We report a case of medically unexplained quadriplegia following lumbar decompression and discuss the management of this type of problem.

Case history

A 56-year-old woman with seronegative polyarthritis presented with a four-year history of lower back pain and right-sided sciatica. On examination she had reduced power and impaired sensation in her right leg. Magnetic resonance imaging (MRI) of the spine revealed anterior degenerative slip of L4 on L5 with moderate overall reduction in canal calibre and osteophyte encroachment in the right lateral recess at that level, causing compression of the right L5 nerve root. The facet joints were subluxed and there was prominent high T2 signal between the articular surfaces. She underwent an uncomplicated L4/5 posterior spinal decompression and instrumented fusion and initially made an uneventful postoperative recovery.

Five days after surgery she woke up quadriplegic. Examination initially revealed absent power (0/5 by MRC power grading). However, it was noted that the patient was able to use her limbs to help turn in the bed. In addition, the patient was able to protect herself when her paralysed arm was dropped over her face. It was possible to demonstrate brief contractions of near-normal power with encouragement. Reflexes were all present and the plantar responses were down going. There was altered sensation in all four limbs but no clear sensory level. Sphincters function was intact. Computed tomography (CT) of her brain was normal and we suspected functional weakness. The patient’s symptoms improved gradually over the course of two weeks with simple reassurance and encouragement. By the time she was discharged she had made a complete recovery.

Discussion

Neurological symptoms with little or no underlying organic disease are relatively common, accounting for approximately one-third of all new patients seen in neurology outpatient clinics. Several names have been historically applied to such symptoms including ‘hysterical’, ‘non-organic’, ‘psychogenic’, ‘somatisation’, ‘conversion disorder’ and ‘medically unexplained’. The term ‘functional weakness’ is used here in view of recent imaging data suggesting neural correlates for some of these symptoms, and because it is less offensive to patients, providing a framework for explaining their disease and planning treatment. Patients with functional symptoms come to the attention of clinicians either because significant functional symptoms overlay suspected organic disease or more rarely when a functional presentation mimics an emergency such as spinal cord or cauda equina compression.

The concept of somatization implies that the mechanism that generates the disability is subconscious and involuntary. Distinguishing this from a conscious, factitious disorder (malingering) is very difficult but factitious disorders are thought to be much rarer. In our experience, working from the starting assumption that the problem is subconscious produces better therapeutic results. Patients with functional disease may be just as disabled and often more distressed than their counterparts with organic disease.

Furthermore, there is a growing body of evidence that many functional symptoms are treatable once recognized. The assessment of functional neurological symptoms and their subsequent
management have received relatively little attention in the literature.

**Assessment**

History-taking in patients with suspected functional symptoms serves both to aid diagnosis, and to plan subsequent management. Important ‘red flags’ suggesting functional symptoms are a long list of physical symptoms, and a history of previous functional symptoms such as such as irritable bowel syndrome or chronic fatigue. Obtaining a patient’s medical case-notes is particularly helpful in revealing the latter, as patients may have forgotten previous problems, or sense that providing this information will lead to their presenting symptoms being taken less seriously. Depression, anxiety and panic attacks are also more common in patients with functional symptoms than with organic disease and should be sought in the history. Enquiring about psychological symptoms can make a patient defensive so such questions may be better left until the end of the clinical assessment; broaching this area by asking the patient how the illness has affected them from a psychological point of view will usually be acceptable.

Alongside these diagnostic aids it is also useful to assess the ideas, concerns and expectations of patients with suspected functional symptoms. This information is helpful in allowing an individualized explanation and planning further management.

As in many patients with functional symptoms, we were not able to elicit any of the ‘red flags’ described in the reported case. Neurological diagnosis in such patients is, therefore, particularly reliant on the physical examination both to rule out organic disease and to look for positive physical signs that suggest the symptoms might be functional. In this instance it was important to rule out postoperative Guillain-Barre syndrome or an incidental cord lesion, so it was reassuring to find that objective physical signs, like the tendon reflexes and plantar responses, were normal.

Careful preliminary observations may demonstrate inconsistencies suggestive of functional weakness. We noted, for example, that while the patient described was quadriplegic when directly examined, she was able to use her limbs when turning in her bed. In addition, a number of specific tests have been developed to detect non-organic weakness. ‘Hoover’s sign’, initially described over a century ago, is arguably the most useful of these tests and has been found in controlled studies to have good sensitivity and specificity. It relies on the principle that flexion at one hip joint is accompanied by involuntary extension of the contralateral joint. The examiner cups the heel of the more affected leg and notes the strength of hip extension. They then note the strength of hip extension when the patient is asked to flex the contralateral hip. Discrepancy between voluntary hip extension (which is often weak) and involuntary hip extension (which should be normal) suggests functional paresis. A recent variation of this test is the ‘abductor sign’ which is based on the principle that the hip abductors work in concert. Here, the examiner may find voluntary hip abduction to be weak, but involuntary hip abduction to be normal when asking the patient to abduct the contralateral hip. Although very useful, these signs relying on synergistic muscle activity should be interpreted cautiously. First, these signs are appropriate only in cases of predominantly unilateral weakness and can lead to false-negatives if applied to patients with bilateral symptoms. Secondly, cortical neglect, or pain in the affected limb, may produce greater weakness in direct compared with indirect testing leading to false-positives.

Several other signs have been used to help diagnosis in functional disease. ‘Collapsing weakness’, when light touch causes a limb to collapse from an instructed position, is a common finding among patients with functional disease. Normal power can often be achieved transiently with encouragement by the examiner. Alternatively, the force applied to the limb can be gently and imperceptibly increased until normal. However, interpreting this sign requires considerable clinical experience; a number of small validity studies have found it to be a relatively poor discriminator between functional and organic symptoms, and similar weakness can be seen in myasthenia gravis and in patients who have both an organic neurological disorder and a non-organic one (so called ‘functional overlay’). The ‘arm drop’ test, which was positive in our case, is usually performed when the examiner drops
the affected arm above the patient's face; in patients with non-organic weakness the patient's hand misses their face. Although supported anecdotally, there are no published studies to support its use.7,12

Investigations may be performed to supplement the history and examination of patients with functional disease, particularly in ruling out organic disease. Had the weakness persisted we had planned to perform an MRI C-Spine and electrophysiological studies to assess the possibility of an incidental cord lesion or postoperative Guillain-Barre syndrome. Care must be taken, however, to avoid over-investigation of functional symptoms with costly, potentially unpleasant and dangerous techniques such as lumbar puncture, angiography or nerve and muscle biopsy.13,14

Management

In this case the patient's functional symptoms resolved with reassurance alone. There are a number of important elements for clinicians to consider when discussing functional neurological symptoms with patients15: Doctors should reassure the patient that they believe their symptoms to be genuine while explaining that although the patient does not have a 'structural' problem, they do have 'functional' problem. They should emphasize that this is common and reversible, and further stress that self-help will be an important part of recovery. Physiotherapy may also be useful in these circumstances. Often this is all that is required.

Patients with functional symptoms that do not resolve with reassurance may be referred to neurologists or liaison psychiatrists for further treatment. Studies of functional neurological symptoms have lagged behind other functional symptoms but there is evidence to support the effectiveness of cognitive behavioural therapy and antidepressant drugs in the treatment of a range of other somatic symptoms such as fatigue, fibromyalgia and irritable bowel syndrome.4,5

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