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Guillian Barre Syndrome- Anaesthetic Management of Tibialis Posterior Tendon Transfer Surgery for Foot Drop

KEYWORDS: Guillain-Barré syndrome (GBS), Tendon Transfer Surgery, Sciatic and Femoral Nerve Block

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ABSTRACT *Guillain-Barré syndrome (GBS) an acute autoimmune demyelinating polyneuropathy usually presents with ascending motor weakness, sensory and autonomic dysfunction frequently following a prodromal illness (usually a respiratory or gastrointestinal infection. GBS can cause significant morbidity requiring long hospital inpatient stay and significant periods of rehabilitation. Approximately 10–15% of patients require assistance with long-term residual disability like foot drop. Severe autonomic instability and sensitivity to skeletal muscle relaxants poses a challenge for anaesthetic management of these patients when they come for surgery. We, here report a case of GBS with residual foot drop coming for tendon transfer surgery.*

A 34 yrs, 48 kg, female with bilateral foot drop came for tendon transfer, one limb at a time. She gave history of upward moving paralysis of the body, finally requiring two months ventilatory support along with tracheostomy, about 6 years ago. Now she has recovered with residual bilateral foot drop leading to difficulty in walking. Old documents were not available as patient lost it in public transport bus. She did not have any other significant medical or surgical history. On examination her pulse, blood pressure, cardiovascular and respiratory examinations were within normal limits. Central nervous system examination revealed Steppage gait,

Examination	Right	Left
Motor- Hip	5/5	5/5
Knee	5/5	5/5
Ankle- dorsiflexion	0/5	0/5
Plantar flexion	5/5	5/5
EHL	0/5	0/5
FHL	0/5	0/5
Sensory	Normal	Normal
Reflexes- Knee	Absent	Absent
Ankle	Absent	Absent
Plantars	âââ	âââ
Tone	Hypotonia	Hypotonia
Distal pulsations	+	+
Straight Leg Raising Test	Free	Free
No bowel bladder involvement		

Pulmonary Function Tests- Early small airway obstruction
Haemoglobin- 10.7 gm%, TLC- 5400 /mm3 (N-56, L-34, E-6, M-4, B-0), Urine- NAD, BSL- 85 mg%, BUN- 13 S. Creatinine- 0.7. S. Electrolytes- 136/4.7/104, LFTs- WNL

NCV- Bilateral popliteal nerves unexcitable at ankle, Tibial nerve motor conduction – increased distal latency, attenuated amplitude, decreased conduction velocity, Bilateral sural and superficial peroneal nerve action potential absent,

Impression- Mixed sensory motor poly-radiculoneuropathy of Lower Limb

EMG- Active denervation with re-innervation in bilateral abductor hallucis and active total denervation in extensor digitorum brevis muscles. Rest of the muscles- chronic partial denervation

Impression- Diffuse axonopathy
Choice of Anaesthesia-
Since General Anaesthesia is highly unpredictable due to susceptibility to neuro mascular blocking agents patient could go again on post op ventilation, it was discussed with the patient and decided to avoid it. After sub arachnoid block worsening of GBS have been reported in some cases. Also autonomic deregulation can be massive leading to severe intractable hypotension. The use of neuraxial anesthesia is questionable because it might influence the course of the disease. Only a few cases have been reported in which neuraxial block was successfully used in pregnant women with GBS (1–3). The risks of neurologic status worsening after regional anesthesia and alternative analgesia options was discussed with the patient. So, plan of giving Right side Femoral and Sciatic nerve blocks was made.

After securing IV line and attaching monitor (NIBP, SpO2, ECG), patient was sedated with 1.0 mg Midazolam i.v. Initialy, , patient was in supine position and right side femoral nerve block was given for tourniquet application with nerve stimulating needle with PNS after eliciting rectus femoris contractions at 0.4 mA and 20 ml of drug (0.5 % bupivacaine 8 ml, 2% Xylocaine+Adrenaline 6 ml, 50 ml Tramadol, 5 ml 0.9% NS) was given.

Then, left lateral position was given for posterior approach sciatic nerve block. After identifying the landmarks and marking the points, 9 cm long nerve stimulating needle was used along with PNS to elicit the plantar extensor response upto 0.5 mA and 25 ml of drug (0.5 % bupivacaine 8 ml, 2% Xylocaine+Adrenaline 6 ml, 50 ml Tramadol, 10 ml 0.9% NS) was given.

Surgery started under tourniquet after adequate block level was confirmed. Intra-operatively her vitals were stable and was given Inj. Fentanyl 50 microgram for anxiety. The surgery went off uneventful and patient was shifted to recovery room.

DISCUSSION
Guillain-Barré syndrome (GBS) is an acute autoimmune demyelinating polyneuropathy first described in 1859. The overall incidence of GBS worldwide is 1.1–1.8 cases per 100,000 per year, Males> Females, peaks in young adults and the elderly. There is an association with precedent infections in 70% of cases that are predominantly respiratory and gastrointesti-

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nal in origin. Progressive ascending motor weakness starting in the lower limbs ascending upto respiratory muscles and cause respiratory failure. Sensory symptoms may include pain, numbness and paraesthesia. Pain commonly affects the lower back and may be severe. On clinical examination a flaccid areflexic paralysis is found. Muscle wasting usually occurs within two weeks of the onset of symptoms and can be severe. Autonomic dysfunction is common and may cause arrhythmias, swings in blood pressure, urinary retention, paralytic ileus and hyperhydriasis. If severe this may be associated with sudden death. [4]

GBS subtypes
Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)- most common. [4]

Acute motor axonal neuropathy (AMAN)- more common in Japan and China, has an association with precedent infection with *Campylobacter jejuni*. [4]

Acute motor and sensory axonal neuropathy (AMSAN)- It is more severe. [4]

Miller Fisher syndrome (MFS) presents with ataxia, areflexia and ophthalmoplegia. Antiganglioside antibodies to GQ1b are found in 90% of patients

Chronic inflammatory demyelinating polyradiculoneuropathy- Slowly progressive or relapsing course. [4]

Urea and electrolytes are usually normal but may have evidence of the syndrome of inappropriate ADH secretion (SIADH) or renal dysfunction. ALT and gamma GT may be raised in 33% of patients. Creatine kinase may be raised. ESR is usually raised and CRP is sometimes elevated. Anti-GM1 is positive in 25% of patients and is associated with a worse outcome. Anti-GD1a is associated with AMAN subtype of GBS. Anti-GQ1b is associated with Miller-Fisher syndrome. Serology for *Campylobacter jejuni*, Cytomegalovirus, Epstein-Barr virus, Herpes simplex virus, *Mycoplasma pneumonia and* HIV antibodies should be considered. Stool cultures looking for evidence of gastrointestinal infections particularly *Campylobacter jejuni*.

Lumbar puncture- Cell count and glucose are usually normal with a raised protein, although the latter may also be normal in first two weeks. Nerve conduction studies- Findings depend on subtype of GBS. The majority show demyelinating pattern while some patients may show evidence of axonal loss with little or no demyelination.

Respiratory function tests- These may show reduced vital capacity, maximal inspiratory and expiratory pressures. Arterial blood gases may indicate progressive respiratory failure.

MANAGEMENT
Supportive-
Airway and respiratory
Around 30% of patients with GBS require ventilatory support. Clinical markers suggestive of the need for ventilatory support include bulbar weakness, inability to lift the head, upper limb weakness and tachypnoea. [2,4] Tracheostomy should be considered if prolonged respiratory support is likely to be needed. Respiratory physiotherapy can be invaluable.

Anaesthetic considerations
Cases of GBS after epidural and general anaesthesia has been reported. [12, 13]

Suxamethonium is absolutely contraindicated in patients with GBS. There have been a number of case reports of severe hyperkalaemia, life threatening arrhythmias, and cardiac arrest after its administration. [6]. Also, they could be hypo or hypersensitive to vecuonium. [11]

Cardiovascular
Autonomic dysfunction occurs in around 70% of patients and may be life-threatening. Monitoring of the ECG, blood pressure and fluid balance is advisable. The most common arrhythmia seen is sinus tachycardia but various other ECG changes have been observed including atrial and ventricular tachyarrhythmias, prolonged QT interval, atrioventricular blocks and even asystole. [4] Blood pressure may fluctuate between severe hypertension and hypotension. Orthostatic hypotension is common. Care should be taken when treating extremes of blood pressure with vasoactive drugs as patients may be particularly sensitive to their effects. Intubated patients with autonomic dysfunction may develop instability after tracheal suction. [4]

Gastrointestinal
Good nutrition for patients with bulbar weakness, and those who are sedated and mechanically ventilated. Autonomic dysfunction may lead to paralytic ileus. This may be treated with prokinetic agents such as metoclopramide or erythromycin.

Neurological
Neuropathic pain is common and occurs in around 50% of patients. Non-opioid analgesics (paracetamol, NSAIDs) in combination with opioid analgesia should be instituted initially. Adjunctive treatments such as anticonvulsants (e.g. gabapentin or carbemazepine), and tricyclic antidepressants may be effective.

Venous thromboembolism prophylaxis
Immobile patients are at very high risk of deep vein thrombosis and pulmonary emboli. Low molecular weight heparin in combination with either pneumatic compression devices or anti-embolism stockings, are recommended until patients are able to walk unaided.

Psychological
High incidence of depression among patients with GBS. If available, it is important for the patient and their family to have access to support groups. [4]

Rehabilitation
40% of patients who suffer from GBS will need admission for inpatient rehabilitation like limb positioning and posture as limb weakness can lead to compression nerve palsies, pressure sores and contractures. [4]

Immunomodulatory
Intravenous immunoglobulin
Intravenous immunoglobulin (IVIg) is an effective treatment for GBS and has been demonstrated to be comparable to plasma exchange in accelerating recovery. It is most effective if administered within two weeks of the onset of symptoms. It is more widely available, less labour intensive and has less side effects. IVIg contains pooled donor IgG antibodies, blocks Fc receptors and thus interrupts antibody mediated cell destruction. Side effects of IVIg- Nausea, headache, erythroderma, fluid overload, deranged liver function tests, venous thromboembolism, acute renal failure and anaphylaxis. [3,10]

Plasma exchange
More beneficial when commenced within one week of the onset of symptoms, but can be beneficial up to thirty days after the onset of illness. [9]Contraindications- Coagulopathy, overwhelming sepsis, haemodynamic instability and shock. [4,8,9]

Corticosteroids
No evidence that they improve recovery or affect long-term prognosis. [7]

PROGNOSIS
Most patients with GBS recover fully but this may take many

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months of intensive therapy. 15% of patients suffer persistent disability. 10% are unable to walk unaided at one year. There may be a recurrence in 2–5% of cases. The mortality from GBS ranges from 2–12%. [4,5]

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