EM CASE OF THE WEEK.

BROWARD HEALTH MEDICAL CENTER DEPARTMENT OF EMERGENCY MEDICINE



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September 2016 | Vol 3 | Issue 4

Subarachnoid Hemorrhage (SAH)

A 55-year-old male with a past medical history of autosomal dominant polycystic kidney disease, coronary artery disease, hypertension and smoking 1 pack per day for the last 30 years presents to the ED with a sudden and severe headache. Patient described it as the worst headache of his life. The headache presented in the bifrontal area and within the following minutes it spread over his head and neck. Patient denied any symptoms of nausea, vomiting, vision changes, or loss of consciousness. On physical exam, he had mild nuchal rigidity, blood pressure of 163/74 mm Hg, pulse rate of 94/min and regular, and respiration rate of 17/min. Laboratory results were as follows: platelet count 195,000/uL, INR 0.9, serum creatinine 1.3 mg/dL. CT of the head without contrast was normal. What is the next best step in management?

- A. CT brain with contrast
- B. Lumbar puncture
- C. Empiric treatment with vancomycin, ceftriaxone, ampicillin
- D. MR angiography of the head and neck
- E. MRI of the brain



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Ruptured aneurysm in a 46-yearold patient with ADPKD

- A CT showing diffuse subarachnoid hemorrhage and hydrocephalus
- B 3T magnetic resonance angiogram showing ruptured basilar bifurcation aneurysm
- C 3D rotational angiography confirmed the presence of a wide neck aneurysm
- D The aneurysm following treatment using endovascular coiling

EM Case of the Week is a weekly "pop quiz" for ED staff.

The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

BROWARD HEALTH MEDICAL CENTER

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September 2016 | Vol 3 | Issue 4

The correct answer is B. At presentation the patient had a sudden severe onset of headache, which is most concerning for subarachnoid hemorrhage. When subarachnoid hemorrhage is suspected and the head CT comes back negative, proceed with a lumbar puncture to determine if there are erythrocytes in the CSF. Autosomal dominant polycystic kidney disease accounts for 5%-10% of chronic kidney failure. Patients present with bilaterally enlarged kidneys composed entirely of cysts between 3 to 4 cm. Cysts arise anywhere in the nephron and compress adjacent parenchyma. Later in the disease, there is interstitial inflammation and fibrosis. About 40% of patients die of hypertensive or coronary heart disease, and about 15% from ruptured berry aneurysms in the Circle of Willis or hypertensive brain hemorrhage.

Discussion

Excluding head trauma, the most common cause of SAH is a ruptured berry aneurysm. Berry aneurysms occur in 2% of the population, with 20-30% of patients having multiple aneurysms. 90% of aneurysms occur in the anterior circulation. SAH present with a sudden, severe headache, often with transient loss of consciousness at onset. Vomiting is also common. Bleeding may affect adjacent tissue and produce focal neurologic deficits. A progressive third nerve palsy, usually involving the pupil, along with headaches suggests a posterior communicating artery aneurysm.

The initial clinical manifestations of SAH can be graded using the Hunt-Hess Scale or World Federation of Neurosurgical Societies Scale. Initial studies include CT without contrast, LP, cerebral angiography, ECG and coagulation studies. The appropriate therapy for SAH depends in part upon the severity of hemorrhage. Level of consciousness on admission, patient age, and the amount of blood on initial head computed tomography (CT) scan are the most important prognostic factors. Early identification is essential to reduce mortality and morbidity rates.



(via http://emedicine.medscape.com/article/1198462-

Emergency Department Management of SAH

Grades I or II: The emergency department is limited to diagnostic and supportive therapy. Sedation should be used judiciously. IV access should be obtained and patients' neurologic functions monitored.

Grades III, IV or V: These patients have an altered neurologic exam. Intubation of obtunded patients can help avoid aspiration. Thiopental and etomidate are the optimal induction agents in SAH during intubation. Thiopental should be used in hypertensive patients while etomidate should be used on normotensive patients. Target the pCO2:30-35 mmHg to reduced ICP. Excessive hyperventilation may be harmful to areas of vasospasm. Avoid sedation if possible; however, excessive agitation from pain can cause an increase in ICP and thus sedation may be needed in these cases.

Early intervations may be needed if herniation is suspected. Osmotic agents such as mannitol reduce ICP by 50% in 30 minutes and peak after 90 minutes. They last 4 hours. Loop diuretics, such as furosemide, also decrease ICP without increasing serum osmolality. IV steroid therapy to control brain edema is controversial and debated. Obtain neurosurgical consultation for definite treatment of subarachnoid hemorrhage. The International Subarachnoid Aneurysm Trial demonstrated improved outcomes with endovascular coiling therapy compared to surgery. However, some aneurysms have a morphology not amenable to endovascular treatment, and therefore surgery is still an important option.

For a list of educational lectures, grand rounds, workshops, and didactics please visit **BrowardER.com** and **click** on the "Conference" link.

All are welcome to attend!



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Subarachnoid Hemorrhage Classifications

Subaracimola Hemorriage Classifications		
Grade	Hunt-Hess Scale	WFNS Scale
I	Mild headache, Normal mental status No CN or motor findings	GCS 15, no motor deficit
II	Severe Headache Normal mental status No focal deficits +/- CN deficit	GCS 13-14, no motor deficit
III	Somnolent, confused Motor deficit or CN deficit	GCS 13-14, with motor deficit
IV	Depressed level of consciousness or focal deficit	GCS 7-12, with or w/o motor deficit
V	Patient posturing Comatose Flaccid	GCS 3-6, with or w/o motor deficit

Medical management of SAH should include monitoring of electrolytes and osmolality. Hyponatremia frequently develops several days after a SAH and an oral salt plus IV normal saline or IV hypertonic saline may be used. Anticonvulsants may be used until the aneurysm is treated. Blood pressure should be carefully controlled while preserving cerebral blood flow, in order to decrease the risk of re-rupture until the aneurysm is repaired.

Vasospasm is the leading cause of mortality and morbidity following initial rupture. This may develop by day 4 and continue through day 14, leading to focal ischemia and possibly stroke. Medical treatment with nimodipine (60 mg PO q4h) improves outcomes by preventing ischemic injury. Cerebral perfusion may be improved in symptomatic vasospasm by increasing mean arterial pressure with vasopressors such as phenylephrine or norepinephrine. Intravascular volume can be expanded with crystalloids. This is referred to as "Triple-H therapy" (hypertension, hemodilution, and hypovolemic). If vasospasm persists despite optimal medical therapy, intra-arterial vasodilators and angioplasty of the cerebral vessels can be effective.

Take Home Points

- Excluding head trauma, the most common cause of SAH is a ruptured berry aneurysm.
- About 15% of patients with ADPKD die from ruptured berry aneurysms in the Circle of Willis or hypertensive brain hemorrhage.
- Vasospasm is the leading cause of mortality and morbidity following initial berry aneurysm rupture.
- Vasospasms may develop by day 4 and continue through day 14, leading to focal ischemia and possibly stroke.
- Medical treatment with nimodipine improves outcomes of vasospasms by preventing ischemic injury.



ABOUT THE AUTHOR

This month's case was written by Raul Fernandez. Raul is a 4th year medical student from LECOM. He did his emergency medicine rotation at BHMC in September 2016. Raul plans on pursuing a career in internal medicine after graduation.

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