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Intracranial Hemorrhage Imaging

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Intracranial hemorrhage is a medical event frequently encountered in the clinical practice of radiology that has significant potential for patient morbidity and mortality. The expedient and accurate identification of intracranial hemorrhage as well as elucidation of the underlying cause can assist in optimizing the care of these patients. In this review, we attempt to familiarize the reader with the imaging appearance of multiple types of intracranial hemorrhage, both intra-axial and extra-axial and utilizing both computed tomography and magnetic resonance imaging, as well as to provide a framework for assessment of the underlying cause of the hemorrhage.

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Introduction

Intracranial hemorrhage is an often devastating event characterized by acute extravasation of blood products into 1 or multiple intracranial compartments. Nontraumatic intracranial hemorrhage has an incidence of 25 per 100,000 people per year and a mortality rate approaching 40% within 1 month of presentation.[1] Even in patients who survive an episode of intracranial hemorrhage, many are left with permanent neurologic deficits, contributing to the significant morbidity of this condition.

The purpose of this review is to familiarize the reader with the wide differential diagnosis of intracranial hemorrhage, utilizing an imaging based framework broadly divided between macroscopic hematomas and petechial or microhemorrhages, in addition to distinctions based on intracranial compartments and traumatic vs nontraumatic presentations. A summary of our recommended diagnostic approach can be seen in Figure 1. Imaging techniques, the physical principles underlying them, and imaging signs useful in the evaluation of intracranial hemorrhage will also be discussed.

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Physical Principles

Macroscopic Hemorrhage

Computed Tomography

Computed tomography (CT) is the most efficient technique for screening patients who present with acute neurologic deficits and in whom acute intracranial hemorrhage is a consideration. Acute intracranial hemorrhage appears on CT as a region of increased density owing to the linear relationship between attenuation and hematocrit, predominately owing to hemoglobin concentration. Acute hemorrhage in a patient with a hematocrit of 45% will be approximately 15-25 Hounsfield units (HU) greater in density than gray and white matter, respectively.[2] During the first 48-72 hours after the initial hemorrhage, the density of the extravasated blood will increase owing to clot formation and retraction, with extrusion of serum and a relative increase in hemoglobin concentration. Subsequently, as progressive liquefaction and lysis occur within the clot, the CT attenuation values decrease and the region of hemorrhage becomes isodense to brain parenchyma over the ensuing 2 weeks. After approximately 2 months, the hemorrhage will have typically resolved completely, leaving behind a variable degree of cystic encephalomalacia or gliosis.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is the study of choice to further characterize intracranial hemorrhage, offering greater sensitivity in the detection of hemorrhage during all stages of hematoma evolution as well as the ability to more accurately assess the temporal evolution of hemorrhage. MRI also allows

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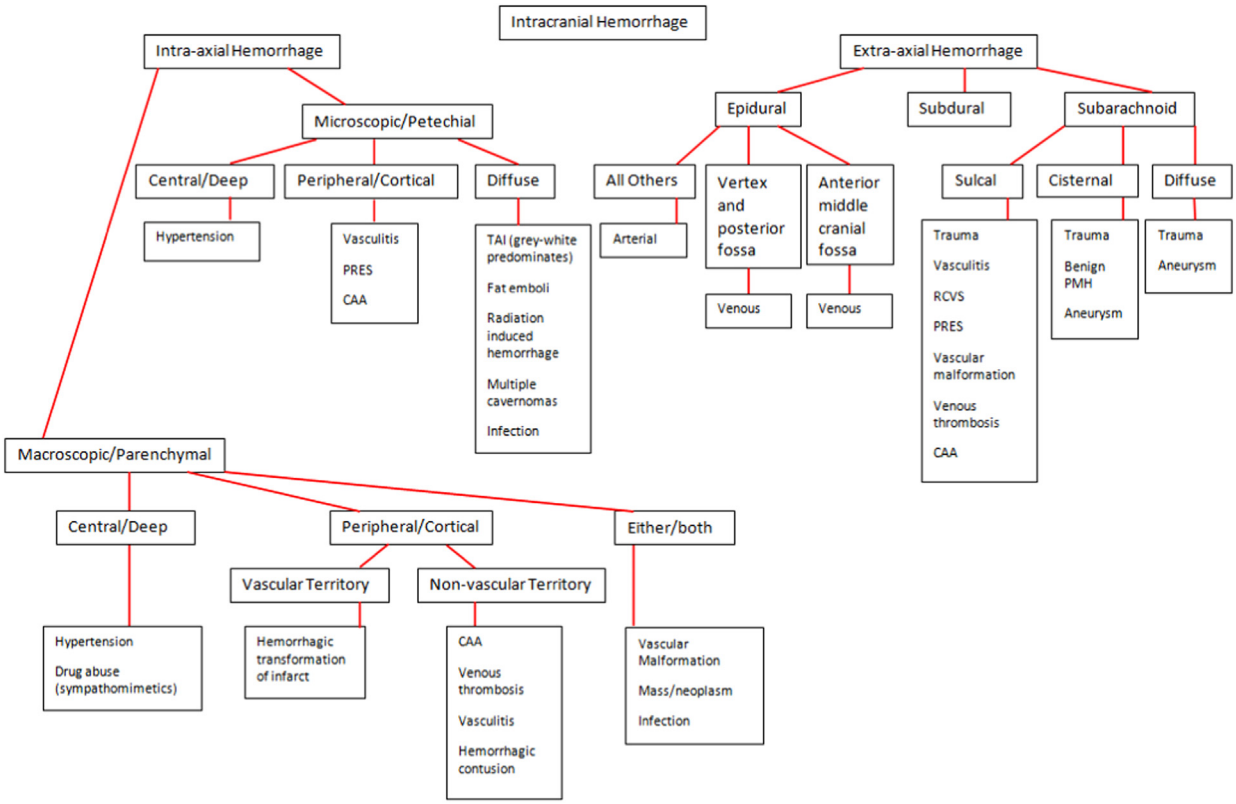

Figure 1 Image analysis algorithm. (Color version of figure is available online.)

more specific investigation of the etiology of intracranial hemorrhage.

The hemoglobin molecule undergoes conformational changes based upon oxygen binding, and these changes in conjunction with susceptibility effects from iron within the hemoglobin molecule and proton-electron dipole interactions influence the signal characteristics of blood on MRI. Hyperacute hemorrhage (<6 hours old) contains oxyhemoglobin, a diamagnetic substance, and is isointense to mildly hyperintense on T1-weighted images (T1WI) and hyperintense on T2-weighted images (T2WI) relative to cerebral white matter, mostly owing to the globins surrounding the heme. Acute hemorrhage (6-72 hours old) contains deoxyhemoglobin, a paramagnetic substance, which increases the local magnetic field strength and results in phase dispersion and associated T2 shortening. These effects are proportionate to the time to echo, and are thus significantly less pronounced on T1WI. Therefore, acute hemorrhage appears isointense on T1WI and hypointense on T2WI relative to cerebral white matter. Early subacute hemorrhage (3 days-1 week old) contains paramagnetic methemoglobin within still intact red blood cell membranes, which results in hyperintense signal on T1WI relative to cerebral white matter secondary to proton-electron dipole interactions; while susceptibility effects still dominate the T2 signal characteristics as seen in acute hemorrhage, resulting in hypointense signal on T2WI. After approximately 1 week, red blood cell membranes undergo lysis, and the previously intracellular paramagnetic methemoglobin enters the extracellular space resulting in significantly decreased susceptibility effects while proton-electron dipole interactions persist. These changes cause late subacute hemorrhage (1 week-1 month old) to appear hyperintense on both T1WI and T2WI relative to cerebral white matter. After further evolution of hemorrhage, resorption of fluid and clot occurs leaving behind hemosiderin from degraded methemoglobin. Hemosiderin contributes to pronounced susceptibility effects and causes chronic hemorrhage (>1 month old) to appear hypointense on both T1WI and T2WI relative to cerebral white matter. These changes are summarized in Figure 2. It is important to note that this expected pattern of temporal evolution applies only to intra-axial hemorrhage and may not be valid for extra-axial hemorrhage. The signal changes of extra-axial hemorrhage are influenced by differing oxygen tension and increased fluid content within the local environment.

Gradient echo (GRE) and newer susceptibility weighted imaging (SWI) highlight the susceptibility effects associated with intracranial hemorrhage- these appear as areas of hypointense signal beginning at the outer margin of a hematoma and progressing centrally. These changes become more pronounced at higher field strengths. Fluid attenuated inversion recovery (FLAIR) images are highly effective in demonstrating extra-axial, and more specifically, subarachnoid hemorrhage, as the presence of blood products within the cerebrospinal fluid (CSF) results in alteration of relaxation times and failure of the inversion pulse to null the fluid signal. These changes result in hyperintense FLAIR signal in regions of hemorrhage on a background of hypointense nulled CSF. These changes can be more reliably demonstrated with the absence of confounding hyperintense CSF pulsation artifacts