

8. Intraparenchymal Hemorrhage

On MR, hemorrhage in the brain follows a regular well-defined temporal progression of changes in signal intensity. Oxyhemoglobin (hyperacute) progresses to deoxyhemoglobin (acute), to intracellular methemoglobin (early subacute), then extracellular methemoglobin (late subacute), and eventually to hemosiderin/ferritin (chronic). Deoxyhemoglobin and methemoglobin are paramagnetic, while hemosiderin/ferritin is superparamagnetic, influencing the signal intensity appearance. Oxyhemoglobin (hyperacute hemorrhage) has the signal intensity of fluid, moderate high on T2- and moderate low on T1-weighted scans (Fig. 8.1 A,B, asterisk). This imaging appearance is relatively nonspecific. Within hours, however, deoxyhemoglobin (acute hemorrhage) is evident with distinctive low signal intensity on T2-weighted scans (Fig 8.1 C, asterisk). Continued clot resorption as illustrated in this patient leads, long term, to the formation of a hemosiderin cleft (Fig. 8.1 D). Deoxyhemoglobin however does not have a unique appearance on T1-weighted scans, on which it appears isointense to mildly hypointense. Methemoglobin (subacute hemorrhage) has distinctive high signal intensity on T1-weighted scans, and bleeds can further be subdivided temporally into intracellular and extracellular methemoglobin. Initially, in the intracellular phase, blood will be high signal intensity on a T1-weighted scan and low signal intensity on a T2-weighted scan (the latter due to a susceptibility effect). With red

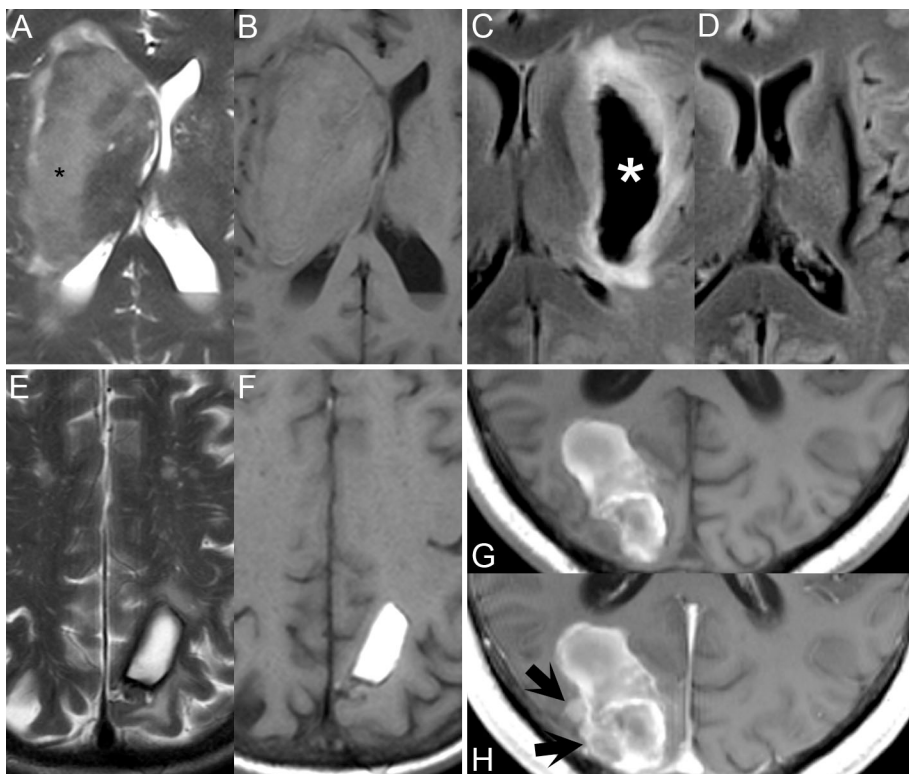


Fig. 8.1

blood cell lysis, methemoglobin becomes extracellular in location, with distinctive high signal intensity on both T1- and T2-weighted scans (Fig. 8.1 E,F). With a further elapse of time, methemoglobin is converted into hemosiderin, with chronic hemorrhage thus exhibiting pronounced low signal intensity on T2-weighted scans again due to susceptibility effects. The appearance of a chronic parenchymal hemorrhage on MR also depends upon whether the central fluid collection is resorbed or not. If resorbed, a hemosiderin cleft will be left (Fig. 8.1 D). If not resorbed, there will be a central fluid collection with high signal intensity on both T1- and T2-weighted scans, surrounded by a hemosiderin rim. With the passage of years, the fluid collection may change in appearance on T1-weighted scans from high to low signal intensity. Although the appearance of chronic hemorrhage on MR, with low signal intensity on T2-weighted scans, is generally well known, the relative sensitivity of different pulse sequences is often not as well understood. Blood products with susceptibility effects are best visualized on GRE (T2* weighted) scans. Also, it is important to note that the evolution of parenchymal hemorrhage on MR does not always follow the characteristic pattern described. Additional factors can be very important, including dilution, clotting, and hematocrit. One key to the recognition of parenchymal hemorrhage, not discussed in detail, is the presence of edema surrounding the hematoma (Fig. 8.1 A,C), which is seen in the hyperacute, acute and early subacute stages.

An important caveat in the imaging of blood products is that not all parenchymal hemorrhages are benign in etiology. IV contrast administration may demonstrate an otherwise benign-appearing hemorrhage, as seen in the subacute phase in Figure 8.1 G (pre-contrast T1WI), to be associated with a neoplastic lesion (Fig. 8.1 H, post-contrast, black arrows). Other features suggestive of neoplasia include the delayed evolution of blood products, particularly in the deoxyhemoglobin stage (from hypoxia), heterogenous SI, and a lack of the complete low SI rim seen in various stages of benign hemorrhage.