Pearls and Pitfalls in Neuroradiology of Cerebrovascular Disease

The Essentials with MR and CT

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Purpose

The focus of this educational exhibit, presenting pearls and pitfalls in imaging of the brain, is also in a more broad sense that of clinical Neuroradiology itself. Recognition of characteristic findings on both MR and CT of the disease processes we are likely to encounter in clinical practice is key. The basis for this presentation is clinical case material, specifically excellent images and characteristic findings, demonstrating the points in question, from both modalities.
Approach/Methods

Of the four major disease categories (neoplastic, vascular, inflammatory, and congenital), vascular disease is the most common. Vascular disease itself is divided into two major subgroups, ischemia and structural vascular anomalies (aneurysms and vascular malformations). Cerebrovascular disease is often accompanied by intracranial hemorrhage, with the imaging appearance of the latter thus also important to understand. This exhibit presents 18 carefully chosen vascular disease imaging 'cases', focusing on less common presentations, the pitfalls in recognition and dating of hemorrhage, and important "Aunt Minnie" findings involving the vessels themselves and lesions therein.
Cerebral Ischemia

With regard to ischemia, several of the less commonly involved arterial territories are illustrated, specifically including the PCA, ACA, PICA and SCA. The spectrum of lacunar infarcts is then presented, along with the imaging appearance of intraparenchymal hemorrhage (including pitfalls in terms of temporal evolution, on both CT and MR). This section concludes with an illustration of the sometimes confusing appearance of late subacute infarction on MR, together with an illustration of extension of an infarct in the setting of a prior chronic infarct.
There is both vasogenic edema and cytotoxic edema, as demonstrated by respectively high signal intensity on the T2- and the diffusion-weighted scans, in the entirety of the left PCA distribution. In this instance, infarcts of the same time frame are also seen in the left thalamus (white arrow), which is supplied by the posterior thalamoperforators. There is also hemorrhage (black arrow) within the lesion, seen with low signal intensity due to a T2* effect.
ACA infarction, early subacute

On FLAIR, there is abnormal high signal intensity in the medial frontal lobe (ACA territory) on the right, involving both gray and white matter, with mild mass effect upon the frontal horn of the lateral ventricle. There is corresponding abnormal hyperintensity on DWI, reflecting cytotoxic edema (confirmed to be restricted diffusion on the ADC map). This anatomic distribution lies within, and forms part of, the ACA territory.
There is abnormal high signal intensity on T2- and diffusion-weighted axial scans in the left PICA territory. The ADC map however does not demonstrate restricted diffusion, thus identifying the findings on DWI to represent “T2 shine through”. The absence of a true diffusion change dates the infarct to be more than 1 week old, thus late subacute in time frame.
A portion of the superior cerebellar artery territory is noted to be involved on the left, with abnormal high signal intensity on both T2- and diffusion weighted scans. The high signal intensity on T2-weighted scans denotes vasogenic edema, with the findings on DWI – specifically restricted diffusion, confirmed on the ADC map (low signal intensity) - consistent with cytotoxic edema. TOF MRA reveals the left SCA to be small in caliber and truncated.
In the first patient, on DWI, high signal intensity is seen both in the lentiform nucleus (globus pallidus and putamen) and the caudate head, a common imaging appearance. The caudate head lesion is also shown on FLAIR to be high signal intensity, dating this lesion as acute to sub-acute in time frame. The caudate infarct, however, cannot be differentiated on the basis of signal intensity on the FLAIR scan from the marked accompanying periventricular white matter gliosis.

In the second and third patients, respectively, thalamic (white arrow) and pontine lacunar infarcts are seen with high signal intensity on DWI. Note the sharp demarcation, vertically along the median raphe, of the pontine infarct, a common imaging appearance. In the fourth patient, a lateral medullary infarct (white arrow) is seen with hyper-intensity on both DWI and T2-weighted scans.
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Early subacute infarction of the caudate head and lentiform nucleus

Both structures are supplied by the lateral lenticulostriate arteries, which originate from the MCA. The caudate nucleus is also supplied by the medial lenticulostriate arteries, which arise from the ACA. The recurrent artery of Heubner, which supplies the anteromedial caudate nucleus and the anteroinferior internal capsule, is the largest of the medial lenticulostriate arteries, and originates near the anterior communicating artery. This early subacute lacunar infarct is better visualized, in terms of the specific region involved, on MR. The infarct manifests both vasogenic and cytotoxic edema.
Hypertensive hemorrhage

A large acute parenchymal hematoma is seen on the initial CT in this patient (left upper image), with its epicenter in the right putamen. There has also been extravasation of blood into the ventricular system, with hemorrhage seen in the frontal horns, third ventricle, and atria of the lateral ventricles. On the follow-up CT 3 weeks later, the hematoma is smaller and is in transition from hyperdense to isodense to brain. On the MR obtained after an additional 3 weeks, the hematoma is high SI on both T2- and T1-weighted scans (consistent with methemoglobin), with a hemosiderin rim seen on the T2-weighted scan and a thin peripheral rim of enhancement (black arrow) post-contrast. Both are characteristic features on MR in a subacute hematoma, with the enhancement blending into the margin of the high signal intensity hemorrhage.
Parenchymal hemorrhage on MR, the spectrum of appearance

Initially, but for a very short time, a parenchymal hemorrhage contains oxygenated hemoglobin and is seen as a fluid collection, with slight hyperintensity on a T2-weighted scan. A large, basal ganglia, hyperacute hemorrhage (black asterisk) illustrates this appearance. There is a small amount of associated vasogenic edema and marked mass effect upon the right lateral ventricle. In the next patient, FLAIR scans are shown both on presentation and long term follow-up. This large, acute, left external capsule hematoma evolves from a fluid collection containing deoxyhemoglobin (white asterisk, with low signal intensity on T2-weighted scans), to a hemosiderin lined cleft (also low signal intensity). Note the associated vasogenic edema and mass effect in the acute stage.
Parenchymal hemorrhage on MR, the spectrum of appearance

The third patient illustrates a subacute, extracellular methemoglobin hematoma, with high signal intensity on both T2- and T1-weighted scans, in the left parietal region. Note the peripheral hemosiderin rim, with low signal intensity, already present on the T2-weighted scan. The fourth patient illustrates on an unenhanced T1-weighted scan a methemoglobin hematoma in the right occipital lobe. This case emphasizes the importance of looking for the cause of a hemorrhage, as the post-contrast scan reveals the associated enhancing metastasis (black arrows).
Late subacute, enhancing, PICA infarct

There is slight hyperintensity on DWI, which proved to be T2-shine through (not true restricted diffusion) in a portion of the arterial territory of PICA on the right. Hyperintensity is noted on the axial FSE T2-weighted scan in this region, with only subtle increased signal intensity on the coronal FLAIR. On the post-contrast coronal scan there is enhancement of the associated gyri, somewhat wedge shaped in appearance and predominantly involving gray matter.
New infarction bordering a chronic infarct

The axial FLAIR and FSE T2-weighted scans reveal extensive chronic deep white matter ischemia, together with generalized cortical atrophy and more focal atrophy in the left MCA posterior division, reflecting a prior chronic infarct. There is also abnormal high SI adjacent to the largest area of cystic change, which could simply reflect gliosis due to this old infarct. However, the diffusion-weighted image demonstrates prominent abnormal high SI (confirmed on the ADC map to be restricted diffusion) circumferential and medial to this region, consistent with an early subacute infarct (representing extension of the prior infarct).
Aneurysms, Vascular Malformations, and Other Lesions

Transitioning to the topic of vessel abnormalities, characteristic imaging findings including pulsation artifacts and flow voids are illustrated, together with partial thrombosis. The exhibit concludes with discussion of several less common entities, all with characteristic and easily recognizable imaging presentations, including specifically myxomatous emboli, carotid cavernous fistula, and venous thrombosis with venous infarction.
ACA aneurysm

Note the prominent pulsation artifact in the first image (a FSE T1-weighted scan at 3 T), seen propagating in the left-right dimension. On the postcontrast image (a short TE GRE scan), the aneurysm is well depicted, other than partial volume effects (due to the 4 mm slice thickness). This 2D scan is relatively immune to pulsation artifacts due to the short TE (2.9 msec in this instance). Thick MIP axial and coronal sections from a 3D TOF exam depict the aneurysm well, with both A2 segments originating from the aneurysm, and absence of the left A1 segment.
A flow void is noted in the region of the anterior communicating artery (ACOM) on an axial T2-weighted image, with signal intensity corresponding to arterial flow on an axial source image from a 3D TOF MRA. CE MRA images further confirm the aneurysm, with the right A1 segment of the ACA noted to be very small in diameter. DSA confirms that the aneurysm has a wide neck, an important finding for treatment planning.
A predominantly hyperintense lesion is noted on the right, anterior to the pons, causing mild adjacent deformity. Additional scans identify the abnormal high SI to represent methemoglobin in a SCA aneurysm that is predominantly thrombosed and has only a small, residual patent component (medially). The TOF exams, with thick coronal and axial MIPs illustrated, visualize both the methemoglobin (with slight hyperintensity) and the small patent portion of the aneurysm. DSA identifies the patent lumen of the aneurysm, but not the much larger thrombosed component.
Several small lesions with peripheral hemosiderin are noted on the axial T2-weighted scan, one with marked associated vasogenic edema. Comparison of axial source images from a TOF exam, both prior to and with contrast enhancement, reveals a small focus of enhancement within one of the previously noted lesions. A lateral projection from DSA confirms multiple peripheral cerebral aneurysms – due to myxomatous emboli, with several visualized in the presented thick section, coronal MIP images from an enhanced TOF MRA.
The images are all from a single patient. In part 1, the axial CT reconstructed with a bone algorithm reveals complete opacification of the sphenoid sinus (by blood in this instance) in this trauma patient, together with a fracture of the wall of the sinus on the left (white arrow). Fractures were also noted of the left occipital bone and petrous apex (not shown). The axial CT and MR through the orbits demonstrate an enlarged superior ophthalmic vein (white asterisk) on the left. This is seen as a flow void (dark) on the MR.
Carotid cavernous fistula, traumatic

In part 2, prominent flow voids are seen within, together with engorgement of, both cavernous sinuses (white arrows). The frontal DSA projection from a left internal carotid artery injection reveals the shunt into the left cavernous sinus, with filling as well of the contralateral cavernous sinus and retrograde filling of the left superior ophthalmic vein (black arrow). There is prominent filling of the inferior petrosal sinuses bilaterally (black asterisks).
Venous thrombosis with venous infarction

Vasogenic edema is noted in the right temporal lobe (arrow), but specifically not in an arterial vascular distribution. There is a small parenchymal hemorrhage, with low signal intensity on the T2-weighted scan, seen within the infarct on the axial image. This venous infarct was the result of dural sinus thrombosis, with clot (asterisk) well depicted on multiple images, including specifically within the superior sagittal sinus on coronal images and the right transverse sinus on the axial T1-weighted scan.
Quiz case (presentation)
Quiz case (additional imaging)
Recurrent MCA aneurysm following surgical clipping. On the unenhanced CT, there is a question of a focal abnormal high-density lesion adjacent and just lateral to the surgical clip, placed 13 years earlier for a proximal MCA aneurysm. Post-contrast, a round 16 mm diameter, enhancing lesion is noted, consistent with a recurrent aneurysm. A second aneurysm is identified on DSA, a 4 mm multi-lobulated aneurysm of the left PCOM. The latter is best depicted (arrow) on the volume rendered projection. Both aneurysms were occluded with platinum microcoils, with DSA presented both prior to and following coiling.
Some other problems today...

• Time pressures
  – image acquisition
  – scan interpretation
• Proliferation of imaging techniques
• Increasing scanner complexity
• Need for standardization
  – imaging plane
  – acquired scans
• Decrease in reimbursement
Use of automatic image alignment prospectively is strongly recommended.

The angle between the straight axial plane and the midline is crucial in imaging examinations. Inconsistent head positioning of the patient by the technologist can lead to the difficult interpretation of the images. To assess the merit of a standardization of imaging plane, and tilted axial MR scans were compared in the midline and found to be superior for imaging available or when an abnormality in the posterior fossa is suspected.

Standardization of imaging plane
Standardization of the axial imaging plane, enabling improved evaluation of follow-up exams. Watershed infarcts are presented on FLAIR the week following clinical presentation, and at 3 months. The upper row of images are the original axial images, and the lower row reformatted to match imaging plane. By standardization of the displayed imaging plane the larger deep white matter infarct on the right (white arrow) can now be easily identified on the follow-up exam, and its evolution assessed. Comparison of a cortical lesion in the superior parietal gyrus on the left (black arrow), with standardization of the presented plane, reveals extensive resolution of edema with only a pinpoint area of gliosis remaining.
Pearls and pitfalls regarding the interpretation of MR and CT in patients with cerebrovascular disease are presented, focusing on entities seen in clinical practice, but occurring less frequently and thus often leading to confusion. This selected group of cases covers the less commonly involved arterial territories (for ischemia), the varied appearance of hemorrhage, and key imaging findings in aneurysms and vascular malformations.