orphanaesthesia

Anaesthesia recommendations for

Transverse myelitis

Disease name: Transverse myelitis

ICD 10: G37.3

Synonyms: -

Disease summary: Transverse myelitis (TM) refers to a group of inflammatory spinal cord disorders with motor, sensory, and autonomic dysfunction [1,2]. It has an incidence of up to 8 in 1 000 000, with a bimodal age predilection in in the second and fourth decades of life [2,3]. Females and males are equally affected [4]. Approximately one third of cases are idiopathic, but it also may arise in association with infections, drug/toxin exposure, or underlying autoimmune disorders including multiple sclerosis. TM has occasionally been attributed to neuraxial anaesthesia [5-10]. and general anaesthesia [11]. The condition is characterised by accumulation of inflammatory cells in a localised region of spinal cord, with focal demyelination.

TM presents with a well-demarcated sensory level corresponding to the site of the spinal cord lesion, with associated weakness of distal spinal cord segments as well as bowel and bladder dysfunction [2,4]. The level of the lesion is most often in the thoracic or cervical spine. Neuropathic pain and allodynia are also common. Deficits are usually bilateral. The time from symptom onset to clinical nadir is usually within 3 weeks. An acute period of flaccid paralysis lasting up to 6 weeks is followed by the onset of upper motor neuron symptoms such as spasticity and hyper-reflexia. Autonomic dysreflexia and orthostatic hypotension have also been described [1].

In the acute phase, patients may be treated with systemic corticosteroids, intravenous immunoglobulin, plasma exchange, or immunomodulating medications (i.e., mitoxantrone, methotrexate, mycophenolate, rituximab, azathioprine, cyclophosphamide) [1,12,13]. Neurologic recovery predominantly occurs in the first 3 months after presentation, but many patients have a protracted course over years [2,4]. Up to 25% of patients experience a recurrence of TM. Only one third of patients fully recover without residual sequelae [3].

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Typical surgery

For prolonged respiratory insufficiency, tracheostomy may be required. Patients with TM may require surgery for similar indications as the general population.

Type of anaesthesia

There are few published care reports of anaesthetic management in TM. General anaesthesia with balanced anaesthetic maintenance is typically preferred. Epidural and spinal anaesthesia have been occasionally reported [14,15].

As with other neurologic disorders, the use of neuraxial anaesthesia or peripheral nerve blocks is not strictly contraindicated, though the relative risks and benefits of these approaches should be carefully weighed [16].

Necessary additional pre-operative testing (beside standard care)

Careful neurologic examination and detailed documentation of sensory and motor deficits is required. Prior history of autonomic dysreflexia episodes should be elicited, along with their triggers and symptoms, such as flushing, headache, diaphoresis, and hypertension.

Complete blood count, coagulation profile, type and screen should be obtained, as should standard liver and renal function tests.

Particular preparation for airway management

Gastroparesis and constipation may be seen in TM, possibly placing them at risk for aspiration. Rapid sequence induction should be considered.

Particular preparation for transfusion or administration of blood products

Patients with acute TM may be treated by plasma exchange.

Otherwise, no special considerations for blood product transfusion or administration.

Particular preparation for anticoagulation

Prophylactic anticoagulation is recommended when patients have limited mobility, and this may impact the patients' eligibility for certain regional anaesthesia techniques.

Particular precautions for positioning, transportation and mobilisation

Patients with residual neurologic dysfunction after acute TM may be liable to spasticity, osteoporosis, and pressure ulcers, so careful positioning and padding are essential [1].

Interactions of disease and anaesthesia medications

It is controversial whether patients acutely affected by or recovered from TM are at risk for disease recurrence when neuraxial anaesthesia is administered. Thus, the risks and benefits of epidural or spinal anaesthesia should be considered carefully.

As in other denervation injuries, administration of succinylcholine may be associated with significant hyperkalaemia, so this must be avoided, starting 24 to 48 hours after onset of the injury. It is unclear when succinylcholine may be used again safely after the acute phase of TM, but a period of at least one year seems prudent [17]. Patients with TM are sensitive to the effects of non-depolarising neuromuscular blocking agents and may have prolonged weakness after a single induction dose, therefore, these medications should be used judiciously. It is essential to employ neuromuscular monitoring to guide the use of paralytics. Sugammadex may be given to ensure complete reversal of muscle strength.

Anaesthetic procedure

There are few published case reports of anaesthetic management in patients with TM [18].

General anaesthesia has been described, using standard intravenous induction agents: propofol, thiopental, non-depolarising neuromuscular blocking agents, opioids, and lidocaine. Balanced anaesthesia maintenance with volatile anaesthetics, nitrous oxide, and intravenous opioids is effective. Endotracheal intubation is most commonly utilised, but supraglottic airways have also been described [19]. Maintenance of spontaneous breathing and avoidance of paralytics may be considered.

It is unclear whether neuraxial anaesthesia exacerbates TM. One benefit of neuraxial anaesthesia is avoidance of autonomic dysreflexia in patients with complete loss of spinal cord function below their level of injury, and this should be weighed against the potential risk of inciting additional neurologic damage. Other regional anaesthesia techniques should similarly be considered in light of relative potential benefits and risks.

Particular or additional monitoring

Neuromuscular monitoring is essential when patients require neuromuscular blocking agents. Other standard anaesthetic monitors should be used as appropriate (e.g., electrocardiogram, non-invasive blood pressure, pulse oximetry, capnography, gas analyser).

Invasive blood pressure monitoring should be considered when patients are at risk for autonomic dysreflexia.

Possible complications

Because patients with TM are treated acutely with high-dose corticosteroids, adrenal insufficiency should be considered in the event of perioperative haemodynamic instability. Liver dysfunction may be seen with administration of immunomodulatory medications used in TM.

The presence of neurogenic shock in the early course of the condition may cause hypotension and bradycardia.

Functional spinal cord transection above T6 may predispose patients to autonomic dysreflexia with resultant severe hypertension [20]. Autonomic dysreflexia has been reported as early as 72 hours after spinal cord injury and is due to a loss of descending inhibition over sympathetic spinal cord reflexes distal to the spinal cord lesion. Episodes are precipitated by nociceptive stimuli below the sensory level. The severe hypertension associated with autonomic dysreflexia may lead to intracranial haemorrhage and death, if left uncontrolled.

Post-operative care

Post-operative neurologic examination and documentation is recommended, particularly if there is subjective complaint of changes in neurologic status from baseline.

Patients with respiratory insufficiency may need prolonged post-operative ventilatory support in the intensive care unit.

Disease-related acute problems and effect on anaesthesia and recovery

Autonomic dysreflexia, adrenal insufficiency, respiratory insufficiency, and sensitivity to non-depolarising neuromuscular blocking agents, as described previously. Avoidance of succinylcholine may be necessary.

Ambulatory anaesthesia

Patients fully recovered from TM can likely safely undergo ambulatory anaesthesia. The degree of residual neurologic dysfunction should be considered, as should the time since recovery from TM.

Obstetrical anaesthesia

It is unclear whether pregnancy has any effect on TM. There are few reports of obstetrical anaesthesia care for patients with TM. Patients with ongoing sensory loss may not be able to detect uterine contractions, while labour progression and contractions may trigger autonomic dysreflexia.

General anaesthesia is the most common approach [14,18,20,21]. Standard aspiration precautions for pregnant patients should be observed (e.g., non-particulate antacid, H_2-receptor antagonist, metoclopramide, proton-pump inhibitor, rapid sequence induction, cricoid

pressure). Additional airway adjuncts should be available given dynamic airway changes during labour and poor maternal apnoea tolerance.

Effective neuraxial analgesia and anaesthesia can mitigate the risk of autonomic dysreflexia during labour or caesarean delivery, and may outweigh the theoretical risk of exacerbating TM [22,23]. While neuraxial anaesthesia has been safely performed in patients with TM and other demyelinating diseases (e.g., multiple sclerosis), this practice remains controversial and should be considered carefully.

One case report described caesarean delivery with ketamine sedation alone, for a patient with TM who was insensate up to T8 [2,4]. However, this approach is not generally recommended.

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Please note that this recommendation has not been reviewed by an anaesthesiologist and a disease expert but by two anaesthesiologists instead.