13. Ischemia and Infarction I

Cerebrovascular disease is the most common neurologic disease encountered by the practicing radiologist. Diffusion-weighted MRI (DWI) demonstrates a high sensitivity for ischemia within 15 to 30 minutes of symptom onset. DWI exploits the differences in the diffusibility of intra- and extracellular water. The lack of availability of adenosine triphosphate (i.e. ATP) in ischemic tissue results in the failure of sodium-potassium exchange pumps. Pump failure results in increased intracellular sodium and thus water. This increase in intracellular water without an increase in overall tissue water is known as cytotoxic edema. Intracellular water is relatively restricted in its diffusion by the cell membrane and intracellular organelles, and this restriction is visualized as high SI on DWI scans. While DWI is sensitive for the detection of cytotoxic edema, it is also a T2WI. Thus an area of increased SI on DWI may represent either pure restriction of diffusion or additional components of increased SI from its T2-weighted component ("T2 shine through"). Thus an area of high SI on DWI must be correlated with an ADC map on which a true diffusion restriction appears as low SI. In the absence of DWI abnormality, ADC maps need not be examined. DWI detects brain ischemia much earlier than FLAIR or T2WI on which ischemia is visualized commonly after 6 hours and almost uniformly after 24 hours of symptom onset. These sequences detect the vasogenic edema present in later stages of brain ischemia. Vasogenic edema occurs when edematous cells lyse and vascular endothelium is destroyed, resulting in the release of water into the extracellular space. Increasing extracellular edema may lead to mass effect and compression of adjacent vessels, thus extending the original infarct. FLAIR images, in which CSF SI is suppressed, are preferred over conventional T2WI for the visualization of vasogenic edema, particularly in periventricular and sulcal locations.

Stokes may be characterized by timeframe as hyperacute, acute, subacute, and chronic. Hyperacute strokes are defined as being less than 6 hours after symptom onset. Although these may not be commonly seen clinically due to delays in patient presentation, the detection of hyperacute strokes is essential as intravenous thrombolytic intervention is only effective if initiated within 3 hours of ischemic insult. As demonstrated in Figure 13.1 A, a hyperacute stroke is not accompanied by SI changes on T2WI or FLAIR (illustrated) scans. DWI, however, clearly demonstrates a hyperacute stroke as an area of high SI (Fig. 13.1 B, arrow), which is confirmed as a true diffusion restriction by a corresponding region of decreased SI on an ADC map (Fig. 13.1 C). An acute stroke is defined as being from 6 to 24 hours from symptom onset. These are typically well-seen on DWI, FLAIR, and T2WI (Fig. 13.1 D) as areas of increased SI. Because a multitude of other entities may appear

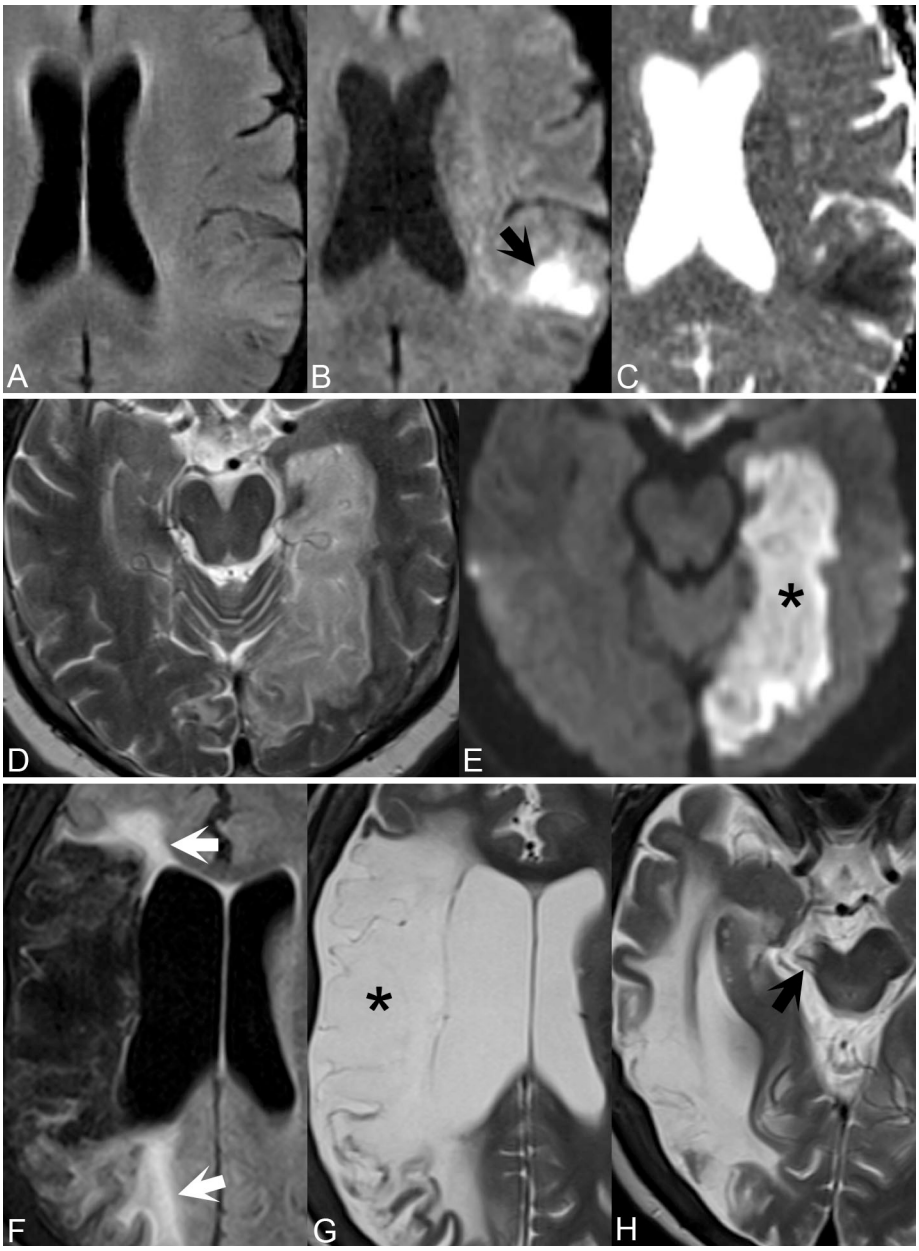

Fig. 13.1

similar on T2WI and FLAIR, DWI may be useful to distinguish the restricted diffusion typical of stroke (Fig. 13.1 E, asterisk) from neoplasia or other entities in which the diffusibility of water often remains unchanged. The appearance of subacute (24 hours to 6 weeks) stroke is dominated by vasogenic edema which correlates with high SI on T2WI and low SI on T1WI. SI on DWI begins to decline in the first week following stroke and typically normalizes by week two. ADC values likewise normalize within the first week and increase thereafter, owing to increased water diffusibility allowed for by the destruction of neurons. Resorption of extracellular edema typifies the chronic phase of stroke (after 6 weeks). Gliotic changes are prominent, correlating with increased SI on FLAIR (Fig. 13.1

F, white arrows) and T2WI (Fig. 13.1 G) and decreased SI on T1WI. Cystic encephalomalacia results from the replacement of infarcted brain parenchyma with free water. These areas are distinguished from gliosis by their isointensity with CSF on all pulse sequences (Fig. 13.1 F-G, asterisk). Figure 13.1 G also demonstrates widened cortical sulci and ex vacuo ventricular dilatation, which provide further MRI evidence of parenchymal atrophy/destruction. Wallerian (anterograde) degeneration of the myelinated axons distal to the site of ischemic injury may be appreciated on MRI as asymmetry of the cerebral peduncles (Fig. 13.1 H, black arrow) resulting from the loss of axonal mass ipsilateral to the infarct. The involved cerebral peduncle may also demonstrate gliotic changes (Fig. 13.1 H, black arrow) with increased SI on T2WI.

The distribution of supratentorial strokes corresponds with the vascular supplies of the anterior (ACA), middle (MCA), and posterior (PCA) cerebral arteries. The MCA supplies the majority of the lateral surface of the cerebrum, the insula, and the anterior and lateral parts of the temporal lobes. It is the most commonly infarcted vascular territory. MCA infarctions most commonly involve either the anterior or posterior (Fig. 13.1 A-C) portion of the MCA distribution but may involve the entire territory of the artery (Fig. 13.1 F-H). The MCA is divided into named segments (M1-M4). The M1 segment is proximal to the bi- or trifurcation of the artery, with M2, M3, and M4 being the subsequent insular, opercular, and cortical segments. The PCA is the second most common vascular distribution involved in ischemic stroke and supplies the occipital lobe along with the medial parietal and temporal lobes. The posterior communicating artery divides the PCA into proximal (P1) and distal (P2) segments. Figure 13.1 D-E demonstrates an infarction involving the entire distribution of the PCA including its medial temporal component. Infarcts in the distribution of the ACA account for less than 3% of cases. This is due in part to the access of the ACA to the circulation of its contralateral counterpart via the anterior communicating artery. The ACA supplies the anterior two thirds of the medial cerebral surface, the corpus callosum, and 1 cm of superomedial brain over the convexity. ACA infarctions involving the callosum may be confused with lymphoma on MRI with the latter also demonstrating restricted diffusion due to its dense cellularity. Clinical correlation and the progression of the lesion's appearance over time aid in differential diagnosis. Thrombosis of the distal portion of the internal carotid artery may, in individuals without effective cervical collateral vasculature or an incomplete circle of Willis, result in infarction of both the ACA and MCA territories.

While contrast administration in some centers may not be used routinely in the evaluation of stroke, often ischemic lesions are visualized on enhanced studies. The vessels supplying the region of an infarct may enhance in large strokes of 1 to 3 days of age. Enhancement of

the meninges adjacent to a large territorial infarct is rare but may be seen in infarcts 2 to 6 days old. When flow is re-established, endothelial damage and loss of the BBB lead to parenchymal enhancement within the infarct in either an early intense or progressive pattern. Early intense enhancement occurs within 2 to 3 days and may predominate in cases of incomplete ischemia whereby contrast material is still delivered to ischemic tissues even at early stages of infarction. Progressive enhancement is first seen at 1 week and develops in a gyriform (if cortical) or uniform (if non-cortical) pattern. Because of the time required for contrast to infiltrate the parenchymal space, scans obtained 5 to 10 minutes postinjection best demonstrate parenchymal enhancement. Disruption of the blood brain-barrier and thus parenchymal enhancement rarely persists beyond 8 weeks.