14. Ischemia and Infarction II

Lacunar infarcts are small deep parenchymal lesions involving the basal ganglia, internal capsule, thalamus, and brainstem. The vascular supply of these areas includes the anterior choroidal artery of the supraclinoid portion of the internal carotid artery, the lenticulostriate branches of the anterior (ACA) and middle (MCA) cerebral arteries, the thalamoperforating branches of the posterior cerebral arteries (PCA), and the paramedian branches of the basilar artery. These infarcts are associated with chronic hypertension and may present clinically with pure motor or sensory deficits. On MRI lacunar infarcts appear as focal slitlike or ovoid areas, progressing in appearance similarly to the infarcts described in Chapter 13. The lenticulostriate branches of the MCA originate prior to the artery's bi- or trifurcation (M1 segment) to supply the basal ganglia and the anterior limb of the internal capsule. Thus ischemic disease involving the MCA prior the branching of the lenticulostriate arteries may involve its hemispheric distribution along with deep gray and white matter structures. A similar phenomenon is seen with the thalamoperforating arteries of the PCA. Figure 14.1 A demonstrates acute ischemia of the lentiform nucleus, which consists of the globus pallidus (medially) and the putamen (laterally), shown on a DWI scan. Note that the susceptibility effects of iron within the globus pallidus result in this structure's low SI on T2WI (such as DWI) at 3 T (Fig. 14.1 A). Figure 14.1 B demonstrates in the same patient an acute infarction of the head of the caudate as seen on DWI. The head of the caudate is supplied by the recurrent artery of Heubner, which arises from the ACA to also supply the anterior limb of the internal capsule and part of the putamen. The anterior choroidal artery, arising from the supraclinoid internal carotid, also supplies portions of the caudate, along with the posterior limb of the internal capsule and parts of the thalamus and cerebral peduncles. DWI is particularly useful in the evaluation of lacunar strokes, due to the tendency of chronic small vessel ischemia to obscure the appearance of high SI infarcts on FLAIR (Fig. 14.1 C) and T2WI. Such lesions are clearly differentiated from chronic ischemic changes on DWI (Fig. 14.1 B). Lesions of multiple sclerosis (see Chapter 18) and hypertensive encephalopathy are similarly easily distinguished from acute and early subacute lacunar infarcts.

The thalamoperforating arteries arise from the P1 segment of the PCA. These supply the medial ventral thalamus and the posterior limb of the internal capsule. An infarction of this region, as seen on DWI, is denoted by a white arrow in Figure 14.1 D. Dilated perivascular spaces (DPVS) surrounding vessels coursing through the brain may also masquerade as lacunar infarcts on T2WI because of their high SI and location. DPVS are seen in the basal ganglia, periatrial and supraventricular white matter, and at the midbrain junction of the

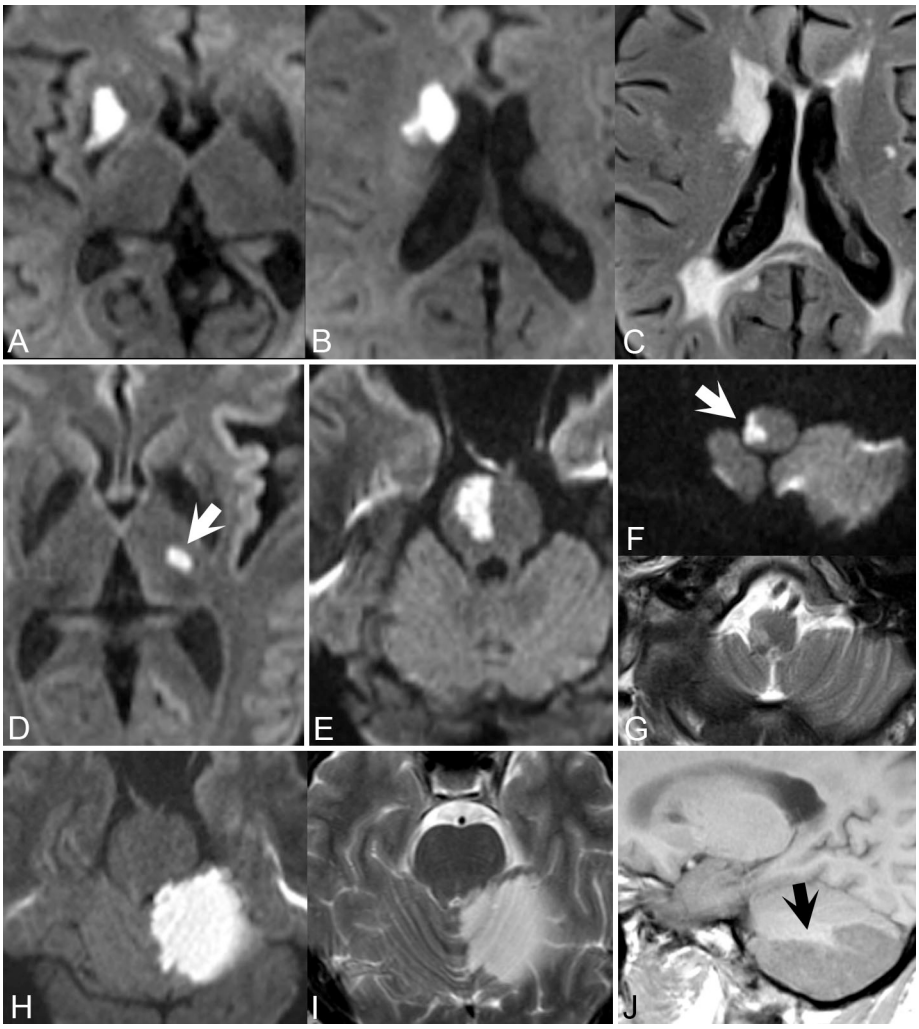

Fig. 14.1

substantia nigra and cerebral peduncle. Unlike lacunar infarcts, they are isointense to CSF on FLAIR and do not demonstrate restricted diffusion.

The vertebrobasilar arterial system supplies the posterior fossa. Of brainstem strokes, isolated infarctions of the midbrain are rarest. The midbrain derives its vascular supply from perforating arteries arising from the posterior communicating artery and the PCA. The PCA arises as a branch of the basilar artery, while the posterior communicating arteries connect the carotid and vertebrobasilar systems by linking the MCA and PCA. Pontine strokes are more common. The paramedian branches of the basilar artery supply the medial aspect of the pons—an infarction of which is demonstrated in Figure 14.1 E on DWI. The lateral pons is supplied by the anterior inferior cerebellar artery (AICA) caudally and the superior cerebellar artery rostrally. Infarctions of the lateral medulla result in a constellation of symptoms known as Wallenberg syndrome. These strokes (as seen on DWI and T2WI in Figure 14.1 F, white arrow and G, respectively) are particularly devastating and result from

interruption of blood supply to the posterior inferior cerebellar artery (PICA). Occlusions of the perforating branches of the basilar artery or vertebral arteries result in medial medullary infarcts.

PICA, AICA, and the superior cerebellar artery supply the cerebellum. The superior cerebellar artery arises from the basilar artery just prior to its termination as the PCA. Figure 14.1 H and I demonstrate an infarct affecting the entirety of the superior cerebellar artery's distribution on diffusion and T2-weighted images, respectively. Figure 14.1 J demonstrates an infarct of the PICA territory, seen with low SI (black arrow) on this sagittal T1-weighted scan. PICA is the largest intracranial branch of the vertebral artery and supplies the posterior inferior portion of the cerebellum and the lateral medulla. Infarctions of PICA may also involve the cerebellar tonsils. AICA arises from the basilar artery just subsequent to its origin at the junction of the vertebral arteries and supplies the anterior inferior portion of the cerebellum and the lateral pons.

Distinct underlying etiologies of stroke may result in differing MRI appearances. Thrombotic, embolic, hemodynamic, and venous (see Chapter 20) infarctions all occur. Arterial thrombotic infarctions are most common, usually resulting from atherosclerotic narrowing and eventual occlusion of vessel lumen. The extent of infarction is determined by the location and extent of obstruction (proximal lesions are less likely to develop infarctions), the availability of collateral circulation, and the integrity of the systemic circulation. Thrombotic infarctions tend to be sharply-demarcated, wedge-shaped lesions confined to a single arterial distribution and extending to the cortical surface. The appearance of the cerebellar infarct in Figure 14.1 H, I typifies that of a thrombotic infarction of the superior cerebellar artery. This appearance of a cerebellar infarction is uncommonly encountered in clinical practice, however, as cerebellar infarctions appear more frequently as multiple, chronic, punctate lesions. Differential considerations for an early thrombotic infarct on DWI include hyperacute hemorrhage, neoplasm, and abscess. None of these localize to an arterial territory, while the former undergoes a classic progression in MRI appearance as described in Chapter 8. Neoplastic lesions and abscesses are usually centered in white matter, whereas thrombotic infarctions involve gray matter as well. Cortically extending neoplasms also demonstrate edema projecting in a finger-like pattern with ill-defined margins and an associated centrally-enhancing mass. If enhancing, thrombotic infarctions should do so in a wedge-shaped pattern. Embolic infarctions, the source of which is most frequently the heart, are often multiple and may simultaneously affect more than one arterial distribution. The presence of septic emboli is suggested by the concurrent presence of infarction and abscess. Occluding emboli tend to fragment and lyse between days 1 and 5, re-establishing normal circulation. Luxury perfusion or hyperemia

following embolic lysis may lead to hemorrhagic conversion of the infarct due to a higher perfusion pressure. These hemorrhages usually appear petechial in nature and cortical in location, although a large hematoma may occasionally develop. The MR appearance of a hemorrhagic infarct varies with the stage of its blood products. When hemorrhagic stroke is suspected clinically, GRE scans should be obtained due to their high sensitivity for paramagnetic blood products (such as deoxyhemoglobin) and the important implications that the presence of hemorrhage has for the clinical management of stroke. Hemodynamic infarctions occur because of the failure of the heart to pump sufficient blood to oxygenate the brain. Watershed regions at the margins of major arterial distributions—areas with the lowest perfusion pressure—are most frequently involved in these infarctions. Watershed areas include the junctions of the distributions of the anterior, middle, and posterior cerebral arteries. The parieto-occipital watershed region—at the junction of all three of the aforementioned arteries—is especially susceptible to injury. Within the cerebellum, the major watershed area is at the junction of the superior cerebellar and the posterior inferior cerebellar arterial territories.