Aneurysmal subarachnoid hemorrhage complications

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A greater proportion of aneurysmal subarachnoid hemorrhage patients, are surviving their initial hemorrhagic event but remain at increased risk of a number of complications, including delayed cerebral ischemia, epilepsy, nosocomial infections, cognitive impairment, shunt-dependent hydrocephalus, and shunt related complications [1].

Intracranial complications including delayed cerebral ischemia (vasospasm), aneurysm rebleeding, and hydrocephalus form the targets for initial management. However, the extracranial consequences including hypertension, hyponatremia, and cardiopulmonary abnormalities can frequently arise during the management phase and have shown to directly affect clinical outcome.

Although the intracranial complications of SAH can take priority in the initial management, the extracranial complications should be monitored for and recognized as early as possible because these complications can develop at varying times throughout the course of the condition. Therefore, a variety of investigations, as described by this article, should be undertaken on admission to maximize early recognition of any of the extracranial consequences. Furthermore, because the extracranial complications have a direct effect on clinical outcome and can lead to and exacerbate the intracranial complications, monitoring, recognizing, and managing these complications in parallel with the intracranial complications is important and would allow optimization of the patient's management and thus help improve their overall outcome [2].

Intracranial hemorrhage

Aneurysmal subarachnoid hemorrhage is complicated by intracerebral hemorrhage in 20—40 %, by intraventricular hemorrhage in 13-28%, and by subdural blood in 2-5% (usually due to posterior communicating aneurysm when over convexity, or distal anterior intracerebral artery (DACA) aneurysm with interhemispheric subdural).

The intracranial effects of aSAH causing death and disability are from vasospasm, direct effects of the initial bleed, increased intracranial pressure (ICP) and rebleeding [3].

Fever is a common occurrence (70%) especially in poor grades, contributes to adverse outcome and may not always respond to conventional treatment.

Persistent hyperglycemia (>200 mg/dl for >2 consecutive days) increases the likelihood of poor outcome after aSAH.

Management of patients following aSAH includes four major considerations:

(1) prediction of patients at highest risk for development of DCI,

(2) prophylactic measures to reduce its occurrence,

(3) monitoring to detect early signs of cerebral ischemia,

(4) treatments to correct vasospasm and cerebral ischemia once it occurs [4].

Brain injury

Brain injury following aneurysmal subarachnoid hemorrhage.

Vasospasm

see Vasospasm after aneurysmal subarachnoid hemorrhage.

Delayed cerebral ischemia

see Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage

Rebleeding

see Aneurysm rebleeding

Non-convulsive status epilepticus

Development of Non-convulsive status epilepticus in patients suffering SAH might correlate with poor prognosis. Even when medical treatment is successful and no EEG abnormalities are detected, the long-term outcome remains poor [5].

Pulmonary complications

Subarachnoid hemorrhage (SAH) is often accompanied by pulmonary complications, which may lead to poor outcomes and death.

Sympathetic activation of the cardiovascular system in aneurysmal subarachnoid hemorrhage not only triggers the release of atrial and brain natriuretic peptides it can also lead to increased pulmonary venous pressures and permeability causing hydrostatic pulmonary edema [6].

see Neurogenic pulmonary edema.

Cardiac complications

see Cardiac Complications After Subarachnoid Hemorrhage

Acute kidney injury

Acute kidney injury

Hyponatremia

see Hyponatremia after aneurysmal subarachnoid hemorrhage

Hypokalemia

Hypokalemia is a common electrolyte disorder in the intensive care unit. Its cause often is complex, involving both potassium losses from the body and shifts of potassium into cells.

We present a case of severe hypokalemia of sudden onset in a patient being treated for subarachnoid hemorrhage in the surgical intensive care unit in order to illustrate the diagnosis and management of severe hypokalemia of unclear cause. The patient received agents that promote renal potassium losses and treatments associated with a shift of potassium into cells. Ibanez et al. outline the steps in diagnosis and management, focusing on the factors regulating the transcellular distribution of potassium in the body [7].

Hydrocephalus

see Hydrocephalus after aneurysmal subarachnoid hemorrhage.

Intraventricular hemorrhage

The clinical outcome after aneurysm rupture is at least in part determined by the severity of IVH. Knowledge of the effect of IVH may help guide physicians in the care of patients with aneurysmal bleeding [8].

Cognitive disorder

see Cognitive disorder after subarachnoid hemorrhage.

Neuropsychiatric disturbance

Aneurysmal subarachnoid hemorrhage neuropsychiatric disturbance.

Deep-Vein Thrombosis

Overall rates of VTE (Deep-Vein Thrombosis Deep-vein thrombosis or PE), Deep-vein thrombosis, and PE were 4.4%, 3.5%, and 1.2%, respectively. On multivariate analysis, the following factors were associated with increased VTE risk: increasing age, black race, male sex, teaching hospital, congestive heart failure, coagulopathy, neurologic disorders, paralysis, fluid and electrolyte disorders, obesity, and weight loss. Patients that underwent clipping versus coiling had similar VTE rates. VTE was associated with pulmonary/cardiac complication (odds ratio [OR] 2.8), infectious complication (OR 2.8), ventriculostomy (OR 1.8), and vasospasm (OR 1.3). Patients with VTE experienced increased non-routine discharge (OR 3.3), and had nearly double the mean length of stay ($p<0.001$) and total inflation-adjusted hospital charges ($p<0.001$). To our knowledge, this is the largest study evaluating the incidence and risk factors associated with the development of VTE after aSAH. The presence of one or more of these factors may necessitate more aggressive VTE prophylaxis [9].

Short course (<48h) administration of EACA in patients with aneurysmal subarachnoid hemorrhage is associated with an 8.5 times greater risk of Deep-Vein Thrombosis (Deep-vein thrombosis) formation [10].

Routine compressive venous Doppler ultrasonography is an efficient, noninvasive means of identifying Deep-Vein Thrombosis (Deep-vein thrombosis) as a screening modality in both symptomatic and asymptomatic patients following aneurysmal SAH. The ability to confirm or deny the presence of Deep-vein thrombosis allows one to better identify the indications for chemoprophylaxis. Prophylaxis for venous thromboembolism in neurosurgical patients is common. Emerging literature and anecdotal experience have exposed risks of complications with prophylactic anticoagulation protocols. The identification of patients at high risk-for example, those with asymptomatic Deep-vein thrombosis-will allow physicians to better assess the role of prophylactic anticoagulation [11].

Deep-Vein Thrombosis (Deep-vein thrombosis) formation most commonly occurs in the first 2 weeks following aSAH, with detection in a cohort peaking between Days 5 and 9. Chemoprophylaxis is

associated with a significantly lower incidence of Deep-vein thrombosis [12].

Prevention

Patient should be ideally monitored in the NICU for at least 1st 24 h after surgery. Anticonvulsants, osmotherapy and nimodipine must be continued. Hydrocephalus, vasospasm, seizures, and electrolyte disturbances can occur necessitating close observation and prompt management. One of the major challenges in the management of aSAH is identifying potential or ongoing perfusion deficits. Ischemic insults can occur following ictus, or due to raised ICP, hypotension and vasospasm. Early identification and appropriate treatment of postictal intracranial (ICP, TCD flow velocities) and cardiovascular (cardiac output, ECG, BP, CVP) changes is possible in dedicated NICU and is crucial for improving outcomes. Heuer et al. observed that raised ICP (>20 mmHg) occurred in >50% of patients after aSAH and was associated with poor outcomes. Factors associated with raised ICP included poor clinical and radiological grades of aSAH, intraoperative brain swelling, parenchymal and intraventricular bleed and rebleeding.

Seizure

see Seizure after aneurysmal subarachnoid hemorrhage.

Cytotoxic Lesions of the Corpus Callosum

Cytotoxic Lesions of the Corpus Callosum.

Nonocclusive mesenteric ischemia

Koizumi et al. evaluated the incidence of NOMI in patients with subarachnoid hemorrhage (SAH) due to ruptured aneurysms, and they present the clinical characteristics and describe the outcomes to emphasize the importance of recognizing NOMI.

Observations: Overall, 7 of 276 consecutive patients with SAH developed NOMI. Their average age was 71 years, and 5 patients were men. Hunt and Kosnik grades were as follows: grade II, 2 patients; grade III, 3 patients; grade IV, 1 patient; and grade V, 1 patient. Fisher grades were as follows: grade 1, 1 patient; grade 2, 1 patient; and grade 3, 5 patients. Three patients were treated with endovascular coiling, 3 with microsurgical clipping, and 1 with conservative management. Five patients had abdominal symptoms prior to the confirmed diagnosis of NOMI. Four patients fell into shock. Two patients required emergent laparotomy followed by second-look surgery. Four patients could be managed conservatively. The overall mortality of patients with NOMI complication was 29% (2 of 7 cases).

NOMI had a high mortality rate. Neurosurgeons should recognize that NOMI can occur as a fatal complication after SAH [13].

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