



A Female with a Severe, Sudden Headache

Marc Dorfman, MD, FACEP, MACP

EM Residency Program Director
Department of Emergency Medicine
Resurrection Medical Center
Chicago, IL

Case Presentation

In February of 2004, the Chicago Fire Department responded to the home of a 52-year-old female with Altered Mental Status. EMS reported that the patient developed a severe headache of sudden onset. She then took aspirin to relieve the headache, started slurring her speech, and then collapsed. Pre-hospital vitals were BP-210/100, HR-48, RR 20, glucose 141, and Glasgow Coma scale 7.

The patient presented to the ED unresponsive, opening eyes spontaneously, and a depressed gag reflex. Her vitals were BP-195/99, P-52, and RR-16. There was no apparent trauma. The pupils were 2 mm, reactive, and deviated to the left. She did not respond to verbal stimuli; however she did withdraw to painful stimuli. The patient was hyper-reflexive on the left side. The remainder of the physical exam was within normal limits. Per the patient's husband, the patient had no previous medical or surgical history. She had no history of drugs or alcohol use. The patient was not on any medication except for the aspirin she had taken that day, and she did not have any allergies. The husband stated the patient had been in good health until the sudden onset of symptoms.

What is the most likely diagnosis in this patient? What is her optimal ED Rx?

Key Clinical Questions and Learning Points

What are the most common etiologies, locations, and symptoms of ICH?

Hypertension is a common etiology of ICH whether it is essential, the result of eclampsia, or caused by the use of sympathomimetics. The six main locations of hypertensive ICH are: putamen, thalamus, lobar, caudate, brainstem, and cerebellar. Amyloid deposition is another common cause of ICH. Other etiologies include aneurysm, trauma, vascular malformation (avm, cavernous hemangiomas), tumor, coagulopathy, and vasculitis. The symptoms and clinical signs of ICH are determined by the location and size of the ICH. The most common symptoms include alteration of consciousness (50%), nausea and vomiting (50%), headache (40%), seizures (7%), and focal neurological deficits. The neurological deficit may help diagnose the location of the ICH:

Putamen-Contralateral hemiparesis, contra lateral sensory loss, contra lateral conjugate gaze paresis, aphasia, neglect

Thalamus-Contra lateral sensory loss, contra lateral hemiparesis, gaze paresis, miosis, aphasia or confusion

Lobar-Contralateral hemiparesis or sensory loss, contra lateral gaze paresis, aphasia, neglect, or apraxia

Caudate nucleus-contra lateral hemiparesis, contra lateral gaze paresis, confusion

Brain stem-facial weakness, decreased level of consciousness, gaze paresis, miosis, autonomic instability

Cerebellum-Ataxia, ipsilateral facial weakness, ipsilateral sensory loss, gaze paresis, miosis, decreased level of consciousness

The best diagnostic test in the Emergency Department is a CT scan.

What are the goals of BP management, with which antihypertensive?

The goals of blood pressure management should be based on individual factors such as previous hypertension, and increased intracranial pressure. In treating blood pressure the physician is balancing two opposing theories. On the one hand elevated blood pressure needs to be lowered to decrease the ongoing bleeding of small arterioles which is increasing the volume of the hemorrhage, and possibly worsening the outcome. Conversely, overaggressive treatment of blood pressure may decrease cerebral perfusion pressure and lead to increased brain injury. Definite recommendations from the AHA are (1) keep the mean arterial pressure below 130 mm Hg in persons with a history of hypertension. (2) If there is an ICP monitor cerebral perfusion pressure (MAP-ICP) should be kept > 70 mm Hg. Furthermore, it is recommended to avoid hypotension. If the Systolic BP is less than 90, start pressors. The most commonly used agents are Labetolol and Nitroprusside. Nitroprusside is a vasodilatory agent and can theoretically increase cerebral blood flow and thereby intracranial pressure. This had not been demonstrated in a clinical study. There are no prospective randomized placebo-controlled studies in which investigators have compared the effects of lowering blood pressure on immediate clinical status.

What are the optimal strategies for managing ICP?

ICP is a major contributor to mortality after ICH, so controlling it is considered essential. The therapeutic goal for elevated ICP is an ICP < 20 mm Hg, and a cerebral perfusion pressure > 70 mm Hg. Managing elevated ICP can be done in several way including intubation, osmotherapy, blood pressure management, and surgery. Elevating the head of the bed to 30 degrees is one of the simplest ways to decrease ICP. The patient may be intubated for airway protection and to monitor the PCO_2 which should be kept between 30-35. Although lowering the PCO_2 to improve outcome has not definitely been proven, it acutely lowers ICP by cerebral vasoconstriction. Sedation and paralytic agents may be added to an intubated patient. These will prevent the patient from increasing intrathoracic and venous pressures associated with coughing, suctioning, or “bucking” the vent and raising ICP. Nondepolarizing agents such as Vecuronium or Pancuronium are the preferred agents. Osmotic diuresis with Mannitol should be given. If the above measures are unsuccessful, an induced barbiturate coma or therapeutic hypothermia may be tried. Both work by reducing cerebral blood flow and volume. Barbiturate coma and hypothermia should be viewed as last ditch efforts and not part of a standard algorithm. If there is the expertise available, an ICP

monitor should be placed to directly measure the ICP. It is recommended that an ICP monitor should be placed in patients with a GCS of < 9, and all patients whose condition is thought to be deteriorating secondary to an elevated ICP.

What other ICH treatments are available to the ED physician?

Fluid management is important in the setting of ICH. The goal is euvolemia, so the patient should have a Foley catheter, and isotonic crystalloid solutions should be used. Electrolytes should be checked and replaced as needed. Acidosis and alkalosis should be corrected. Seizures may result from damaged neurons, therefore prophylactic anti-seizure medication should strongly be considered. This is especially true in patients with a lobar hemorrhage in which the rate of seizures is 35%. Fever management is important in patients with ICH. Elevated temperatures can increase the degree of ischemic injury. Previous studies have demonstrated increased morbidity and mortality in patients with stroke and sustained temperature elevation. The goal is to maintain normal body temperature. This is best done with Acetaminophen or a cooling blanket.

Which ICH patients need surgery?

Surgical candidates include: (1) Cerebellar hemorrhage > 3 cm who are deteriorating or who have brain stem compression and hydrocephalus from ventricular obstruction, (2) Vascular malformation if the lesion is surgically accessible and the patient has a chance for a good outcome, and (3) young patients with a moderate or large lobar hemorrhage who are clinically deteriorating. Patients with small hemorrhages <10 cm³ or minimal neurological deficits may be managed medically. Patients with a GCS less than 4 should be treated medically because they either die or have extremely poor functional outcome that cannot be improved by surgery.

How does hemorrhage volume growth over time effect mortality?

The old concept of ICH is that the bleeding was completed in a few minutes. Recently this concept has changed. It is now felt that bleeding occurs over several hours. It is the continued bleeding that may lead to poor neurological outcome. A few studies have been published regarding increases in hemorrhage volume with a prospective study by Brott; Early Hemorrhage Growth in Patients with Intracerebral hemorrhage: published in the journal Stroke in 1997. The article found that hemorrhages grew about 33% in at least 38% of the patients in the first 24 hours. The conclusion was that substantial early hemorrhage growth in patients with ICH is common and associated with neurological deterioration. There have been other studies with similar results.

What are the new therapies being tested for this disease process?

Annually, more than 20,000 people in the United States die of an intracranial hemorrhage, with the 30 day mortality rate about 50%. The American Heart Association published practice guidelines in 1999 for the management of ICH. In the guidelines they emphasized a desperate need for randomized controlled trials. With the emergence of the concept of increased volume of hemorrhage as a cause of neurological deterioration in ICH, a therapy to decrease bleeding is being studied. There is a phase II study showing that Factor rVIIa (commonly used in Hemophilia) is safe to give in the setting of ICH, and may limit bleeding and therefore the size of an ICH. A phase III trial is needed to elucidate if Factor rVIIa has a role in the management of ICH.

Patient Case Outcome

Emergency Department Course

10 minutes after arrival the patient was felt to be getting worse. She lost her gag reflex, and stopped moving. The patient underwent rapid sequence intubation with Lidocaine 100mgs, Etomidate 20 mgs and Succinylcholine 100 mgs. The patient was then paralyzed with Pancuronium 7 mgs. Following ET tube confirmation the patient was sent to the CT scanner.

The patient's CT scan showed a large left temporal-parietal hemorrhage with intraventricular and subarachnoid hemorrhage. There was a 5 mm midline shift and cerebral edema. Upon return from CT scan the patient's BP-207/109, P-70, and RR-13. The patient was started on a Nipride drip. The patient was given Mannitol 150 ccs of a 20% solution and Decadron 10 mg IV. Neurosurgery was consulted and they saw the patient in the ED. Neurosurgery placed an extraventricular drain in the ED without a change in the patient's status.

Patient Outcome

Neurosurgery discussed treatment options with the family and the decision was made to take the patient to the operating room for clot evacuation. Because of the size of the hemorrhage, the patient was given a poor prognosis. Prior to surgery, the patient's BP-119/79 and the pulse was 94. On Post Op Day #1 brain wave studies failed to show activity. The patient was declared dead, and extubated. The patient's organs were donated.