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Question: 1152

A 34-year-old woman presents to the emergency department with a 3-day history of progressive, bilateral sensorineural hearing loss, encephalopathy presenting as acute confusion with paranoid delusions, and multiple small branch retinal artery occlusions visible on fundoscopy. Brain MRI demonstrates multiple small, punched-out lesions in the central corpus callosum that are hyperintense on T2-weighted and FLAIR sequences. Lumbar puncture reveals a mild lymphocytic pleocytosis and elevated protein. What is the most appropriate initial disease-modifying therapeutic approach for this patient?

A. Therapeutic anticoagulation with unfractionated heparin followed by long-term warfarin therapy
B. Wide-field retinal photocoagulation combined with high-dose oral prednisone therapy
C. Intravenous tissue plasminogen activator followed by dual antiplatelet therapy with aspirin and clopidogrel
D. High-dose intravenous methylprednisolone followed by oral prednisone and cyclophosphamide
E. Intravenous immunoglobulin administration followed by chronic plasma exchange therapy

Answer: D

Explanation: The clinical triad of encephalopathy, branch retinal artery occlusions, and bilateral sensorineural hearing loss is highly specific for Susac syndrome, an immune-mediated microangiopathy targeting the precapillary arterioles of the brain, retina, and cochlea. The pathognomonic MRI finding of small, punched-out lesions in the central corpus callosum, often resembling "snowballs," reflects the selective involvement of the microvasculature supplying this region. Because the underlying pathophysiology involves an autoimmune-mediated endothelial injury rather than thromboembolism, standard stroke therapies like thrombolysis or anticoagulation are ineffective. Aggressive immunosuppressive therapy is required to prevent irreversible neurological deficit, vision loss, and deafness. The standard induction regimen for severe presentations involves high-dose intravenous corticosteroids paired with potent immunosuppressive agents such as cyclophosphamide or rituximab to arrest the immune-mediated destruction of the microvascular endothelium.

Question: 1153

A 64-year-old woman is brought to the clinic by her partner, who reports a rapid decline in her cognitive abilities over the past four months. She has transitioned from being fully independent to requiring total care for basic activities of daily living. On examination, she is disoriented, has severe startle myoclonus elicited by unexpected auditory stimuli, a progressive cerebellar ataxia, and bilateral pyramidal signs. An electroencephalogram demonstrates periodic synchronous sharp-wave complexes at a frequency of 1 Hz. A lumbar puncture is performed, and cerebrospinal fluid analysis is notable for a normal cell count and protein level, but is positive for the 14-3-3 protein and has an extremely elevated level of total tau protein. What is the most definitive method to confirm the diagnosis of this patient's underlying condition?

A. Repeat electroencephalography showing burst-suppression patterns
B. Brain biopsy demonstrating spongiform encephalopathy
C. Genetic testing for mutations in the presenilin-1 gene
D. Real-Time Quaking-Induced Conversion assay of cerebrospinal fluid
E. High-resolution magnetic resonance imaging showing cortical ribboning

Answer: B

Explanation: The patient's clinical presentation of rapidly progressive dementia, myoclonus, ataxia, and pyramidal signs, combined with periodic synchronous sharp-wave complexes on EEG and a positive 14-3-3 protein, is classic for sporadic Creutzfeldt-Jakob disease, a transmissible spongiform encephalopathy. While the Real-Time Quaking-Induced Conversion (RT-QuIC) assay provides extremely high sensitivity and specificity in vivo, the gold standard and most definitive method for confirming the diagnosis remains a brain biopsy or post-mortem brain tissue examination demonstrating the characteristic triad of spongiform vacuolation, neuronal loss, and astrogliosis, along with prion protein deposition. Presenilin-1 mutations are linked to early-onset familial Alzheimer's disease.

Question: 1154

A 58-year-old man undergoes an unenhanced head CT scan following a minor motor vehicle

accident, which incidentally reveals a 7-mm, well-circumscribed, lobulated unruptured aneurysm arising from the anterior communicating artery. He has a history of well-controlled hypertension and a 20 pack-year smoking history, though he quit 5 years ago. He has no family history of subarachnoid hemorrhage or aneurysms. When counseling this patient utilizing data from the Phases score and the Unruptured Intracranial Aneurysm Treatment Score (UIATS), which of the following statements provides the most accurate estimation of management strategy and risk stratification?

A. His 5-year rupture risk is approximately 15%, making immediate endovascular coil embolization mandatory regardless of comorbidities
B. The PHASES score accounts for aneurysm size, site, patient age, hypertension, geographic region, and history of prior SAH
C. The patient's past smoking history permanently negates any benefit derived from a conservative observational approach
D. The anterior communicating artery location carries a lower rupture risk than a similar-sized internal carotid artery aneurysm
E. The UIATS framework strongly recommends conservative management for all anterior circulation aneurysms measuring under 10 mm

Answer: B

Explanation: The PHASES score is a validated tool used to predict the 5-year rupture risk of unruptured intracranial aneurysms based on six easily assessable predictors: Population (geographic region), Hypertension, Age, Size of aneurysm, Earlier subarachnoid hemorrhage from another aneurysm, and Site of aneurysm. Aneurysms located at the anterior communicating artery or posterior circulation carry a higher rupture risk than those located on the internal carotid artery for any given size. The UIATS provides a comprehensive framework balancing patient-specific and aneurysm-specific factors to guide management decisions (repair versus observation), rather than issuing blanket restrictions based purely on size or location. A 7-mm anterior communicating artery aneurysm typically yields a modest 5-year rupture risk (well under 15%), making a nuanced discussion regarding observation versus treatment essential.

Question: 1155

A stroke unit implements an intensive educational program for nursing staff focused on the early identification of neurological decline using the NIH Stroke Scale. To rigorously evaluate the statistical significance of the pre-intervention versus post-intervention rates of rapid response team activation for stroke patients, the quality officer plans to analyze data collected monthly over one year before and one year after the intervention. Which of the following statistical methods is most appropriate for analyzing this type of sequential, time-dependent quality improvement data?

A. An interrupted time-series analysis to evaluate changes in both the level and the trend over time
B. A multivariable logistic regression model excluding the time variable to avoid confounding factors
C. A simple Pearson correlation coefficient comparing the time elapsed to the absolute activation counts
D. A standard independent two-sample Student t-test of the pre- and post-intervention annual means
E. A standard chi-square test of independence combining all twenty-four months of data into a single pool

Answer: A

Explanation: In quality improvement research, data are collected sequentially over time, making them subject to secular trends and autocorrelation. An interrupted time-series analysis is a quasi-experimental design that is highly robust and considered the gold standard for evaluating the impact of a distinct population-level intervention implemented at a specific point in time. It allows investigators to statistically evaluate whether the intervention produced a significant immediate change in level, a change in the long-term trend, or both, while controlling for pre-existing trends. A standard t-test or a simple chi-square test pools the data, ignoring the critical element of time and chronological trends, which can lead to erroneous conclusions. A Pearson correlation coefficient only measures a linear relationship between two continuous variables and cannot model an intervention effect. Logistic regression that excludes time would fail to account for the fundamental time-dependent structure of the quality improvement data.

Question: 1156

A 78-year-old female with a history of mild cognitive impairment presents with an acute, spontaneous lobar intracerebral hemorrhage in the left occipital lobe. She has no history of hypertension. A gradient-echo (GRE) or T2*-weighted susceptibility brain MRI reveals multiple, asymptomatic microbleeds restricted exclusively to the lobar cortical and subcortical regions, sparing the basal ganglia, pons, and cerebellum. This constellation of clinical and imaging findings is most indicative of which underlying vascular pathology?

A. Septic embolic panarteritis
B. Primary central nervous system vasculitis
C. Sneddon syndrome
D. Hypertensive arteriolosclerosis
E. Cerebral amyloid angiopathy

Answer: E

Explanation: Cerebral amyloid angiopathy (CAA) is a major cause of spontaneous lobar intracerebral hemorrhage in elderly individuals. It is histopathologically characterized by the progressive deposition of beta-amyloid peptide within the walls of small- to medium-sized arteries and arterioles of the cerebral cortex and leptomeninges. According to the Modified Boston Criteria, the presence of multiple microbleeds restricted strictly to lobar (cortical and subcortical) locations in a patient aged 55 or older, without another identifiable cause, provides a diagnosis of probable CAA**.** Hypertensive microbleeds, by contrast, characteristically involve deep structures like the basal ganglia, thalamus, and brainstem.

Question: 1157

A 31-year-old woman with a history of episodic migraine with aura presents with a new type of headache. Over the past 3 weeks, she has experienced daily episodes of sudden, severe, throbbing pain over her left forehead and eye. Each episode lasts between 2 and 5 minutes and recurs 15 to 20 times throughout the day and night. The pain is accompanied by prominent tearing and redness of her left eye, as well as rhinorrhea on the left side. She notes that the attacks can sometimes be triggered by turning her head to the left. She has tried

sumatriptan and ibuprofen with no relief. Which of the following medications is the most effective and specific treatment for this patient's condition?

A. Indomethacin
B. Topiramate
C. Verapamil
D. Lithium carbonate
E. Oxygen via high-flow mask

Answer: A

Explanation: The patient's clinical presentation is consistent with paroxysmal hemicrania, a trigeminal autonomic cephalgia characterized by frequent (typically > 5 per day), short-lasting (2 to 30 minutes) attacks of severe unilateral pain accompanied by prominent ipsilateral cranial autonomic symptoms. A defining feature of paroxysmal hemicrania, which distinguishes it from cluster headache, is its absolute and dramatic response to therapeutic doses of indomethacin (usually 75 mg to 150 mg daily). Oxygen and verapamil are highly effective for cluster headaches but are not the primary specific treatments for paroxysmal hemicrania. Lithium can be used for chronic cluster headache. Topiramate is a migraine preventive and does not demonstrate the rapid, absolute response required to satisfy the diagnostic criteria for paroxysmal hemicrania.

Question: 1158

A 74-year-old woman with a history of stage III chronic kidney disease, type 2 diabetes, and severe peripheral vascular disease presents with recurrent, stereotypic 5-minute episodes of left leg weakness and numbness. Carotid ultrasound shows mild non-stenotic plaques. The clinician suspects an intracranial or extracranial large-vessel steno-occlusive lesion within the posterior circulation or right anterior circulation. Given her compromised renal function (estimated glomerular filtration rate of 32 mL/min/1.73m2), the team wishes to evaluate the intracranial vasculature while minimizing the risks of contrast-induced nephropathy or nephrogenic systemic fibrosis. Which diagnostic imaging modality and specific protocol provides the highest-resolution structural mapping of the intracranial arterial tree without requiring the administration of any intravenous gadolinium or iodinated contrast media?

A. Transcranial color-coded duplex sonography through the transtemporal and transforaminal windows exclusively
B. Contrast-enhanced 3D cranial computed tomography angiography with pre-procedural sodium bicarbonate hydration
C. Three-dimensional time-of-flight magnetic resonance angiography (3D TOF-MRA) of the head
D. Two-dimensional phase-contrast magnetic resonance venography
E. High-resolution vessel wall MRI using an interleaved T2-weighted turbo spin-echo sequence

Answer: C

Explanation: Three-dimensional time-of-flight magnetic resonance angiography (3D TOF-MRA) is a non-contrast MRI technique that visualizes vascular structures without exogenous contrast agents. It relies on the flow-related enhancement of flowing blood protons. As stationary tissue protons within the imaged slab become saturated by repeated, rapid radiofrequency pulses, their signal is suppressed. Fresh, unsaturated blood protons flowing into the imaging volume from outside the saturated slab possess full longitudinal magnetization, appearing bright and providing high signal-to-noise ratio contrast against the dark background. 3D TOF-MRA provides high-resolution anatomical maps of the circle of Willis and major intracranial branches, making it the ideal diagnostic choice for identifying high-grade intracranial stenoses or occlusions in patients with advanced chronic kidney disease where iodinated contrast (risk of contrast-induced nephropathy) or gadolinium contrast (risk of nephrogenic systemic fibrosis with older agents or general volume caution) should be avoided.

Question: 1159

A 45-year-old man with a long-standing history of alcohol use disorder is admitted to the hospital for acute pancreatitis. Thirty-six hours after admission, he experiences two generalized tonic-clonic seizures separated by 20 minutes, returning to his baseline clear sensorium between them. His vital signs show a temperature of 37.8°C, pulse of 112 bpm, and blood pressure of 155/92 mmHg. He is tremulous, anxious, and diaphoretic. Laboratory

results reveal normal serum glucose, calcium, and magnesium levels. What is the most appropriate management strategy for this patient's seizures?

A. Perform an urgent lumbar puncture to rule out bacterial meningitis
B. Administer scheduled intravenous lorazepam or chlordiazepoxide based on CIWA protocol
C. Schedule an urgent outpatient brain MRI and routine EEG
D. Initiate a maintenance regimen of oral phenytoin 300 mg daily
E. Administer an intravenous loading dose of levetiracetam 1500 mg

Answer: B

Explanation: The patient's seizures are occurring within the classic 6 to 48-hour window following cessation of alcohol intake, accompanied by autonomic hyperactivity, diagnosing alcohol withdrawal seizures. These are provoked seizures caused by transient neurochemical imbalances, specifically GABA receptor downregulation and NMDA receptor upregulation. Long-term maintenance therapy with antiseizure medications like phenytoin or levetiracetam is not indicated or effective for alcohol withdrawal seizures. The treatment of choice consists of benzodiazepines, such as lorazepam, diazepam, or chlordiazepoxide, managed using a symptom-triggered protocol like the Clinical Institute Withdrawal Assessment for Alcohol. Neuroimaging and lumbar puncture are unnecessary unless focal signs, prolonged postictal state, or persistent fever develop.

Question: 1160

A 58-year-old male with a history of mechanical mitral valve replacement is admitted for an acute ischemic stroke secondary to a occlusion of a branch of the left middle cerebral artery. His initial head CT shows a small infarct without hemorrhage. He is currently taking warfarin, and his INR on admission is 2.5. According to current evidence-based guidelines and consensus regarding the management of therapeutic anticoagulation in the acute phase of ischemic stroke, what is the most appropriate timing to resume warfarin therapy?

A. Within 4 to 14 days, tailored to infarct size and individual hemorrhagic transformation risk

B. Postpone indefinitely and transition the patient permanently to dual antiplatelet therapy
C. Within 24 hours of symptom onset regardless of the size or appearance of the infarct
D. Immediately upon admission combined with an intravenous heparin infusion bridge
E. Within 48 hours provided a repeat CT scan demonstrates complete resolution of the ischemia

Answer: A

Explanation: Resuming therapeutic anticoagulation in patients with prosthetic valves who suffer an acute ischemic stroke requires balancing the risk of recurrent cardioembolism against the risk of hemorrhagic transformation of the recent infarct. Current practice consensus suggests delaying the initiation or resumption of oral anticoagulation for 4 to 14 days. The precise timing is highly individualized, taking into heavy consideration the volume of the infarcted tissue, the presence of asymptomatic petechial hemorrhage on imaging, and the control of systemic blood pressure.

Question: 1161

A 45-year-old man presents with an acute, spontaneous intracerebral hemorrhage centered in the left caudate nucleus, measuring 12 mL on head CT, with significant extension of blood into the lateral and third ventricles causing acute obstructive hydrocephalus. His blood pressure is 155/90 mmHg, and he has no history of hypertension, trauma, or drug use. A digital subtraction angiogram (DSA) is performed and reveals a vascular malformation located near the ependymal surface of the lateral ventricle. The angiogram demonstrates a distinct "medusa head" appearance, characterized by multiple small, radiating radicles or anomalous veins draining into a single, enlarged central collector vein that empties into the deep venous system. What is the most accurate diagnosis of this vascular malformation?

A. Cavernous malformation (cavernoma)
B. Developmental venous anomaly (DVA)
C. Capillary telangiectasia
D. Arteriovenous malformation (AVM)
E. Dural arteriovenous fistula (DAVF)

Answer: B

Explanation: The angiographic appearance of multiple small, radiating, tufted venous radicles draining into a large, dilated central venous collector column is classic and pathognomonic for a developmental venous anomaly (DVA), also known as a venous angioma. This vascular arrangement is often described as a "medusa head" or "caput medusae." DVAs are congenital variations in normal venous drainage and are generally benign, low-flow lesions. While they are usually asymptomatic, they can occasionally be associated with cavernous malformations or undergo thrombosis, which can rarely precipitate hemorrhage or venous infarction, particularly when located in an intraventricular or subependymal location.

Question: 1162

A 64-year-old man presents with a left hemispheric ischemic stroke and global aphasic symptoms. During the discharge planning meeting, his wife expresses profound distress, stating, "He was the primary breadwinner, and I don't know how I am going to care for him at home, manage our finances, and cope with the fact that he cannot talk to me anymore." Which of the following represents the most comprehensive and supportive response by the vascular neurologist?

A. Advise her to focus solely on his physical therapy exercises and let the hospital social workers handle all financial concerns.
B. Reassure her that most patients with global aphasia recover their speech completely within 3 to 6 months of intensive therapy.
C. Acknowledge the overwhelming nature of her burden and coordinate a multi-disciplinary meeting with social work, and case management.
D. Recommend that she start taking an antidepressant medication to help her cope with the emotional strain of caregiving.
E. Inform her that she may need to consider placing him in a permanent long-term care facility if she cannot handle the stress.

Answer: C

Explanation: Stroke recovery introduces extensive psychological, financial, and logistical burdens for caregivers. An expert vascular neurologist addresses these multi-faceted challenges by acknowledging the emotional strain and actively involving a multi-disciplinary support network (social work for finances, speech therapy for communication strategies, and case management for home care resources). This provides structural support rather than generic reassurance or premature institutionalization.

Question: 1163

A clinical research coordinator is reviewing the documentation required by the Food and Drug Administration (FDA) for an upcoming multi-center clinical trial testing an investigative endovascular reperfusion device. The sponsor must file an application to legally distribute the device for human clinical investigations. Which of the following acronyms correctly represents the specific regulatory application required by the FDA before clinical trials can begin on a significant-risk medical device?

A. IDE (Investigational Device Exemption)
B. HDE (Humanitarian Device Exemption)
C. PMA (Premarket Approval)
D. 510(k) Premarket Notification
E. IND (Investigational New Drug)

Answer: A

Explanation: An Investigational Device Exemption (IDE) allows a significant-risk investigational device to be used in a clinical study in order to collect safety and effectiveness data. This exemption is required by the FDA before human clinical trials can commence for any new device that has not been cleared or approved for that specific indication. An IND is used for pharmaceutical agents, a 510(k) is for devices substantially equivalent to already marketed predicates, and a PMA is the final approval application for marketing significant-risk devices based on completed trial data.

Question: 1164

A 48-year-old man presents with a 2-day history of a severe, sharp, left-sided neck pain that radiates to his occiput, which began after an intense chiropractic cervical manipulation session. Twelve hours after the pain started, he developed a drooping left eyelid and a smaller left pupil. Today, he experienced two separate episodes of transient weakness in his right hand lasting 15 minutes each. On examination, he has left-sided ptosis and miosis, but no cranial nerve deficits or limb weakness at the time of evaluation. What is the most appropriate definitive diagnostic imaging test to confirm the suspected underlying pathology?

A. Catheter-based digital subtraction angiography of the cerebral vessels
B. High-resolution ultrasound of the bilateral carotid and vertebral arteries
C. MRI of the brain with diffusion-weighted imaging and fluid-attenuated sequences
D. Non-contrast computed tomography scan of the head and cervical spine
E. CT angiography of the head and neck from the aortic arch to the vertex

Answer: E

Explanation: The clinical triad of unilateral neck/occipital pain, ipsilateral Horner syndrome (ptosis and miosis), and contralateral transient ischemic attacks (right hand weakness) following mechanical trauma (chiropractic manipulation) is highly specific for a left vertebral artery dissection. CT angiography from the aortic arch to the vertex is an excellent, non-invasive, rapid, and highly sensitive definitive diagnostic modality to visualize the arterial wall lumen, intraluminal flaps, or tapering occlusions associated with cervical artery dissections. Catheter-based angiography is invasive and rarely needed as an initial diagnostic tool given modern CTA/MRA capabilities. Ultrasound has limited sensitivity for the distal or intracranial segments of the vertebral arteries. Brain MRI is useful for identifying parenchymal ischemia but is less optimal than CTA or dedicated neck MRA for visualizing the dissection vessel wall itself. Non-contrast CT cannot visualize vascular dissection.

Question: 1165

A 68-year-old man with a history of hypertension and tobacco use presents with acute onset of vertigo, dysphagia, left-sided facial numbness, and right-sided truncal ataxia. On examination, he has left-sided ptosis and miosis, diminished pain and temperature sensation on the left side of his face and the right side of his body, and hoarseness with poor elevation of the left palate. A brain MRI confirms an acute infarct. Neurovascular imaging reveals an occlusion. Which of the following vascular structures is most likely occluded to produce this specific constellation of clinical findings?

A. Left posterior inferior cerebellar artery
B. Left anterior inferior cerebellar artery
C. Right superior cerebellar artery
D. Right anterior spinal artery
E. Right paramedian pontine perforator

Answer: A

Explanation: The clinical scenario describes lateral medullary syndrome (Wallenberg syndrome) on the left side. The key features include ipsilateral Horner syndrome (ptosis and miosis from damage to the descending sympathetic tract), ipsilateral facial loss of pain and temperature (spinal trigeminal nucleus and tract), contralateral body loss of pain and temperature (lateral spinothalamic tract), dysphagia and hoarseness (nucleus ambiguus involving cranial nerves IX and X), and ipsilateral hemiataxia (inferior cerebellar peduncle). This specific region of the dorsolateral medulla is supplied by the posterior inferior cerebellar artery (PICA) or the vertebral artery from which it originates. Occlusion of the left PICA accounts perfectly for this constellation of signs. Occlusions of the other listed arteries would result in different brainstem syndromes, such as lateral pontine syndrome (AICA) or medial medullary syndrome (ASA).

Question: 1166

A 48-year-old woman with an acute right middle cerebral artery ischemic stroke is treated with intravenous alteplase. Two hours after the infusion, she complains of severe headache and develops acute onset of bradycardia and hypertension (Cushing's triad). A stat non-

contrast head CT reveals a large parenchymal hematoma (PH2) with a 10 mm midline shift. The vascular neurology resident panics and tells the patient's husband, "The clot-busting medicine we gave her caused her brain to bleed, and she is likely going to die because of what we did." How should the attending vascular neurologist address this communication crisis?

A. Confirm the resident's statement to the husband and advise him to contact a medical malpractice attorney immediately.
B. File a legal non-disclosure agreement to prevent the family from discussing the resident's statements outside the hospital.
C. Reassign the resident to another service immediately and refuse to allow them to communicate with any future stroke families.
D. Tell the husband that the resident was completely wrong and that the hemorrhage was caused by the patient's high blood pressure alone.
E. Take the husband aside, apologize for the resident's bluntness, and clarify that intracranial hemorrhage is an inherent.

Answer: E

Explanation: Managing a communication crisis where a trainee has inappropriately framed a known complication as an act of negligence requires immediate damage control by the attending. The attending must provide honest, objective disclosure of the complication (intracranial hemorrhage occurs in approximately 6% of alteplase cases) while contextualizing it as an inherent risk of a necessary, life-saving intervention, rather than an error or malpractice, thereby restoring a professional and therapeutic relationship.

Question: 1167

A 29-year-old previously healthy woman presents with a three-day history of progressive headache, subjective fevers, altered mental status, and a new onset of focal seizures affecting her left upper extremity. Brain magnetic resonance imaging demonstrates T2-hyperintensity and restricted diffusion isolated within the right medial temporal lobe, insular cortex, and inferior frontal lobe. Cerebrospinal fluid analysis reveals a lymphocytic pleocytosis, elevated protein, and normal glucose. Polymerase chain reaction confirms the

presence of Herpes Simplex Virus 1 DN**A.** Which of the following mechanisms best explains the strict anatomical tropism displayed by this virus during central nervous system infection?

A. Selective binding to endothelial cell adhesion molecules found exclusively in temporal capillaries
B. Microglial activation restricted to areas expressing high densities of chemokine receptor 5
C. Oligodendrocyte vulnerability mediated by regional differences in myelin basic protein expression
D. Vulnerability of cortical neurons expressing specific acetylcholine receptor subtypes in the uncus
E. Retrograde axonal transport via the olfactory or trigeminal nerves to limbic structures

Answer: E

Explanation: Herpes simplex virus 1 encephalitis characteristically demonstrates a striking tropism for the temporal and frontal lobes, particularly the limbic system. This anatomical localization is due to the route of entry and propagation of the virus. The virus typically establishes latency in the trigeminal ganglion or enters the central nervous system via the olfactory mucosa. Retrograde and anterograde axonal transport along the olfactory tracts or the meningeal branches of the trigeminal nerve provides direct access to the orbitofrontal and medial temporal cortices, leading to localized necrotizing encephalitis.

Question: 1168

A 61-year-old man with end-stage renal disease on hemodialysis presents to the emergency department with progressive lethargy, asterixis, and generalized myoclonus over the past 48 hours. He missed his last two scheduled dialysis sessions. His laboratory workup is notable for a blood urea nitrogen level of 142 mg/dL and a serum creatinine of 11.4 mg/dL. Electroencephalography reveals generalized, symmetric, synchronous three-Hz triphasic waves over a diffuse background slowing. What is the primary neurochemical mechanism responsible for the central nervous system dysfunction seen in this condition?

A. Downregulation of central postsynaptic NMDA receptors due to high serum creatinine levels

B. Microvascular endothelial cell death caused by severe, systemic metabolic acidosis
C. Intracellular accumulation of glutamine causing astrocyte swelling and cerebral edema
D. Impairment of the blood-brain barrier leading to a massive influx of urea into neurons
E. Accumulation of middle molecules and guanidino compounds causing GABA receptor inhibition

Answer: E

Explanation: Uremic encephalopathy is a complex metabolic syndrome that occurs in patients with acute or chronic renal failure. The precise neurochemical pathogenesis involves the accumulation of various retained uremic toxins, which include urea, guanidino compounds, uric acid, and middle molecules. Guanidino compounds, such as guanidinosuccinic acid and methylguanidine, act as endogenous neurotoxins by inhibiting the activity of γ-aminobutyric acid (GABA)-A receptors and activating NMDA receptors. This imbalance between inhibitory and excitatory neurotransmission alters the cerebral metabolic rate for oxygen and glucose, leading to cortical hyperexcitability, which manifests clinically as asterixis, myoclonus, seizures, and characteristic triphasic waves on electroencephalography. It contrasts with hepatic encephalopathy, where glutamine accumulation in astrocytes is the primary driver.

Question: 1169

A 63-year-old woman with a history of hypertension, type 2 diabetes, and severe peripheral arterial disease presents with an acute ischemic stroke presenting as a mild left arm weakness and numbness (NIHSS score 2). Her non-contrast head CT is completely normal. She arrives 6 hours after symptom onset and is not a candidate for thrombolysis. A CT angiogram demonstrates a 55% stenosis of the right intracranial internal carotid artery. The clinical team decides to initiate dual antiplatelet therapy for secondary prevention based on the POINT and CHANCE trial criteria.

A. Initiate aspirin 81 mg daily and clopidogrel 75 mg daily after a clopidogrel 300 mg loading dose
B. Initiate aspirin 325 mg daily and prasugrel 10 mg daily without a loading dose
C. Initiate clopidogrel 75 mg daily as monotherapy and defer dual antiplatelet therapy

D. Initiate oral warfarin titrated to an INR of 2.0-3.0 and add aspirin 81 mg daily
E. Initiate aspirin 81 mg daily and ticagrelor 90 mg twice daily after a ticagrelor 180 mg loading dose

Answer: A

Explanation: For patients with an acute minor ischemic stroke (NIHSS score ≤3) or high-risk TIA who present within 24 hours of symptom onset, standard guidelines recommend the initiation of short-term dual antiplatelet therapy (DAPT) with aspirin and clopidogrel to reduce the risk of early recurrent stroke. The standard protocol derived from the CHANCE and POINT trials involves administering a loading dose of clopidogrel (300 mg to 600 mg) followed by 75 mg daily, combined with low-dose aspirin, maintained for 21 to 90 days. Prasugrel is absolutely contraindicated in patients with a history of stroke or TIA due to an unacceptably high risk of intracranial hemorrhage. While aspirin plus ticagrelor is an alternative regimen validated in the THALES trial (especially for patients with intracranial stenosis), aspirin plus clopidogrel remains the primary first-line standard unless a clopidogrel resistance or hypersensitivity is known. Full anticoagulation with warfarin combined with aspirin is not indicated for intracranial atherosclerosis or lacunar strokes, as trials like WASID and SAMMPRIS showed that it significantly increases major bleeding risks without providing superior ischemic protection compared to antiplatelet therapy.

Question: 1170

A 62-year-old female is evaluated in the clinic for an asymptomatic, severe 80% stenosis of the left internal carotid artery, discovered incidentally during evaluation for positional vertigo. She has no history of transient ischemic attacks or strokes. She has well-controlled hypertension and takes low-dose aspirin. A discussion regarding the choice between carotid endarterectomy (CEA) and carotid artery stenting (CAS) is initiated. Based on data from major randomized clinical trials evaluating asymptomatic carotid disease, which of the following statements represents the most accurate comparison of outcomes between CEA and CAS in asymptomatic patients?

A. CEA is indicated only if the stenosis progresses to 100% complete occlusion on consecutive imaging studies

B. CAS is associated with a lower 30-day stroke or death rate compared to CEA across all age groups
C. CAS is associated with a significantly lower rate of periprocedural myocardial infarction compared to CEA
D. CEA and CAS have identical long-term ipsilateral stroke prevention rates, but CAS carries a higher periprocedural stroke risk, particularly in patients over 70 years old
E. Medical management alone has been proven superior to both CEA and CAS in all modern clinical trials

Answer: D

Explanation: Data from large trials such as CREST and ACT I demonstrate that in asymptomatic patients with high-grade carotid stenosis, both CEA and CAS offer durable, comparable long-term protection against ipsilateral stroke. However, during the periprocedural period, CAS carries a slightly higher risk of minor stroke, which becomes more pronounced in elderly individuals (typically older than 70 years) due to increased vascular tortuosity and calcified plaque burden. Conversely, CEA carries a higher risk of periprocedural myocardial infarction and cranial nerve injury.

Question: 1171

A 38-year-old woman with a history of intravenous drug use presents with a subacute history of progressive right-sided weakness, cognitive slowing, and left-sided headaches over the past three weeks. On examination, she is cachectic and has a prominent right hemiparesis and expressive aphasia. Her CD4+ T-lymphocyte count is 42 cells/mm3. A brain MRI reveals confluent, asymmetric, non-enhancing T2/FLAIR hyperintensities involving the subcortical white matter of the left frontal and parietal lobes, notably involving the U-fibers, without mass effect. Cerebrospinal fluid PCR is positive for JC virus DN**A.** Which of the following cellular targets is primarily infected by this pathogen, leading to the observed pathology?

A. Microglial cells causing widespread neuroinflammation
B. Neurons within the deep layers of the cerebral cortex
C. Endothelial cells disrupting the blood-brain barrier

D. Astrocytes causing secondary microvascular thrombosis
E. Oligodendrocytes leading to active demyelination

Answer: E

Explanation: Progressive multifocal leukoencephalopathy is a demyelinating disease of the central nervous system caused by the lytic infection of oligodendrocytes by the JC polyomavirus in severely immunocompromised individuals. The destruction of oligodendrocytes, which are responsible for producing and maintaining myelin in the central nervous system, leads to patchy, confluent areas of demyelination. This is characteristically seen on neuroimaging as asymmetric white matter lesions that involve the subcortical U-fibers and lack mass effect or significant contrast enhancement.

Question: 1172

A 60-year-old man is evaluated in the stroke clinic for a progressive, stuttering course of dysarthria-clumsy hand syndrome over the past month. A brain MRI reveals a small, acute infarct in the paramedian left pons, along with multiple older lacunar infarcts in the internal capsule and thalamus. A carotid duplex ultrasound shows minimal stenosis at the bifurcation. Autonomic function testing is requested to differentiate between primary degenerative autonomic failure and vascular-induced dysautonomia arising from strategic brainstem lesions. During the continuous beat-to-beat blood pressure and heart rate monitoring during a 70-degree head-up tilt table test, the patient experiences a sudden drop in systolic blood pressure of 35 mmHg within 2 minutes of tilting, without an appropriate compensatory increase in heart rate (the heart rate increases by only 4 beats per minute). Plasma norepinephrine levels are measured in both the supine and tilted positions. Which laboratory profile confirms a central, pre-ganglionic autonomic deficit as opposed to a peripheral, post-ganglionic autonomic neuropathy?

A. Supine norepinephrine level is normal to low-normal, and fails to increase by at least 50% upon tilting
B. Supine norepinephrine level is normal, but drops to zero immediately upon orthostatic challenge
C. Supine norepinephrine level is markedly elevated (> 1200 pg/mL); tilt position causes a

five-fold increase in norepinephrine

D. Supine norepinephrine level is completely undetectable, and remains undetectable after intravenous administration of edrophonium

E. Supine norepinephrine level is normal, and increases by $> 100\%$ upon standing, accompanied by severe tachycardia

Answer: A

Explanation: Neurogenic orthostatic hypotension (nOH) is characterized by a sustained reduction in systolic blood pressure (≥ 20 mmHg) within 3 minutes of orthostatic stress due to impaired baroreflex-mediated sympathetic vasoconpletion. In central, pre-ganglionic autonomic disorders (such as those caused by strategic brainstem vascular lesions or multiple system atrophy), the post-ganglionic sympathetic post-junctional neurons remain intact but lack central driving inputs. Consequently, the supine plasma norepinephrine level is typically within the normal reference range because basal peripheral sympathetic tone is preserved. However, upon tilting or standing, the central nervous system cannot trigger additional norepinephrine release, resulting in a failure to increase plasma norepinephrine levels by at least 50% from baseline. Conversely, in peripheral, post-ganglionic failure (like pure autonomic failure or advanced diabetic neuropathy), supine norepinephrine levels are abnormally low because the peripheral terminals themselves are lost or depleted.

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