Possible SAQ questions for patient with Weakness

What other conditions can mimic GBS in the critically ill patient?

Intracranial/Spinal Cord abnormalities – Meningitis, Brainstem encephalitis, Transverse Myelitis, Cord compression

Anterior horn cell – Polio, Westnile Virus

Spinal Nerve root – Compression, Inflammation, Malignancy

Peripheral Nerve – CIDP (chronic inflammatory demyelinating polyneuropathy), Drug-induced neuropathy, Porrphyria, Critical illness neuropathy, Vasculitis, Drug intoxication, heavy metal poisoning, Beri-Beri (Vit B1 deficiency), Metabolic (hypoK, HypoMag, HypoPhos, Hypoglycaemia)

NM Junction – Myasthenia Gravis, Organophosphate poisoning

Acute Rhabdomyolysis, Dermatomyositis

What are the distinguishing features on clinical examination between a neuropathy and a myopathy?

	Neuropathy	**Myopathy**
Site of weakness	Distal weakness	Usually proximal
Sensory	May have concomitant sensory symptoms and signs	Usually pure motor
Reflexes	Reflexes lost early	Reflexes preserved till late
Fasciculations	Fasciculations may be present	Not typical
Contractures	Contractures not a feature	Contractures present
Myocardial dysfunction	Not a typical feature	May have accompanying cardiac dysfunction with the dystrophies
Atrophy	Present	Absent until late
CK level	Normal	Elevated
Nerve conduction	Slowed	Normal
EMG	Fibrillations and fasciculations	Small motor units

MRI	Subtle, near normal appearance	Enhancement of affected muscle
Muscle biopsy	Normal looking muscle	Irregular necrotic fibers

List 6 organisms causing sensitising infections leading to GBS?

- Campylobacter jejuni
- Mycoplasma
- CMV
- Influenza A
- Parainfluenza,
- Varicella-zoster
- Epstein–Barr virus
- HIV

List and justify your key tests for this patient?

- VBG
- FBE UEC CMP Glucose CK
- ECG
- CXR
- PEFR (peak flow < 250L/min)
- Consider LP (non-urgent) - raised protein level with normal WCC (only seen after 5-7 days)
- viral PCR/ antibodies
- stool culture for *Campylobacter*
- mycoplasma antibodies

What are the general / broad management decisions in a patient with GBS?

- General – pain relief, eye care, DVT prophylaxis,
- Monitor – check respiration every 1-2 hrs, monitor haemodynamics, swallowing, pupils/ileus
- Early ICU involvement with intubation and haemodynamic support
- IV Ig

What are the indications for ICU admission?

- Requiring intubation for airway protection or respiratory failure
- Labile HR or BP – needing haemodynamic support
- Rapidly progressive disease
- Associated severe co-existing illness or comorbidities

Why do we intubate such patients?

- They are too weak to support a satisfactory minute volume
- They are too weak to cough
- Their airway protection is lost when bulbar cranial nerves become involved

What are the respiratory markers to indicate need for intubation?

Spirometry - FVC

- An FVC less than 20ml/kg (i.e. 1400ml for a normal 70kg male) is quoted by LITFL as a trigger for ICU admission,
- Interestingly, these people may actually have a higher FVC while supine rather than sitting up, much like the patients with a high spinal injury.

MIPs - Maximum Inspiratory Pressures

- If the MIP is less than 30cmH2O, they need a tube.
- This demonstrates respiratory muscle weakness

MEPs - Maximum Expiratory Pressures

- If the MEP is less than 40cmH2O, they need a tube.
- As well as demonstrating respiratory and abdominal muscle weakness, this finding also suggests that the patient will be unable to generate enough pressure to cough effectively.

Peak Expiratory Flow

- A PEF rate less than 250 L/min seems to be associated with a need for mechanical ventilation
- Like the MEP, it is an indirect suggestion that the ability to cough has become impaired.

Guillian-Barre versus Acute Transverse Myelitis

Guillian-Barré	Acute Transverse Myelitis
Pathophysiology	
Acute inflammatory demyelinating peripheral neuropathy, associated with infection such as: • Antecedent viral illness; usually with diarrhoea • EBV • HSV • *Campylobacter jejuni* • HIV	Autoimmune inflammation of the spinal cord; may be idiopathic or associated with other illnesses: • Usually occurs as a postinfectious complication • Can fall within the spectrum of coexisting MS • Can coexist with acute disseminated encephalomyelitis • Autoimmune diseases are associated (eg. SLE, scleroderma, etc)
Typical features of history	
• Sub-acute onset • Ascending pattern of clinical signs	• Often, very rapidly progressing • Weakness nadir is achieved within 4 hours in some cases (though some take as long as 21 days)
Power	
• Bilaterally decreased • Symmetrical • Weakness ascends over time	• Bilaterally decreased • Symmetrical • Weakness remains at and below the level of the lesion • "Pyramidal" preference: flexors of the legs and the extensors of the arms
Tone	
• Flaccid • Later, remains flaccid	• Initially flaccid • Later, hypertonic spasticity
Reflexes	
• Diminished or absent • Later, remain diminished	• Depressed initially • Hyperreflexia subsequently
Cranial nerves	
• Usually, not involved	• Usually, not involved

• Miller Fischer variant involves (usually, medullary) cranial nerves	• When it forms a part of the MS spectrum, there may be optic neuritis
Autonomic features	
• Usually present	• Not usually involved, unless the level of the lesion is high • High lesions may present with spinal shock
Sensory findings	
• Sensation usually preserved or oly mildly affected	• Sensation is usually absent • There is usually a distinct symmetrical sensory level
CSF features	
• Raised protein • Usually no white cells • Antibodies (Anti-GM1) or GQ1b antibodies in the Miller Fischer variant	• Raised protein • Lymphocytosis
Nerve conduction studies	
• Marked slowing, conduction block	• Reduced amplitude sensory nerve action potential (SNAP) • Pathological F-wave responses • Decreased conduction velocity of motor and sensory nerves.
Electromyography	
• Abnormal spontaneous activity, reduced recruitment, normal MUPs (early in disease). Later, reduced	• Reduced amplitude of motor (MUP) action potentials
MRI	
• Noncontrast MRI is essentially normal • Gadolinium reveals surface thickening and contrast enhancement on the conus medullaris and the nerve roots of the cauda equina	• Noncontrast MRI reveals cord oedema at the level of the lesion (but in 40%, looks totally normal) • Gadolinium-enhancing signal abnormality extending over one or more cord segments.

	• Lesions ccupy most of the transverse diameter of the cord (2/3rds)

References

Uptodate: May 2019, Guillain-Barré syndrome in adults: Treatment and prognosis

Oh's Intensive Care Manual

Derangedphysiology.com notes on GBS vs Transverse Myelitis

LITFL.com CCC compendium