Intracranial hemorrhage: principles of CT and MRI interpretation

Abstract Accurate diagnosis of intracranial hemorrhage represents a frequent challenge for the practicing radiologist. The purpose of this article is to provide the reader with a synoptic overview of the imaging characteristics of intracranial hemorrhage, using text, tables, and figures to illustrate time-dependent changes. We examine the underlying physical, biological, and biochemical factors of evolving hematoma and correlate them with the aspect on cross-sectional imaging techniques. On CT scanning, the appearance of intracranial blood is determined by density changes which occur over time, reflecting clot formation, clot retraction, clot lysis and, eventually, tissue loss. However, MRI has become the technique of choice for assessing the age of an intracranial hemorrhage. On MRI the signal intensity of intracranial hemorrhage is much more complex and is influenced by multiple variables including: (a) age, location, and size of the lesion; (b) technical factors (e.g., sequence type and parameters, field strength); and (c) biological factors (e.g., pO2, arterial vs venous origin, tissue pH, protein concentration, presence of a blood-brain barrier, condition of the patient). We discuss the intrinsic magnetic properties of sequential hemoglobin degradation products. The differences in evolution between extra- and intracerebral hemorrhages are addressed and illustrated.

Keywords Brain • Hemorrhage • CT • MRI • Hematoma
Extracerebral hemorrhages comprise epidural hemorrhage (EDH), subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH), and intraventricular hemorrhage (IVH) [4]. Both EDH and SDH are most commonly caused by craniocerebral trauma. The origin of SAH can be traumatic (superficial contusion of the cerebral gyri) or non-traumatic (aneurysm rupture, arteriovenous malformation, secondary extension from an intracerebral hemorrhage). The same holds true for IVH.

In this article, we review the basics of CT and MRI interpretation of intracranial hemorrhage. We also analyze the evolution of the hematoma over time and its effect on the imaging findings.

**Computed tomography**

In reviewing the principles of CT interpretation of ICH, the basic physics of X-ray imaging must be taken into account. Attenuation, defined as the removal of X-ray photons from the beam, occurs in biologic tissues. The attenuation properties of a tissue are linked to their atomic number and physical density. In other words, attenuation of the X-ray beam is determined by the density of the electron clouds in the tissues it traverses [5]. The attenuation properties of intracranial blood are determined by the aggregation of globin molecules in the hematoma [6]. There is a linear relationship between CT attenuation, protein content (mainly hemo-

globin), and hematocrit [7]; however, artifacts located close to the skull base can easily mimic hemorrhage on spiral-CT scans [8].

Immediately after the hemorrhage, freshly extravasated blood exhibits a markedly heterogeneous appearance with mixed density values in the range of 40–60 Hounsfield units (HU; Fig. 1a) [9, 10]. This is due to the formation of a complex, inhomogeneous mass that contains red blood cells (RBCs), white blood cells (WBCs), and small platelet clumps interspersed with protein-rich serum. Parts of the hematoma exhibit density values, which are only moderately higher than the density value of the adjacent brain parenchyma; therefore, a hyperacute EDH may be difficult to distinguish from the adjacent cortical gray matter.

During the early hours of hemorrhage, the CT density values within the hematoma rapidly increase up to 60–80 HU (Fig. 1b). This is due to the formation of a meshwork of fibrin fibrils and globin molecules. The globin (protein) component of the hemoglobin has a high density [11]. Moreover, due to incipient clot retraction, the hematocrit may increase to 90%, thereby further augmenting the density. These phenomena explain why, during the first week, intracranial hematomas appear on non-contrast CT scans as well-demarcated hypodense lesions [12].

In large hematomas, a horizontal fluid–fluid level is observed in the hyperacute and acute phase. This effect is called the “hematocrit effect” [13]. The dependent area, which has higher CT attenuation values, is believed to represent sedimented cellular elements of blood. The supernatant portion, which has a lower CT density, presumably represents blood serum (Fig. 2).

As the hematoma matures, clot retraction ensues. This increases the attenuation coefficient to 80–100 HU in the center of the hematoma. A hypointense halo appears around the central nidus due to serum extrusion and reactive vasogenic edema (Fig. 3).

This stage is short-lived because soon proteolysis begins, and the hematoma protein is degraded and ab-