Imaging reveals signs of intracranial hemorrhage

November 01, 2005
By NeedsFixing [1]

Intracranial hemorrhage is one of the most common causes of acute focal neurological deficit in children and adults. Early and reliable identification of its age and cause is essential when choosing the correct treatment and estimating the patient's prognosis and outcome. Neuroimaging options include ultrasound, CT, MRI, and digital subtraction angiography. A thorough understanding of hematoma evolution and its appearance on neuroimaging studies is essential for determining the age of the hemorrhage. The radiologist or neuroradiologist should also be familiar with the various causes of intracranial hemorrhage.

Intracranial hematomas follow well-described, predictable pathophysiological processes of evolution and resorption (Figures 1 and 2). Five distinct evolutionary phases can be identified: hyperacute (less than 12 hours after onset), acute (12 hours to two days), early subacute (two to seven days), late subacute (eight days to a month), and chronic (more than one month). Factors that influence hematoma evolution include location (intra- or extra-axial, gray versus white matter), size (punctuate versus confluent, unifocal versus multifocal), etiology (arterial versus venous, trauma versus hypertension), and temporal occurrence of hemorrhage (acute event versus multistaged, recurrent hemorrhage). Biological factors, such as the patient's general physical condition, associated systemic diseases, hematocrit, tissue oxygenation level, pH level, and protein concentration (hemoglobin) may also influence hemorrhage evolution, along with medical treatment and interventions.

Hyperacute hematomas consist of a liquid mixture of red and white blood cells, platelet thrombi, and protein-rich serum. Red blood cells keep their normal biconcave shape, while intracellular hemoglobin remains oxygenated. The blood clot will retract over the following hours, expelling serum. Vasogenic edema may develop within adjacent brain tissue in the case of an intra-axial hemorrhage.

Red blood cells lose energy progressively during the acute stage. They shrink, due to dehydration, although the red blood cell membrane remains intact. Intracellular oxygenated hemoglobin also deoxygenates during this stage, increasing the amount of intracellular deoxygenated hemoglobin. An oxidative denaturation transforms deoxygenated hemoglobin into methemoglobin over the following few days. The red blood cell membrane is still intact at this early subacute stage, leading to an accumulation of intracellular methemoglobin. It is not until the late subacute phase that progressive denaturation of the membrane releases methemoglobin into the extracellular space. Macrophages and astroglial cells phagocytize the hematoma in the chronic stage. Extracellular methemoglobin is converted into hemosiderin and ferritin, which is stored within the macrophages. Intra-axial hematomas leave a cystic, fluid-filled, or collapsed brain defect.

IDENTIFYING SIGNS
Ultrasound, CT, MRI, and DSA are well-established sensitive methods of identifying and evaluating intracranial hemorrhages. Imaging appearance and characteristics vary with the stage of hematoma resolution, enabling estimation of the age of the hematoma (Figure 2). Extension, location, and correlation with clinical history usually make it possible to determine the hemorrhage's exact etiology.

Ultrasound is performed mainly on neonates, using the anterior fontanel as an acoustic window to the cranial vault. CT is the most frequently used modality for acute and follow-up examinations of intracranial hemorrhage. Evaluation is rapid, and scanners are widely available and accessible. CT
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Published on Physicians Practice (http://www.physicianspractice.com)

usually provides sufficient information to guide acute treatment. MRI is typically used as a second-line imaging modality. Its multiplanar capabilities, different tissue contrasts such as T1-, T2-, and T2*-weighted sequences (Figures 3 and 4), and functional techniques such as MR angiography and diffusion-weighted imaging provide valuable information for the diagnostic workup of complex intracranial hemorrhages (Figure 5). Its higher sensitivity to tissue changes also permits better evaluation of the exact extent of injury.

DSA is used in the rare cases for which ultrasound, CT, and MRI cannot provide a satisfactory explanation of the hemorrhage. DSA is used mainly in combined diagnostic and therapeutic procedures such as embolization of arteriovenous malformations.

CT imaging characteristics depend on the degree of x-ray attenuation of the hemorrhage, which hinges on its temporal resolution. Hyperacute-phase hematomas are isodense to healthy brain tissue. Progressive blood clot retraction increases hemorrhage density during the acute and early subacute phases, while progressive red blood cell lysis during the late subacute phase decreases the density of the hematoma. Progressive resorption of intra-axial hematoma in the chronic phase results in a hypodense brain defect filled with cerebrospinal fluid. Hematoma density is essentially determined by the degree of clot retraction, hematocrit, hemoglobin fraction, and protein concentration. The oxygenation level or metabolic state of the hemoglobin molecule is not a factor. Injection of contrast can help identify an isodense hematoma. Peripheral rim enhancement, however, may mimic focal brain tumors or infarctions. The degree of CSF dilution alters the density of extracerebral (subarachnoid, subdural) hematomas. Dilution of the hematomas by CSF may accelerate hematoma resolution.

MRI signal intensity is affected by differing magnetic effects of products formed during hematoma breakdown, hemoglobin oxygenation level, magnetic field strength, and chosen imaging sequence. In short, the oxidation state of iron within hemoglobin changes as the molecules complete their transformation from intracellular oxygenated hemoglobin to hemosiderin. These various oxidation states of iron produce different signal intensities on MRI and allow estimation of the age of the hematoma. The hematoma will be iso- or hypointense on T1-weighted MRI and hyperintense on T2-weighted MRI during the hyperacute phase. It will be iso- or hypointense on T1-weighted MRI in the acute phase but hypointense on T2-weighted imaging. Both T1- and T2-weighted imaging reveal a hypointense lesion in the early subacute phase, moving to a hyperintense lesion in the late subacute phase. Chronic-stage hematomas appear hypointense on T1-weighted MRI and hyperintense with a hypointense peripheral rim on T2-weighted MRI.

Transient disruption to the blood-brain barrier during hematoma evolution causes peripheral rim enhancement if contrast is injected. T2* gradient-echo sequences, which are very sensitive to paramagnetic susceptibility effects of hemosiderin, may increase the diagnostic accuracy of MRI. Fluid-attenuated inversion recovery (FLAIR) sequences suppress the signal intensity of free water and thus facilitate identification of blood within CSF spaces. This is especially helpful in subarachnoid hemorrhages. DSA identifies hematomas only indirectly. The diagnosis is suggested if visualized intracranial vessels are displaced or an active contrast leak is displayed. Disruption along the blood-brain barrier during the chronic phase may cause a contrast blush along the hematoma. DSA cannot estimate the age of a hematoma.

Few studies have systematically studied the evolution of hematomas on ultrasound. Exact differentiation between the phases of hematoma evolution is limited. Acute hematomas may appear iso- or hyperechogenic, and blood clot retraction can reduce the signal on follow-up. Hematomas may dissolve completely in the chronic phase, leaving a hypoechoic CSF-filled brain defect.

**COMMON CAUSES**

Intracranial hemorrhages result from a variety of intrinsic and/or extrinsic factors. Extrinsic factors include accidental head trauma and surgical interventions. Intrinsic factors, which are numerous, include hemorrhagic stroke, hypertensive hemorrhages due to atherosclerosis, rupture of an aneurysm or an arteriovenous malformation, various hematologic disorders, anticoagulation treatment, intracranial neoplasms, and cerebral amyloid angiopathy. Detailed identification of the location and extension of the hemorrhage provides valuable information about its possible etiology and guides treatment.

Epidural hematomas, located between the skull and underlying dura mater (Figure 6A), usually result from accidental head injury with skull fracture. Tears of the middle meningeal artery result in arterial epidural hematomas, and tearing of the dural sinuses may result in a venous lesion. The difference in etiology (arterial or venous) determines imaging appearance on CT and especially on MRI. Epidural hematomas are not diluted by CSF unless a coexisting dural tear connects the epidural
space with the subdural or subarachnoidal space. Subdural hematomas are located between the dura mater and arachnoideal membrane (Figure 6B). They are usually of venous origin, resulting from a tear to veins bridging the subdural space, and may occur post-trauma or spontaneously. They may also be diluted by CSF if an arachnoideal tear occurs. Subdural hematomas are typically seen in patients with an enlarged subdural and/or subarachnoidal space due to cerebral atrophy. They are also found in cases of nonaccidental head injury in children, including shaken babies.

Subarachnoid hematomas occur between the arachnoideal membrane and pia mater (Figure 6C). The most common cause is aneurysm rupture, as most parts of the intracranial arteries run through the subarachnoid space. Subarachnoid hematomas can also occur post-trauma if the cortical veins or arteries are ruptured, possibly in combination with a cortical laceration. They are diluted rapidly by CSF. Patients typically describe these hematomas as "life's worst headache."

Intraventricular hemorrhages are located within the ventricular system (Figure 6D). They may arise from complications to a subarachnoid hemorrhage if a "reflux" of blood from the subarachnoid space enters the ventricular system. Intraventricular hemorrhage can also result from a ruptured intraparenchymal hematoma, but rarely from a hemorrhaging intraventricular tumor (e.g., plexus papilloma).

Intracerebral hematomas are located within the brain parenchyma (Figure 6E) and can occur at any site, supra- or infratentorial, within gray and/or white matter. Location, distribution, size, number of hemorrhages, and coexisting lesions provide important clues to the etiology of intracerebral hematomas.

Neuroimaging plays an important role in identifying and locating intracranial hemorrhage in the acute setting and in determining the age and etiology of the hematoma to guide treatment. Ultrasound, CT, MRI, and DSA all contribute to the diagnostic workup of intracranial hematomas. CT is the most commonly used primary imaging modality, with MRI as a second-line tool. MR imaging should include several pulse sequences, particularly a T2*-weighted sequence because of its high susceptibility to blood products. We strongly recommend performing MRA at the same time to exclude vascular anomalies and/or malformations. Ultrasound is reserved for neonates and very young children. DSA is rarely used for diagnostic purposes but plays a critical role in neurovascular interventions. Functional MRI techniques, especially diffusion- and perfusion-weighted imaging, will be used more routinely to estimate the risk of secondary complications and hemorrhage recurrence, monitor treatment results, and indicate likely prognosis.

Unexplained spontaneous intracerebral hemorrhage should be followed up with additional examinations after partial or complete resolution of the hematoma. Follow-up may reveal a causative lesion that was initially overlooked due to compression by the hematoma.

DR. HUISMAN is radiologist-in-chief and chair of the department of diagnostic imaging at the University of Zurich Children's Hospital in Switzerland. He is a board-certified radiologist, pediatric radiologist, and diagnostic neuroradiologist.

REFERENCES

Disclosures:

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