Intracranial Hemorrhages
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It is estimated that approximately 800,000 strokes occur each year in the United States, with intracranial hemorrhages accounting for 20% of the strokes. About 1% to 2% of patients seek treatment in EDs with a primary complaint of headache, and it is thought that 1% of these patients may have subarachnoid hemorrhage (SAH). If a filter of “worst headache of my life” is used, that estimate increases to up to 12% of patients with SAH. Approximately 500,000 moderate and severe traumatic head injuries occur each year in the United States. One percent to 2% of these patients have epidural hematomas, with estimates of an additional 5% to 25% having acute subdural hematomas.

PATHOPHYSIOLOGY

Intracranial hemorrhage is the umbrella term used to encompass the many types of bleeding within the cranial vault (Figs. 103.1 to 103.5 and Table 103.1). Intraparenchymal hemorrhage implies blood within the substance of the brain. When the hemorrhage is not clearly secondary to a detectable cause, it is deemed a spontaneous hemorrhage. This term is often used synonymously with the terms hypertensive hemorrhage and, when anatomically appropriate, intracerebral hemorrhage. Intraparenchymal hemorrhages may also occur in the brainstem or cerebellum. SAH literally describes blood in the subarachnoid space, and if nontraumatic, a vascular lesion such as an aneurysm is the implied cause of the bleeding. SAH and intraparenchymal hemorrhage may coexist. Intraventricular hemorrhage means that blood is visualized within the ventricles by cranial CT, and it is most often present with other types of intracranial hemorrhage. Intracranial hemorrhages outside the brain substance are referred to as extraaxial hemorrhages and include both subdural hematomas and epidural hematomas. Although uncommon exceptions exist, extraaxial hemorrhages almost always have a traumatic etiology. The term hemorrhagic stroke might literally describe abrupt symptoms with any of the previously mentioned hemorrhages, but it is sometimes used in a more restrictive sense to describe hemorrhagic changes in an area of ischemic stroke to the point of being visible on cranial CT; this is termed hemorrhagic transformation of ischemic stroke.

KEY POINTS

- “Head bleed” is an oversimplified term for intracranial hemorrhage because different types of hemorrhages have different causes, signs and symptoms, diagnostic strategies, and therapies.
- Descriptions of intracranial hemorrhage should include the anatomic location, estimation of size, presence of midline shift, and whether the hemorrhage is thought to be spontaneous or secondary to another process.
- Treatment recommendations regarding blood pressure management and anticonvulsant therapy remain controversial and lack strong evidence-based support.

EPIDEMIOLOGY

Estimates of the incidence of intracranial hemorrhage in patients seen in the emergency department (ED) are difficult because the findings include isolated headache, stroke syndromes, and head trauma, but a few generalities may be made.

PERSPECTIVE

Intracranial hemorrhages have different clinical manifestations ranging from subtle to catastrophic. The efforts of the emergency physician (EP) are directed toward identifying the correct diagnosis, confirming the diagnosis by cranial computed tomography (CT) or other diagnostic tests, and providing supportive care. Liberal consultation plus collaboration with specialists is required. Strong evidence of the best course of action regarding basic treatment issues such as blood pressure management and the use of anticonvulsants is lacking. Definitive therapy is most often in the hands of consulting and admitting physicians.

It is tempting to place any intracranial hemorrhage in the diagnostic category head bleed. Different types of intracranial hemorrhage may have different causes, different natural histories, different diagnostic strategies, different treatments, and frequently different prognoses. It is important to delineate the various types of intracranial hemorrhage so that correct diagnostic steps and therapeutic interventions can be performed.
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Intracranial bleeding may be of either arterial or venous origin. Because of the closed nature of the cranial vault, any increase in intracranial volume from bleeding may result in increased intracranial pressure (ICP) and decreased cerebral perfusion pressure (CPP). As a mass expands, some initial compensation occurs in the form of diminished intracranial vascular and cerebrospinal fluid volume. However, at some point the compensatory mechanisms fail and ICP will dramatically rise with a further increase in size of the mass. A key concept is CPP, the effective blood pressure exerted on the intracranial contents. CPP is equal to mean arterial pressure (MAP) minus ICP:

\[ \text{CPP} = \text{MAP} - \text{ICP} \]

If ICP increases abruptly or if MAP falls, CPP decreases and central nervous system ischemia may follow and exacerbate the neuronal injury.

Hemorrhages also cause injury by direct tissue destruction or compression of adjacent structures. Edema formation around a hematoma may further increase the mass effect. For example, with cerebellar hemorrhages, tissue damage may cause the initial symptoms, but increased ICP and rapid progression to coma result from compression of the adjacent brainstem.

Spontaneous intraparenchymal hemorrhages (e.g., intracerebral hemorrhages, lobar hemorrhages, hypertensive hemorrhages) are most often associated with chronic hypertension. Cerebral amyloid angiopathy is increasingly being recognized.
as a contributing process in the elderly. Chronic excessive alcohol use is also a risk factor. Hemorrhage usually originates from rupture of small penetrating branch arteries of the vessels at the base of the brain (Fig. 103.6).

Serial cranial CT demonstrates that many intracerebral hemorrhages expand over the course of several hours. The initial hemorrhage may infiltrate the white matter with little direct destruction, but continued hematoma expansion, white matter edema, additional hemorrhage from surrounding vessels, and the development of hydrocephalus may all contribute to increased ICP and secondary neuronal injury. The frequency of anticoagulant-associated intracerebral hemorrhage is increasing. Warfarin therapy does not appear to increase hematoma volume initially, but it does increase the risk for later hematoma expansion.

SAH literally means “blood in the subarachnoid space.” Trauma is the most common cause. Spontaneous, or nontraumatic, SAH has an entirely different differential diagnosis. About 80% of spontaneous SAHs are caused by rupture of saccular (berry) aneurysms of the intracranial vessels, which are commonly located near intracranial arterial bifurcations of the circle of Willis (Fig. 103.7). Aneurysms are often named after the vascular site of origin, such as the anterior communicating artery or middle cerebral artery. Aneurysms that develop following vascular infection from endocarditis are termed mycotic aneurysms. Some aneurysms also cause symptoms without rupture from a mass effect or from emboli originating within the aneurysm. Rupture of an intracranial aneurysm abruptly raises ICP and leads to the onset of symptoms. The bleeding may be confined to the subarachnoid space, or a hematoma may extend into the brain substance and create an intraparenchymal hemorrhage, which in turn may rupture into the ventricles. Vasospasm of the vascular tree related to the aneurysm typically takes hours to develop and may worsen regional ischemia.

Arteriovenous malformations are another cause of intracranial hemorrhage of both the subarachnoid and intraparenchymal anatomic subtypes. These arteriovenous shunts vary in their anatomy, and many patients have saccular aneurysms as well. Lesions with deep venous drainage and high pressure in the feeding vessels are at increased risk for bleeding. Cavernous angiomas are low-pressure vascular lesions associated with small hemorrhages.

Closed head injury may cause diffuse or localized subarachnoid bleeding. Cerebral contusion is a loosely defined term that describes the CT appearance of low density consistent with edema and often with some hemorrhage within that region.

An epidural hematoma usually reflects arterial bleeding into the epidural space following injury to a meningeal artery. A common mechanism is a skull fracture in the temporal area with associated laceration of the middle meningeal artery. The arterial pressure hematoma may increase in size until tamponade occurs as a result of resistance of distorted intracranial structures and increased ICP (at the expense of CPP).

Subdural hematomas reflect bleeding from small vessel sources and from diffuse brain injury with hemorrhage accumulating over the surface of the brain. Again, distortion of the cranial contents may occur, as well as increased ICP. The cortical atrophy that occurs with aging is thought to make the bridging vessels from the cortex to the dura increasingly susceptible to rupture from even trivial trauma in the elderly.

Less common causes of intracranial hemorrhage include dural sinus thrombosis with venous infarction and
Intracranial hemorrhages of any type may be associated with a continuum of changes in mental status ranging from mild headache to agitation, confusion, and coma. Seizures and stroke symptoms are also common findings.

Patients with a large intracranial hemorrhage typically have a diminished level of consciousness with or without a focal neurologic deficit. Intracerebral hemorrhages are responsible for about 20% of acute strokes. It is not possible at the bedside to reliably distinguish between an ischemic stroke and an intracerebral hemorrhage. Patients with a diminished level of consciousness often have a larger hemorrhage and increased ICP or distortion of the thalamic and brainstem reticular activating system.

With increased ICP, the Cushing triad of hypertension, bradycardia, and irregular respirations may be present, but this is not specific for intracranial hemorrhage. If able to speak, many patients complain of headache and nausea. Depending on the region of brain injured, the examiner will often find corresponding neurologic deficits. With hemispheric lesions, the picture may be similar to ischemic stroke—that is, patients with a large left cerebral hemorrhage may have right-sided hemiparesis and aphasia. Other stroke syndromes of neglect, visual field defects, and cortical sensory abnormalities may be present. With frontal lesions, conjugate eye deviation toward the side of the lesion is common. Large hematomas with mass effects may present the clinical picture of uncal herniation with diminished consciousness and third nerve dysfunction.
of SAH have led to the derivation of preliminary decision rules, which remain under study. It is important to identify the diagnosis at the time of an initial or “sentinel” hemorrhage or warning leak. The sentinel hemorrhage may be manifested as a transient headache or confusional episode. If the patient complains of headache of abrupt onset or that the headache is different from the kind that the patient usually experiences, the possibility of SAH exists. Patients in whom the diagnosis is made at subsequent visits often have worse outcomes.

An expanding unruptured aneurysm may be accompanied by cranial nerve abnormalities. Typically, this is a third nerve paresis with asymmetric pupils and impairment of extraocular movement. The pupillary reflex may or may not be impaired.

With SAH and increased ICP, cardiac arrhythmias and changes on the electrocardiogram (ECG) consistent with myocardial ischemia may at times confound the diagnosis. Usually, these patients have severe neurologic symptoms, but cases in which the arrhythmia overshadows the clinical findings are reported as well.

Intracerebral hemorrhage is the most common manifestation of arteriovenous malformations and accounts for roughly half the presentations. Other manifestations include seizures and focal neurologic deficits.

A history of trauma suggests the possibility of an extraxial hematoma. Progression of symptoms or deterioration in level of consciousness mandates investigation for an expanding mass lesion. With epidural hematoma, the classic description (present in only a minority of patients) is a transient loss of consciousness followed by an alert or lucid interval and later by progressively decreased level of consciousness. Headache out of proportion to the head blow or the presence of persistent vegetative symptoms such as nausea and vomiting is the more common manifestation. As the mass progresses, the neurologic findings may progress. Altered mental status following trauma is the typical clinical scenario.

Chronic large subdural hematomas may be found during evaluation of patients for altered mental status or headaches. Occult manifestations of intracranial injury are more common in the elderly.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

The major differential diagnosis of intracranial hemorrhage with focal neurologic signs or symptoms is ischemic stroke. Both processes may be associated with an abrupt onset of symptoms and focal neurologic deficits. Intracranial neoplasms are also in the differential diagnosis. However, the spectrum of findings in patients with intracranial hemorrhage is wide. Seizures are not a frequent initial complaint of patients with intracranial hemorrhage, although they do occur with enough frequency to include intracerebral hemorrhage and SAH in the differential diagnosis of seizures. Abnormal decerebrate posturing, which may occur with intracranial hemorrhage, is sometimes mistaken as seizure activity, particularly if it is brief and repetitive. Though countereintuitive, infectious processes such as encephalitis and meningitis may at times also have an abrupt onset of symptoms. If altered mental status is the initial finding, all the causes of altered mental status should be included in the differential
Intracranial Hemorrhages

The existence of an isodense hematoma must be inferred from cortical sulcus markings that do not reach the cranium. Clinically, the terms acute, subacute, and chronic are used to reflect the change in appearance on the CT scan, from hyperdense to isodense and finally hypodense. Nonhomogeneous density may be observed in some cases and is an indication of acute or acute on chronic bleeding. The possibility of hyperacute bleeding should always be kept in mind, with the areas of rapid bleeding appearing relatively hypodense within a larger, more dense area on CT (see Fig. 103.4).

**QUESTION 2—WHERE IS THE HEMORRHAGE?**

If hemorrhage is present, it is then described as external to the brain substance (extraaxial), within the substance of the brain (intraaxial or intraparenchymal), or visible in the intraventricular and subarachnoid or cisternal spaces. Extraaxial hematomas have two basic types of appearance. Subdural hematomas are most often crescentic (see Fig. 103.3), whereas epidural hematomas have a typical lens-shaped pattern (see Fig. 103.4). An intraparenchymal hemorrhage may be located in the cerebrum (intracerebral hemorrhage) or in subcortical or brainstem structures. Intracerebral hemorrhages of hypertensive origin tend to be situated in deep white matter or the basal ganglia or are confined to one lobe of the brain (lobar) (see Fig. 103.1). These hemorrhages tend to have a stereotypic pattern, and deviation from these patterns may suggest an uncommon cause of the hemorrhage.

Cerebellar hemorrhages may be midline or hemispheric (Fig. 103.12) and may cause brainstem compression. SAH may be detected by high density in the suprasellar or perimesencephalic cistern or by blood in the cortical sulci, where ordinarily there should be low-density images from the cerebrospinal fluid signal. Depending on the degree of hemorrhage, SAH may be obvious (Fig. 103.13) or relatively subtle (see Fig. 103.2). Intraventricular hemorrhage (literally “blood within the ventricles”) may result from rupture of an intracerebral hemorrhage into the ventricular system, from trauma, or from SAH (Fig. 103.14).

**QUESTION 1—IS BLOOD PRESENT?**

Acute blood appears white or hyperdense on non–contrast-enhanced CT scans (Fig. 103.10). Some intracranial structures such as the dura or choroid plexus may calcify and at times simulate hemorrhage. As blood ages, it becomes increasingly low density or dark (Fig. 103.11). There is a time during this evolution when blood is nearly the same CT density as brain parenchyma and is therefore termed isodense. Rarely, the existence of an isodense hematoma must be inferred from cortical sulcus markings that do not reach the cranium. Clinically, the terms acute, subacute, and chronic are used to reflect the change in appearance on the CT scan, from hyperdense to isodense and finally hypodense. Nonhomogeneous density may be observed in some cases and is an indication of acute or acute on chronic bleeding. The possibility of hyperacute bleeding should always be kept in mind, with the areas of rapid bleeding appearing relatively hypodense within a larger, more dense area on CT (see Fig. 103.4).

**Fig. 103.10** Computed tomography scan: acute epidural hematoma. Note the high density of the hemorrhage and the lens-shaped clot.

**Fig. 103.11** Computed tomography scan: chronic subdural hematoma (left hemispheric and bifrontal section). Low density is consistent with the presence of hemorrhage for some days to weeks.

Cranial CT is the current initial imaging test of choice for evaluation of intracranial hemorrhage because of the ability of non–contrast-enhanced cranial CT to demonstrate the presence of acute hemorrhage. Cranial CT is readily available in most U.S. EDs. Expert interpretation of cranial CT scans is sometimes not as readily obtainable, however, and EPs should be familiar with the basics of CT interpretation as it applies to immediate patient care.

In patients with suspected SAH, CT is very sensitive in detecting acute hemorrhage, with estimates of 95% or better.\(^ {11,13} \) Sensitivity starts to diminish as time from the hemorrhage increases, and CT sensitivity is estimated to be less than 50% 7 days after the event.

A suggested approach to analyzing CT scans (see Chapter 74) and a useful structure for communicating with consultants can be determined by asking the following series of simple questions:

1. Is blood present?
2. Where is the hemorrhage?
3. How much blood is present, and what is the effect?
4. What is causing the bleeding?
Fig. 103.12  Computed tomography scan: acute hemispheric cerebellar hemorrhage. The expanding mass in the posterior fossa places the brainstem at risk for compression.

Fig. 103.13  Computed tomography scan: acute subarachnoid hemorrhage. Blood is present in the suprasellar cistern and over the hemispheres.

Fig. 103.14  Computed tomography scan: intraventricular blood from extension of deep intracerebral hemorrhage. The right lateral and third ventricles are filled with blood casts.

**QUESTION 3—HOW MUCH BLOOD IS PRESENT, AND WHAT IS THE EFFECT?**

Some quantification of the hemorrhage should follow. For extraaxial hemorrhage, the greatest thickness of the hematoma is easily estimated from the ruler on the CT scan. Volumetric estimation of intraparenchymal hematomas may be estimated from information present on the cranial CT scan, although this is not usually done frequently by EPs. Because most hemorrhages are ellipsoid, the formula $ABC/2$ may be used to estimate the volume, where $A$ is the greatest hemorrhage diameter on CT, $B$ is the diameter 90 degrees to $A$, and $C$ is the approximate number of CT slices with hemorrhage multiplied by slice thickness.$^{14}$ Of more importance is any effect that the hematoma is having on adjacent structures. This may be estimated qualitatively by noting any compression on the ventricular system and the amount of shift of midline structure, as well as by CT signs of herniation (subfalcine, uncal, tonsillar herniations).

**QUESTION 4—WHAT IS CAUSING THE BLEEDING?**

Some cranial CT scan patterns of intracranial hemorrhage are sufficiently typical that an etiologic diagnosis may be suspected. For example, in a middle-aged or elderly patient with a spontaneous intracerebral hemorrhage in the deep white matter, the term hypertensive hemorrhage may be used. The same hemorrhage in a much younger patient might suggest a vascular lesion such as an arteriovenous malformation as the cause. One must remember that the specific cause of a hemorrhage seen on a CT scan is garnered from pattern recognition and is speculative to some degree.

Conventional angiography with selective injection of contrast material has traditionally been used when vascular lesions such as aneurysms or arteriovenous malformations are suspected. CT angiography with intravenously administered radiographic contrast material is increasingly being used instead of conventional angiography. Selection of direct vascular imaging is determined by a radiologist, neurologist, or neurosurgeon. (Discussion of this modality is outside the scope of this chapter.)

The role of magnetic resonance imaging (MRI) in current emergency medicine practice is evolving. In some centers, magnetic resonance angiography or venography is used, although again this is done in consultation with the admitting physicians or services. The traditional view is that MRI is inferior to CT when acute intracranial hemorrhage is suspected; however, recent literature suggests that with some technical adaptations, MRI may readily detect hemorrhages.

Lumbar puncture can increase the sensitivity for the detection of SAH in patients with negative or equivocal results on CT scanning. The common procedure is to collect cerebrospinal fluid in four tubes and obtain a cell count in tubes 1 and 4. Findings consistent with SAH include the presence of xanthochromia and a red blood cell count that does not diminish.
from tube 1 to tube 4. Xanthochromia from the breakdown of red blood cells may take more than 12 hours to develop and may not be present when lumbar puncture is performed soon after the onset of symptoms. The most common method of determining xanthochromia in the clinical laboratory is visual inspection, although some studies show that spectrophotometry is superior. Lumbar puncture performed to exclude SAH sometimes reveals unexpected diagnoses such as meningitis. Basic laboratory work should include coagulation studies and platelet counts if hemorrhage is suspected.

**TREATMENT**

Supportive care including appropriate management of the ABCs—airway, breathing, and circulation—is of course important. The decision to perform endotracheal intubation is based on the judgment of the physician who assesses the patient’s ability to protect the airway. It is recommended that certain steps be taken for rapid-sequence induction in patients with intracranial hemorrhage or other conditions with suspected increased ICP, including the use of lidocaine and a defasciculating dose of a paralytic agent, although rigorous proof of efficacy is lacking. In the past, hyperventilation was recommended with the goal of reducing abnormally increased ICP. Again, evidence is lacking, but the consensus is that hyperventilation beyond that needed to reduce PaCO₂ to only a small degree (PaCO₂ of 30 to 35 mm Hg) is not indicated.¹⁵

Blood pressure management in the setting of intracranial hemorrhage is controversial. In multiple-trauma patients with central nervous system injury, hypotension is associated with a poor outcome. In patients with intracerebral hemorrhage, the risk of expanding a hematoma associated with sustained hypertension must be weighed against the risk of impairing cerebral perfusion if blood pressure is reduced. A 2010 study suggested a trend toward more favorable outcomes in patients with aggressive blood pressure reduction but admitted that the study was underpowered and called for further investigation.¹⁶ In patients with established intracerebral hematoma and hypertension, consensus at this time is to use intravenous agents that can be titrated, such as nitroprusside, labetalol, esmolol, or carvedilol, if needed, to maintain blood pressure with an MAP of less than 130 mm Hg. Systolic blood pressure greater than 180 mm Hg or diastolic blood pressure higher than 105 mm Hg on two readings taken 5 minutes apart are the criteria recommended for intervention.¹⁵

In patients with SAH, there is also no clear evidence-supported management strategy. Hypertension should be avoided in patients with a ruptured aneurysm by administering intravenous titratable agents, as described previously. Some experts argue that relative hypotension should be induced based on the theory that a ruptured aneurysm is at risk for rebleeding in the presence of hypertension. Once the aneurysm is secured by interventional techniques, blood pressure is allowed to return to normal levels. The calcium channel antagonist nimodipine is recommended to reduce the chance of ischemia from vasospasm.⁵,⁶

Management of increased ICP is conjectural if its existence is unknown. However, basic steps such as elevating the head of the bed, keeping the head midline, and avoiding painful stimulation are clearly indicated. Hyperventilation with the goal of reducing ICP is currently out of favor. Steroids have no proven benefit in patients with intracranial hemorrhage. Use of osmotic agents such as mannitol in cases in which a herniation syndrome is present may be useful as a temporizing measure when decompressive neurosurgical therapy is planned.

ICP monitoring may be useful in the ED but should be performed under the direction of a neurosurgeon or neurointensivist. Ventriculostomy may be performed in the ED by a neurosurgeon.

If a patient with intracranial hemorrhage has a seizure, the use of anticonvulsants is clearly indicated. Phenytoin and fosphenytoin are the current drugs of choice. Increasingly, levetiracetam seems to be used in these settings, though rigorous evidence for superiority is lacking. Use of anticonvulsants is common in patients with intracerebral hematomas who have not had seizures, although their efficacy is not based on evidence. Likewise, use of anticonvulsants in patients with SAH who have not had seizures is controversial.

Hyperglycemia and hyperthermia are associated with neuronal injury and should be avoided if possible and treated if present. No specific guidelines exist for the clinician at this time. If a coagulopathy is detected, appropriate treatment should be initiated (see the following discussion on warfarin coagulopathy).

In patients with intracranial hemorrhage, most activity in the ED is directed at diagnosis. Treatment is governed by the type of hemorrhage, its cause, and any associated medical and surgical conditions. Definitive treatment is under the direction of the consulting and admitting physicians, and the EP should work in concert with them. In most institutions, SAH and traumatic hemorrhage will be managed by neurosurgeons. Intracerebral hemorrhage is also often managed by neurosurgeons, but institutional management patterns vary. Intensive care and monitoring are necessary in many cases.

For deeply comatose patients, intensive supportive care is indicated in the short term. Evidence of the development of hydrocephalus on CT should lead to consideration of ventriculostomy. Drainage of cerebrospinal fluid and other supportive measures may be guided by continuous measurement of ICP by a variety of invasive techniques.

Cerebellar hemorrhage is an emergency requiring removal of the hematoma and relief of brainstem compression, which offers the possibility of good recovery in selected cases.¹ Surgery for removal of supratentorial intracerebral hematomas is controversial and not generally recommended.¹⁷ Other inpatient supportive measures for intracerebral hemorrhage might include prophylaxis for thromboembolic events.

Patients with an acute intracerebral hematoma who are taking warfarin should receive fresh frozen plasma (FFP) and vitamin K as soon as possible to correct the coagulopathy. The best dosing regimens are not known, but for patients with a prolonged international normalized ratio, a reasonable recommendation is 5-10 mg of vitamin K (administered intravenously over a 10-minute period) plus 10 mL/kg of FFP administered as soon as possible. Type AB FFP is available in some blood banks and can speed administration time by eliminating the time needed for cross-matching. Institutional recommendation may vary. Time to treatment of warfarin-associated coagulopathy in patients with intracerebral hemorrhage has been found to be the most important determinant of 14-hour reversal of anticoagulation.¹⁸
Antiplatelet therapy has been noted to be associated with clinical deterioration.

Acute administration of recombinant activated factor VII limits expansion of hematomas, but it has limited use for thromboembolic complications. Other procoagulant pharmaceutical preparations are under study.

In recent years the trend has continued for early surgical intervention for SAH—that is, intervention to isolate the aneurysm or occlude it within 1 to 2 days of bleeding. Until the aneurysm is secured, the consensus is that blood pressure should be lowered with parenteral medications if necessary. A recent study reported that intervention to endovascular coiling may lead to better outcomes in selected patients with ruptured aneurysms.

Treatment of arteriovenous malformations is complex, controversial, and outside the practice of EPs other than providing supportive care as outlined previously. The risk for rebleeding is less than that with saccular aneurysms. Should seizures occur, anticonvulsant medication should be administered. Additional diagnostic vascular studies besides cranial CT are required and may include angiography, CT angiography, and MRI. Treatment may include radiotherapy, embolization of the arteriovenous malformation, resection, or any combination of these modalities.

All but the smallest epidural hematomas must be treated by craniotomy and surgical evacuation plus inspection of the bleeding site to secure hemostasis. Treatment of subdural hematomas depends on the size and chronicity of the hematoma, the general medical condition of the patient, and signs and symptoms referable to the hematoma. In some patients, chronic subdural hematomas may be of large size with seemingly minimal or no effect on the patient; the problem is underlying atrophy, with the subdural hematoma filling the void. Acute subdural hematomas are generally evacuated if they are causing any mass effect, but at times this may be difficult to assess because the underlying brain is usually injured and edematous.

In hemorrhages complicating abscesses, tumors, or other conditions, treatment is generally directed at the underlying lesion.

**FOLLOW-UP, NEXT STEPS IN CARE, AND PATIENT EDUCATION**

If the facility does not have the necessary specialty care, the patient should be transferred after stabilization to an appropriate facility. The urgency of transport corresponds roughly with the clinical condition of the patient; for example, patients with a headache, a chronic subdural hematoma, and no other findings may probably be transferred electively. The necessity for transfer of stable alert patients with minimal symptoms and minute traumatic hemorrhages detected on CT has not been studied. The notable exceptions in which emergency transport is indicated include acute SAH, cerebellar hemorrhage, and epidural hematoma when the natural history includes early clinical deterioration.

**SUGGESTED READINGS**


Runchev S, McGee S. Does this patient have a hemorrhagic stroke? Clinical findings distinguishing hemorrhagic stroke from ischemic stroke. JAMA 2010;308:2280-6.

**REFERENCES**

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