Hemorrhagic stroke is the second most common form of acute strokes. Patients can develop a hemorrhagic stroke due to many risk factors which include hypertension, cerebral amyloid angiopathy, neoplastic diseases and cerebral aneurysms. Hemorrhagic strokes can be classified into subarachnoid or intracerebral hemorrhage depending on the site of the hematoma formation. The semiology is usually similar to ischemic stroke; therefore, adequate brain imaging is very important for proper diagnosis and management.

Definition of Hemorrhagic Stroke
A stroke/cerebrovascular accident (CVA) is a disease that is characterized by rapid appearance of focal deficit of the brain with/without higher cerebral dysfunctions such as aphasia, hemisensory loss and visual field defects. It can either be an ischemic stroke (more than 85% of cases) or a hemorrhagic stroke (less than 15% of cases).

The simplest definition of hemorrhagic stroke is the acute neurologic injury that happens as a consequence to intracranial bleeding. Per this definition, the bleeding might be either within the parenchyma, also known as intracerebral hemorrhage, or in the cerebrospinal fluid surrounding the brain, known as subarachnoid hemorrhage. Epidural and subdural hemorrhage are not considered as part of the hemorrhagic stroke spectrum.

Classification of hemorrhagic strokes

Per the definition above, the bleeding might be:
1. Within the parenchyma, also known as intracerebral hemorrhage
2. In the cerebrospinal fluid surrounding the brain, known as subarachnoid hemorrhage.

NB: Epidural and subdural hemorrhage are not considered as part of the hemorrhagic stroke spectrum.

Hemorrhagic strokes are also classified based on the area of the brain involved:
- The basal ganglia (66%)
- Thalamus
- Cerebellum.

Hemorrhagic stroke may also be classified based on the vessel involved into:
- Anterior circulation stroke.
- Posterior circulation stroke.
- Large vessel disease.
- Small vessel disease.

The most common stroke is an ischemic stroke, while the second most common cause
is a hemorrhagic stroke. **Hemorrhagic stroke is implicated in about 15% of the cases of all strokes.** Mortality rates associated with intracerebral hemorrhage are considered as significantly higher compared to ischemic stroke.

Mortality related to hemorrhagic stroke seems to be highest in the first immediate period after the event up to three weeks. Current estimates put mortality risk at a similar rate for both hemorrhagic and ischemic strokes at 3 months post the cerebrovascular accident.

**Hypertension** is considered the most common cause of spontaneous intracerebral hemorrhage in adults. **Cerebral amyloid angiopathy, cerebrovascular malformations, arteriovenous malformations and neoplastic disease** are other risk factors for hemorrhagic stroke.

**Cerebral aneurysms** are more commonly associated with **subarachnoid hemorrhage** rather than intracerebral hemorrhage; however, up to 30% of bleeding aneurysms might cause intracerebral hemorrhage.

**Risk factors are:**
- Hypertension
- AVMs
- Cocaine use
- Amyloid angiopathy
- Metastatic tumors

**Etiology of Hemorrhagic Stroke**

Several diseases might be complicated by hemorrhagic stroke. The most common etiology of primary intracerebral hemorrhage is hypertension. **Prolonged hypertension** increases the pressure on the small arteries arising from the middle cerebral, thalamic and pontine arteries. This increased pressure leads to **intimal hyperplasia, degeneration and eventually necrosis of the small arteries.** At some point, the small arteries can rupture and intracerebral bleeding happens.
People older than 60 years of age are at risk of developing cerebral amyloid angiopathy, another condition that is commonly associated with hemorrhagic stroke. Beta-amyloid is deposited in small and medium sized cerebral arteries, especially in the basal ganglia. The affected blood vessels degenerate and undergo necrosis, which puts them at risk of rupture. If the vessels rupture, the patient will develop an intracerebral hemorrhage.

Developmental venous anomalies, arteriovenous malformations, capillary telangiectasias and cavernous malformations all share the one common pathological feature, i.e. abnormal fragile blood vessels. These blood vessels can bleed and cause an intracerebral hemorrhage. Larger arteriovenous malformations that have deep venous drainage, and are in the eloquent cortex, are very likely to bleed.

Neoplastic tissue is known to be hypervascular and these blood vessels are usually pathological. They are fragile, can undergo necrosis, and put the patient at risk of intracerebral bleeding. The most common cause of neoplastic related intracerebral hemorrhage is metastatic brain disease from melanomas, lungs, kidneys and thyroid gland.

Pituitary adenoma, glioblastoma and ependymoma are the most common primary brain tumors to be associated with intracerebral hemorrhage.

Hemorrhagic infarction is defined as the transformation of an ischemic infarct into a hemorrhagic infarct. The blood vessels are within the ischemic brain tissue, are fragile, and can rupture. If this happens, the patient might develop a hemorrhagic infarction. A parenchymal hematoma might form which can have drastic consequences on the clinical outcome of the patient.

Aneurysmal rupture is more likely to be associated with subarachnoid hemorrhage rather than intracerebral hemorrhage. Patients usually are younger and describe a sudden-onset severe headache.
Clinical Presentation of Hemorrhagic Stroke

Patients who develop subarachnoid hemorrhage and are still conscious, usually describe an extremely severe headache that is classically described as the worst headache in their lives. In addition to the severe headache, patients usually have nausea or vomiting. Focal neurological deficits and impaired consciousness are other common findings of hemorrhagic stroke. Seizures due to cortical irritation by the blood can occur. Patients can also develop neck rigidity and fever, signs suggestive of meningism.

Intracerebral hemorrhage can cause headaches and nausea, but more often differentiating intracerebral hemorrhage from ischemic stroke based on the clinical picture is not possible. Focal neurological deficits, impaired consciousness, and the sudden onset of the semiology can be seen in both ischemic and hemorrhagic strokes.

Because of the overlap of the symptoms between hemorrhagic and ischemic strokes, prompt and early brain imaging is very important in differentiating the two conditions because the treatment plan is different.

Diagnostic Workup for Hemorrhagic Stroke

Computed tomography without contrast remains the first-line diagnostic imaging modality in the evaluation of the acute stroke patient despite the recent advances in magnetic resonance imaging. Computed tomography imaging in acute hemorrhagic stroke due to hypertension shows an oval or round hyperintense mass.

If the patient presents very early in the disease process, the mass might be heterogeneous due to the ongoing bleeding. Patients presenting in the subacute stage can have an isodense mass instead of the typical hyperdense mass seen in acute hemorrhagic stroke.

Patients who undergo magnetic resonance imaging with a new device that is fast
enough for emergencies can have a hyperintense or isointense mass in the hyperacute and acute stages. In the subacute stage, up to one week after the hemorrhagic event, patients can still have a hyperintense mass on magnetic resonance imaging. At the chronic stage, the picture becomes similar to chronic ischemic stroke, i.e. hypointense.

Computed tomography imaging is also helpful in the diagnostic work-up of patients presenting with subarachnoid hemorrhage. The typical picture of subarachnoid hemorrhage on computed tomography is that of hyper-attenuation that is confined to the subarachnoid space. The hyperintensity is present within the sulci, fissures and the basal cisterns. Magnetic resonance imaging can also show hyperintensity in the subarachnoid space in case of subarachnoid hemorrhage.

**Treatment of Hemorrhagic Stroke**

The management of hemorrhagic stroke is dependent on the hematoma size, hematoma expansion rate and the computed tomography angiography sign.

Patients with large intracerebral hematomas are at the highest risk of immediate and 30-day mortality. The medical management mainly consists of anticonvulsants to prevent seizures, anti-hypertensive drugs to lower the blood pressure in case of hypertension, and osmotic diuretics. The decision to lower the systolic blood pressure in acute intracerebral hemorrhage to 180 mmHg or below or 140 mmHg or below is still under extensive research.

Patients with posterior fossa intracerebral hemorrhage usually have a worse prognosis, therefore, early surgical intervention for decompression is recommended. Patients who have hematoma expansion on repeated imaging might need Factor VII infusion.

Patients who have small 1 to 2 mm foci of intense enhancement inside the hematoma on computed tomography angiography are most likely having ongoing bleeding and might benefit from embolization therapy in addition to better control of their blood pressure. Additionally, these patients are at an increased risk of hematoma expansion, hence the decision to monitor them more closely with repeated brain imaging is justifiable.

Management of subarachnoid hemorrhage is largely dependent on blood pressure control and decreasing the intracranial pressure by osmotic diuresis.

Patients with subarachnoid hemorrhage due to head trauma usually have a slightly better prognosis compared to aneurysmal subarachnoid hemorrhage. Patients should receive good analgesia, but opioids should be avoided if possible to avoid masking of neurological signs of ongoing bleeding.

**References**


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