EASILY MISSED?

Constipation in people with learning disability

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Chronic constipation is surprisingly common in people with learning disability. Communication difficulties, the vagueness of the symptoms, and the presence of other priorities often mean that carers and clinicians miss looking for this problem.[1]

Why is it missed?

The usual trigger for doctors to consider this diagnosis is the specific mention of constipation by the patient, or the communicated history of an abdominal symptom. Patients with learning disability may be unable to communicate these, to either their doctor or carer. In addition to more intuitive presentations such as overflow diarrhoea or urinary incontinence, pain from constipation may present as distress, sleep disturbance, or behavioural changes, including self harm.[6] According to expert opinion, recognition of constipation in children with disability is commonly delayed, with symptoms often present for months or years beforehand.[7] In a retrospective cohort of hospital deaths caused by intestinal obstruction, people with learning disability and constipation presented late and with minimal signs and symptoms.[8]

Why does it matter?

In patients who are less able to indicate the site of pain, or perhaps the very existence of pain, it is important to consider this very treatable cause of unnecessary suffering. Chronic constipation is also associated with urinary and faecal incontinence (further increasing the risk of social stigma), chronic nausea, rectal prolapse, anal fissures, haemorrhoids, the need for manual evacuation, and hospital admission for faecal impaction.[3]

When people with learning disability were asked to rate their health problems, those listing constipation were more likely to have their health rated as poor.[9] Learning disability predicted fatal intestinal obstruction, and chronic constipation was a risk factor for this in one hospital based study.[8]

Case scenario

A 48 year old woman with mild intellectual impairment and epilepsy (for which she takes phenytoin) presented with left sided abdominal pain. She had mentioned this before but had denied constipation or diarrhoea. We found an archived radiograph showing moderate faecal loading. When pressed, she admitted not knowing what "constipation" meant and, in fact, had been manually evacuating but was too ashamed to tell anyone.

How common is it?

In a randomly selected institutionalised adult population of 215 people with learning disability (IQ <50), 149 cases (69.3%) had constipation[2]

In children with severe disabilities, estimates of the prevalence of constipation vary from 26% to more than 50%[3]

People with learning disability are more likely to be taking drugs that are associated with constipation, such as anticonvulsants, benzodiazepines,[2] and antipsychotics

Other factors associated with constipation in people with learning disability include reduced physical mobility and refusal to eat[2]

Some causes of learning disability, whether inherited (such as Down's syndrome) or acquired (such as cerebral palsy), are strongly associated with constipation. In one study, 74% of children at a cerebral palsy outpatients clinic had chronic constipation.[4] In comparison, a Canadian study of the general adult population showed a prevalence of around 15%[5]

How is it diagnosed?

Clinical features

Actively consider constipation as a diagnosis in patients with learning disability. A focused history from the patient or carer regarding bowel habit (including faecal overflow) and abdominal pain is required, with careful attention to ensuring that the patient understands. We suggest that constipation be specifically considered during all learning disability checks as part of directed enhanced services in the United Kingdom.[10] Faeces may be palpable on abdominal examination, and rectal examination may show impacted faeces or rectal complications such as fissures or prolapse.

Investigations

Abdominal radiography, although usually unnecessary in the general population, is often confirmatory and can be a useful investigation in people with learning disability.[7]

How is it managed?

Management principles include emptying the bowel and maintaining soft, lubricated stools in the long term (months, or even years if necessary). A small randomised controlled trial in neurologically impaired children showed that increasing dietary fibre significantly increases stool frequency and reduces painful defecation.[11] Effective agents include polyethylene glycol (which provides benefit over long periods for chronic constipation, including

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)

KEY POINTS

- People with learning disability are prone to constipation, often because of medication, reduced mobility, or refusal to eat
- They may not communicate the typical symptoms to doctors and carers and sometimes present with apparently unrelated behavioural change; untreated constipation may cause unnecessary problems and occasionally even death
- Doctors should actively consider and exclude the diagnosis of constipation in people with learning disability using history, abdominal examination, and, where necessary, abdominal radiography
- Although ideally treatment should include increasing dietary fibre, laxatives are usually needed, sometimes for long periods

faecal impaction),[12] psyllium, and lactulose (on the basis of a systematic review of 20 commonly used agents).[13] Rectal suppositories or enemas are necessary only occasionally and should be considered a last resort in children.[14] Three before and after studies recommend regular toileting and behavioural treatments including rectal sensory biofeedback during defecation and psychological support,[12] which may be especially important in people with learning disability. If available, health promotional materials with pictures and simple language are particularly helpful for those with learning disability.[15]

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A PATIENT'S JOURNEY

Dopa responsive dystonia

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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.

Karen Low helps Teresa Harding tell the story of her life with dopa responsive dystonia, which was not diagnosed until she was in her mid twenties

I was six when they sent me to the children's hospital. The doctor told my mother, "I don't know why you've brought this child here—you're doing it for attention and you've wasted my time." My mother says that if they had watched me walk out to the car they would have seen the problem, but they just thought I was attention seeking. In my old notes it says that I had a psychological problem.

My legs continued to deteriorate. I struggled to walk and sometimes collapsed in the street. I would gradually become worse as the day went on and my father often had to carry me. This continued until I was nine when my parents arranged a private appointment with a neurologist. They felt this was the only way to make someone see what was happening.

The consultant admitted me to Frenchay Hospital, Bristol, for observation. The floors were very slippery so I found it very difficult to walk and my feet pulled inward. Initially, my parents were told I had muscular dystrophy; but then the doctors decided that this was the wrong diagnosis because my muscles were not wasting away. I remained under the care of the neurologist for the next nine years, during which time I had five years of physiotherapy that caused my legs to become weaker, rather than stronger. Various tests, including a brain scan, found nothing wrong. I had iron callipers on both legs, and then I progressed to plastic splints. My posture became very poor and I continued to struggle to walk. On every annual visit I was told I was "unique" and that they did not know what was wrong with me.

At 18 I had a kidney infection and could not stop vomiting, so my GP gave me antibiotics and an anti-sickness injection. Things immediately went from bad to worse. For several hours my ankles pulled inwards, my whole body went stiff, and I was unable to relax. My doctor did not understand

A DOCTOR'S PERSPECTIVE

The form of dopa responsive dystonia described here is inherited as an autosomal dominant trait and is caused by mutations in a gene for guanosine-5-triphosphate (GTP) cyclohydrolase (GCH1). This enzyme is essential for normal dopamine synthesis. The symptoms and signs of dopa responsive dystonia are varied, but onset of dystonia in early childhood with marked variation in symptoms throughout the day is typical. For this reason, there might be a mismatch between the child's symptoms and the signs that are evident to a doctor at a particular time. A range of other neurological symptoms and signs have been described in this condition including tremor, spasticity, and ataxia. Many people with dopa responsive dystonia are misdiagnosed with other neurological conditions, but the diagnosis can be confirmed in several ways, including DNA analysis. Dopa responsive dystonia is often completely curable with levodopa (L-dopa) replacement. Some patients respond quickly to a low dose of L-dopa, whereas others need a larger dose and respond more slowly.

Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non progressive disturbances that occurred in the developing fetal or infant brain. Not all conditions that affect posture and movement and start in early childhood are a type of cerebral palsy. The prevalence of cerebral palsy varies throughout the world but is about 2 in 1000 live births in developed countries. The prevalence of DRD is unknown, but is a much rarer condition. In 14 years as a consultant paediatric neurologist I have diagnosed five new cases. There are likely to be many undiagnosed patients in the UK.

Magnetic resonance brain imaging is crucial in evaluating the cause of a patient's cerebral palsy. Although normal brain imaging does occur in some people with cerebral palsy, this raises the possibility of an alternative diagnosis. The condition starts in early childhood and does not fluctuate markedly as the day goes by. Although the functional abilities of a child with CP might change over the years—for instance losing the ability to walk independently in adult life—a marked progression in symptoms should raise the possibility of an alternative diagnosis. Cerebral palsy does not usually recur within families. A first degree relative with cerebral palsy should certainly lead to the diagnosis being questioned. A growing list of rare genetic and metabolic disorders, can masquerade as cerebral palsy. Accurate diagnosis is important because some of these conditions are treatable—or even curable—and some might recur in families.

No doctor can be omniscient. There will always be patients with rare conditions that the doctor is not familiar with. Keeping an open mind about potential diagnoses and listening carefully to the patient's story is important.

Philip Jardine

what had happened. I had no strength in my legs at all, was reduced to crawling, and had lost my sense of balance. I was off sick from work as a telephonist for six months and went from 161 lbs to 108 lbs. When I saw the neurologist for my annual appointment he said, "Whatever happened to you?" I told him, but nobody could explain my illness and there was nothing more he could do for me. My condition still baffled medical staff and that year they wrote me off.

Living with my disability

I am quite an outgoing person and I had brilliant friends throughout school who really helped me. I have never let my illness get to me and nobody has been nasty about it, but I did suffer because I couldn't do the same activities as everyone else. I could never go on school trips or to school camp, because in those days facilities to help disabled people did not exist.

So I lived with it—dragging my feet and not being able to walk without holding on to something or somebody for support. Most of the time I was forced to use a wheelchair.

In 1990 I moved to Clevedon with my future husband and had my first son. I had been told I would be unable to conceive, so it was a shock when I became pregnant. I love being a mum, but my disability made it difficult to begin with. Social services provided me with home help five days a week to assist me with the baby and housework.

My lowest point was after I had had my first son. I had gone over on my ankle many times. It was so painful and weak that I couldn't look after my son properly, so I went to my GP.

I was sent back to Frenchay Hospital and saw a doctor who asked about my legs rather than my ankle injury, which was difficult to understand. He wanted to know how far I could walk and how things were during the day and the evening. When I asked him the reasons for these questions, he said "If we can get you on the right medication, it can change your life."

The turning point

How right he was. I was told that I had a condition called dopa responsive dystonia. I was started on co-beneldopa 125 mg, one daily for the first week, increasing to six daily

LESSONS TO BE LEARNT

The bad

I felt quite let down by the medical profession in my younger years. It felt like no one listened to my parents, really looked at me, or understood the gravity of the situation.

Having my symptoms labelled as "psychological" is still particularly upsetting.

In my younger years I was unable to participate in many activities that other people my age could do and sometimes felt excluded. Nowadays disabled facilities are so much better and allow for people with disabilities to be much more included.

The good

I have now met doctors who believe in listening to mothers' instincts and paying attention to what the patient is telling them. This would be my advice for doctors reading this—always listen to what the patient is telling you.

One of the most important things was that someone could give my problem a name and could then do something to treat it.

I have now met doctors who believe in excellent communication. My current specialist is so accessible, he listens to all my concerns and doesn't even mind if I contact him occasionally with email queries, and he always gets back to me very quickly.

I hope that I can help other people who have this condition. I have become actively involved with the Dystonia Society and speaking to families who are newly diagnosed.

ADDITIONAL RESOURCES

The Dystonia Society (www.dystonia.org.uk)—A UK wide charity providing support, advice, and information for anyone affected by dystonia

Action for Dystonia, Diagnosis, Education & Research (ADDER) (www.actionfordystonis.co.uk)—UK based group aiming to advance education, promote treatment, and offer support for patients

Contact a Family (www.cafamily.org.uk/Direct/d51.html)—Dystonia page of Contact a Family, the UK site that allows users to contact other families in similar positions. Specifically aimed at families with disabled children

Dystonia Support 4 U (www.dystonia-support4u.com/about.php)—UK based support group

The European Dystonia Federation (www.dystonia-europe.org)—This portal includes links to websites about dystonia in different European countries

Dystonia Bulletin Board (www.dystonia-bb.org/forums/asd/)—Sponsored by the Dystonia Medical Research Foundation, provides support and discussion for patients, family members, and caregivers with all forms of dystonia

within six weeks. After taking the first tablet I noticed an instant effect. I felt as though I had strength in my legs for the first time in years. I then stabilised at two tablets daily and continued to improve slowly over the next months. Gradually my balance and posture improved and my body began to relax. In April 1992 I realised my dream of walking down the aisle, with my splints still in place under my wedding dress. On our honeymoon I discarded my splints and started the difficult process of teaching myself to walk again.

My second son was born in April 1994. During my pregnancy my consultant advised that I reduce my medication, which I did without complications. I increased the dose again after the birth.

I am now 45 years old—I walk normally and have a very active family life. My great love is line dancing. I was always afraid of the illness returning and I've had a few blips that really worried me. I asked the consultant if I would get used to the tablets and whether the condition would come back. He reassured me that should that ever happen; I can just have my medication increased.

I still class myself as disabled—I still have the condition and if I don't take the medication I can't walk. On some occasions when dancing I have suddenly thought I had better sit down or I would fall over.

Since my diagnosis other family members have been tested. Now we know that my mother, my eldest son, both my aunts, and some of their children have the condition. They are all being treated for it. If Dr Jardine hadn't put in the effort to investigate my family they would never have known that they have dopa responsive dystonia as well.

I continue to take co-beneldopa and will for the rest of my life. It is a small price to pay for the life I thought I would never have.

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10-MINUTE CONSULTATION

Acute cough in adults

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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 healthy, non-smoking 54 year old woman consults with a severe acute cough. It started two weeks ago with symptoms of a common cold, but she is worried about its duration and would like something to "clear it up."

What you should cover

- Ask about the duration and nature of cough, dyspnoea, wheezing, thoracic pain, upper respiratory tract symptoms, smoking, and use of drugs. Explore restrictions in activities as a result of the cough.
- Are the symptoms caused by a self limiting or serious illness? Acute cough (less than three weeks) is most commonly caused by self limiting infections of the respiratory tract. Consider other common causes: (passive) smoking, bronchial hyper-reactivity, use of angiotensin converting enzyme inhibitors, postnasal drip, and gastro-oesophageal reflux disease (unlikely with no history of heartburn or regurgitation).
- Consider pneumonia—factors associated with an increased risk include use of oral glucocorticosteroids, heart failure, chronic obstructive pulmonary disease (COPD), insulin dependent diabetes, serious neurological disorder, rheumatoid arthritis, Parkinson's disease, cancer, and older age (especially over 80).
- Asthma must be considered but is unlikely with no history of respiratory or atopic problems. COPD should be considered in smokers. Persisting paroxysms of cough, sometimes with vomiting, suggest whooping cough. Assess for less common causes, including Q fever, pulmonary embolism, tuberculosis, heart failure, and malignancy.

What you should do

Physical examination and additional diagnostic testing

Gauge degree of illness by assessing fever, tachypnoea, tachycardia, and level of consciousness. CRB-65 scoring (new onset mental confusion, respiratory rate >30/min, blood pressure: systolic <90 mm Hg or diastolic <60 mm Hg, age ≥65) may be useful.

Examine the pharynx for signs of upper respiratory tract infection; perform lung auscultation and percussion. However, clinical signs and symptoms do not reliably differentiate atypical from typical pneumonia.

For suspected lower respiratory tract infection, near patient measurement of C reactive protein (CRP) can help

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differentiate serious disease from self limiting disease and has more diagnostic value than erythrocyte sedimentation rate and leucocyte count. A low C reactive protein (<20 mg/l) rules out pneumonia and can help in withholding antibiotics.

Microbiological tests are not routinely recommended in primary care. Chest radiography should be performed if serious illness (malignancy, heart failure) is suspected, although it has a limited role in primary care for detecting pneumonia on logistical and economic grounds.

If whooping cough is suspected and the patient lives close to a newborn infant or pregnant woman, antipertussis toxin IgG antibodies should be measured (single raised titre is 99% specific for diagnosis).

Advice and treatment

Patients often worry about coughs and expect antibiotics. Eliciting their main concerns and expectations, and their view on antibiotics, ensures the patient's agenda is dealt with and "opens up" the conversation about antibiotics.

A symptomatic and prognostic diagnosis is better than an aetiological or anatomical one, because aetiology in acute cough is often not known. Discuss the association between smoking and symptoms, and consider a smoking intervention (see NICE guidance).

Dextromethorphan is an effective antitussive that suppresses cough reflex (number needed to treat (NNT) 3-8). Codeine has no greater efficacy but more side effects. Encourage self management strategies including rest, analgesics, antipyretics, and regular fluids. Home remedies such as lemon and honey may alleviate symptoms. Patients report some effect of expectorants, mucolytics, antihistamines (or decongestants), and inhaled bronchodilators, but little evidence supports or discourages their use. Potential benefits must be weighed against the risk of side effects. Antibiotics are not indicated in cough caused by upper respiratory tract infection and offer limited benefit in terms of cough resolution (NNT 6) in acute bronchitis. Potential benefits (improvement in mean duration of cough of less than one day) and adverse effects (nausea, diarrhoea, rash) must be balanced. Antibiotics are likely to be beneficial for community acquired pneumonia or lower respiratory tract infection with a potentially complicated course—follow local guidelines on first choice of agent. British Thoracic Society guidelines for community acquired pneumonia recommend amoxicillin 500 mg three times daily for seven days or, in case of penicillin hypersensitivity, doxycycline or clarithromycin. In whooping cough, treatment may not affect the patient's symptoms, but a seven day course of erythromycin within 21 days of onset reduces the period of infectivity and may prevent transmission.

Consider hospital referral for patients who have lower respiratory tract infection with signs of severe illness, age over 80, comorbidity, or suspected pulmonary embolism or malignancy. CRB-65 scoring can help determine severity.

Create a safety net by discussing factors that should prompt reconsultation (such as haemoptysis, fever lasting more than four days, increasing dyspnoea and fast breathing, decreasing consciousness, and confusion) and back up your advice with an information leaflet (box).

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USEFUL READING

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RECOMMENDED RESOURCES FOR PATIENTS

European Centre for Disease Control (www.antibiotic.ecdc.europa.eu)—Patient information brochure on acute cough, colds, and flu

European Centre for Disease Prevention and Control (http://ecdc.europa.eu/en/eaad/antibiotics/Pages/messagesForPublic.aspx)—Advice on using antibiotics responsibly

NHS Direct (www.cks.nhs.uk/patient_information_leaflet/chest_infection_adult)—Patient information leaflet NHS on chest infection

ANSWERS TO ENDGAMES, p 541. For long answers go to the Education channel on bmj.com

STATISTICAL QUESTION

Describing the spread of data I

Answers *a*, *b*, and *c* are true; *d* is false.

PICTURE QUIZ **More than skin deep?**

1 The different types of melanoma are superficial spreading melanoma, lentigo maligna, acral lentiginous melanoma, and nodular melanoma.
2 Breslow's thickness measures in millimetres the distance between the upper layer, or granulous layer, of the epidermis and the deepest point of the tumour's penetration, allowing staging of malignant melanomas. The thinner the Breslow's thickness, the better the chance of a cure.
3 The computed tomography and radiograph images show multiple polypoid filling defects in the proximal jejunum.
4 The investigation of choice to facilitate histological evaluation is small bowel enteroscopy and biopsy.
5 The most likely diagnosis is superficial spreading metastatic malignant melanoma.