade.[12-14] There is also evidence of outbreaks of other forms of invasive serotype 1 disease in many countries.[2 15 16] In contrast to most other invasive serotypes, carriage of serotype 1 is rarely detected in the nasopharynx of either adults or children, suggesting short duration of carriage or high virulence.[17 18]

There have been no reports of outbreaks of pneumococcal pneumonia among UK children, but they have been reported in other countries.[7 19] We describe an outbreak of serotype 1 pneumococcal pneumonia among young children in a school in northeast England.

Case reports

Clinical summary

Three cases of pneumococcal pneumonia in young children were initially reported in a primary school in North Tyneside. The dates of onset of illness were between 10 and 13 October 2006, and all three children, aged 4-5 years, attended the same reception class at the school and all were admitted to hospital with radiologically confirmed lobar pneumonia. The course of the disease was uncomplicated in two children, whose blood cultures were negative for *Streptococcus pneumoniae*. The third child had a positive blood culture and developed thoracic empyema (despite appropriate antibiotic treatment) requiring surgical drainage and mini-thoracotomy.

Public health action

After the notification of these three linked cases, public health action was taken to prevent further transmission of infection. The outbreak control team sought specialist advice from the Health Protection Agency Centre for Infections and arranged for all classroom contacts (64 pupils and staff) and household contacts (13) to receive rifampicin chemoprophylaxis. The uptake rate for classroom contacts was 97% (62/64) and for household contacts was 100%.

Further developments

Two further children from the school then presented on 15 and 20 November 2006, with clinical and radiological features of lobar pneumonia. Neither required inpatient hospital treatment, and both had received rifampicin chemoprophylaxis in response to the first cluster of cases.

Serotype 1 pneumococcus was detected in all five cases with a serotype-specific antigen detection assay from urine, but only one of the cases was positive for *S pneumonia* on blood culture. The serotype-specific assay, performed at the Health Protection Agency Centre for Infections, can detect 13 of the most common serotypes (1, 3, 4, 5, 6A, 6B, 7F/A, 8, 9V, 14, 18, 19A, 19F, and 23F)[20 21] and was developed from an enzyme linked immunosorbent assay (ELISA).[22] A pleural fluid sample from the child with a positive blood culture was also positive for serotype 1 using the serotype-specific assay. This assay has been validated but is not yet commercially available.[21]

The five urine samples were also positive on testing with the commercially available Binax NOW *S pneumoniae* antigen testing kit, which is reported to have 94% specificity and 86% sensitivity compared with blood culture results.[23] The two hospitalised children with negative blood culture results for *S pneumoniae* were also tested for respiratory viruses and mycoplasma, with negative results.

As there was evidence of ongoing transmission, the five infected children and their classroom and

household contacts were offered the 23-valent pneumococcal polysaccharide vaccination (Pneumovax II), which includes serotype 1 antigen, and 77/83 (93%) received it. (None of the children had previously received the 7-valent pneumococcal conjugate vaccine.) Throat swabs were also obtained from the cases and contacts to test for carriage of the outbreak strain. Of the 81 people who provided swabs, one had evidence of carriage of *S pneumoniae* serotype 1 and was treated with azithromycin (12 mg/kg/day, once daily for five days) to eliminate it. No new cases of pneumococcal disease were reported after these further interventions.

Discussion

The incidence of complications associated with pneumococcal pneumonia, including necrotising pneumonia, parapneumonic effusion, thoracic empyema, and lung abscess has increased dramatically in UK children over the past decade.[12-14] The reasons for this are still largely unknown, but it is at least partially related to the emergence of *S pneumoniae* serotype 1 as the dominant serotype. Whether this is due to changes in the host, organisms, or environment or as part of a naturally occurring cycle of dominant serotypes has yet to be determined.[15 24]

In September 2006 the 7-valent pneumococcal conjugate vaccine was introduced for routine vaccination of UK children, but the cohort of children in this outbreak had not received it at the time of the outbreak. The vaccine contains antigen for serotypes 4, 6B, 9V, 14, 18C, 19F, and 23F (which account for 70-80% of invasive isolates from European children), but it does not include antigen for serotype 1. After the vaccine's introduction into the US vaccination programme in 2000, the incidence of pneumococcal parapneumonic empyema has continued to increase in US children, because of a non-significant increase in serotype 1 disease and the emergence of disease related to other serotypes not covered by the vaccine.[25] Enhanced pneumococcal surveillance has been in place in England and Wales for several years now in order to monitor changes in serotype prevalence after the introduction of the pneumococcal conjugate vaccine. Paediatric vaccination with a conjugate vaccine containing serotype1 antigen is not available in the UK, but such vaccines are at an advanced stage of development. It may become necessary to introduce such vaccines to prevent serotype 1 pneumococcal disease.

Antibiotic prophylaxis is not normally recommended for close family contacts of index cases of pneumococcal pneumonia. At the time of outbreak, there were no national guidelines available for the public health management of outbreaks of pneumococcal pneumonia. However, the Health Protection Agency's Vaccine Programme Board has convened an expert group to develop evidence based guidelines.

When further cases were found in this North Tyneside outbreak, throat swabs were obtained to identify carriage of serotype 1 pneumococcus. Ideally, nasopharyngeal swabs should have been done, but this is an unpleasant procedure for children, and throat swabs were taken instead. This may have reduced the carriage detection rate. The one person who was found to be carrying serotype 1 pneumococcus was treated with azithromycin, which is effective in reducing nasopharyngeal carriage of pneumococci and was used to halt an outbreak of pneumococcal pneumonia in US marines.[8]

This report highlights the importance of early detection and notification of suspected outbreaks of pneumococcal disease to enable the early implementation of control measures. Healthcare workers and other professionals such as teachers have a vital role in reporting suspected outbreaks to public health professionals. The investigation of this outbreak was facilitated by the use of a non-invasive, multiplex, serotype-specific antigen detection assay, which identified the same serotype in all cases. The use of such tests should be considered as a diagnostic option, especially when blood samples are negative on culture testing.

This outbreak of serotype 1 pneumococcal pneumonia in children may support the need for immunisation against serotype 1 disease. Introduction of a vaccine that includes serotype 1 antigen should be considered if there is evidence that serotype 1 disease is an increasing public health problem.

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Contributors: F-MK was chair of the outbreak control team that managed this outbreak and is guarantor for this article. DAS clinically managed one of the cases. F-MK and DAS had the idea for the article, AG performed the literature search and prepared the first draft of the article. The other authors contributed to the literature search and commented on the initial drafts of the manuscript.

Competing interests: RCG has been reimbursed by manufacturers of pneumococcal vaccines Wyeth and GSK (GlaxoSmithKline) for attending conferences, and his laboratory has received research funding from Wyeth. CLS has been funded by Wyeth for attending international conferences. MS has received funding from vaccine manufacturers to attend conferences and meetings. DAS has received research funding from Wyeth and funds for attending advisory boards and speaking from Wyeth and GSK. ELS has participated in a research project funded by Wyeth.

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Great minds don't always think alike

One of the well worn mnemonics used in assessing Alzheimer's disease focuses on the four A's—aphasia, amnesia, agnosia, and apraxia. I have always relied on this tool as a useful diagnostic guide. The dictionary definition of the fourth A, apraxia, is "inaction" from the Greek praktos (to do, to practice). This inaction is due to the loss of ability to recognise objects or their uses. "My mother can't seem to dress on her own anymore. She puts her shirt on backwards and puts her arms in the pant legs. When I give her a pen, she turns it over and over but doesn't know what to do with it."

My diagnostic mantra when examining patients with a memory complaint is, "Pretend you are holding a toothbrush in your hand and show me how you brush your teeth." Patients without apraxia will make a loose fist, as if holding a toothbrush, and then move their fist from right to left and up and down in front of their teeth. Patients with Alzheimer's disease, unable to think abstractly and imagine a toothbrush in their hand, use their index finger as a toothbrush.

While answering some questions about Alzheimer's disease posed by my 97 year old grandmother, I started with a description of the four A's. Getting to apraxia, I said, "Mama, try this." I launched into my familiar request awaiting the up and down motions of her fist.

"Is this what you mean?" my grandma asked as she pretended to remove her dentures and move her fist from side to side over her imaginary choppers.

"Exactly," I responded, as we broke into hearty laughter at the cleverness of an experienced mind to defy standard categorisations.

I also met my match with the physics professor who raised his fist as expected but then left it motionless, suspended in mid-air opposite his teeth. "Professor," I urged, "show me how you brush your teeth."

He paused, calmly responding to my impatience. "But, doctor," he countered, "I have an electric toothbrush."

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