Musculoskeletal Complications of Neurological Conditions

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Introduction

Physically disabling neurological conditions can result in secondary musculoskeletal complications that limit patients' activities even further. These secondary complications can develop at any stage after the onset of a neurological illness, and the ability of clinicians to recognise and treat these complications will improve patients' functioning. Rehabilitation physicians have traditionally come from a neurological or rheumatological background, and current training in rehabilitation medicine includes rotations in both neurology and rheumatology. Rehabilitation physicians are therefore well placed to assess and treat patients with neurological conditions who have developed secondary musculoskeletal complications.

The impairments caused by neurological conditions can result in direct degenerative complications in skeletal and soft tissues. Complications may also arise secondarily to the neurological condition or its treatment. As trauma is a common cause of neurological deficits, patients who have sustained trauma often present with concomitant musculoskeletal injuries. Finally, the sudden onset of neurological symptoms may herald an underlying rheumatological condition.

Direct Degenerative Complications

Musculoskeletal problems can be a direct consequence of the neurological condition resulting from decreased or increased muscle activity around a joint. For instance, imbalance of the muscles of the rotator cuff in patients with stroke can contribute to post-stroke shoulder pain[1]. The condition is more common in patients who had degenerative shoulder conditions prior to their stroke. Post-stroke shoulder pain is, however, multifactorial and its treatment requires a concerted multidisciplinary and multimodality approach that is beyond the scope of this article.

Neck and shoulder pain can also be a feature of a spinal cord injury (SCI)[2]. This is typically seen in mid-cervical lesions where spasticity in the trapezii muscles elevate the shoulders without opposition from antagonists, the so-called "coat-hanger" syndrome. This painful condition is generally preventable by early upper limb passive range of movement exercises and judicious use of anti-spasticity medications.

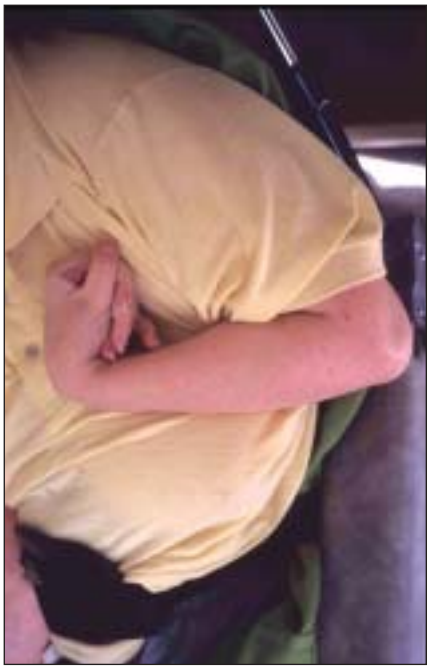

Figure 1. Wrist dislocation secondary to traumatic brain injury induced muscle spasticity.

Neurological conditions also result in patients putting extra stress across upper limb joints, which are not designed to take body weight. Intra-articular pressure in the shoulders can be as much as five times body weight when transferring from chair to chair, for example. Appropriate transferring technique with the use of a transfer board can alleviate these pressures and prevent further complications.

Intense upper limb muscle spasticity, as seen in traumatic brain injury (TBI), for instance, is frequently associated with wrist joint dislocation (figure 1). Treatment involves management of pain and spasticity by appropriate splinting, therapy and medication.

Wrist problems can also occur when patients use wheelchairs or crutches for mobility. Prolonged wrist extension when holding the handle of an elbow crutch or gripping a wheelchair rim can result in carpal tunnel syndrome. Indeed, the advice of SCI teams to patients in the past was to relieve pressure on their ischial tuberosities by pushing up on their wheelchair rims every 15 minutes. This resulted in a generation of SCI patients with iatrogenic carpal tunnel syndrome. Management is similar to that in idiopathic carpal tunnel syndrome, with rest, splinting and steroid injection as appropriate. If operative management is required it is necessary to arrange alternative mobility aids for patients, such as powered wheelchairs, while they are recovering from the surgery.

The widespread increased spasticity and dystonic movements associated with cerebral palsy frequently result in degenerative joint disease[3,4]. As a further complication, asymmetrical development of the spine in patients with hemiplegia or diplegia produces scoliosis, which can progress onto myelopathy[5,6]. For adults with cerebral palsy, joint pain is a common limiting factor with 67% to 84% of patients reporting large joint pain. Early detection and treatment of spinal and large joint degeneration particularly during growth spurts can reduce the degenerative complications associated with cerebral palsy.

It is not all bad news however, there is well-documented evidence of reduction in rheumatoid arthritis, osteoarthritis, gout and scleroderma in the parts of the body affected by neurological impairments[7–9]. The complex neuroendocrine basis of these conditions can be altered by neurological conditions resulting in reduced joint inflammation.

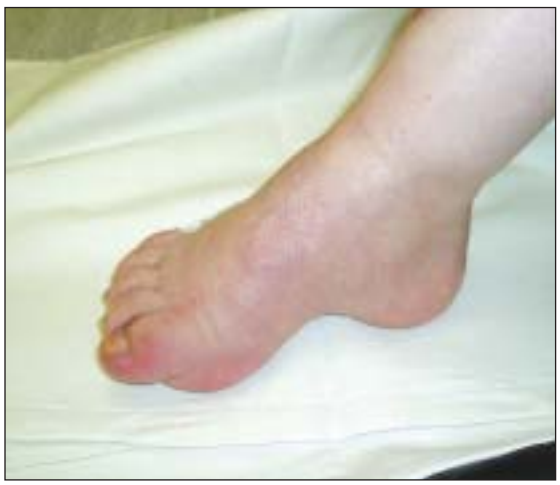

Figure 2. Ankle, tarsal and metatarsal neuropathic joints in a patient with diabetes mellitus.

Secondary to Neurological Injury

A wide range of secondary musculoskeletal problems can occur due to the neurological impairments caused by the original neurological condition. The prototypical condition in this respect is the neuropathic or Charcot joint. This is seen most commonly in the UK in patients with diabetic neuropathy, who develop neuropathic tarsal, metatarsal and ankle joints (figure 2)[10]. Upper limb neuropathic joints are seen in patients with syringomyelia. Worldwide, neurosyphilis and leprosy are also common causes of neuropathic joints. The aetiology of the joint destruction is due to repeated trauma to the anaesthetic limb, which is not protected by nociceptive and propioceptive mechanisms. Neuropathic joints are swollen and unstable, with relatively less pain than might be expected, although rarely pain free as commonly reported. Radiologically, subchondral lucencies are the earliest signs, progressing to microfractures, articular irregularities and increased uptake on the delayed phase of an isotope bone scan. Treatment is, generally, prevention of the neuropathy and orthotic protection of the neuropathic joint. More recently, bisphosphonates have been demonstrated to be useful in the amelioration of joint destruction and pain[11].

Another bone disease directly related to the patient's neurological condition is heterotopic ossification (HO). This is a condition of uncertain aetiology characterised by an imbalance between bone formation and resorption. New bone is laid down, generally in the soft tissues surrounding a large joint, within the area of impaired neurology. For patients with paraplegia, it is mainly in the hips and knees, but for patients with tetraplegia, the shoulders and elbows can also be involved (figure 3). The incidence of heterotopic ossification in spinal cord injury is 20% to 30% within two to five months of injury[12]. The diagnosis is established by a combination of raised bone specific alkaline phosphotase and increased uptake on isotope bone scan[13]. However, these tests are rarely entirely sensitive or specific, and ultrasonography, computed tomography or magnetic resonance imaging have all been demonstrated to be useful in the early detection of HO (figure 4)[14]. Treatment is conservative in the first instance with early immobilisation of the joint and high dose non-steroidal anti-inflammatory drugs. Bisphosphonates have also been reported to be useful and my current treatment preference is oral alendronate, weekly for six months. If these measures are unsuccessful, surgery followed by radiotherapy may be required to release an ossified joint[15].

Impairments of muscle function may result in muscle changes including muscle contractures, fibrosis and atrophy. Changes will also occur in tendons, ligaments and joint capsules. Contractures affect 9% of patients with SCI and are increased by spasticity, concomitant TBI, pressure sores and delay in transfer for rehabilitation[16]. Prevention includes proper positioning, therapy and splinting in conjunction with spasticity management. Treatment has largely been unsuccessful, with surgical release of contracted muscles being the only effective therapy.

Musculoskeletal infections are also more common in patients with neurological conditions. Osteomyelitis and septic arthritis have been reported in up to 13% of patients following SCI. Some of these infections have been related to overlying pressure sores. For patients with TBI, increased muscle spasticity, unexplained pyrexias and decreased cognitive ability can be indicators of underlying bone or joint sepsis.

Osteoporosis is another musculoskeletal complication that can be directly attributable to the immobilisation caused by neurological impairments. High doses of steroids used for immunosuppression, may also be implicated in bone loss. For patients with SCI, bone mass is reduced to 70% of normal within six months of injury. The bone loss is principally from the femoral shafts, which may be injured in subsequent minor falls or trauma. Whilst fractures that occur concomitantly with a SCI are best treated operatively, fractures in patients with established SCI and osteoporosis should be managed conservatively[2].

Associated with Neurological Injury

Musculoskeletal injuries that occurred at the same time as the neurological lesion, in patients injured as a result of trauma, may complicate a patient's recovery. Thirty to forty percent of patients with TBI will have sustained musculoskeletal injuries, including spinal column injuries, at the time of the original injury. Long bone fractures are present in 20% of patients with traumatic SCI. Secondary injuries may also occur as a result of seizures or falls in patients with impaired balance. Bony healing is relatively unaffected in patients with neurological injuries; however, closed operative reduction of fractures is the preferred method of treatment as it reduces barriers to rehabilitation. For instance, self-propulsion in a wheelchair can be achieved much earlier without the encumbrance of an external fixator on a fractured radius and ulna. Bone healing is relatively unaffected early after neurological impairment; however, callus formation may be over-exuberant due to heterotopic ossification, impairing joint mobility if the fracture site is close to a joint.

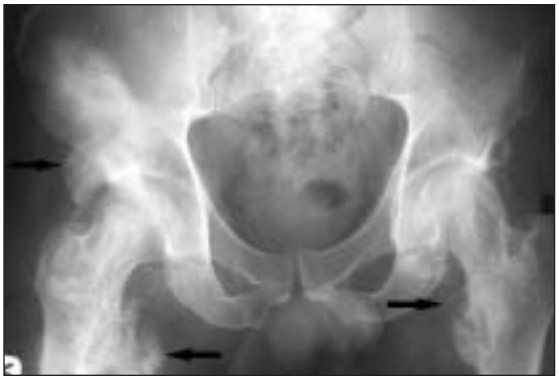

Figure 3. Heterotopic ossification seen on a pelvic radiograph of a 40 year old man with T4 paraplegia. Arrows indicate ossification at both hips and right iliac crest.

Figure 4. Computed tomography of the pelvis demonstrating ossification in the iliopsoas muscles bilaterally.

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Rheumatological Neurology

Finally, a patient presenting with neurological symptoms may be found to have an underlying musculoskeletal condition. Not only does rheumatoid arthritis (RA) result in significant musculoskeletal disability, it is also one of the commonest causes of secondary neurological impairment[17]. There is an increased risk of ischaemic stroke, which can be explained by a combination of increased diastolic blood pressure, and increased fibrinogen levels and plasma viscosity[18]. This is in addition to the effect of the antiphospholipid syndrome, which commonly accompanies rheumatological conditions. RA is also associated with neurological entrapment syndromes, ranging from atlanto-axial subluxation (approximately 5% of patients with RA)[19] to compressive neuropathies at the elbow and wrist.

Ankylosing spondylitis (AS) can be a direct cause of a compressive myelopathy. Patients with AS are also at increased risk of SCI following minor trauma. AS may be a contributory factor in cerebral ischaemia, particularly posterior circulation infarcts, if the vertebral arteries are compromised by spondolytic bone.

Marfan's syndrome can present with a similar range of neurological impairments. Aortic valve disease will predispose patients to cerebral ischaemia, while aortic dissection can result in paraplegia at T4, if the cord's vascular supply is compromised.

Conclusion

The impact of musculoskeletal problems on the function of patients with neurological conditions must be appreciated and treated. With the majority of musculoskeletal problems, preventative strategies, which are key components of the multidisciplinary rehabilitation programme, are most effective. Treatments must focus on improving patients' function and preventing secondary disability.

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