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This quiz is being published on behalf of the Education Committee of the SNACC.
1. 67/Y old woman with a Past Medical History of Hypertension, Diabetes Mellitus, NSTEMI (cardiac cath four years back with no obstruction, on Aspirin), breast cancer s/p L mastectomy and chemotherapy, last round 3 years ago, and tobacco use who presented to ED with sudden onset 10/10 frontal headache that lasted 30 minutes with associated bilateral hearing loss. CT scan is significant for diffuse SAH in suprasellar cistern and bilateral sylvian fissures. All the following are effects of a subarachnoid hemorrhage EXCEPT-

A. **Raised intracranial pressure**
B. **Impaired autoregulation.**
C. **Impaired cerebrovascular response to carbon-dioxide**
D. **Normal perfusion pressure breakthrough**

Go to Q 2
A. Raised intracranial pressure (ICP)

ICP increases rapidly after a Subarachnoid hemorrhage and may approach the levels of the systemic blood pressure. This phase lasts minutes and is thought to limit the amount of blood leakage through the ruptured aneurysm. With recurrent rupture of the aneurysm, ICP increases further from mass effect, cerebral edema, or hydrocephalus due to a blocked aqueduct.

Ref: Cottrell and Young's Neuroanesthesia; Ryan Pong Arthur M. Lam; Chapter 13, 218-246
Patients with SAH have an impairment of auto-regulation and a rightward shift in the lower limit of auto-regulation. A new neurologic deficit can occur with a decrease in blood pressure and a subsequent reversal of the deficit with a pharmacologic increase in blood pressure. Hence induced hypotension in these patients can be deleterious.
C. Impaired cerebrovascular response to carbon-dioxide

Impaired cerebrovascular response (CVR) to hyperventilation is frequent after SAH, particularly in patients with a poor clinical grade. Loss of normal CVR identifies patients at high risk for delayed cerebral ischemia.

Normal perfusion pressure breakthrough (NPPB) is a term used in situations of resection of arteriovenous malformations of the brain. The intra-operative appearance of diffuse bleeding from the operative site or brain swelling in the postoperative period have been attributed to NPPB. NPPB is attributed to cerebral hyperemia due to repressurization of previously hypotensive regions. This is not present in SAH.
2. All the following are systemic effects of a subarachnoid hemorrhage **EXCEPT**-

A. Hypervolemia
B. Hyponatremia
C. Pulmonary edema
D. EKG changes

Go to Q3
The intravascular volume status has been found to be abnormally low in 36% to 100% of patients with SAH, and the level of hypovolemia correlates with the clinical grade. The reasons for hypovolemia are multifactorial and probably include bed rest, supine diuresis, negative nitrogen balance, decreased erythopoiesis, and iatrogenic blood loss. Hypovolemia may exacerbate vasospasm and is associated with cerebral ischemia and infarction and hence hypervolemia was considered a therapeutic option in the past.
B. Hyponatremia

The etiology of hyponatremia is still a matter of debate. The syndrome of inappropriate antidiuretic hormone secretion has been implicated, treatment for which consists of fluid restriction. However, the preponderance of data now suggests that the hyponatremia is related to release of natriuretic peptides—which has been referred to as cerebral salt wasting syndrome. Hyponatremia is common in subarachnoid hemorrhage and is associated with longer length of stay, but not increased mortality.
C. Pulmonary edema

It is estimated that up to 23% of patients with SAH develop some form of pulmonary edema, with 2% to 8% being attributed to neurogenic pulmonary edema. Of those patients who develop neurogenic pulmonary edema, mortality has been reported as high as 50%. The clinical presentation of neurogenic pulmonary edema includes dyspnea, tachypnea, tachycardia, cyanosis, pink frothy sputum, and/or crackles on auscultation of lung fields. Radiographic imaging reveals bilateral diffuse alveolar infiltrates. The pathophysiology behind neurogenic pulmonary edema is not fully understood but may be related to excess sympathetic nervous system activity.

Electrocardiographic (ECG) abnormalities occur in 40% to 100% of patients with SAH. These abnormalities include sinus bradycardia, sinus tachycardia, atrioventricular dissociation, and bradycardia-tachycardia to more serious and potentially life-threatening rhythms such as ventricular tachycardia and fibrillation. A prolonged QT interval may occur in 20% to 41% of patients predisposing them to dangerous ventricular arrhythmias. Atrial arrhythmias including fibrillation and flutter occur in 4% of patients and have been found to be associated with a higher risk of severe disability and death.

3. Which of the following is true regarding the myocardial effects of subarachnoid hemorrhage?

A. Cardiac troponins(CKMb) are usually not increased.
B. Akinesis of Left ventricular apex on ECHO.
C. Prognosis for reversibility of ventricular dysfunction is poor.
D. Surgery should be delayed in a situation of EKG changes.

Go to Q 4
A. Cardiac troponins (CKMb) are usually not increased.

After SAH, damage to the myocardium can be indicated by an increase in circulating levels of cardiac Troponin I (cTi) found in 17% to 68% of patients although the elevation is much less than that related to myocardial infarction. Elevation of cTi has been found to be associated with regional wall motion abnormalities and left ventricular dysfunction.

Takotsubo cardiomyopathy has been reported in aneurysmal subarachnoid hemorrhage and typically, ventricular dysfunction secondary to SAH is associated with involvement of LV (left ventricle) apex and sparing of the basal segments. The mechanism of myocardial dysfunction is thought to be due increased release of catecholamines from the myocardium.
C. Prognosis for reversibility of ventricular dysfunction is poor.

The prognosis of SAH-induced ventricular dysfunction is good and generally reversible.

D. Surgery should be delayed in a situation of EKG changes

As with any surgical procedure, the decision to proceed with surgery should be based on a risk-benefit analysis. Since the EKG changes reflect the severity of neurologic damage, and have not shown to contribute materially to perioperative morbidity or mortality, the decision to operate should not be influenced by these EKG changes.

4. All the following could be useful to prevent early re-bleeding of aneurysms EXCEPT;

A. Hypervolemia.
B. Systolic pressure reduction of < 140mHg.
C. Early aneurysm obliteration (24-48 hours).
D. Antifibrinolytic usage.
Hypervolemic and hypertensive therapy is used when vasospasm occurs but can potentially contribute to early re-bleeding if used inadvertently. Other concerns are worsening of cerebral edema and pulmonary edema with hypervolemia.
B. Systolic pressure reduction of < 140mHg

Re-bleeding may be attributable to uncontrolled hypertension. A titrable agent like nicardipine is preferable. Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATACH-II), a multi-center, randomized, controlled, phase III trial, has enrolled 1000 subjects and will evaluate intensive systolic blood pressure reduction to 140 mm Hg using nicardipine compared with 180 mm Hg in the control arm.
Early aneurysm repair should be undertaken when possible and reasonable to prevent rebleeding (high quality of evidence, strong recommendation)

Critical Care Management of Patients Following Aneurysmal Subarachnoid Hemorrhage: Recommendations from the Neurocritical Care Society’s Multidisciplinary Consensus Conference. Neurocrit Care (2011) 15:211–240
D. Antifibrinolytic usage

An early, short course of antifibrinolytic therapy prior to early aneurysm repair (begun at diagnosis; continued up to the point at which the aneurysm is secured or at 72 h post-ictus, whichever is shorter) should be considered (Low Quality Evidence; Weak Recommendation).

Antifibrinolytic therapy is relatively contraindicated in patients with risk factors for thromboembolic complications (Moderate Quality Evidence; Strong Recommendation).

Patients treated with antifibrinolytic therapy should have close screening for deep venous thrombosis (Moderate Quality Evidence; Strong Recommendation).
5. The patient experiences focal neurologic deficits with a decrease in Glasgow coma scale from 11 to 9. Delayed cerebral ischemia (DCI) is attributed to the following EXCEPT

A. **Cortical spreading depolarization**
B. **Dysfunctional cerebral autoregulation**
C. **Microthrombi**
D. **Cerebral salt wasting syndrome**
Cortical spreading depolarization (CSD) is described as self-propagating tissue depolarization waves that lead to ischemia and are associated with increase in extracellular potassium levels, compromise of the blood-brain barrier, formation of vasogenic and cytotoxic edema, and change in regional cerebral blood flow. This can lead to delayed cerebral ischemia and neurologic deficit.

Defective cerebral autoregulation is seen early after SAH. Measures of dynamic cerebral autoregulation have been correlated with the development of angiographic vasospasm and, more critically, to the development of DCI.
C. Microthrombi

On aneurysm rupture, an inflammatory reaction is initiated involving reduced nitric oxide levels and increased fibrin and platelet aggregation leading to a hypercoagulable state. Microthrombi may form and contribute to DCI.
Cerebral salt wasting is common after SAH and cause hyponatremia. However, it does not cause delayed cerebral ischemia.