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**Certification-Board**

# NCC

*Certified in NeuroCritical Care (ABEM)*



Question: 710

A 55-year-old man, admitted with a Hunt and Hess grade 4 aneurysmal subarachnoid hemorrhage (SAH), is now post bleed day 5 with increasing Transcranial Doppler (TCD) velocities. Mean arterial pressure (MAP) is 76 mmHg on norepinephrine 12 mcg/min, up from 5 mcg/min 24 hours earlier, intracranial pressure (ICP) is 12 mmHg, and heart rate (HR) is 110/min with sinus rhythm on volume control mechanical ventilation receiving 7 mL/kg tidal volume (TV) with positive end-expiratory pressure (PEEP) of 10 mmHg. White blood cell (WBC) count is 11,000 with a new infiltrate on chest x-ray (CXR); and serum creatinine has increased to 1.5 mg/dL. Fluid balance over the past 24 hours is 2 L positive with good urine output. In addition to sending blood cultures, lactate, and starting antibiotics, how can volume status be assessed to determine whether additional fluid is needed for adequate resuscitation?

- A. Bolus 2 L normal saline (NS) over 2 hours and assess change in
- B. Perform a straight leg raise and assess for change in cardiac
- C. Place a Swan-Ganz catheter given the presence of shock and
- D. Measure inferior vena cava size change during inspiration and
- E. Insert arterial line and measure pulse pressure or stroke volume
- F. Arterial blood gas (ABG) reveals a PaO<sub>2</sub>/FiO<sub>2</sub> ratio of 250.

Answer: B

Explanation:

Our patient is in septic shock, which requires timely acquisition of cultures, lactic acid, initiating antibiotics, and fluid resuscitation. This question is focused on how to best determine whether a patient in shock has been adequately resuscitated—a critical question, since too little fluid leads to inadequate preload, reduced cardiac output (CO) and oxygen delivery, and consequently tissue hypoperfusion. Conversely, excessive volume loading, without further increase in CO, would lead to hemodilution and tissue edema. Fluid overload has been associated with worse outcomes in critically ill patients, and current guidelines recommend frequent assessment for ongoing need for resuscitation prior to fluid administration. Fluid responsiveness can be assessed by several methods. Traditional static measures of cardiac preload, such as central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) are unreliable for predicting fluid responsiveness. Dynamic measures of cardiac preload, such as stroke volume variation (SVV) and pulse pressure variability (PPV) require an arterial line, and are excellent measures of volume responsiveness in shock patients - with some notable exceptions: spontaneous respiration; tidal volume (TV) <8 mL/kg; PEEP >5; low lung compliance (i.e., ARDS); and when arrhythmias are present. Our patient was spontaneously breathing, limiting the diagnostic accuracy of SVV/PPV. Variation in inferior vena caval size during mechanical ventilation, as measured by echocardiography, is another means of assessing fluid responsiveness, but seems to be less sensitive and specific than SVV/PPV, with similar limitations. A third technique involves an *autotransfusion*, by passively raising the legs to 40 degrees in a supine patient, which has been shown to be a sensitive and specific assessment for volume responsiveness, potentially even in spontaneously breathing patients, during low-tidal ventilation, and with cardiac arrhythmia.

Question: 711

An 18-year-old man admitted to the hospital with a gunshot wound to the head and intractable intracranial hypertension is being deescalated from hyperosmolar therapy with hypertonic saline and potassium is 2.0 mEq/L, despite having received approximately 120 mEq of potassium chloride in replacement that day, and the urine output

has been consistently greater than 200 mL/hour. Additional pertinent labs include Na 153 mEq/L, Mg 1.5 mEq/L, and HCO<sub>3</sub> 20 mEq/L. You notice a change on the telemetry monitor and order a stat ECG. What is the likely cause of this abnormality?

- A. Acute myocardial infarction (MI)
- B. Acute pulmonary embolism
- C. Hypokalemia
- D. Acidosis
- E. Hypothermia
- G. stat ECG

Answer: C

Explanation:

These ECG changes are typically seen with severe refractory hypokalemia. The earliest ECG changes associated with hypokalemia are a decrease in T-wave amplitude followed by a ST segment depression and T-wave inversions. Subsequently, the PR interval may be prolonged with an increase in amplitude of the P wave. U waves (a positive deflection after the T wave) may be seen and in severe hypokalemia may fuse with the T wave to form giant U waves as seen in the ECG. A pseudo-prolonged QT interval may be seen, which is actually a QU interval in the absence of a T wave. The most common cause of hypokalemia is renal losses after diuretic use (more commonly thiazide than loop or osmotic diuretics), especially if two diuretics acting on different parts of the tubular system are used. Patients who develop secondary hypoaldosteronism from liver disease, congestive heart failure (CHF), or nephrotic syndrome are also at risk. Antibiotics such as penicillins and aminoglycosides can promote potassium loss. In the neurocritical care setting, patients receiving mannitol or hypertonic saline infusions have been noted to develop hypokalemia because of the diuretic effect and as a consequence of the high-sodium load reaching the collecting ducts. For this reason some centers use potassium-sparing diuretics in conjunction with mannitol to avoid symptomatic hypokalemia in the setting of intracranial pressure (ICP) management. Hypokalemia can be refractory if concomitant hypomagnesemia is not corrected. The exact etiology of this is not known, but it may be multifactorial. Low intracellular magnesium levels may increase potassium wasting from the collecting tubules, especially in the setting of additional factors such as an increase in distal sodium delivery. ECG changes in hypomagnesemia include an increased PR and QT interval, widened QRS complex, and flattened T wave. The findings can be identical to those seen in hypokalemia; however, in this case, the patient's potassium level is much lower than his magnesium level and, given the refractory nature of his disease, hypokalemia is the more likely cause. Hypothermia at 35°C (95°F) is associated with sinus tachycardia. As the core body temperature drops further (<90°F or 32°C), sinus bradycardia with prolongation of PR interval, QRS widening, and QT interval are seen. Osborn waves (an upward deflection after the QRS complex) are seen below 86°F or 30°C.

Question: 712

A 44-year-old morbidly obese woman with hypertension and diabetes is found slumped over in a bathroom stall. At the scene, her blood pressure (BP) is documented at 180/90 with a pulse of 78; her blood sugar is 180 mmol/L. CT scan of the head is shown. What is the likely cause of this patient's findings?

- A. Hypertensive intracerebral hemorrhage (ICH)
- B. Embolic stroke with hemorrhagic conversion
- C. Venous sinus thrombosis
- D. Aneurysmal subarachnoid hemorrhage (SAH)
- E. Hemorrhagic encephalitis

Answer: D

Explanation:

This axial CT scan of the head shows an intraparenchymal hemorrhage (IPH) with a sylvian SAH and a small subdural hematoma (SDH), with the outline of an aneurysm visible lateral to the IPH. Approximately 20% of patients with aneurysmal rupture have associated IPH, occurring more commonly in patients with anterior communicating artery (AComm), distal anterior cerebral artery, and distal middle cerebral artery (MCA) aneurysms. These patients present with callosal, interhemispheric, and sylvian/temporal hematomas. In addition, patients may have concurrent intraventricular hemorrhage (IVH) and SDH. Rarely, patients may present with IPH, IVH, or SDH without any evidence of SAH. In these cases, a history of thunderclap headache, absence of risk factors for IPH or IVH, and absence of a history of trauma should raise the suspicion of a saccular aneurysm. Likely causes of the absence of SAH are the location of the dome of the aneurysm close to the ventricular system or brain parenchyma and delayed presentation after the hemorrhage, with radiographic resolution of subarachnoid blood. Another theory is that the IPH/IVH may represent a rebleed after the subarachnoid space has been scarred down by a sentinel hemorrhage.

Question: 713

A 29-year-old C5-C6 quadriplegic patient is being prepared for transfer to a rehabilitation facility when you are paged for with the condition?

- A. Permanent pacemaker
  - B. PRN intravenous (IV) atropine
  - C. PO albuterol
  - D. PO theophylline
  - E. All of the above
  - G. following findings
- What is an appropriate long-term therapy for this

Answer: E

Explanation:

Patients with acute spinal cord injury suffer a myriad of complications of which pulmonary and cardiac are the most common. Among the cardiac complications, shock and hypotension occur early and are usually the reason for ICU admission in the first week after injury. Sinus bradycardia, dysrhythmia, and cardiac arrest can occur subsequently, usually within the first 14 days after injury. Acute spinal cord injury above T6 can disrupt the descending sympathetic pathways to the intermediolateral cell column in the T1-L2 spinal cord. This results in loss of supraspinal sympathetic control and unopposed parasympathetic activity in the respiratory and cardiac systems in quadriplegic patients, making them prone to prolonged episodes of bradycardia, pauses, and intermittent heart blocks, especially when suctioned or turned. Atropine should be readily available for patients with bradycardia, and 0.5 to 1 mg should be administered when symptomatic or prophylactically before suctioning. Oral albuterol and theophylline can be used to increase resting heart rate, although there are no randomized trials to support this. Some patients with significant heart block or recurrent cardiac arrests may require a permanent pacemaker.

Question: 714

Answer: C

Explanation:

The history of ovarian cancer and the diffuse leptomeningeal enhancement are suggestive of meningeal carcinomatosis. Meningeal carcinomatosis is seen in 1% to 5% of patients with solid tumors (commonly breast, nonsmall-cell lung cancer, and melanoma), 5% to 15% of patients with leukemia/lymphoma (leukemic/lymphomatous meningitis), and 1% to 2% of patients with primary brain tumors. Neurologic symptoms

usually localize to several regions of the neuraxis, with those involving the spinal cord and cauda equina being most common, followed by cranial neuropathies and hemispheric dysfunction. Patients often present with signs of hydrocephalus and elevated intracranial pressures (ICP). Diagnosis is made by having a high index of suspicion, meningeal enhancement (often most prominent in the basal meninges, dorsal spinal column, and cauda equina), cerebrospinal fluid (CSF) monocytosis, and isolating tumor cells in CSF on cytology. Although the latter is the gold standard, tumor cells are isolated in only 50% of samples on the first LP, with the yield increasing to 80% on the second puncture. There is no added benefit of subsequent LPs. Treatment is mostly palliative, with median survival of 2 to 3 months. Whole brain radiation therapy and intrathecal chemotherapy are the mainstays of treatment and may prevent further neurologic deterioration and improvement in quality of life. Supportive treatments such as antiepileptic medications, pain medications, and occasionally corticosteroids in patients with concomitant parenchymal disease should be prescribed. Miller-Fisher syndrome is a demyelinating cranial and peripheral neuropathy, which is a variant of Guillain-Barré syndrome (GBS) and is defined by a triad of areflexia, ophthalmoplegia, and ataxia. Patients classically do not have signs of encephalitis or encephalopathy. Anti-GQ1b antibodies are present in 90% of patients. Bickerstaff brainstem encephalitis presents with ataxia, ophthalmoplegia, hyperreflexia, and alteration of consciousness. Hyperintense lesions are seen in the midbrain, pons, and medulla on MRI. A significant number of patients have evidence of concurrent axonal GBS, suggesting that this may be another variant of the disease. The progression of symptoms and MRI findings preclude pontine infarction, and the EEG does not support a diagnosis of NCSE.

Question: 715

A 67-year-old woman was transferred from an outside facility without hospital with complaints of nausea, vomiting, ataxia, and progressive lethargy. The patient had a history of ovarian cancer in remission for 3 years and was otherwise healthy. CT scan and MRI blood cell (WBC) count of 15 (40% N, 20% L, and 40% M), red blood cell (RBC) count 250, glucose 40, and protein 100. All bacterial and viral cultures and polymerase chain reactions (PCRs) were negative, and she was empirically treated with acyclovir. Upon your evaluation, the patient was comatose with sluggish pupillary response, dysconjugate gaze, absent oculoccephalic reflexes, extensor posturing bilaterally, and brisk reflexes. She was breathing repeatedly and showed WBC 18 (100% M), RBC 50, glucose 28, and protein 150. Serum glucose was 150 mg/dL. EEG revealed generalized delta and theta activity without rhythmicity. Brain MRI with contrast is shown on the right. What is the most likely cause of the patient's condition?

- A. Miller-Fisher syndrome
- B. Bickerstaff brainstem encephalitis
- C. Meningeal carcinomatosis
- D. Pontine infarction
- E. Nonconvulsive status epilepticus (NCSE)

Answer: C

Explanation:

The history of ovarian cancer and the diffuse leptomeningeal enhancement are suggestive of meningeal carcinomatosis. Meningeal carcinomatosis is seen in 1% to 5% of patients with solid tumors (commonly breast, nonsmall-cell lung cancer, and melanoma), 5% to 15% of patients with leukemia/lymphoma (leukemic/lymphomatous meningitis), and 1% to 2% of patients with primary brain tumors. Neurologic symptoms usually localize to several regions of the neuraxis, with those involving the spinal cord and cauda equina being most common, followed by cranial neuropathies and hemispheric dysfunction. Patients often present with signs of hydrocephalus and elevated intracranial pressures (ICP). Diagnosis is made by having a high index of suspicion, meningeal enhancement (often most prominent in the basal meninges, dorsal spinal column, and cauda equina), cerebrospinal fluid (CSF) monocytosis, and isolating tumor cells in CSF on cytology. Although the latter is the gold standard, tumor cells are isolated in only 50% of samples on the first LP, with the yield increasing to 80% on the second puncture. There is no added benefit of subsequent LPs. Treatment is mostly palliative, with median survival of 2 to 3 months. Whole brain radiation therapy and intrathecal chemotherapy are the mainstays of

treatment and may prevent further neurologic deterioration and improvement in quality of life. Supportive treatments such as antiepileptic medications, pain medications, and occasionally corticosteroids in patients with concomitant parenchymal disease should be prescribed. Miller-Fisher syndrome is a demyelinating cranial and peripheral neuropathy, which is a variant of Guillain-Barré syndrome (GBS) and is defined by a triad of areflexia, ophthalmoplegia, and ataxia. Patients classically do not have signs of encephalitis or encephalopathy. Anti-GQ1b antibodies are present in 90% of patients. Bickerstaff brainstem encephalitis presents with ataxia, ophthalmoplegia, hyperreflexia, and alteration of consciousness. Hyperintense lesions are seen in the midbrain, pons, and medulla on MRI. A significant number of patients have evidence of concurrent axonal GBS, suggesting that this may be another variant of the disease. The progression of symptoms and MRI findings preclude pontine infarction, and the EEG does not support a diagnosis of NCSE.

Question: 716

A 60-year-old patient with diabetes and hypertension is transferred to your ICU from another facility, where he was being treated for urosepsis. The patient has been on stable, low-dose vasopressors for 4 days, is on antibiotics, and appears adequately volume replete. He has not received nutrition since his admission to the hospital 5 days ago because he was on vasopressors. You would:

- A. Insert a feeding tube and advance to full dose tube feeds despite
- B. Order total parenteral nutrition (TPN)
- C. Order peripheral parenteral nutrition (PPN)
- D. Start a dextrose solution; it should provide enough calories!
- E. Start trophic feeds through the gut at 10 to 20 mL/hour while he

Answer: A

Explanation:

Initiation of enteral nutrition (EN) in critically ill patients is not always clear cut. It is preferable to use EN in critically ill patients as it reduces infectious complications, promotes enterocyte health while maintaining a strong mucosal barrier, and has a lesser stress response than parenteral nutrition (PN). However, there is concern that initiating EN in a patient with hemodynamic compromise and possible splanchnic vasoconstriction may promote nonocclusive mucosal ischemia (NOMI) due to increased oxygen demand. Turza et al. recommend a four-stage approach to initiating EN in patients requiring vasopressors: a. Evaluate the patient's medical and nutritional history. Patients with multiple vascular risk factors may be predisposed to NOMI, while those with poor nutritional response or high metabolic demand will benefit from early nutrition. b. Evaluate the current physiologic state. Low-dose, nonescalating vasopressors in patients who are volume resuscitated and able to maintain a mean arterial pressure (MAP) greater than 60 should not deter the initiation of EN. Alternately, patients with dropping urine output, worsening lactate levels, and base deficit and those requiring frequent transfusions may not be able to tolerate EN. c. Establish gastrointestinal (GI) access and pick appropriate tube feeds. The use of formulas with lower osmolarity (<700 mOsm) and fiber content and simpler sugars will reduce metabolic demands, improve transit time, and reduce dysmotility, thereby alleviating factors which may result in the bacterial overgrowth that worsens NOMI. d. Postinitiation monitoring includes serial abdominal examinations and gastric residual checks. Laboratory monitoring of lactate, white blood cell counts, and hemoglobin levels or radiologic testing may be performed if there is concern for ileus. At times, a combination of trophic EN and supplemental PN is adopted as an intuitive compromise. This has been shown to increase the risk of nosocomial infections in a retrospective review of trauma patients.

Question: 717

A 40-year-old woman is admitted to the hospital after witnessed cardiac arrest for 20 minutes. Bystanders performed CPR until she was initiated after admission to the hospital, and the patient was noted to have frequent

myoclonic jerks during rewarming. Brainstem reflexes were intact and the patient had flexor posturing to painful stimulation. MRI brain revealed scattered diffusion-weighted imaging changes in the cortex but no damage to the deep structures. EEG was performed and is shown in the following

- A. Have a family discussion regarding irreversible brain damage
- B. Load with an antiepileptic agent until myoclonic jerks stop since
- C. Continue EEG monitoring and titrate antiepileptic agents until
- D. Load with antiepileptic drugs until clinical myoclonus stops and
- E. Recool the patient and rewarm more slowly next time

Answer: C

Explanation:

The EEG is consistent with postanoxic status epilepticus (PSE). Although this diagnosis usually portends a poor prognosis, the administration of therapeutic hypothermia (TH) to postcardiac arrest patients has made the prediction of outcomes based on American Academy of Neurology (AAN) criteria more difficult. In a prospective study by Rossetti et al., three clinical variables were demonstrated to have higher false-positive rates compared to the AAN guidelines: incomplete brainstem reflexes, myoclonus, and absent motor responses to pain. Early lack of reactivity on continuous EEG, prolonged periods of discontinuity, epileptiform discharges or seizures, and absent cortical responses on somatosensory evoked potentials (SSEP) were strongly associated with mortality. PSE is independently associated with a poor outcome after anoxic injury. However, in the presence of brainstem reflexes, SSEP responses, and EEG reactivity can have a favorable outcome if the condition is treated as status epilepticus (SE).

Question: 718

A 60-year-old woman with a history of hypertension presented with a cerebellar intraparenchymal hemorrhage (IPH), fourth ventricle intraventricular hemorrhage (IVH), hydrocephalus with partial brainstem dysfunction, and somnolence on examination. She was emergently taken for surgical decompression and did well. A conventional angiogram was subsequently performed and is shown in the following images. Your next steps in management include all of the following, except:

- A. Wean external ventricular drain (EVD) and tightly control blood
- B. Use intrathecal tissue plasminogen activator (tPA) to allow quick
- C. Take a detailed family history
- D. Send genetic testing
- E. Continue outpatient, staged management of this condition

Answer: B

Explanation:

The patient has multiple arteriovenous malformations (AVMs) warranting a detailed family history and genetic workup. Intrathecal tPA is contraindicated in this setting. Intracranial vascular malformations include developmental venous anomalies (DVA), capillary telangiectasias, AVM, and cavernous malformations, each of which have different natural histories and treatment options. Capillary telangiectasias are usually angiographically occult lesions, detected incidentally on contrast-enhanced MRI or CT scans, and are rarely symptomatic. They are benign, thin-walled capillaries surrounded by normal brain parenchyma, do not need treatment or follow-up, and account for 4% to 12% of all vascular malformations. De novo development has been reported. DVA also known as venous angiomas are congenitally enlarged, thickened, hyalinized venous vessels draining normal brain tissue. They are the most common intracranial vascular malformation, with a prevalence of

2.5%, discovered mostly incidentally and often associated with cavernous malformations. A causative link between the two has been suggested, with DVAs being a precursor to cavernous malformations. DVAs can be detected on MRI, CT scan, and angiogram as a single dilated vein or caput medusa. They usually have a benign clinical course with low morbidity and mortality and do not warrant treatment. ò AVMs are a collection of abnormal blood vessels, comprising arteries, veins, and an intervening collection of abnormal vessels called the nidus. They are congenital lesions with an incidence of 1 per 100,000, often presenting as an intracerebral hemorrhage (ICH) in the third to fourth decade. The bleeding risk varies according to size, location, draining pattern, and so on. Magnetic resonance angiography (MRA) and CT angiography (CTA) can both visualize AVMs; however, conventional angiography remains the gold standard for diagnoses, formulation, and implementation of a treatment plan which may include a combination of surgery, embolization, and radiation therapy. ò Cavernous malformations are acquired or congenital lesions formed by endothelium-lined vascular spaces without intervening brain parenchyma. They can be seen on CT and MRI as ôpopcornö-shaped dystrophic calcifications or hemosiderin deposits from prior hemorrhages. They are angiographically occult and most commonly present with seizures. Treatment is not always warranted, but surgical resection is often necessary if associated with recurrent hemorrhages or intractable epilepsy. ò Dural arteriovenous fistulas are malformations located in the meninges fed by dural arteries and drained by dural or leptomeningeal venous channels. The most common location is the transverse sinus. They can develop after trauma, venous thrombosis, or spontaneously, and account for 10% to 15% of all cranial malformations. Clinical presentation includes headache, neurologic deficits, bruits, and hemorrhage, with a cortical drainage pattern consistent with more aggressive behavior. Sixvessel cerebral angiogram remains the gold standard in diagnosing a fistula. Treatment includes endovascular or surgical interventions, with surgery being the more definitive approach.

Question: 719

A 35-year-old, 2 days postpartum woman had sudden onset of thethe ED was 180/90, and she was somnolent but had a nonfocalneurologic examination. Opening pressure was normal on lumbarpuncture (LP), and cerebrospinal fluid (CSF) was clear with normalchemistries and cell count. Imaging studies are shown in thefollowing images. What is the most likely diagnosis?

- A. Eclampsia/preeclampsia
- B. Posterior reversible encephalopathy syndrome (PRES)
- C. Venous sinus thrombosis
- D. Reversible cerebral vasoconstriction syndrome (RCVS)
- E. SheehanÆs syndrome

Answer: D

Explanation:

RCVS unifies a group of disorders with similar clinical and radiographic characteristics, such as CallûFleming syndrome, benign angiopathy of the central nervous system (CNS), migrainous angiitis, drug-induced cerebral angiopathy (selective serotonin reuptake inhibitors [SSRIs], marijuana), and postpartum angiopathy (hormonal changes, serotonergic surge). Typically, patients present with a thunderclap headache with or without focal neurologic signs, normal CSF analysis without evidence of CNS inflammation, exclusion of other causes of sudden severe headache (aneurysm or vascular abnormalities), and presence of segmental cerebral arterial vasoconstriction on catheter, CT, or magnetic resonance angiography (MRA), which resolves within 12 weeks. Cortical (nonaneurysmal) subarachnoid hemorrhage (SAH) is the most common radiographic finding. The gold standard for diagnosis is detecting the presence and subsequent resolution of segmental vasoconstriction on conventional angiography. In most cases the vascular changes resolve without treatment. Calcium channel blockers (nimodipine, verapamil), glucocorticoids, and intravenous (IV) magnesium have been tried with limited success. Although the course is usually benign, severe vasospasm has been reported resulting in transient ischemic attacks (TIAs), seizures, and ischemic and hemorrhagic infarctions. Intraarterial milrinone and verapamil as well as angioplasty have been attempted with some success in severe or refractory cases. Preeclampsia is defined as pregnancy-induced hypertension with proteinuria, but there is no information about urine studies in this case and the condition is not



associated with the imaging findings of cerebral vasoconstriction. Eclampsia, a life-threatening condition occurring in pregnancy or early puerperium, is the occurrence of a tonic-clonic seizure in the setting of preeclampsia. Treatment includes IV magnesium to a goal of 4 to 7 mEq/L (4 g IV loading dose, then 1 g/hour), BP control with IV hydralazine or labetalol, and emergent termination of pregnancy. If the patient continues to have seizures, the dose of magnesium may be increased (with close observation for respiratory failure or heart block). IV anticonvulsants and mechanical ventilation can be initiated in refractory cases. PRES is characterized by headache, confusion, seizures, and visual loss with acute subcortical and cortical edema on MRI. Although classically seen in the parietooccipital lobes, brainstem, and cerebellum, the edema can extend as far anteriorly as the temporal and frontal lobes. PRES can be seen in the setting of malignant hypertension, eclampsia, hypercalcemia, and due to drugs such as tacrolimus and cyclosporine. Sheehan's syndrome, or postpartum pituitary necrosis, is a complication of postpartum hypovolemic shock resulting in panhypopituitarism. The most common presenting symptom is galactorrhea.

Question: 720

An 18-year-old G1P0 woman presented with a severe headache behind her right ear, followed by a left-sided tingling sensation and monitoring and treatment while she continues to have frequent complex partial seizures. On examination, she is awake, but somnolent with left-sided hemiparesis. Imaging studies are shown

- A. Eclampsia/preeclampsia
- B. Posterior reversible encephalopathy syndrome (PRES)
- C. Cerebral venous sinus thrombosis (CVST)
- D. Reversible cerebral vasoconstriction syndrome (RCVS)
- E. Sheehan's syndrome

Answer: C

Explanation:

CVST is a rare disorder affecting 3 to 4 adults per million annually. With the increasing use of oral contraceptives (OCP) in the past few decades, the disease has become more prevalent in adult women of child-bearing age with an almost sixfold increase in the risk of CVST among OCP users. This is followed by patients with inherited thrombophilia, hypercoagulability associated with pregnancy and puerperium, and head and neck infections. Presentation is varied and dependent on the location and extent of venous involvement. Headache is a ubiquitous presenting complaint, accompanied by seizures in 47% and paresis in 43% of patients. The majority of patients have an indolent course with symptoms developing over days to months. Rarely, they may resemble an arterial infarction but with a waxing and waning course. Focal edema and infarctions are often seen when cortical veins are involved. Larger infarctions and hemorrhages are associated with worsening mental status and coma. Abnormal signal change in the venous sinus on MRI with concomitant loss of flow on magnetic resonance venography (MRV) is diagnostic. Treatment includes systemic anticoagulation with weight-based low molecular weight heparin or unfractionated heparin with transition to vitamin K antagonists to a goal International Normalized Ratio (INR) of 2 to 3 for 3 to 6 months. Patients with a history of deep vein thrombosis (DVT) or recurrent CVST will need indefinite treatment. Intraparenchymal hemorrhage (IPH) is not a contraindication to anticoagulation in this population. Local administration of endovascular thrombolysis has been reported, but there is insufficient efficacy or safety data available to justify its utility in patients who are not refractory to systemic anticoagulation. Decompressive craniectomy can be performed in the setting of malignant cerebral edema with reasonable outcomes. Over 80% of the patients have favorable recovery. Mortality of 7% to 13% is seen within the first month, usually due to cerebral edema in the acute phase or due to underlying cause on subsequent follow-up.

Question: 721

A 65-year-old alcoholic with traumatic brain injury (TBI), bifrontal contusions, and right-sided epidural hematoma

with midline shift and uncal herniation is admitted to your ICU. After initial decompression, intracranial pressure (ICP) remained within normal limits. Four days postoperatively, the patient is now off all sedation, withdraws on the right, and flexes on the left. What is the cause of his poor mental status?

- A. Bifrontal injury
- B. Diffuse axonal injury
- C. Persistent effects of midline shift after initial injury
- D. Nonconvulsive status epilepticus (NCSE)
- E. All of the above

Answer: E

Explanation:

The patient has suffered a severe head injury with multiple contusions, cerebral edema, and herniation. Individually, each of these conditions can result in a persistent comatose state. In addition, the patient is having nonconvulsive focal seizures, which may be contributing to the encephalopathy. The growing recognition of nonconvulsive seizures (NCS) in the critically ill population and the need for treatment has been a topic of debate in recent years. Are these seizures a cause of the encephalopathy or simply a manifestation of the dying brain? Similarly, should they be aggressively treated with the hope of resolution of coma or are they a hallmark of irreversible brain injury and a poor prognostic sign? Although case reports supporting both arguments exist, these questions are yet to be answered in a randomized controlled trial. Additionally, many reports of NCSE lump together patients who are delirious with patients who are deeply comatose in the setting of NCS, making it all the more difficult to establish prognosis. Until the availability of further evidence, the best way to approach these situations is to look at the entire clinical picture. Aggressive treatment, with its risks, may be warranted if the clinical picture looks worse than can be explained by the level of injury. Similarly, in the setting of a devastating injury, administration of further sedatives to treat focal NCSE may not be worthwhile.

Question: 722

A 46-year-old man is admitted to your ICU after facial assault with a penetrating object through his orbit. You are called to the bedside on postop day 1 after removal of the object because the patient is in severe pain. Upon your examination, the eye is swollen, injected, movements are difficult to assess because of pain. He has light perception on visual acuity, which is unchanged from the time of presentation. What is the most likely diagnosis?

- A. Orbital cellulitis
- B. Orbital hematoma
- C. Cavernous sinus thrombosis
- D. Carotid-cavernous fistula (CCF)
- E. Orbital compartment syndrome
- G. and pulsatile The pupil is minimally reactive and extraocular

Answer: D

Explanation:

The patient has a CCF, which is an abnormal communication between the arterial and venous blood within the cavernous sinus and is characterized by pain, chemosis, pulsatile proptosis, ocular bruit, and progressive vision loss. The most common form of CCF is a direct communication between the internal carotid artery (ICA) and cavernous sinus (type A) usually as a result of trauma (young males) or aneurysm rupture (older women). Traumatic CCFs are the most common type, accounting for 75% of all CCFs, and occur in 0.2% of all head trauma and 4% of basilar skull fractures. Conventional angiogram is the gold standard for diagnosis. Endovascular

transvenous embolization of the fistula while maintaining patency of the ICA is the mainstay of treatment, with greater than 80% cure rates at 1 year. Symptoms of chemosis and proptosis usually resolve within hours to days of intervention, while cranial nerve (CN) palsies may persist for a few weeks. Visual loss may or may not be reversed depending on the degree of blindness at presentation and the underlying cause. Cavernous sinus thrombosis is a close differential diagnosis and usually presents with ptosis, chemosis, proptosis, CN palsies, vision loss, and a dilated, sluggishly reactive pupil. The most common etiology is infectious with direct spread from the nose, sinuses, or teeth. Diagnosis is made by clinical findings, MRI, and magnetic resonance venography (MRV), and treatment includes intravenous (IV) antibiotics and close monitoring for complications such as meningitis, vision loss, sepsis, or septic emboli. Orbital cellulitis is a bacterial infection of the tissues surrounding the eye, including eyelids, eyebrows, and cheeks, resulting in swelling of the eyelids, pain with eye movements, fever, and decreased vision if not treated promptly. Orbital hematomas can be preseptal or postseptal. Preseptal hemorrhages are usually posttraumatic and benign, resulting in extensive ecchymoses of the eyelids. Postseptal hemorrhages may occur due to trauma, surgical intervention, arteriovenous malformations, or bleeding diathesis, among other causes, and can lead to orbital compartment syndrome with increase in intraocular pressures and vision loss from orbital nerve compression.

Question: 723

A 78-year-old man with a history of hypertension, hyperlipidemia, and mild hearing loss is brought to the hospital by ambulance after a motor vehicle accident. He is intubated in the ED for airway protection and undergoes massive blood transfusion for hemorrhagic shock. He is admitted to the ICU. Which of the following reduces the risk of delirium in the ICU?

- A. Benzodiazepine-induced coma
- B. Mechanical ventilation
- C. Polytrauma
- D. Metabolic acidosis
- E. Sedation with dexmedetomidine

Answer: E

Explanation:

A systematic review identified eleven risk factors for developing delirium in the ICU. These included age, dementia, hypertension, poly-trauma, emergency surgery prior to ICU admission, sedative-induced coma, delirium on the day prior, use of mechanical ventilation, metabolic acidosis, multi-organ failure and APACHE II score. Factors that were clearly associated with reduction in delirium were dexmedetomidine. It is unclear if this was a physiological effect of the drug itself or because dexmedetomidine use was associated with less benzodiazepine use.

Question: 724

A 65-year-old man with a history of hypertension and hyperlipidemia is found down at home by his son at 6 p.m. after he does not show up at his granddaughter's birthday. He was last seen; vital signs are blood pressure (BP) 178/92 mmHg, heart rate (HR) examination, the HR is irregularly irregular. He is awake but aphasic with right lower facial droop and flaccid right arm. His initial National Institutes of Health Stroke Scale (NIHSS) score is 18. A noncontrast CT scan of the brain shows loss of grey-white matter differentiation in the left frontal lobe and hypoattenuation of CT angiogram of the head shows an occlusion of the first segment of the middle cerebral artery (MCA); and perfusion CT estimates an infarct size of 28 mL. At baseline, the patient is independent with next step in management?

- A. Start aspirin 325 mg. Patient is not eligible for either intravenous
- B. Initiate IV thrombolysis with alteplase based on neuroimaging
- C. Proceed with endovascular thrombectomy based on mismatch
- D. Initiate IV thrombolysis with alteplase followed by endovascular
- E. Need to calculate the ratio of the volume of ischemic tissues on

Answer: C

Explanation:

The patient is out of 4.5-hour time window for IV thrombolysis. However, he is a candidate for endovascular thrombectomy based on the recent DWI or CTP Assessment with Clinical Mismatch in the Triage of Wake-Up and Late Presenting Strokes Undergoing Neurointervention with Trevo (DAWN) trial. In this trial, patients with occlusion of the intracranial internal carotid artery (ICA) or proximal middle cerebral artery (MCA) who had last been known to be well 6 to 24 hours earlier and showed evidence of salvageable brain tissue were randomly assigned to thrombectomy plus standard care (the thrombectomy group) or to standard care alone (the control group). Enrolled patients had good premorbid baseline defined as modified Rankin scale (mRS), 0 to 1. Infarct volume was assessed on diffusion-weighted imaging (DWI) sequence of brain MRI or perfusion CT scan using the automated RAPID software. Presence of salvageable brain tissue was based on a mismatch between severity of their neurological deficits and the volume of infarcted brain, and not the ratio of the volume of ischemic tissue at risk to infarct volume: DAWN trial measured utility-weighted mRS at 90 days as the primary outcome. In contrast to mRS, a lower score in utility-weighted mRS indicates a better outcome (ranges from 0 = death to 10 = no deficits). At 90 days, the utility-weighted mRS was 5.5 for the thrombectomy group compared to 3.4 in the control group, and 49% of patients in the thrombectomy group achieved functional independence versus only 13% in the control group. These translate to number-to-treat (NTT) of 2 and 2.8 for less disability and functional independence at 90 days, respectively. Rate of procedure-related complications was very low. Serious adverse events including mortality and stroke-related death at 90 days as well as and symptomatic intracerebral hemorrhage (ICH) were similar between the two groups.

Question: 725

A 72-year-old woman with a past medical history of diabetes, hypertension, hyperlipidemia, and atrial fibrillation on warfarin presents to the ED with a new severe dysarthria, left-sided weakness, and neglect (National Institutes of Health Stroke Scale [NIHSS] score 17). Her vital signs are blood pressure (BP) 169/83 mmHg, heart rate (HR) 87/min, respiratory rate (RR) 18/min, and SpO<sub>2</sub> 93%. She was last seen 12 hours prior to presentation. MRI of the brain shows an acute stroke in the territory of the right middle cerebral artery (MCA) without evidence of intracranial hemorrhage. Infarct volume on diffusion-weighted imaging (DWI) is measured 48 mL; and volume of ischemic tissue on MRI perfusion scan is calculated as 110 mL. Magnetic resonance angiography (MRA) treatment for her stroke?

- A. Start intravenous (IV) alteplase infusion based on clear evidence
- B. Start heparin drip with a goal partial thromboplastin time (PTT)
- C. Start warfarin for secondary stroke prevention
- D. Immediate endovascular thrombectomy
- E. Start aspirin 325 mg

Answer: D

Explanation:

The patient is not a candidate for IV alteplase as she presents out of 4.5-hour treatment time window. However, she is a candidate for endovascular thrombectomy. In DEFUSE 3 trial, patients 6 to 16 hours after they were last known to be well who had a proximal MCA or internal carotid artery (ICA) occlusion, an initial infarct size of less

than 70 mL, and a ratio of the volume of ischemic tissue on perfusion imaging to infarct volume of 1.8 or more were randomly assigned to thrombectomy plus standard medical therapy (thrombectomy group) or standard medical therapy alone (control group). Thrombectomy was associated with better functional outcomes (odds ratio, 2.77) and lower mortality (14% vs. 26% in the control group) without a difference in symptomatic intracranial hemorrhage or serious adverse events.

Question: 726

A 70-year-old man is admitted to the neurointensive care unit for aneurysmal subarachnoid hemorrhage (SAH). He is intubated on mechanical ventilation. On day 3 of admission, he develops septic shock. He is fluid resuscitated and broad-spectrum antibiotics with early and aggressive treatment, 12 hours later, he remains hemodynamically unstable necessitating escalating doses of norepinephrine ( $>1$  mg per hour) to maintain a systolic blood pressure. Which intervention has been shown to decrease vasopressor requirement and mortality in septic shock?

- A. Switch antibiotics to linezolid plus meropenem
- B. Add vasopressin infusion
- C. Add intravenous (IV) infusion of hydrocortisone 200 mg per day
- D. Add oral fludrocortisone 50 mcg daily
- E. Add IV hydrocortisone 50 mg 4 times a day plus fludrocortisone
- G. pressure (SBP)  $>90$  mmHg

Answer: E

Explanation:

Two recent trials (ADRENAL and APROCCHSS) studied the effects of adjunctive glucocorticoid therapy and glucocorticoid + mineralocorticoid therapy in patients with septic shock. In the former study, 3,800 patients with septic shock who were undergoing mechanical ventilation were randomized to receive hydrocortisone (at a dose of 200 mg per day) or placebo for 7 days (or until death or discharge from the ICU). Although patients in the hydrocortisone group had faster resolution of shock (3 days vs. 4 days in the control group), no significant between-group difference was found in mortality. In APROCCHSS, the effect of hydrocortisone plus fludrocortisone in patients with septic shock was compared to the placebo. Mortality was significantly lower in the hydrocortisone-plus-fludrocortisone group than in the placebo group at ICU and hospital discharge, and at 90 and 180 days. In addition, hydrocortisone plus fludrocortisone therapy increased the number of vasopressor-free and organ-failure-free days without increasing the rate of serious adverse events except for hyperglycemia.



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