Non-traumatic neurological emergencies presenting to the intensive care unit

There is little evidence for the management of non-traumatic neuromuscular disorders, so the best approach to treatment is often unclear. This article discusses some of the more common disorders – Guillain–Barré syndrome, acute transverse myelitis and myasthenia gravis – and looks at their critical care management.

Traumatic neurological injury is common-place on the critical care unit and management normally follows widely published guidelines. However, the treatment of other neuromuscular disorders is supported by a sparse evidence base and, as a result, there may be confusion as to the best approach to therapy. This article outlines some of the more common disorders – Guillain–Barré syndrome, acute transverse myelitis and myasthenia gravis – and describes their subsequent critical care management. It must be emphasized that all patients should have regular advice and involvement of the neurological team, ideally with additional advice from a specialist centre with experience in the critical care management of these conditions.

Guillain–Barré syndrome

Epidemiology

This is a rare condition with an incidence of 1–2:100 000. Rates are generally higher with advancing age, in men and in caucasians. Important triggers of Guillain–Barré syndrome include non-specific respiratory and gastrointestinal infections as well as cytomegalovirus infection.

Pathophysiology

Guillain–Barré syndrome is an acute demyelinating polyradiculopathy. Various forms exist, including the Miller–Fisher variant presenting with ophthalmoplegia and ataxia. Acute inflammatory demyelinating polyneuropathy is the most common form of Guillain–Barré syndrome in the United States and Europe, causing approximately 85–90% of cases.

Myelin in the peripheral nerves is the target of an immune attack in acute inflammatory demyelinating polyneuropathy. Specific antigens on invading pathogens are shared with nerves and immune responses are then directed against these antigens, producing antibodies that cross-react with myelin to cause demyelination. Common triggers include viruses (such as influenza), bacteria (commonly campylobacter), certain immunizations, surgery and bone marrow transplantation. Demyelination is thought to start at the level of the nerve roots, leading to electrophysiological conduction slowing and conduction blocks with resultant muscle weakness. Multifocal, patchy, widespread peripheral nerve demyelination follows, causing increasing paralysis. Peripheral nerve remyelination occurs relatively rapidly over several weeks to months. However, in a small percentage of patients, there is significant concurrent axonal degeneration with markedly delayed and incomplete recovery (Jacobs et al, 1998).

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Clinical features

An illness 7–10 days earlier normally precedes the development of Guillain–Barré syndrome. Guillain–Barré syndrome normally presents as an ascending progressive, fairly symmetric muscle weakness accompanied by absent or depressed deep tendon reflexes. Weakness begins in the arms or facial muscles in about 10% of patients. Severe respiratory muscle weakness necessitating ventilatory support develops in 10–30%. Paraesthesias in the hands and feet accompany the weakness in more than 80% of patients. There can be cranial nerve involvement, including the extra-ocular muscles and pupils. If this occurs, then remaining residual weakness is more likely. Sensory abnormalities on examination are usually mild (Hughes and Rees, 1997).

Dysautonomia occurs in 70% of patients, manifesting as tachycardia (most common), bradycardia and other arrhythmias, urinary retention, labile blood pressure, orthostatic hypotension, ileus and loss of sweating. Severe autonomic dysfunction is important to recognize since this can be associated with sudden death (McLeod, 1993).

Diagnosis

The diagnosis of Guillain–Barré syndrome is normally based on clinical observations. Diagnostic tests may confirm Guillain–Barré syndrome and provide guidance for further treatment.

CSF may show elevated protein levels >400 mg/litre (although 10% of cases may have normal protein levels) and >10 mononuclear cells/mm^3, with other cell counts normal.

Bloods tests should include urea and electrolytes (to test for syndrome of inappropriate antidiuretic hormone), liver function tests (often elevated), erythrocyte sedimentation rate (almost always elevated), and antiganglioside antibodies (Miller–Fisher variant may have anti-GQ1b antibodies).

With advice from your microbiology department send tests for antibodies to Epstein–Barr virus, cytomegalovirus, human immunodeficiency virus, *Campylobacter jejuni*, herpes simplex virus, and *Mycoplasma pneumoniae*. Stool culture for *C. jejuni* is also recommended.

Magnetic resonance imaging of the brain or spine is sensitive but non-specific (may see spinal nerve root enhancement with gadolinium). Selective anterior nerve root enhancement is strongly associated with Guillain–Barré syndrome (cauda equina roots are enhanced in 83% of patients).

Respiratory function is discussed below, but guides assessment of disease severity and the need for intervention.

Nerve conduction studies may suggest conduction delays as a result of demyelination, with a decrease in motor action potentials and conduction blocks. The extent of decreased action potentials correlates with prognosis. Muscle biopsy may distinguish Guillain–Barré syndrome from a primary myopathy, but is usually unnecessary.

An electrocardiogram may provide useful information about possible autonomic dysfunction which may present as heart block (secondary or complete), or sinus bradycardia.

Critical care management

Supportive care

The critical care team become involved primarily when bulbar weakness and/or respiratory failure occur together. Pulmonary aspiration secondary to severe bulbar weakness may result in rapid deterioration of the patient's condition. Autonomic instability and other comorbidities may also require level II or III care. Many patients are initially admitted to the intensive care unit for close monitoring of respiratory and haemodynamic indices.

Airway and breathing

About 40% of patients will have respiratory symptoms on presentation, and 30% will require intubation and mechanical ventilation because they have inspiratory muscle weakness, but weakness of abdominal and accessory muscles, retained secretions and atelectasis also contribute. Close respiratory monitoring with frequent measurement of vital capacity and negative inspiratory force should be started on admission and continued while weakness is progressing. Indications for intubation include forced vital capacity <20 ml/kg, maximum inspiratory pressure <30 cmH_2O or maximum expiratory pressure <40 cmH_2O. Mechanical ventilation will be needed in >85% of patients who have four of the following: time of onset to admission <7 days, inability to cough, inability to stand, inability to lift the elbows, inability to lift the head or increased levels of liver enzymes (Teitelbaum and Borel, 1994).

Weaning from mechanical ventilation should be guided by improvement in muscle power and improvement in serial pulmonary function tests. Tracheostomy should be performed after 2 weeks if pulmonary function tests do not show any significant improvement from baseline, but can be deferred for another week if they show improvement.

Circulation

Central venous pressure line insertion may be indicated to permit monitoring of fluid status and use of vasopressor or inotropic drugs. Arterial line insertion is advised in order to monitor blood pressure and arterial blood gases closely.

Autonomic dysfunction: Dysautonomia occurs in 70%. Consequently, close monitoring of blood pressure, fluid status and cardiac rhythm is essential to the management of patients with Guillain–Barré syndrome. Monitoring should be continued until ventilatory support is no longer necessary or until recovery is underway in patients not needing mechanical ventilation.

Arrhythmias: These frequently occur during suctioning. Sustained sinus tachycardia occurs in 37% of patients requiring no treatment, and severe arrhythmias occur in 4%.

Orthostatic hypotension: This occurs in 19% of cases and should be treated with maintenance of intravascular volume. Vasopressor or inotropic support may also be required. Quadriplegic patients should not be left unattended in the sitting position without assessment of orthostatic hypotension. Drugs with hypotensive side effects should be avoided if possible. Other causes of hypotension, such as pulmonary embolism, bleeding or electrolyte disturbance, must be excluded.

Hypertension: Paroxysmal hypertension occurs in 24% of cases and prolonged hypertension in 3%. Episodes of severe hypertension (mean arterial pressure >125 mmHg) can be treated with labetalol, esmolol or nitroprusside (Winer, 2002).

Other therapies

Bowel and bladder care

Ileus and urinary retention are common. Daily abdominal auscultation is recommended to monitor for bowel silence and the development of ileus. Opioid drugs should be prescribed with caution. Promotility agents are contraindicated in patients with dysautonomia.

Pain control

Neuropathic pain occurs in 40–50% of patients during the course of Guillain–Barré syndrome, often requiring treatment. Simple analgesics or non-steroidal anti-inflammatory drugs may be tried, but they often do not

provide adequate pain relief. Appropriate narcotic analgesics may be used, but require careful monitoring for adverse effects in cases of autonomic denervation. For long-term management of neuropathic pain, tricyclic antidepressants, tramadol, gabapentin, carbamazepine or pregabalin may be useful.

Anticoagulation

There is a significant risk of deep vein thrombosis and pulmonary embolism as these patients are immobile. Thromboprophylaxis should be commenced early (once-daily regimen of subcutaneous enoxaparin 20–40 mg as well as thromboembolic deterrent stockings) and continued until the patient is able to walk independently.

Disease-modifying treatment

The choice between plasma exchange and intravenous immunoglobulins depends upon local availability, patient-related risk factors, contraindications and preference (Koski and Patterson, 2006). Evidence from the Plasma Exchange/Sandoglobulin Guillain–Barré Syndrome Trial Group (1997) showed that the effects of intravenous immunoglobulins and plasmapheresis are comparable and there was no advantage in using combined therapy. There is little evidence for use of corticosteroids, so they are no longer recommended. Because of their ease of administration and wide availability, intravenous immunoglobulins are frequently the preferred treatment. Plasmapheresis may cause hypotension and is contraindicated in patients with underlying cardiac comorbidities (Van Koningsveld and Van Doorn, 2005).

Rehabilitation

Acute phase rehabilitation should include an individualized programme of gentle strengthening, involving isometric, isotonic, isokinetic, and manual and progressive resistive exercises. Rehabilitation should emphasize proper limb positioning, posture and orthotics as well as nutrition.

Prognosis

The majority of patients will recover within weeks, but 30% will still have weakness after 3 years and 3% will relapse. The overall mortality rate is around 3–4% (El Mhandi et al, 2007).

Acute transverse myelitis

Epidemiology

The disease has two peak incidences: between the ages of 10–19 years, then again between the ages of 30–39 years. There have been few population-based studies of acute transverse myelitis and there appears to be no real link with any identifiable patient characteristics. There is no significant seasonal or annual fluctuation in frequency.

Pathophysiology

Acute transverse myelitis has a variety of causes. In 33% of cases, there is an antecedent history of prior infection, more commonly among younger patients (*Table 1*). The disease is often seen during recovery from primary infections (Berman et al, 1981).

Prior exposure to infective agents may cause a derangement of the immune system, initiating an indirect autoimmune attack on the spinal cord, rather than a direct attack by the organism. This is known as molecular mimicry.

Certain toxic agents (carbon monoxide, lead and arsenic) can cause a type of myelitis in which acute inflammation (followed by haemorrhage and possible necrosis) destroys the entire circumference (myelin, axis cylinders and neurons) of the spinal cord (Krishnan et al, 2004).

A variant, acute disseminated encephalomyelitis may present and be difficult to distinguish from multiple sclerosis. Acute disseminated encephalomyelitis typically follows a prodromal viral illness, often presents with fever, stiff neck and CNS disturbance, and is more likely to be widespread (including ataxia) with impaired consciousness. Optic neuritis is more often bilateral when it occurs in acute disseminated encephalomyelitis and myelopathy is usually complete, accompanied by areflexia. Multiple sclerosis typically is monosymptomatic

Table 1. Possible infective agents responsible for acute transverse myelitis

Bacterial infections	Syphilis
	Abscess
	Mycoplasma
	Lyme disease
Viral infections	Encephalomyelitis
	Poliovirus
	Herpes zoster
	Herpes virus B
	Rabies
	Human immunodeficiency virus
Autoimmune	Systemic lupus erythematosus
	Sjögren's syndrome
	Sarcoidosis
	Vasculitis
Vaccinations	Bacillus Calmette–Guérin
	Smallpox
	Polio
Other	Parasitic infection
	Fungal infection
	Acute multiple sclerosis

(e.g. optic neuritis or a subacute myelopathy) and has a relapsing, unremitting course.

Clinical features

Acute transverse myelitis can present in a similar fashion to Guillain–Barré syndrome, but cranial nerve palsies are very rarely seen with acute transverse myelitis. Symptoms usually occur over a period of days. Inflammation interrupts motor and sensory pathways causing limb weakness, sensory disturbance, bowel and bladder dysfunction, back pain and radicular pain (pain in the distribution of a single spinal nerve). Patients may also describe a band-like tightness or pain over their chest or abdomen.

Almost all patients will develop leg weakness of varying degrees of severity. The arms are involved in a minority of cases, depending on the level of spinal cord involvement.

There is often sensory loss below the level of the lesion. Pain and temperature sensation are lost, but there may be preservation of vibration and proprioception.

Diagnosis

Magnetic resonance imaging of the spinal cord or brain is done using gadolinium enhancement. If there is no mass lesion outside or within the spinal cord and no signs of demyelination within the brain suggestive of multiple sclerosis, the cause is likely to be acute transverse myelitis or a vascular cause.

On analysis of CSF, pleocytosis (often a mildly raised white cell count) or raised immunoglobulin G:albumin ratio may be seen. Autoimmune antibodies should be measured to eliminate an autoimmune cause such as systemic lupus erythematosus or Sjögren's syndrome.

Microbiology screen is needed on blood, CSF and sputum cultures. A routine blood screen should be sent, along with human immunodeficiency virus, vitamin B_{12} and syphilis testing.

Critical care management

Management of acute transverse myelitis is similar to that of Guillain–Barré syndrome. Critical care involvement usually occurs when respiratory symptoms are severe.

The airways, breathing, circulation approach should be followed, as with Guillain–Barré syndrome. Intubation and mechanical ventilation may be required in severe cases. Arterial line insertion is advised to monitor blood pressure and arterial blood gases closely. Central venous pressure monitoring may be necessary.

Other therapies

Corticosteroids are effective if given in the acute phase as methylprednisolone 1 g for 3–5 days (Hughes and Rees, 1997). Plasma exchange is of proven benefit in patients with severe transverse myelitis or in those who failed to respond to steroids (Rodriguez et al, 1993). Although there are no randomized control data available there is evidence from a small trial in 1976 (Rosen and Vastola, 1976) that cyclophosphamide may improve outcome in this area. A larger and more up to date study is required for this treatment.

Prognosis

Over 30% of patients with acute transverse myelitis will make a good recovery, and 30% will make a partial recovery generally beginning within 1–3 months (Berman et al, 1981).

Myasthenia gravis

Epidemiology

The prevalence of myasthenia gravis is approximately 1:20 000 among the western population, with a female preponderance of 3:2. There are two age peaks: women in the third decade and men in the fifth or sixth decade. There may be a genetic predisposition linked with certain HLA sub-types (Bell et al, 1986).

Pathophysiology

Myasthenia gravis is an autoimmune disorder that targets post-synaptic acetylcholine receptors within the neuromuscular junction. This causes blockage and degradation of receptors, which manifests as weakening muscle contractions with exercise. Understanding this process enables targeting of treatment against the acetylcholinesterase enzyme responsible for breakdown of acetylcholine within the neuromuscular junction after a nervous impulse. Treatment results in increased availability of acetylcholine within the junction and hence a decrease in fatiguability.

Up to 75% of patients have an abnormality of the thymus, and 25% of these have a thymoma. The disease status will often remain stationary after a thymectomy despite the removal of the neoplastic effects of the thymoma.

Clinical features

Common symptoms include weakness of proximal muscles, worsening with exercise. Ocular muscles are the most frequently affected muscle group, causing ptosis and diplopia which classically worsens towards the end of the day. Pupils are never involved and 20% of patients with myasthenia gravis will only have ocular involvement. The next most commonly involved muscle group is that controlling bulbar function, and these patients present with dysarthria and dysphagia. Sensation and reflexes are normal.

Diagnosis

Signs and symptoms can be further supported with a number of tests, although a normal test result does not exclude myasthenia gravis (Benatar, 2006).

In the tensilon test a short-acting acetylcholinesterase (pyridostigmine) is administered which will lead to a dramatic improvement of muscle weakness. An intravenous test dose of 2 mg is given, then after 30–60 seconds, a further 8 mg is given. There should be an improvement in muscle function (increased forced vital

capacity and/or maximal inspiratory pressure, or ocular movement) within 5–10 minutes, and reduced or absent fatiguability. Atropine and full resuscitation equipment should be available in case of a cholinergic crisis.

Approximately 85% of myasthenia gravis patients have acetylcholine receptor antibodies, and the test for this has a sensitivity of 80–96%. In patients who have a negative test result, typically those with isolated ophthalmic involvement, 40–70% have antibodies against muscle-specific tyrosine kinase.

Single fibre electromyography studies show increased jitter in muscle fibres.

Chest imaging is needed to rule out a malignant thymus gland as the cause.

Myasthenia gravis is associated with other autoimmune diseases therefore it is worth testing for antinuclear antibodies, antiparietal cell antibodies and antithyroid antibodies.

Critical care management

There are two life-threatening crises associated with myasthenia gravis that are likely to require critical care involvement. In a myasthenic crisis patients present with severe muscle weakness and respiratory failure, pyrexia with tachycardia and severe difficulty swallowing (Juel, 2004). A cholinergic crisis usually occurs after excessive anticholinergic drug administration, leading to weakness, respiratory failure, bulbar palsy, lacrimation, hypersalivation, diarrhoea, vomiting, miosis and sweating. Treat with atropine and stop the drug that precipitated it (Saperstein and Barohn, 2004).

Airway and breathing

A myasthenic crisis may manifest as respiratory failure or inability to clear secretions. Serial measurements of forced vital capacity or maximum inspiratory pressures should be performed. Forced vital capacity should be measured in the supine and sitting positions. Forced vital capacity <20 ml/kg, maximum inspiratory pressure ≥40 mmHg or greater than 30% decrease in either of these values heralds impending respiratory failure (Rabinstein and Wijdicks, 2003). This may require intubation and a period of artificial ventilation (Mazia et al, 2003).

KEY POINTS

- Post-infective neuropathies are becoming increasingly common indications for intensive care admission.
- The clinician should be vigilant for acute deterioration of these patients, particularly the development of respiratory compromise.
- Critical care should be involved early in order to escalate care as necessary.
- Most patients make a full recovery, but some are left with longer lasting, often permanent, sequelae.
- Recovery depends upon a multidisciplinary approach.

Any precipitating factors should be sought and treated to aid the patient's recovery. Such factors include occult infection, electrolyte imbalance, cholinergic crisis, thyroid problems, menstruation and certain medications. Drugs that may precipitate a crisis include aminoglycosides, beta-blockers, steroids and antiarrhythmics.

Cholinergic crisis must be suspected if a patient received anticholinesterase medications and presents with diarrhoea and fasciculations.

Circulation

Arterial line insertion is advised to monitor blood pressure and arterial blood gases closely. A central venous pressure line may also be required.

Disability and CNS

Any neuromuscular blocking agents should be avoided as they have profound, prolonged effects in myasthenia gravis.

Other therapies

Plasmapheresis usually produces a response within 48 hours, allowing time for other therapies to be instigated. Use a dose of 50 ml/kg/session over three to seven sessions. Beware that plasmapheresis increases sensitivity to anticholinesterase drugs and also can cause hypotension, arrhythmias and hypercoagulability. If patients have concurrent cardiovascular disease plasmapheresis may be contraindicated (Schneider-Gold et al, 2005).

Intravenous immunoglobulins give a similar rapid response to plasmapheresis but can be used in patients with cardiovascular disease contraindicating plasmapheresis. They are typically continued over five consecutive days at a dose of 400 mg/kg/day. Beware of fluid overload. Response is usually maximal at 2 weeks and persists for several weeks after administering.

For longer term immunosuppression corticosteroids, azathioprine and ciclosporin can be used in patients who fail to respond to acetylcholinesterase inhibitors. They generally take longer to work but can be started during a period of plasmapheresis (Dalakas, 2004).

Acetylcholinesterase inhibitors block breakdown of acetylcholine, allowing more muscle contraction. In excessive doses they can have fatal effects as a result of a cholinergic crisis, when a patient may experience excessive sweating and respiratory secretions (Richman and Agius, 2003). The most commonly used are pyridostigmine and neostigmine. They should be discontinued in a myasthenic crisis as they can cause increased respiratory secretions and do nothing to alter the course of the crisis.

Conclusions

Neuromuscular conditions can present with and follow a relatively benign course. However, when deterioration occurs, particularly involving the respiratory system, rapid intervention is often required to prevent mortality.

A good working knowledge of some of these diseases, with early critical care involvement can minimize associated morbidity. **BJHM**

Conflict of interest: none.

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Further information

Guillain-Barré patient advocacy group www.gbs.org.uk/index2.shtml

GBS foundation (USA) http://gbs-cidp.org/

Myasthenia Gravis patient advocacy group www.dailystrength.org/c/Myasthenia-Gravis/support-group

Transverse myelitis patient advocacy group www.myelitis.org.uk/