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NEUROLOGY MCQs

for
Postgraduate and Superspecialty Medical Entrance Examinations

Based on 20th Edition of Harrison's Principles of Internal Medicine



Prof. Ajay Mathur

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Based on 20th Edition of Harrison's Prinicples of Internal Medicine

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The Health Sciences Publisher
New Delhi | London | Panama

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Guillain-Barré Syndrome and Other Immune-Mediated Neuropathies

2677. Guillain-Barré syndrome was first described in which year?
N Engl J Med. 2012;366:2294-304

A. 1869
B. 1898
C. 1916
D. 1934

Guillain–Barré syndrome, characterized by acute areflexic paralysis with albuminocytologic dissociation (elevated protein in CSF and normal cell counts), was described in 1916.

2678. Miller Fisher syndrome was first reported in which year?
N Engl J Med. 2012;366:2294-304

A. 1923
B. 1943
C. 1956
D. 1966

Miller Fisher syndrome, characterized by ophthalmoplegia, ataxia & areflexia, was reported in 1956 as a likely variant of Guillain–Barré syndrome, because the CSF of affected patients showed albuminocytologic dissociation.

2679. Feature not typical of Guillain-Barre syndrome is?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Acute onset
B. Males & females are at equal risk
C. Autoimmune
D. Polyradiculoneuropathy

Males are at 1.5-fold higher risk for GBS than females. Adults are more frequently affected than children.

2680. Facial diparesis is present in what proportion of GBS patients?
Harrison's 20th Ed. Chapter 439, Page 3225

A. 10%
B. 20%
C. 50%
D. 80%

Facial diparesis is present in 50% of GBS patients.

2681. Bilateral facial nerve palsy occurs in which of the following?
Harrison's 20th Ed. Chapter 439, Page 3214

A. Leprosy
B. Sarcoidosis
C. Sjogren's syndrome
D. All of the above

Facial palsy that is often bilateral occurs in sarcoidosis and in Guillain-Barre syndrome.

2682. GBS manifests as?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Rapidly evolving areflexic ascending motor paralysis
B. With or without sensory disturbance
C. Autonomic involvement is common
D. All of the above

2683. Which of the following about GBS is false?
Lancet. 2016;388:717-27

A. Annual incidence rate of GBS increases with age
B. Estimated lifetime risk of developing GBS <1 in 1000
C. Post-infectious, rapidly progressive, monophasic disorder
D. None of the above

2684. Which of the following is not typical of Guillain-Barre syndrome?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Fever & constitutional symptoms are absent at onset
B. Lower cranial nerves are frequently involved
C. DTRs disappear within first few days of onset
D. Bladder dysfunction is a prominent feature

Presence of fever & constitutional symptoms at onset cast doubt on the diagnosis of GBS. Transient bladder dysfunction may occur in severe GBS. If bladder dysfunction is a prominent & early in the course, GBS is unlikely.

2685. In GBS, clinical worsening reaches a plateau in how many weeks of onset?
Harrison's 20th Ed. Chapter 439, Page 3225

A. 1 week
B. 4 weeks
C. 8 weeks
D. 12 weeks

Clinical worsening stops and GBS patient reaches a plateau almost always within 4 weeks of onset.

2686. Limited or regional GBS syndromes include?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Acute motor axonal neuropathy (AMAN)
B. Acute motor sensory axonal neuropathy (AMSAN)
C. Miller Fisher syndrome (MFS)
D. All of the above

Limited or regional GBS syndromes are Miller Fisher syndrome, pure sensory forms, ophthalmoplegia with anti-GQ1b antibodies (severe motor-sensory GBS), GBS with severe bulbar & facial paralysis associated with cytomegalovirus (CMV) infection & anti-GM2 antibodies & acute pandysautonomia.

2687. Which of the following is the most common form of GBS?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Acute inflammatory demyelinating polyradiculoneuropathy
B. Acute axonal motor disorder
C. Acute sensory & motor axonal neuropathy
D. Miller Fisher syndrome (MFS)

In Europe & North America ~95% of GBS are acute inflammatory demyelinating polyradiculoneuropathy. ~5% are AMAN & AMSAN.

2688. Nationality of Charles Miller Fisher was?

A. Canadian
B. American
C. British
D. Brazilian

Charles Miller Fisher was a Canadian stroke specialist.

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2689. Which of the following is a demyelinating disorder?
Harrison's 20th Ed. Chapter 439, Page 3226 Table 439-1

A. Acute motor axonal neuropathy (AMAN)
B. Acute motor sensory axonal neuropathy (AMSAN)
C. Miller Fisher syndrome (MFS)
D. All of the above

Acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN) subtypes are axonal variants of Guillain-Barré syndrome (GBS).

2690. Which of the following is a regional variant of GBS?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Pure sensory form
B. GBS with severe bulbar & facial paralysis
C. Acute pandysautonomia
D. All of the above

Regional variants of GBS include pure sensory forms, ophthalmoplegia with anti-GQ1b antibodies as part of severe motor-sensory GBS, GBS with severe bulbar & facial paralysis, sometimes associated with antecedent cytomegalovirus (CMV) infection and anti-GM2 antibodies and acute pandysautonomia.

2691. Organisms involved in antecedent infections in Guillain-Barre syndrome include?
Harrison's 20th Ed. Chapter 439, Page 3225, N Engl J Med. 2012;366:2294-304

A. Campylobacter jejuni
B. Human herpes virus
C. Cytomegalovirus
D. All of the above

~70% of GBS occur 1–3 weeks after an acute infectious process (respiratory or gastrointestinal). Antecedent infection or reinfection with Campylobacter jejuni, human herpes virus infection, CMV, Epstein-Barr virus, HIV, hepatitis E, Zika, chikungunya, Mycoplasma pneumoniae & swine influenza vaccine have been identified. Case clusters are rare. Presence of sialylated lipopolysaccharides on C. jejuni strains is a form of molecular mimicry that promotes autoimmune recognition of sialylated cell surface molecules on axons. Asymptomatic Campylobacter infection also may trigger Guillain-Barre syndrome.

2692. Which of the following is false if preceding infection in GBS case is C. jejuni?
Lancet. 2016;388:717–27

A. A pure motor axonal form of GBS occurs
B. Seropositivity against GM1 & GD1a gangliosides
C. Poorer outcome
D. None of the above

2693. Which of the following vaccinations could predispose to Guillain-Barre syndrome?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Swine influenza vaccine
B. Influenza A vaccine
C. Older type rabies vaccine
D. All of the above

Immunizations with swine influenza vaccine, influenza A vaccines and older-type (Semple) rabies vaccine are implicated as a trigger of GBS. There is no increased risk of GBS with meningococcal vaccinations.

2694. Guillain-Barre syndrome occurs more frequently in patients with following diseases except?
Harrison's 20th Ed. Chapter 439, Page 3225

A. Hodgkin's disease
B. Chronic myeloid leukemia
C. HIV-seropositive individuals
D. Systemic lupus erythematosus

GBS also occurs more frequently in patients with lymphoma (Hodgkin's disease), HIV-seropositive individuals and in patients with SLE.

2695. Which of the following statements is false about GBS?
Harrison's 20th Ed. Chapter 439, Page 3226

A. Both cellular & humoral immune mechanisms involved
B. Analogous to experimental allergic neuritis (EAN)
C. Molecular mimicry mechanism
D. None of the above

2696. Which of the following is the critical event in the development of GBS?
N Engl J Med. 2012;366:2294-304

A. B cells recognize glycoconjugates on C. jejuni
B. Activation of B cells T cell
C. Escape of activated B cells from Peyer's patches into regional lymph nodes
D. All of the above

In the immunopathogenesis of GBS, a critical event in the development of GBS is the escape of activated B cells from Peyer's patches into regional lymph nodes.

2697. Which of the following is a functional region of myelinated axons?
Harrison's 20th Ed. Chapter 439, Page 3226 Figure 439-1

A. Internodes
B. Paranode
C. Juxtaparanodes
D. All of the above

Myelinated axons are divided into four functional regions. They are the nodes of Ranvier, paranodes, juxtaparanodes & internodes.

2698. Which of the following antiganglioside antibody is common in GBS?
Harrison's 20th Ed. Chapter 439, Page 3228 Table 439-2

A. Anti GM1
B. Anti GM2
C. Anti GM3
D. Anti GM4

Anti GM1, an antiganglioside antibody is common in GBS (20–50%), particularly in those preceded by C. jejuni infection.

2699. Which of the following is most relevant to Gangliosides?
N Engl J Med. 2012;366:2294-304

A. Lectin
B. Heparan
C. Ceramide
D. Mannose

Gangliosides are composed of a ceramide attached to one or more sugars (hexoses) and contain sialic acid (N-acetylneuraminic acid) linked to an oligosaccharide core. Gangliosides are important components of the peripheral nerves.

2700. Which of the following is a Ganglioside?
N Engl J Med. 2012;366:2294-304

A. GM1
B. GD1a
C. GT1a
D. All of the above

GM1, GD1a, GT1a, and GQ1b are gangliosides.

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2701. Which of the following gangliosides is strongly expressed at the nodes of Ranvier?

Harrison's 20th Ed. Chapter 439, Page 3228 Table 439-2, N Engl J Med. 2012;366: 2294-304

A. GM1
B. GT1a
C. GQ1b
D. All of the above

Gangliosides GM1 and GD1a are strongly expressed at the nodes of Ranvier, where voltage-gated sodium (Nav) channels are localized. Contactin associated protein (Caspr) & voltage-gated potassium (Kv) channels are present at paranodes and juxtaparanodes respectively. IgG anti-GM1 or anti-GD1a autoantibodies bind to the nodal axolemma, leading to membrane-attack complex (MAC) formation. This results in disappearance of Nav clusters and detachment of paranodal myelin, which can lead to nerve-conduction failure and muscle weakness.

2702. lgG autoantibodies to GQ1b cross-react with which of the following?

N Engl J Med. 2012;366:2294-304

A. GM1
B. GD1a
C. GT1a
D. All of the above

IgG autoantibodies to GQ1b, which cross-react with GT1a, are strongly associated with the Miller Fisher syndrome, its incomplete forms (acute ophthalmoparesis and acute ataxic neuropathy), and its CNS variant, Bickerstaff's brainstem encephalitis (acute ophthalmoplegia, ataxia, and impaired consciousness after an infectious episode).

2703. The anti-GQ1b antibody syndrome includes which of the following?

N Engl J Med. 2012;366:2294-304

A. Bickerstaff's brainstem encephalitis
B. Miller Fisher syndrome
C. Pharyngeal-cervical-brachial weakness
D. All of the above

The anti-GQ1b antibody syndrome includes the Miller Fisher syndrome, acute ophthalmoparesis, acute ataxic neuropathy, Bickerstaff's brainstem encephalitis, and pharyngeal–cervical–brachial weakness.

2704. GQ1b is strongly expressed in which of the following cranial nerves?

N Engl J Med. 2012;366:2294-304

A. Oculomotor nerve
B. Trochlear nerve
C. Abducens nerve
D. All of the above

GQ1b is strongly expressed in the oculomotor, trochlear, and abducens nerves, as well as muscle spindles in the limbs. Glossopharyngeal and vagus nerves strongly express GT1a and GQ1b, possibly accounting for dysphagia.

2705. Which of the following is false about Miller Fisher syndrome?

N Engl J Med. 2012;366:2294-304

A. Anti-GQ1b antibodies are found in > 90% patients
B. IgG titers are highest early in the course
C. Areflexia of limbs without weakness
D. None of the above

Anti-GQ1b IgG antibodies are found in >90% of patients with MFS, and titers of IgG are highest early in the course. Anti-GQ1b antibodies are not found in other forms of GBS unless there is extraocular motor nerve involvement.

2706. Which of the following is not found in Miller Fisher syndrome?

N Engl J Med. 2012;366:2294-304

A. Ataxia
B. Areflexia of limbs
C. Weakness of limbs
D. Ophthalmoplegia

Miller Fisher's syndrome is an ocular variant of Guillain-Barre syndrome. It produces ophthalmoplegia with areflexia and mild ataxia, reflexes are normal. Antiganglioside antibodies (GQIb) can be detected in about 50% of cases.

2707. Which of the following is false about Miller Fisher syndrome?

N Engl J Med. 2012;366:2294-304

A. Rapidly evolving ataxia
B. Areflexia of limbs without weakness
C. Ophthalmoplegia often with pupillary paralysis
D. None of the above

2708. MFS is strongly associated with which of the following antibodies?

N Engl J Med. 2012;366:2294-304

A. GQ1a
B. GQ1b
C. GQ1c
D. GQ1d

Miller Fisher syndrome (MFS) presents as rapidly evolving ataxia & areflexia of limbs without weakness, and ophthalmoplegia, often with pupillary paralysis. MFS is strongly associated with antibodies to ganglioside GQ1b.

2709. Which of the following is a central nervous system variant of Miller Fisher syndrome?

N Engl J Med. 2012;366:2294-304

A. Anti-Hu–associated paraneoplastic encephalitis (PEM)
B. Bickerstaff's brainstem encephalitis
C. Post–infectious brainstem encephalitis
D. All of the above

Antibodies against ganglioside GQ1b have also been detected in patients with Bickerstaff's brainstem encephalitis. Bickerstaff's encephalitis is characterized by ophthalmoplegia and ataxia but is also accompanied by pyramidal and sensory tract findings and cerebrospinal fluid pleocytosis.

2710. **In demyelinating GBS, basis for flaccid paralysis & sensory disturbance is?**

Harrison's 20th Ed. Chapter 439, Page 3227

A. Conduction block
B. Neurodegeneration
C. Apoptosis
D. All of the above

In demyelinating forms of GBS, basis for flaccid paralysis & sensory disturbance is conduction block. Secondary axonal degeneration usually occurs in severe cases.

2711. **CSF findings in Guillain-Barre syndrome include all except?**

Harrison's 20th Ed. Chapter 439, Page 3227

A. Elevated CSF proteins
B. CSF protein elevated by end of first week
C. No pleocytosis
D. CSF is normal when symptoms are present for < 6 hours

CSF is often normal when symptoms have been present for 48 hours. By the end of first week the level of protein is usually elevated without accompanying pleocytosis. Sustained CSF pleocytosis suggests an alternative diagnosis (viral myelitis, unrecognized HIV infection, leukemia or lymphoma with infiltration of nerves or neurosarcoidosis.

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2712. Cytoalbuminologic dissociation best relates to?

Lancet. 2016;388:717–27

A. Normal CSF protein level & CSF TLC > 5 cells/µL
B. Raised CSF protein level & CSF TLC < 5 cells/µL
C. Normal CSF protein level & CSF TLC > 50 cells/µL
D. Raised CSF protein level & CSF TLC < 50 cells/µL

Cytoalbuminologic dissociation implies elevation of CSF protein level above laboratory normal value and CSF total white cell count <50 cells/µL).

2713. Electrodiagnostic feature of GBS is?

Harrison's 20th Ed. Chapter 439, Page 3227

A. Prolonged F-wave latencies
B. Prolonged distal latencies
C. Reduced amplitudes of compound muscle action potentials
D. All of the above

Edx features are mild or absent in the early stages of GBS and lag behind the clinical evolution. In AIDP, the earliest features are prolonged F-wave latencies, prolonged distal latencies and reduced amplitudes of compound muscle action potentials (CMAPs).

2714. Which of the following criteria is used for the diagnosis of Guillain-Barre syndrome?

Harrison's 20th Ed. Chapter 439, Page 3227

A. Amsel criteria
B. CASPAR criteria
C. Research Domain Criteria (RDoCs)
D. Brighton Criteria

Brighton criteria. Brain 137:33,2014.

2715. In CSF of GBS patient, increase in which of the following **reflects axonal damage & predicts a residual deficit?**

Harrison's 20th Ed. Chapter 439, Page 3227

A. Tau
B. Amyloid precursor protein (APP)
C. Chymotrypsin
D. Apolipoprotein E

Tau & 14-3-3 protein levels are elevated early in some cases of GBS. Tau increases in CSF may reflect axonal damage & predict a residual deficit.

2716. Which of the following can cause ptosis?

Harrison's 20th Ed. Chapter 28, Page 190

A. Guillain-Barre syndrome
B. Miller Fisher syndrome
C. Lambert-Eaton syndrome
D. All of the above

2717. Which of the following can cause ptosis without ophthalmoparesis?

Harrison's 20th Ed. Chapter 441, Page 3240 Table 441-1

A. Myotonic dystrophy
B. Congenital myopathies
C. Neuromuscular junction disorders
D. All of the above

2718. Differential diagnosis of Guillain-Barré syndrome includes?

Harrison's 20th Ed. Chapter 439, Page 3228

A. Hypokalemia
B. Polymyositis
C. Porphyria
D. All of the above

Differential diagnosis of Guillain-Barré syndrome include transverse myelitis & neuromyelitis optica, Lyme polyradiculitis, porphyria, vasculitic neuropathy, poliomyelitis, West Nile virus, CMV polyradiculitis, critical illness neuropathy or myopathy, myasthenia gravis, botulism, poisonings with organophosphates, thallium, or arsenic, paralytic shellfish poisoning, severe hypophosphatemia.

2719. Which of the following must be considered in the differential diagnosis of Guillain Barre syndrome?

Harrison's 20th Ed. Chapter 148, Page 1107

A. Adult botulism
B. Tick paralysis
C. Eaton Lambert syndrome
D. All of the above

Adult botulism, myasthenia gravis, stroke syndromes, Eaton Lambert syndrome and tick paralysis must be considered in the differential diagnosis of cases include Guillain Barre syndrome (GBS).

2720. Type of respiratory failure in GBS is?

Harrison's 20th Ed. Chapter 293, Page 2026

A. I
B. II
C. III
D. IV

Type II respiratory failure is a consequence of alveolar hypoventilation and results from the inability to eliminate carbon dioxide effectively due to impaired CNS drive to breathe, impaired strength with failure of neuromuscular function in the respiratory system and increased load(s) on the respiratory system. Reduced strength can be due to impaired neuromuscular transmission as in myasthenia gravis, Guillain-Barre syndrome, amyotrophic lateral sclerosis or respiratory muscle weakness (e.g. myopathy, electrolyte derangements, fatigue).

2721. **What length of time after the first motor symptoms in GBS,** immunotherapy is no longer effective?

Harrison's 20th Ed. Chapter 439, Page 3229

A. 3 days
B. 14 days
C. 30 days
D. 60 days

Each day counts in the treatment of GBS. ~2 weeks after first motor symptoms, immunotherapy is no longer effective.

2722. What percentage of patients with GBS require ventilatory assistance?

Harrison's 20th Ed. Chapter 439, Page 3229

A. ~ 10%
B. ~ 20%
C. ~ 30%
D. ~ 40%

~30% of patients with GBS require ventilatory assistance, sometimes for prolonged periods of time.

2723. Which of the following about treatment of GBS is false?

Harrison's 20th Ed. Chapter 439, Page 3229

A. High-dose IVIg & plasmapheresis are equally effective
B. Combination of IVIg & plasmapheresis offers no advantage
C. After successful treatment of GBS, relapse may occur in II or III week
D. None of the above

IVIg is administered as five daily infusions for a total dose of 2 g/kg body weight. A course of plasmapheresis usually consists of 40–50 mL/kg plasma exchange (PE) four to five times over a week. Glucocorticoids have not been found to be effective in GBS.

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2724. Guillain-Barre syndrome best relates to which of the following?
Harrison's 20th Ed. Chapter 344, Page 2497

A. Autoimmune lymphoproliferative syndrome (ALPS)
B. Hemophagocytic lymphohistiocytosis (HLH)
C. Hyper-IgM (HIGM) Syndromes
D. Wiskott-Aldrich Syndrome

Autoimmune lymphoproliferative syndrome (ALPS) is characterized by nonmalignant T and B lymphoproliferation causing splenomegaly and enlarged lymph nodes. 70% of patients also display autoimmune manifestations like autoimmune cytopenias, Guillain-Barre syndrome, uveitis & hepatitis.

2725. Which of the following statements about Gullain-Barre syndrome (GBS) is false?
Harrison's 20th Ed. Chapter 439, Page 3229

A. Escape of activated B cells from Peyers patches into regional lymph nodes
B. Anti GQ1b IgG antibodies are found in >90% of patients with Miller Fisher subtype of GBS
C. Glucocorticoids are effective in GBS
D. Severe proximal motor & sensory axonal damage is a poor prognostic sign

Glucocorticoids have not been found to be effective in GBS. CIDP responds to glucocorticoids, whereas GBS does not.

2726. Factor that worsen the outlook for recovery in GBS is?
Harrison's 20th Ed. Chapter 439, Page 3230

A. Severe proximal motor & sensory axonal damage
B. Fulminant or severe attack
C. Delay in the onset of treatment
D. All of the above

2727. Patient characteristic consistently related to poor prognostic outcome in GBS is?
Harrison's 20th Ed. Chapter 439, Page 3230, Lancet. 2016;388:717–27

A. Age > 40 years
B. Preceding diarrhea (or C. jejuni infection in past 4 weeks)
C. High disability at nadir
D. All of the above

Factors that worsen the outlook for recovery in AIDP are patients with severe proximal motor and sensory axonal damage, advanced age, a fulminant or severe attack and a delay in the onset of treatment.

2728. What percentage of patients with GBS achieve a full functional recovery?
Harrison's 20th Ed. Chapter 439, Page 3230

A. ~ 65%
B. ~ 75%
C. ~ 85%
D. ~ 95%

~85% of patients with GBS achieve a full functional recovery within several months to a year.

2729. In optimal settings, mortality rate in patients with GBS is?
Harrison's 20th Ed. Chapter 439, Page 3230

A. <1%
B. <3%
C. <5%
D. <7%

In optimal settings, mortality rate in patients with GBS is <5%. Death usually results from secondary pulmonary complications.

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Gold medal recipient at undergraduate and postgraduate levels, Dr Mathur has keen academic interests. This book is an outcome of his passion for medical science and research spanning over the last three decades.

In keeping with the shift in medical pedagogies and intense competition that professional face today, he embarked upon this academic exercise in the year 2007. His books help medical professionals keep abreast with the latest and greatest trends in medicine while also revering the glorious age-old traditions of clinical methods.

Adding yet another edition to his body of work, Dr Mathur gleans great satisfaction from enabling medical students to pursue excellence in their education and becoming responsible professionals of tomorrow. In his latest undertaking, he adjusts his approach to tackle medical education, one specialism at a time. This allows students to delve deeper into the subject of their choice

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