12. Aneurysms

Aneurysms are the most common cause of subarachnoid hemorrhage, other than trauma. Non-ruptured aneurysms are usually asymptomatic but may cause mass effect or – rarely – produce emboli from areas of partial thrombosis. Risk factors include smoking, binge drinking, use of illicit drugs and hypertension, along with collagen vascular, connective tissue, and polycystic kidney diseases. Intracranial aneurysms also frequently occur in the feeding arteries of AVMs, presumably due to increased flow. Aneurysms are broadly classified into fusiform (spindle-shaped) and saccular (spherical) types. Fusiform aneurysms frequently occur secondary to arteriosclerosis and favor the basilar and intracranial carotid arteries. Saccular or berry aneurysms are far more common and result from a congenital defect in the tunica media. Saccular aneurysms involve most frequently the anterior communicating (Fig. 12.1 A, white arrow), posterior communicating (Fig. 12.1 B, C, white arrows), and middle cerebral arteries – the latter primarily at the bifurcation/trifurcation. Flow dynamics at arterial branch points render these favorable locations for aneurysm development.

MR is the non-invasive screening test of choice for the detection and evaluation of intracranial aneurysms. Time-of-flight (TOF) MRA (performed without the use of intravenous contrast) is the specific sequence of choice, with maximum intensity projection (MIP, Fig. 12.1 A, B, and F) or volume rendering (Fig. 12.1 C) used to create images for viewing. While not commonly employed acutely, except in cases with a low clinical probability of hemorrhage or when angiography is contraindicated, MR is also the preferred modality for monitoring previously treated aneurysms. Extreme care must be exercised to avoid imaging a patient with a ferromagnetic aneurysm clip (these were used in the distant past), as doing so may result in death. MRI demonstrates areas of fast vascular flow as signal loss. This occurs when protons within blood do not remain within a selected slice long enough to acquire both the 90- and 180-degree pulses needed to produce a spin echo. Saccular aneurysms are no exception, appearing as an enlarged flow void (Fig. 12.1 D) corresponding with the focally dilated vasculature. Regions of slower-flowing blood within an aneurysm produce high or mixed SI rather than flow voids. The identification of one aneurysm on MR warrants a careful search for additional lesions as aneurysms are multiple in up to one-fourth of cases. Pulsation artifacts, propagating in the phase-encoding direction, may be seen with patent aneurysms, although these are less evident on modern MR imaging than in the past. Identification of this artifact, however, is not a reliable marker for aneurysms in the prepontine cistern as CSF pulsation artifact may simulate a basilar artery aneurysm in this region. Pulsation artifacts are more prominent following contrast

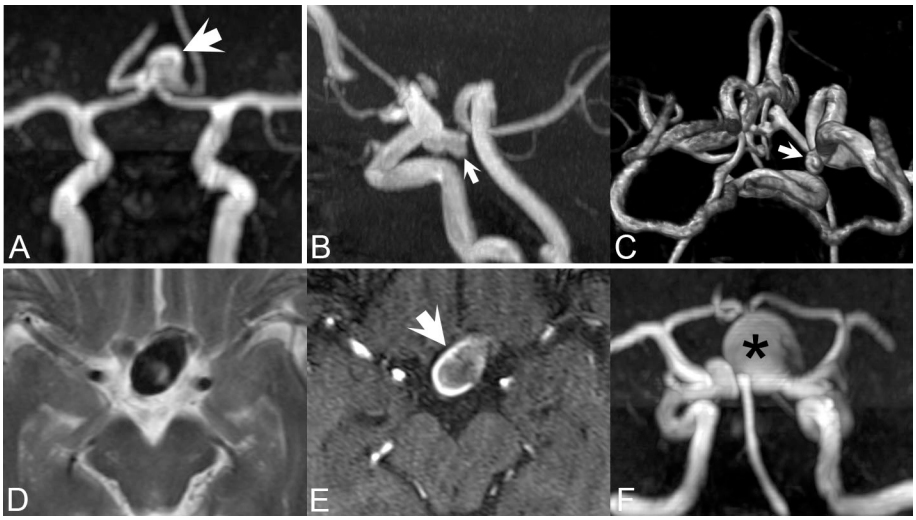

Fig. 12.1

administration. Both normal vasculature and patent aneurysms fill with contrast during the arterial phase (and for a period of time thereafter) and are thus high SI on T1WI post-contrast. Giant aneurysms are defined as having a diameter greater than 2.5 cm (Fig. 12.1 D-F). Giant aneurysms frequently involve the internal carotid artery (as illustrated), MCA, and the tip of the basilar artery. A partially thrombosed giant aneurysm may appear somewhat similar to an intraparenchymal hematoma on MR. In distinction, however, a flow void will be seen within the residual patent lumen, with flow therein demonstrable on many other MR sequences (for example, TOF MRA). In rare instances, different stages of organized clot within the thrombosed portion of an aneurysm will manifest as concentric layers of various SI. The distribution of subarachnoid hemorrhage (and likewise the location of any parenchymal hemorrhage) may suggest the location of the culprit aneurysm. For example, prominence of blood along the midline and within the anterior interhemispheric fissure, with less but symmetrically within the sylvian fissures, is suggestive of an ACOM aneurysm. Conversely, the presence of blood within one sylvian fissure points to an MCA bifurcation aneurysm.

Modern MR scanners easily detect aneuryms as small as 2 mm in diameter, with time-of-flight (TOF) MRA important for this evaluation. Aneurysms less than 3 mm in diameter are thought not to be at risk for rupture. Both the thin-section (source) images (Fig. 12.1 E) and maximum intensity projections (MIPs) - rotated both right to left and tumbled - should be reviewed. MIPs oriented in sagittal (Fig. 12.1 B) and coronal (Fig. 12.1 A, F) planes are illustrated. Volume rendering technique reconstructions may also aid in aneurysm visualization (Fig. 12.1 C). Particularly in internal carotid artery lesions, the neck of an aneurysm may not be visualized. In this case, targeted reconstructions, shorter TEs, and a smaller voxel size (all with TOF MRA) may be helpful.

With TOF MRA, the SI of stationary tissue is suppressed. Rapidly flowing blood is identified due to its relatively high SI. In large aneurysms, however, the flow within may be sufficiently slow to be suppressed. Thus, as demonstrated in Figure 12.1 E (white arrow), large or giant aneurysms may demonstrate heterogenous SI on TOF MRA, with a fast-flowing, relatively small stream of blood—appearing as high SI—flowing into a larger area of relatively stagnant blood—appearing as low SI. Because of this heterogenous signal, MIP images (Fig. 12.1 F, asterisk) may not fully visualize the aneurysm. Comparison of pre- and post-contrast images (with the addition of post-contrast TOF MRA) and the use of phase contrast MRA (PC-MRA) are possible alternatives in this instance. These approaches also allow better differentiation of intra-aneurysmal clot than TOF MRA where high SI clot (from methemoglobin) may not be distinguishable from the high SI of flowing blood.