# Aneurysmal Subarachnoid Hemorrhage: It's more than a Headache

*Quick, accurate diagnosis and treatment of patients with ruptured brain aneurysms and subarachnoid hemorrhage can save lives. Here's how to determine whether your client received the appropriate --and most responsive--care.*

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Every year, more than 30,000 people in the United States suffer a ruptured brain aneurysm resulting in a life-threatening subarachnoid hemorrhage--bleeding around the brain.[1] Thousands of these people will go promptly to their local emergency room or family physician and report that they are experiencing the worst headache of their lives. Unfortunately, many will be discharged without receiving the correct diagnosis or the emergency medical treatment they desperately need.

Failure to diagnose is the most common negligence in the treatment of patients with aneurysmal subarachnoid hemorrhage,[2] and this failure may be as high as 53 percent in patients visiting the emergency room for the first time.[3] Physicians fail to obtain an adequate history, perform a thorough neurological exam, or understand the significance of the history and physical exam findings. Less often, but still with troubling regularity, negligence involves failure to obtain or accurately interpret diagnostic tests.[4]

Delay in diagnosis and treatment has dire consequences. In some cases, even minimal delay can mean the difference between life and death.

To adequately evaluate a potential malpractice case involving the failure to timely diagnose and treat a patient with a ruptured brain aneurysm, you must first gain a basic understanding of brain aneurysms, how they rupture, and the events that follow.

Aneurysms are ballooned areas on arteries. They are often referred to as berry, saccular, or congenital (existing at birth) aneurysms. The most likely cause is a weak area of the artery wall due to a congenital defect combined with degenerative changes that further weaken the tissue.[5]

Aneurysms are typically located on large arteries at the base of the brain and usually occur at branching points. If they stretch and ultimately rupture, arterial pressure forces blood to leak from the aneurysm into the subarachnoid space--the area between the arachnoid membrane and the pia mater, a membrane adherent to the brain. The blood then quickly spreads into the cerebrospinal fluid surrounding the brain and spinal cord.

This results in a sudden increase in intracranial pressure and chemical irritation of the meninges, which leads to a sudden, explosive headache.[6] Headaches caused by ruptured aneurysms may be associated with physical exertion in up to 20 percent of patients.[7]

Significantly more women than men, and more blacks than whites, suffer ruptured brain aneurysms.[8] The risk of rupture increases with age and is increased in people with a family history of intracranial aneurysms.[9] It also increases dramatically during the third trimester of pregnancy[10] and contributes to between 6 percent and 25 percent of maternal deaths.[11] Lifestyle risk factors include smoking, uncontrolled hypertension, heavy alcohol use, and cocaine or amphetamine abuse.[12]

Cerebral aneurysms are more common in people with fibromuscular dysplasia (a disease process which causes narrowing of the arteries) and several hereditary diseases.[13] Aneurysms

may occur as multiples and following the treatment of one aneurysm, another aneurysm that was not previously recognized or one that was known but considered too insignificant to treat may bleed.

Up to 70 percent of patients will experience a sentinel (warning) leak before suffering a larger hemorrhage. Often these patients have a headache but no other signs or symptoms. Half of the patients with sentinel leaks seek immediate attention, but up to 60 percent of them are misdiagnosed and later suffer a larger hemorrhage. If the sentinel leak is diagnosed and treated promptly, a more serious hemorrhage and the associated significant complications can be prevented.[14]

## From triage to treatment

Once you have an understanding of intracranial aneurysms, the next step is a thorough review of your potential client's medical records, typically those from an emergency room and/or primary care physician. Make a list of every sign or symptom of a ruptured brain aneurysm that the prospective client exhibited or reported.

Look for complaints of a severe headache with sudden onset, nausea, vomiting, insensitivity to light, or neck pain. Exam findings may include neck stiffness, an altered state of consciousness (for example, drowsiness, restlessness, or agitation), neurological impairments such as weakness or paralysis, visual problems, abnormal eye movements, or paralysis of eye movements.[15] If an examination of the back of the eye, a funduscopic examination, was conducted, it may reveal retinal or preretinal hemorrhages.[16] About 50 percent of patients with aneurysmal hemorrhage will have no alteration in consciousness, and the majority will lack focal neurological signs.[17]

After a person suffers a hemorrhage, their electrocardiogram may suggest acute myocardial infarction. This may complicate the patient's evaluation and delay the correct diagnosis while an acute heart attack is being ruled out.

In evaluating the case, develop a minute-to-minute chronology of every action taken by the medical professionals, from triage, to initial evaluation, to diagnostic testing, to definitive diagnosis, and through treatment. The exact timing of these actions or any inaction may be a critical factor in deciding whether to accept the case. A delay in treatment that causes a patient's condition to deteriorate may be a significant breach of the standard of care by the attending staff.

The next step in the evaluation process is learning the applicable standard of care for physicians involved in diagnosing and treating this condition. Be alert to the fact that patient complaints of headache are exceedingly common, accounting for about 2 million emergency room visits per year in the United States, but only 1 to 4 percent of all patients who go to the emergency room with a headache have suffered a subarachnoid hemorrhage.[18]

Because headache is such a common complaint, frontline physicians--emergency room doctors, internists, and family practice physicians--are trained to differentiate between headaches due to a hemorrhage and headaches caused by other conditions that are not life-threatening. These physicians are taught to get a detailed history from the patient or an accompanying family member, perform a comprehensive physical exam, and document both. The history should include details such as the time of headache onset; whether it was sudden or more gradual; its severity, location, and quality; the patient's activity when it started; the patient's history of headaches; the progression of the headache; and whether the headache was accompanied by loss of consciousness.

If a patient describes their headache with phrases such as "It is the worst headache ever," "I feel like my head will explode," or "It's unlike any headache I ever had before," the treating physician should suspect a subarachnoid hemorrhage. A thorough general physical exam should be conducted, including a funduscopic exam and a detailed neurological exam.[19] The physician should assess the patient's level of consciousness, orientation, cranial nerves, muscle strength, neck mobility, sensation, gait (if possible), reflexes, and cerebellar function. Changes in level of consciousness may be slight, but the physician must appreciate any alteration, either by direct observation or with information from others.

The physician should know that the patient may not have any of the physical or neurological findings discussed above. About 12 percent of patients who arrive at the emergency room with acute-onset headaches and normal neurological exams have suffered a hemorrhage.[20] Therefore, patients with sudden severe headaches should be thoroughly evaluated for subarachnoid hemorrhage even if they have no other signs or symptoms or the headache abates spontaneously or with pain medication.[21]

Once the differential diagnosis includes subarachnoid hemorrhage, diagnostic testing should immediately follow. Computerized tomography (CT) of the head, without contrast material, is the diagnostic test of choice. It is about 95 percent sensitive for detecting acute subarachnoid hemorrhage if performed within hours of aneurysm rupture. CT is noninvasive, relatively inexpensive, and it can be completed in minutes. CT sensitivity decreases to approximately 50 percent one week after rupture.[22]

When the patient is suspected of having suffered a hemorrhage but the CT scan is normal or inconclusive, a lumbar puncture should be performed to look for blood in the cerebrospinal

fluid. This is an inexpensive and low-risk procedure that can be completed and evaluated in minutes.

A diagnosis of subarachnoid hemorrhage is made on the basis of persistent red blood cells in the sampled cerebrospinal fluid. To determine that the presence of red blood cells was not caused by the lumbar puncture itself, the patient's opening cerebrospinal fluid pressure is measured, and a sample is collected in four consecutive test tubes. Findings consistent with a hemorrhage include an elevated opening cerebrospinal fluid pressure, an elevated red blood cell count that does not diminish between the first and last tube, and xanthochromia--a golden-yellow pigment in the fluid from the breakdown of red blood cells and hemoglobin.[23] Because xanthochromia can take several hours to become appreciable, patients with elevated opening cerebrospinal fluid pressure and persistently bloody fluid should be diagnosed as having suffered a hemorrhage and immediately undergo vascular imaging.

One additional analysis involves distinguishing patients with an aneurysmal hemorrhage from those with a traumatic hemorrhage. For example, a patient who arrives in the emergency room after being involved in a car crash may complain of a headache but may not remember the event or may be too drowsy to recount it. The patient may be unable to tell the physician whether a severe, sudden headache caused the crash or whether the crash caused the headache. If possible, the physician should obtain a history from others present at the scene. A CT scan showing blood at the base of the brain suggests aneurysmal hemorrhage, while blood layering over the top of the brain suggests a traumatic hemorrhage. Angiography should be considered when there is a question regarding the cause of the bleed.

Once a diagnosis of subarachnoid hemorrhage is made, complete diagnostic cerebral angiography should be performed as soon as possible to localize the source of bleeding.

Angiography is the gold standard for identifying cerebral aneurysms. Surgical clipping (in which the neurosurgeon opens the skull and places a clip across the neck of the aneurysm to prevent rupture) or less-invasive endovascular treatment (in which coils are placed in the aneurysm via a catheter) should be performed as soon as possible.

Patients with a hemorrhage should be stabilized immediately with particular concern for airway maintenance, blood pressure treatment, correction of any coagulopathy (abnormal blood clotting), and pain and agitation control. They must be admitted to a critical care unit for monitoring and management.

Treatment involves preventing and managing secondary complications--rebleeding, vasospasm (constriction of large-capacity intracranial arteries), hydrocephalus (enlargement of the ventricles in the brain), hyponatremia (abnormally low sodium levels in the blood), and seizures.[24] Serum electrolyte levels, a complete blood cell count including platelets, and clotting time studies should be done to determine whether the patient has other conditions such as an infection or clotting abnormalities.

Frequent neurologic exams should be performed to evaluate the patient for changes in neurological status or evidence of rebleeding. For untreated ruptured aneurysms, there is a 3-4 percent risk of rebleeding in the first 24 hours, 1-2 percent risk per day in the first month, and a long-term risk of 3 percent per year after 3 months.[25]

Rebleeding may be due to sudden changes in blood pressure rather than absolute blood pressure. Bed rest, analgesics for headaches, and antihypertensive medications are generally recommended. Because blood in the subarachnoid space can cause seizures, patients should be monitored for seizure activity and given antiepileptic prophylaxis. To prevent vasospasm, the calcium channel blocker nimodipine should be used.

## Complications and consequences

The last step in your case evaluation is to know the possible complications and outcomes of a subarachnoid hemorrhage. The primary difficulty with these cases lies in the widely recognized high morbidity and mortality associated with ruptured aneurysms, even in the absence of negligence. Complications--including rebleeding, vasospasm, hydrocephalus, hyponatremia, and seizures--occur frequently and may be severe.

Rebleeding occurs in about 7 percent of all cases, but early surgical or endovascular treatment will usually prevent it. Vasospasm develops in about half of all patients, although the patient may not develop symptoms. Vasospasm can diminish blood flow to the brain in the territory of the constricted arteries, which in turn can cause ischemia or infarction of the brain.[26]

Vasospasm typically occurs between 5 and 14 days after the rupture and gradually resolves over 2 to 4 weeks.[27] If the aneurysm has been treated, ischemia associated with vasospasm can be reduced or prevented using "triple H therapy"[28] or angioplasty to dilate narrowed vessels. However, despite optimal therapy, 15 percent to 20 percent of patients with vasospasm die.[29]

Hydrocephalus following a rupture occurs in about 20 percent of cases. The patient may undergo placement of a ventriculostomy, a procedure to drain cerebrospinal fluid, but this intervention can lead to rebleeding and carries a small risk of infection. In symptomatic patients, chronic hydrocephalus may require a shunt to permanently divert cerebrospinal fluid.

Hyponatremia occurs in 10 percent to 34 percent of patients, and seizures occur in 25 percent of these patients.

As many as 46 percent of the people who survive a ruptured brain aneurysm have long-term cognitive impairment, and one-third of those who survive require lifelong care.[30] The average case fatality rate is 51 percent.[31]

A patient's prognosis largely depends on three factors: the patient's age, level of consciousness on presentation, and the amount of subarachnoid blood seen on the initial CT scan.[32] Younger patients with a good level of consciousness and minimal blood visible on the CT scan tend to do significantly better--and if diagnosed and treated without delay have dramatically better outcomes than patients who present later in the course of a hemorrhage or are subjected to delay in diagnosis and/or treatment.

In a major retrospective study involving patients who were treated at four different tertiary-care hospitals, the rate of rebleeding or other deterioration in patients who were initially misdiagnosed was 48 percent, while the rate in patients correctly diagnosed was under 3 percent.[33] The authors concluded that "in patients in whom correct diagnosis was promptly established, the rates of subsequent deterioration and of untoward outcomes were significantly decreased."[34]

Consider all this information when evaluating a prospective case and review the case with experts to understand and assess their opinions. There will always be a tension between the two dimensions of the case: liability and damages.

For damages, the ideal client will be a young person who presented to the emergency room with a severe sudden-onset headache, a relatively normal neurological exam, and minimal evidence of bleeding on a CT scan. These same facts make the case more difficult in the analysis of liability. The defense will argue that the physician cannot be expected to diagnose a cerebral hemorrhage in a healthy and vigorous patient when the physician sees thousands of patients with

nonsurgical headaches for every one patient with a headache caused by a subarachnoid hemorrhage.

The defense will point out that patients with headache make up 1 percent to 2 percent of all emergency room visits, headache complaints that are not related to hemorrhages are 1,000 times more common than those caused by a ruptured aneurysm, and CT scans and lumbar punctures cannot be performed on every patient who complains of headache.

Conversely, when bringing a case on behalf of an older person who presented with a severe headache and significant neurological deficits, the defense will cite statistics regarding your client's poor prognosis in the absence of negligence. The decision to pursue either case will depend on the specific facts and circumstances of your case.

In today's fast-paced emergency rooms and medical offices, performing a detailed, documented history and physical exam is a dying art. The tendency to rush to a diagnosis largely accounts for frontline physicians' failure to correctly diagnose and treat ruptured aneurysms. Many of these physicians also fail to appreciate the wide spectrum of possible clinical presentations of subarachnoid hemorrhage.

When physicians perform a comprehensive history and physical exam and recognize the variations in clinical presentation of subarachnoid hemorrhage, most patients will be promptly and correctly diagnosed.[35] In light of these facts, when the prospective client is elderly and had severe neurological impairment at the time of presentation, or the delay in diagnosis and treatment was minor, attorneys may choose not to accept the case. Otherwise, a client's case deserves further consideration.

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**1** Jonathan A. Edlow & Louis R. Caplan, *Avoiding Pitfalls in the Diagnosis of Subarachnoid Hemorrhage*, 342 New Eng. J. Med. 29, 29 (2000).

**2** Much less common breaches are related to surgical or endovascular treatment of the aneurysm.

**3** Jose I. Suarez et al., *Aneurysmal Subarachnoid Hemorrhage*, 354 New Eng. J. Med. 387, 388 (2006).

**4** Jonathan A. Edlow, *Diagnosis of Subarachnoid Hemorrhage in the Emergency Department*, 21 Emerg. Med. Clinics of N. Am. 73, 75 (2003).

**5** *Textbook of Clinical Neurology,* 1041 (Christopher G. Goetz ed., Elsevier 2007).

**6** *Subarachnoid Hemorrhage* (Elias A. Giraldo, 2007). Available at http://www.merck.com/mmpe/sec16/ch211/ch211d.html. (Accessed March 12, 2009).

**7** Marx: *Rosen's Emergency Medicine: Concepts in Clinical Practice* (John A. Marx, Robert Hockberger & Ron Walls eds., Mosby 2006). Avaiable at http://www.mdconsult.com/das/book/body/125640014-3/815689265/1365/305.html#4-u1.0-B0-323-02845-4..50106-2--cesec24_5271. (Accessed March 12, 2009).

**8** *Textbook of Clinical Neurology*, *supra* n. 5, at 1042.

**9** *Id.* at 1042.

**10** *Id.* at 1042.

**11** *Id.* at 1042.

**12** *Rosen's Emergency Medicine, supra* n. 7.

**13** These include autosomal dominant polycystic kidney disease, Marfan's syndrome, and Ehlers-Danlos syndrome (type IV). *Id.*

**14** Peter L. Mayer et al., *Misdiagnosis of Symptomatic Cerebral Aneurysm: Prevalence and Correlation with Outcome at Four Institutions*, 27 Stroke 1558, 1562 (1996).

**15** *Rosen's Emergency Medicine, supra* n. 7.

**16** Viewing the retina may reveal bleeding indicative of increased intracranial pressure and subarachnoid hemorrhage.

**17** *Rosen's Emergency Medicine, supra* n. 7.

**18** *Id*.

**19** *Id*.

**20** Edlow, *supra* note 4, at 79.

**21** Edlow & Caplan, *supra* n. 1, at 30-31.

**22** *Id.* at 33.

**23** *Rosen's Emergency Medicine, supra* n. 7.

**24** *Textbook of Clinical Neurology*, *supra* n. 5, at 1043.

**25** *Id*. at 1044.

**26** *Id*. at 1043.

**27** *Id*. at 1043.

**28** *Id.* at 1043. The three Hs are hypertension, hypervolemia, and hemodilution. Hypervolemia is a relative increase in blood plasma volume that results in hemodilution, a reduced concentration of formed elements such as red blood cells in the blood stream.

**29** *Id.* at 1043.

**30** Suarez, *supra* note 3, at 387.

**31** *Id.* at 387.

**32** *Id.* at 387.

**33** Mayer et al., *supra* n. 14, at 1560-61.

**34** *Id.* at 1562.

**35** Edlow & Caplan, *supra* n. 1, at 30-32, 34.