COMPREHENSIVE REVIEW TEST 5: NEUROLOGICAL, HEMATOLOGICAL, AND TOXICOLOGICAL EMERGENCIES

1. A 62-year-old man presents with sudden onset right-sided weakness and aphasia. Symptoms began 90 minutes ago. CT head shows no hemorrhage. What is the most appropriate next step?

A. MRI brain with diffusion-weighted imaging

B. IV alteplase administration

C. Antiplatelet therapy with aspirin 325 mg

D. Observation and repeat CT in 24 hours

2. What is the time window for IV alteplase administration in acute ischemic stroke?

A. Within 3 hours only

B. Within 4.5 hours of symptom onset

C. Within 6 hours

D. Within 12 hours

3. A patient receiving IV alteplase for acute stroke develops sudden headache and neurological deterioration. What is the immediate action?

A. Continue alteplase and administer antihypertensive

B. Stop alteplase and obtain emergent CT head

C. Administer additional alteplase bolus

D. Start antiplatelet therapy

4. A patient with acute ischemic stroke has BP 195/110 mmHg and is a candidate for thrombolysis. What is the BP target before alteplase?

A. <220/120 mmHg

B. <185/110 mmHg

C. <160/90 mmHg

D. <140/90 mmHg

5. A 58-year-old woman presents with the "worst headache of her life" with sudden onset. CT head is negative. What is the next step?

A. Discharge with migraine treatment

B. Lumbar puncture

C. MRI brain

D. Cerebral angiography

6. What CSF finding confirms subarachnoid hemorrhage when CT is negative?

A. Elevated protein

B. Xanthochromia

C. Low glucose

D. Pleocytosis

7. A patient with confirmed subarachnoid hemorrhage from aneurysm rupture develops confusion on day 5. Transcranial Doppler shows elevated velocities. What complication is this?

A. Rebleeding

B. Hydrocephalus

C. Cerebral vasospasm

D. Seizure

8. What medication is used to prevent cerebral vasospasm after subarachnoid hemorrhage?

A. Nimodipine

B. Amlodipine

C. Phenytoin

D. Mannitol

9. A patient presents with severe headache, BP 220/130 mmHg, and CT showing intracerebral hemorrhage with volume 45 mL. According to current guidelines, what is the BP target?

A. SBP <220 mmHg

B. SBP <180 mmHg

C. SBP <140 mmHg

D. SBP <120 mmHg

10. A patient with intracerebral hemorrhage is on warfarin with INR 3.8. What is the most appropriate reversal strategy?

A. Vitamin K alone

B. Fresh frozen plasma alone

C. 4-factor PCC plus vitamin K

D. Protamine sulfate

11. A patient presents with witnessed generalized tonic-clonic seizure activity for 8 minutes that is ongoing. What is the first-line treatment?

A. IV phenytoin

B. IV lorazepam

C. IV phenobarbital

D. IV levetiracetam

12. What defines refractory status epilepticus?

A. Seizure lasting >5 minutes

B. Seizure continuing despite first-line benzodiazepine

C. Seizure continuing despite second-line antiepileptic

D. Seizure requiring general anesthesia

13. A patient with status epilepticus fails lorazepam and fosphenytoin. What is the next step?

A. Repeat lorazepam

B. Intubation and continuous IV midazolam or propofol

C. Oral levetiracetam

D. Observation

14. A patient presents with fever, headache, nuchal rigidity, and photophobia. CSF shows WBC 1,800 with 92% neutrophils, protein 180 mg/dL, and glucose 28 mg/dL. What is the most likely diagnosis?

A. Viral meningitis

B. Bacterial meningitis

C. Tuberculous meningitis

D. Fungal meningitis

15. What is the empiric antibiotic regimen for community-acquired bacterial meningitis in adults?

A. Ceftriaxone alone

B. Vancomycin plus ceftriaxone plus dexamethasone

C. Ampicillin alone

D. Metronidazole plus gentamicin

16. When should dexamethasone be administered in bacterial meningitis?

A. After antibiotics are given

B. Before or with the first dose of antibiotics

C. Only if pneumococcal meningitis is confirmed

D. Dexamethasone is contraindicated in meningitis

17. A patient presents with fever, altered mental status, and temporal lobe abnormalities on MRI. CSF shows lymphocytic pleocytosis. What is the most likely diagnosis?

A. Bacterial meningitis

B. Herpes simplex encephalitis

C. Cryptococcal meningitis

D. Brain abscess

18. What is the treatment for herpes simplex encephalitis?

A. IV acyclovir

B. IV ganciclovir

C. IV amphotericin B

D. IV ceftriaxone

19. A patient with GCS 7 and signs of increased intracranial pressure is deteriorating. CT shows cerebral edema. What is the immediate intervention?

A. IV furosemide

B. IV mannitol or hypertonic saline

C. Hyperventilation to PaCO2 25 mmHg permanently

D. Lumbar puncture

20. What is the mechanism of action of mannitol in reducing intracranial pressure?

A. Decreases CSF production

B. Osmotic diuresis drawing water from brain tissue

C. Direct vasoconstriction

D. Reduces cerebral metabolism

21. A patient presents with ascending weakness starting in the legs, areflexia, and recent history of gastroenteritis. What is the most likely diagnosis?

A. Myasthenia gravis

B. Guillain-Barré syndrome

C. Multiple sclerosis

D. Transverse myelitis

22. What is the indication for intubation in Guillain-Barré syndrome?

A. FVC <20 mL/kg or NIF weaker than -30 cmH2O

B. Any respiratory symptoms

C. Inability to walk

D. Areflexia

23. What is the treatment for Guillain-Barré syndrome?

A. High-dose corticosteroids

B. IV immunoglobulin or plasmapheresis

C. Antibiotics

D. Supportive care only

24. A patient with myasthenia gravis develops respiratory distress and weakness. FVC is 800 mL (predicted 3,000 mL). What is the diagnosis?

A. Cholinergic crisis

B. Myasthenic crisis

C. Asthma exacerbation

D. Pneumonia

25. A patient in myasthenic crisis is being treated. Which medication should be avoided as it can worsen myasthenia?

A. IV immunoglobulin

B. Aminoglycosides

C. Prednisone

D. Pyridostigmine

26. A patient presents with sudden back pain followed by bilateral leg weakness and urinary retention. MRI shows spinal cord compression at T8. What is the diagnosis?

A. Transverse myelitis

B. Guillain-Barré syndrome

C. Spinal cord compression (oncologic emergency)

D. Herniated lumbar disc

27. What is the first-line treatment for malignant spinal cord compression?

A. Emergent surgical decompression for all patients

B. High-dose IV dexamethasone

C. Radiation therapy alone

D. Chemotherapy

28. A patient with known cancer presents with headache, nausea, and papilledema. MRI shows multiple brain lesions with surrounding edema. What medication reduces vasogenic edema?

A. Mannitol

B. Dexamethasone

C. Furosemide

D. Hypertonic saline

29. A patient presents with petechiae, thrombocytopenia (platelet 18,000), microangiopathic hemolytic anemia (schistocytes), fever, renal dysfunction, and neurological changes. What is the diagnosis?

A. DIC

B. ITP

C. TTP

D. HIT

30. What is the treatment for TTP?

A. Platelet transfusion

B. Plasma exchange (plasmapheresis)

C. Heparin anticoagulation

D. Splenectomy

31. Why are platelet transfusions generally avoided in TTP?

A. They are ineffective

B. They may worsen thrombosis and organ damage

C. They cause allergic reactions

D. They interfere with plasmapheresis

32. A patient has platelets 42,000, PT/INR elevated, PTT elevated, low fibrinogen, and elevated D-dimer. Peripheral smear shows schistocytes. What is the diagnosis?

A. TTP

B. DIC

C. ITP

D. Liver failure

33. What is the underlying treatment principle for DIC?

A. Aggressive platelet transfusion

B. Treat the underlying cause

C. Anticoagulation for all patients

D. Plasmapheresis

34. A patient on heparin for 7 days develops platelet count drop from 180,000 to 62,000 with new DVT. What is the most likely diagnosis?

A. Heparin-induced thrombocytopenia (HIT)

B. ITP

C. DIC

D. Dilutional thrombocytopenia

35. What is the 4T score used for?

A. Diagnosing TTP

B. Assessing pre-test probability of HIT

C. Predicting bleeding risk

D. Staging DIC

36. A patient with confirmed HIT requires ongoing anticoagulation. Which agent is appropriate?

A. Low-molecular-weight heparin

B. Warfarin immediately

C. Argatroban or bivalirudin

D. Continue unfractionated heparin at lower dose

37. A patient with sickle cell disease presents with severe pain in chest and back, fever, and new pulmonary infiltrate with hypoxemia. What is the diagnosis?

A. Pneumonia

B. Acute chest syndrome

C. Vaso-occlusive crisis

D. Pulmonary embolism

38. What is the treatment for acute chest syndrome?

A. Antibiotics, oxygen, and exchange transfusion if severe

B. Hydroxyurea only

C. Pain management only

D. Anticoagulation

39. A patient receiving blood transfusion develops fever, hypotension, and dark urine 30 minutes into the transfusion. What is the most likely reaction?

A. Febrile non-hemolytic reaction

B. Acute hemolytic transfusion reaction

C. Allergic reaction

D. TRALI

40. A patient develops acute respiratory distress, hypoxemia, and bilateral infiltrates during blood transfusion with no evidence of fluid overload. What is the diagnosis?

A. TACO (transfusion-associated circulatory overload)

B. TRALI (transfusion-related acute lung injury)

C. Acute hemolytic reaction

D. Anaphylaxis

41. A patient with neutropenic fever (ANC 320, temp 38.6°C) after chemotherapy presents. What is the empiric antibiotic?

A. Oral amoxicillin-clavulanate

B. IV vancomycin alone

C. Anti-pseudomonal beta-lactam (cefepime, piperacillin-tazobactam)

D. IV metronidazole

42. A patient presents with altered mental status, and family reports empty acetaminophen bottles. It has been approximately 4 hours since ingestion. What is the antidote?

A. Flumazenil

B. N-acetylcysteine

C. Naloxone

D. Fomepizole

43. A patient with acetaminophen overdose has AST 8,200 U/L and INR 4.8. Despite late presentation, what should be done?

A. NAC is ineffective after hepatotoxicity develops

B. Administer NAC regardless of timing

C. Supportive care only

D. Immediate liver transplant

44. A patient presents with pinpoint pupils, respiratory rate of 6, and decreased consciousness. Track marks are visible on arms. What is the antidote?

A. Flumazenil

B. Naloxone

C. Atropine

D. Physostigmine

45. After naloxone administration for opioid overdose, the patient awakens but the opioid involved is methadone. What is the concern?

A. Naloxone will not work for methadone

B. Methadone has longer half-life than naloxone; renarcotization risk

C. Methadone causes seizures

D. No concern; patient can be discharged

46. A patient presents with agitation, hyperthermia, tachycardia, dilated pupils, and dry skin after taking unknown pills. What toxidrome is this?

A. Cholinergic

B. Anticholinergic

C. Sympathomimetic

D. Opioid

47. How do you distinguish anticholinergic from sympathomimetic toxidrome?

A. Heart rate

B. Pupil size

C. Skin (dry in anticholinergic, diaphoretic in sympathomimetic)

D. Blood pressure

48. A patient presents with salivation, lacrimation, urination, defecation, GI distress, and emesis (SLUDGE) after pesticide exposure. What is the toxidrome?

A. Anticholinergic

B. Cholinergic

C. Sympathomimetic

D. Serotonin syndrome

49. What is the antidote for organophosphate poisoning?

A. Physostigmine

B. Atropine plus pralidoxime

C. Naloxone

D. Flumazenil

50. A patient on multiple psychiatric medications presents with hyperthermia, rigidity, altered mental status, and autonomic instability. CK is 12,000 U/L. What are the two main diagnostic considerations?

A. Sepsis and meningitis

B. Neuroleptic malignant syndrome and serotonin syndrome

C. Thyroid storm and myxedema coma

D. Heat stroke and malignant hyperthermia

51. A patient recently started on haloperidol presents with high fever, severe rigidity, altered mental status, and elevated CK. What is the diagnosis?

A. Serotonin syndrome

B. Neuroleptic malignant syndrome

C. Malignant hyperthermia

D. Heat stroke

52. What is the treatment for neuroleptic malignant syndrome?

A. Dantrolene and continue antipsychotic

B. Stop offending agent, supportive care, consider bromocriptine or dantrolene

C. Cyproheptadine

D. Cooling measures only

53. A patient on SSRI and tramadol presents with hyperthermia, agitation, tremor, hyperreflexia, and clonus. What is the diagnosis?

A. Neuroleptic malignant syndrome

B. Serotonin syndrome

C. Anticholinergic toxicity

D. Malignant hyperthermia

54. What is the key clinical feature distinguishing serotonin syndrome from NMS?

A. Fever

B. Hyperreflexia and clonus in serotonin syndrome vs. rigidity in NMS

C. Elevated CK

D. Altered mental status

55. A patient presents with metabolic acidosis, elevated anion gap, and osmolar gap after ingesting antifreeze. What toxic alcohol is responsible?

A. Methanol

B. Ethylene glycol

C. Isopropyl alcohol

D. Ethanol

56. What is the antidote for ethylene glycol and methanol poisoning?

A. N-acetylcysteine

B. Fomepizole or ethanol

C. Naloxone

D. Sodium bicarbonate alone

57. A patient presents with tinnitus, hyperpnea, fever, and altered mental status. ABG shows pH 7.48 and 7.32 on repeat (mixed respiratory alkalosis and metabolic acidosis). What ingestion should be suspected?

A. Acetaminophen

B. Opioids

C. Salicylates

D. Benzodiazepines

58. A patient with TCA overdose presents with altered mental status, seizures, and wide QRS complex (140 ms). What is the treatment for cardiac toxicity?

A. Amiodarone

B. Sodium bicarbonate

C. Magnesium sulfate

D. Lidocaine

59. A chronic alcoholic presents with tremor, tachycardia, hypertension, and visual hallucinations 48 hours after last drink. What is the diagnosis?

A. Wernicke encephalopathy

B. Alcohol withdrawal with impending delirium tremens

C. Hepatic encephalopathy

D. Alcohol intoxication

60. What is the first-line treatment for alcohol withdrawal?

A. Haloperidol

B. Benzodiazepines

C. Phenobarbital as first-line

D. Propranolol

Answer Key with Explanations

1. B - This patient has acute ischemic stroke within the 4.5-hour window with no contraindications (CT negative for hemorrhage). IV alteplase should be administered promptly. Time is brain—every minute of delay results in approximately 1.9 million neurons lost. Do not delay for additional imaging if CT excludes hemorrhage.

2. B - IV alteplase is approved within 4.5 hours of symptom onset (or last known well). Originally approved for 3 hours, extended to 4.5 hours based on ECASS III trial. Additional exclusion criteria apply for 3-4.5 hour window (age >80, severe stroke NIHSS >25, oral anticoagulant use, history of both diabetes and prior stroke).

3. B - Sudden neurological deterioration during or after alteplase suggests intracranial hemorrhage. Immediately stop alteplase infusion, obtain emergent CT head, check coagulation studies, and prepare for reversal with cryoprecipitate (for fibrinogen), tranexamic acid, or aminocaproic acid. Hemorrhagic transformation occurs in 6-7% of patients.

4. B - Blood pressure must be <185/110 mmHg before alteplase and maintained <180/105 mmHg for 24 hours after. Labetalol or nicardipine are commonly used. Higher BP increases hemorrhagic transformation risk. If BP cannot be controlled, thrombolysis is contraindicated. Permissive hypertension (up to 220/120) is allowed only if NOT receiving thrombolytics.

5. B - "Worst headache of life" with sudden onset suggests subarachnoid hemorrhage (SAH) until proven otherwise. CT sensitivity decreases with time (nearly 100% at 6 hours, ~90% at 24 hours, ~50% at 1 week). Negative CT requires lumbar puncture to evaluate for xanthochromia or RBCs to rule out SAH.

6. B - Xanthochromia (yellow discoloration of CSF from bilirubin, a hemoglobin breakdown product) confirms SAH when CT is negative. It develops 6-12 hours after hemorrhage and persists for 2-4 weeks. Elevated RBC count alone may represent traumatic tap; xanthochromia distinguishes true SAH from traumatic LP.

7. C - Cerebral vasospasm is the leading cause of morbidity and mortality after aneurysmal SAH. It typically occurs days 3-14 (peak days 7-10) and presents with new neurological deficits or

decreased consciousness. Transcranial Doppler showing elevated velocities (>120 cm/s in MCA) suggests vasospasm. Treat with "triple-H" therapy and intra-arterial interventions.

8. A - Nimodipine (60 mg every 4 hours for 21 days) is the only medication proven to improve outcomes in SAH by reducing delayed ischemic neurological deficits. It's a calcium channel blocker with relative selectivity for cerebral vasculature. It does not prevent angiographic vasospasm but improves outcomes.

9. C - INTERACT2 and ATACH-2 trials evaluated intensive BP lowering in ICH. Current guidelines suggest SBP target <140 mmHg is safe and may improve functional outcomes, particularly when initiated early. Avoid rapid reduction >60 mmHg in first hour. Nicardipine or labetalol are preferred agents.

10. C - Warfarin-related ICH requires rapid reversal. 4-factor PCC (contains factors II, VII, IX, X) reverses INR within minutes and is preferred over FFP (faster, smaller volume, no typing needed). Give with vitamin K 10 mg IV (for sustained reversal as PCC effect wanes). Target INR <1.4 within 4 hours.

11. B - IV benzodiazepines (lorazepam 4 mg IV, or diazepam 10 mg IV, or midazolam 10 mg IM if no IV access) are first-line for status epilepticus. Lorazepam is preferred due to longer duration of anticonvulsant effect. Repeat once if seizures continue. Time is critical—prolonged seizures cause neuronal injury and become increasingly refractory.

12. C - Refractory status epilepticus is defined as seizures continuing despite adequate doses of first-line (benzodiazepine) AND second-line (fosphenytoin, valproate, or levetiracetam) antiepileptic drugs. This occurs in 30-40% of status epilepticus cases and requires anesthetic doses of midazolam, propofol, or pentobarbital with continuous EEG monitoring.

13. B - After failure of benzodiazepine and second-line agent, patient has refractory status epilepticus requiring anesthetic management. Intubate for airway protection, then start continuous IV midazolam, propofol, or pentobarbital titrated to EEG burst suppression. Continuous EEG monitoring is essential. ICU admission mandatory.

14. B - CSF profile strongly suggests bacterial meningitis: high WBC with neutrophil predominance (>80%), elevated protein (>100 mg/dL), and low glucose (<40 mg/dL or CSF/serum

ratio <0.4). Viral meningitis shows lymphocyte predominance with normal glucose. Gram stain and culture will identify organism.

15. B - Empiric therapy for community-acquired bacterial meningitis in adults: vancomycin (for resistant S. pneumoniae) plus ceftriaxone (for S. pneumoniae, N. meningitidis, H. influenzae) plus dexamethasone. Add ampicillin for Listeria coverage in age >50, immunocompromised, or alcoholics. Give antibiotics immediately—don't delay for LP if suspected.

16. B - Dexamethasone (0.15 mg/kg every 6 hours for 4 days) should be given before or with first antibiotic dose, as it reduces inflammation triggered by bacterial lysis. Proven benefit in pneumococcal meningitis, reducing mortality and neurological sequelae. Efficacy is reduced if given after antibiotics. Discontinue if non-pneumococcal.

17. B - Herpes simplex encephalitis (HSE) classically involves temporal lobes, presenting with fever, altered mental status, personality changes, and focal neurological findings. CSF shows lymphocytic pleocytosis, elevated protein, normal glucose, and often RBCs (hemorrhagic). MRI shows temporal lobe hyperintensities. HSV PCR confirms diagnosis.

18. A - IV acyclovir (10 mg/kg every 8 hours for 14-21 days) is the treatment for HSE. Start empirically if HSE is suspected—do not wait for PCR results. Delay in treatment significantly increases mortality and morbidity. HSE has 70% mortality if untreated but <20% with early acyclovir.

19. B - Acute elevated ICP with herniation risk requires immediate hyperosmolar therapy. Mannitol (0.5-1 g/kg IV) or hypertonic saline (23.4% 30 mL or 3% 250 mL bolus) rapidly reduces ICP. Elevate head of bed 30°, maintain euvolemia, avoid hyperthermia. Hyperventilation is temporary bridge only; lumbar puncture is contraindicated with mass effect.

20. B - Mannitol is an osmotic diuretic that creates an osmotic gradient between blood and brain, drawing water from brain parenchyma into the intravascular space. Effect begins within minutes, peaks at 20-60 minutes. Also reduces blood viscosity, improving cerebral blood flow. Monitor serum osmolality (keep <320 mOsm/kg) and renal function.

21. B - Guillain-Barré syndrome (GBS) is acute inflammatory demyelinating polyneuropathy presenting with ascending symmetric weakness, areflexia, and often preceding infection

(Campylobacter most common). Albuminocytologic dissociation (elevated CSF protein with normal cell count) is characteristic. Can progress rapidly to respiratory failure.

22. A - Monitor respiratory function closely in GBS. Indications for intubation include FVC <20 mL/kg (or <1 L), NIF weaker than -30 cmH2O, or >30% decline in FVC. The "20/30/40 rule" predicts need for ventilation: FVC <20, NIF <30, or >40% decline from baseline. Don't wait for hypoxia or hypercapnia.

23. B - IV immunoglobulin (0.4 g/kg/day for 5 days) or plasmapheresis are equally effective and are the treatments for GBS. Start early if patient is non-ambulatory or rapidly progressing. Corticosteroids are NOT effective in GBS (unlike CIDP). Supportive care includes VTE prophylaxis, pain management, and monitoring for autonomic instability.

24. B - Myasthenic crisis is life-threatening weakness with respiratory failure in myasthenia gravis. Precipitated by infection, surgery, medications, or tapering immunotherapy. FVC <1 L or <15-20 mL/kg indicates need for intubation. Distinguished from cholinergic crisis (from excessive anticholinesterase) by edrophonium test or clinical context.

25. B - Many medications can exacerbate myasthenia gravis by affecting neuromuscular junction. Aminoglycosides, fluoroquinolones, macrolides, beta-blockers, magnesium, and neuromuscular blockers should be avoided or used cautiously. Always review medications in MG patients. IVIG and steroids are appropriate treatments.

26. C - Acute spinal cord compression is an oncologic emergency. Back pain followed by weakness, sensory level, and bladder dysfunction (urinary retention is common) suggests cord compression. MRI is diagnostic. Most commonly from metastatic disease (breast, lung, prostate). Permanent paralysis occurs if not treated within 24-48 hours of weakness onset.

27. B - High-dose IV dexamethasone (10 mg bolus then 4-16 mg every 6 hours) is first-line treatment, reducing vasogenic edema around tumor and preserving neurological function. Start immediately upon clinical suspicion, before imaging. Definitive treatment (surgery vs. radiation) depends on tumor type, stability, prognosis, and extent of compression.

28. B - Dexamethasone reduces vasogenic edema around brain metastases by decreasing capillary permeability. Unlike mannitol (osmotic effect for cytotoxic edema and acute ICP crises), steroids

work on the tumor-induced vasogenic edema by stabilizing the blood-brain barrier. Typical dose: 10 mg IV then 4 mg every 6 hours.

29. C - TTP classically presents with pentad: thrombocytopenia, microangiopathic hemolytic anemia (MAHA with schistocytes), fever, renal dysfunction, and neurological changes. However, full pentad is present in <10% of cases. Thrombocytopenia plus MAHA without alternative explanation should prompt urgent consideration of TTP. ADAMTS13 activity <10% confirms diagnosis.

30. B - Plasma exchange (plasmapheresis) is life-saving treatment for TTP. It removes anti-ADAMTS13 antibodies and ultralarge vWF multimers while replacing ADAMTS13. Without treatment, mortality is >90%; with plasma exchange, survival is >80%. Start urgently upon clinical suspicion—don't wait for ADAMTS13 results. Rituximab and steroids are adjunctive.

31. B - Platelet transfusion in TTP may worsen thrombosis by providing additional "fuel" for microvascular thrombosis—described as "adding fuel to fire." Platelets should be avoided except for life-threatening bleeding or invasive procedures. Even with platelet count <10,000, transfusion is generally avoided if no active bleeding.

32. B - DIC shows thrombocytopenia, prolonged PT/PTT (consumption of factors), low fibrinogen (consumption), elevated D-dimer (fibrinolysis), and schistocytes (microangiopathic process). Unlike TTP (isolated ADAMTS13 deficiency), DIC involves widespread activation of coagulation cascade with both thrombosis and bleeding. Always has underlying cause.

33. B - DIC is always secondary to an underlying condition (sepsis, malignancy, trauma, obstetric complications). Treatment focuses on addressing the underlying cause. Supportive transfusion (platelets, FFP, cryoprecipitate for fibrinogen) for bleeding or procedures. Anticoagulation is controversial but may be considered in thrombosis-predominant DIC.

34. A - HIT typically presents 5-10 days after heparin initiation (or earlier if prior heparin exposure) with >50% platelet drop or platelet <150,000 AND thrombotic complication (often despite thrombocytopenia). The 4T score (Timing, Thrombocytopenia, Thrombosis, oTher causes) guides clinical probability. This patient has classic presentation with new thrombosis.

35. B - The 4T score assesses pre-test probability of HIT: Thrombocytopenia severity, Timing of platelet fall, Thrombosis or other sequelae, and oTher causes for thrombocytopenia. Score 0-3 =

low probability (HIT rare, may continue heparin); 4-5 = intermediate; 6-8 = high probability (stop heparin, start alternative anticoagulant, send confirmatory testing).

36. C - In confirmed HIT, stop ALL heparin (including flushes and heparin-coated catheters) and start non-heparin anticoagulant. Argatroban (direct thrombin inhibitor, hepatic metabolism) or bivalirudin (direct thrombin inhibitor) are appropriate. Fondaparinux is an option. Warfarin is contraindicated initially as it can cause venous limb gangrene; start only after platelet recovery.

37. B - Acute chest syndrome (ACS) is a leading cause of death in sickle cell disease. Defined by new pulmonary infiltrate plus one of: chest pain, fever, respiratory symptoms, hypoxemia. May be triggered by infection, fat embolism, or pulmonary infarction. Can rapidly progress to ARDS and multiorgan failure.

38. A - ACS treatment includes supplemental oxygen (maintain SpO2 >95%), empiric antibiotics (covering typical and atypical pathogens—ceftriaxone plus azithromycin/fluoroquinolone), pain management, incentive spirometry, and transfusion. Simple transfusion for mild cases; exchange transfusion for severe (PaO2 <60, extensive infiltrates, rapid progression).

39. B - Acute hemolytic transfusion reaction occurs when recipient antibodies attack donor RBCs (usually ABO incompatibility). Presents with fever, chills, hypotension, hemoglobinuria (dark urine), back/flank pain, and DIC. Stop transfusion immediately, maintain IV access with saline, support hemodynamics, send blood bank workup, and monitor for renal failure.

40. B - TRALI (transfusion-related acute lung injury) presents with acute hypoxemia and bilateral pulmonary infiltrates within 6 hours of transfusion without circulatory overload. Caused by donor antibodies activating recipient neutrophils in pulmonary vasculature. Treatment is supportive (oxygen, mechanical ventilation if needed). Most patients recover within 48-96 hours. Plasma-containing products (FFP, platelets) are highest risk.

41. C - Neutropenic fever (ANC <500 and temperature ≥38.3°C or ≥38.0°C sustained) is a medical emergency requiring immediate empiric antibiotics. Anti-pseudomonal beta-lactam monotherapy (cefepime, piperacillin-tazobactam, meropenem) is first-line. Add vancomycin only for specific indications (catheter infection, skin/soft tissue infection, mucositis, hemodynamic instability, known MRSA).

42. B - N-acetylcysteine (NAC) is the antidote for acetaminophen toxicity. At 4 hours post-ingestion, check acetaminophen level and plot on Rumack-Matthew nomogram. NAC is most effective within 8 hours but should be given if any evidence of toxicity regardless of timing. It replenishes glutathione and may have direct hepatoprotective effects.

43. B - NAC should be administered regardless of time since ingestion when hepatotoxicity has developed. Even with established liver failure, NAC improves outcomes by improving oxygen delivery, reducing cerebral edema, and supporting mitochondrial function. Continue until INR normalizing, liver recovery evident, or transplant. Never withhold NAC based on timing alone.

44. B - Classic opioid toxidrome: pinpoint pupils (miosis), respiratory depression, and decreased consciousness. Naloxone (0.4-2 mg IV/IM/IN, repeat as needed) is the antidote. Start with lower doses in opioid-dependent patients to avoid precipitating severe withdrawal. Goal is adequate respirations, not full alertness. Short duration may require repeat dosing or infusion.

45. B - Methadone has a long half-life (24-36 hours) compared to naloxone (30-90 minutes). After naloxone reversal, renarcotization can occur as naloxone wears off. These patients require extended observation (at least 4-6 hours, longer for methadone/fentanyl patches), repeated naloxone dosing, or naloxone infusion. Never discharge immediately after naloxone reversal.

46. B - Anticholinergic toxidrome: "hot as a hare (hyperthermia), blind as a bat (mydriasis), dry as a bone (dry skin/mucous membranes), red as a beet (flushed), mad as a hatter (altered mental status/agitation), full as a flask (urinary retention)." Caused by antihistamines, TCAs, antipsychotics, atropine, scopolamine. Heart rate elevated, bowel sounds decreased.

47. C - Both cause tachycardia, mydriasis, agitation, and hyperthermia. Key difference is SKIN: anticholinergic causes DRY skin (no sweating—"dry as a bone"), while sympathomimetic causes DIAPHORESIS (sweating). Also, anticholinergic has decreased bowel sounds and urinary retention; sympathomimetic usually doesn't affect GI/bladder.

48. B - SLUDGE/DUMBBBELS mnemonics describe cholinergic toxidrome from acetylcholinesterase inhibitors (organophosphates, carbamates, nerve agents). Excess acetylcholine at muscarinic receptors causes secretions (salivation, lacrimation, urination, defecation), bronchospasm, bradycardia. Nicotinic effects include fasciculations and weakness. Can progress to respiratory failure.

49. B - Organophosphate poisoning treatment: Atropine (blocks muscarinic effects—start 2-4 mg IV, double dose every 5-10 minutes until secretions dry) plus pralidoxime/2-PAM (reactivates acetylcholinesterase before "aging" occurs—give within 24-48 hours). Large atropine doses may be needed (sometimes hundreds of milligrams). Decontaminate patient and protect healthcare workers.

50. B - NMS (from antipsychotics/dopamine antagonists) and serotonin syndrome (from serotonergic drugs) both cause hyperthermia, altered mental status, and autonomic instability. Key differences: NMS has "lead-pipe" rigidity, bradykinesia, slow onset (days-weeks); serotonin syndrome has hyperreflexia, clonus, tremor, rapid onset (hours). Both have elevated CK but higher in NMS.

51. B - NMS is caused by dopamine antagonists (antipsychotics, metoclopramide). Classic tetrad: hyperthermia (often >40°C), severe "lead-pipe" rigidity, altered mental status, and autonomic instability (tachycardia, labile BP, diaphoresis). CK markedly elevated (rhabdomyolysis). Onset typically 1-2 weeks after starting/increasing antipsychotic. Mortality 10-20%.

52. B - NMS treatment: Stop offending agent immediately, aggressive supportive care (cooling, hydration, ICU monitoring), consider bromocriptine (dopamine agonist, 2.5-10 mg TID) and/or dantrolene (muscle relaxant, 1-2.5 mg/kg IV). Monitor for complications: rhabdomyolysis, AKI, DIC, aspiration. Recovery typically 1-2 weeks after drug cessation.

53. B - Serotonin syndrome results from excess serotonergic activity, often from drug combinations (SSRIs + tramadol, MAOIs + SSRIs, etc.). Classic triad: altered mental status, autonomic instability, neuromuscular abnormalities (hyperreflexia, clonus, tremor, myoclonus). Onset rapid (within 24 hours of medication change). Clonus (especially lower extremity) is highly characteristic.

54. B - Key distinguishing features: Serotonin syndrome has hyperreflexia, clonus, myoclonus (neuromuscular excitation); NMS has rigidity, bradykinesia (parkinsonian features). Serotonin syndrome has rapid onset (hours); NMS develops over days-weeks. Both have fever, altered mental status, autonomic instability, and elevated CK.

55. B - Ethylene glycol (antifreeze) causes high anion gap metabolic acidosis from toxic metabolites (glycolic acid, oxalic acid) plus osmolar gap from parent compound. Classic triad: CNS depression, cardiopulmonary symptoms, renal failure with calcium oxalate crystals. Fluorescein in antifreeze may cause urine fluorescence under Wood's lamp.

56. B - Fomepizole (preferred) or ethanol inhibits alcohol dehydrogenase, preventing metabolism of ethylene glycol/methanol to toxic metabolites. Give if: confirmed ingestion, metabolic acidosis, osmolar gap, or ethylene glycol/methanol level >20 mg/dL. Hemodialysis indicated for severe acidosis, renal failure, visual symptoms (methanol), or very high levels.

57. C - Salicylate toxicity causes early respiratory alkalosis (direct medullary stimulation) followed by metabolic acidosis (uncoupling oxidative phosphorylation, accumulation of organic acids). Mixed respiratory alkalosis and metabolic acidosis is characteristic. Other features: tinnitus, hyperthermia, altered mental status, hypoglycemia (CNS), pulmonary edema. Treat with sodium bicarbonate alkalinization and hemodialysis.

58. B - TCA toxicity causes cardiac sodium channel blockade manifesting as QRS widening (>100 ms is concerning, >160 ms associated with arrhythmias). Sodium bicarbonate (1-2 mEq/kg boluses, then infusion to maintain pH 7.50-7.55) overcomes sodium channel blockade and treats acidosis. Also treats hypotension and arrhythmias. Avoid class IA and IC antiarrhythmics.

59. B - Timeline suggests alcohol withdrawal: tremor, autonomic hyperactivity (tachycardia, hypertension, diaphoresis), anxiety at 6-24 hours; hallucinations at 12-48 hours; seizures at 12-48 hours; delirium tremens at 48-96 hours. This patient with visual hallucinations at 48 hours is at high risk for progression to delirium tremens. Requires close monitoring and treatment.

60. B - Benzodiazepines are first-line for alcohol withdrawal, reducing seizure risk, severity of withdrawal, and progression to delirium tremens. Long-acting agents (diazepam, chlordiazepoxide) preferred for smoother withdrawal; lorazepam for hepatic impairment. Use symptom-triggered dosing (CIWA protocol) when possible. Add thiamine to prevent Wernicke encephalopathy.