Reversal of Abnormal Ischemic Vascular Enhancement after Intracerebral Angioplasty

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Summary: We describe ischemia-related cerebral vascular enhancement on contrast-enhanced MR images that resolved after correction of a flow-limiting lesion. A brief literature review, highlighting some of the original work in this area, is included.

Index terms: Arteries, transluminal angioplasty; Brain, ischemia

Many articles have been written about enhancement patterns seen on contrast-enhanced magnetic resonance (MR) images in patients with cerebral infarction. Abnormal arterial enhancement has been described as one of the initial findings in acute ischemia (1). We present a case of marked abnormal cerebral vascular enhancement that resolved after correction of an underlying vascular stenosis.

Case Report

A 58-year-old woman had two episodes of left-sided monocular blindness, each lasting approximately 4 hours. Her medical history included polymyalgia rheumatica, erythema nodosum, and temporal arteritis. Results of a physical examination done at the time of presentation, including a detailed neurologic examination, were normal. CT findings were unremarkable, but a cerebral arteriogram revealed a 5-mm saccular aneurysm of the left anterior choroidal artery and a 4-mm aneurysm involving the posterolateral aspect of the left internal carotid artery at the level of the origin of the superior hypophyseal artery.

A craniotomy was performed, during which the left anterior choroidal artery aneurysm was clipped, and the left internal carotid artery/superior hypophyseal artery was wrapped circumferentially with muslin held in place with a Yasargill miniclip (an ordinary aneurysm clip could not be placed for technical reasons). The patient did well postoperatively and was asymptomatic until approximately 4 months after surgery, when numbness of the face and right arm and expressive aphasia developed.

Arteriography at this time revealed a critical stenosis of the left supraclinoid internal carotid artery (Fig 1A), which was thought to be caused by perivascular fibrosis resulting from the prior surgery. A contrast-enhanced T1-weighted MR image, although limited by the susceptibility artifact in the surgical bed, showed intense vascular enhancement over the left cerebral hemisphere (Fig 1B).

Preparatory to angioplasty, 500,000 units of urokinase were infused locally through a Tracker microcatheter to ensure that the stenosis did not represent an area of mural thrombus. Angioplasty of the area of stenosis was performed successfully with the use of a 2-mm microballoon through an 8F base catheter in the internal carotid artery (Fig 1C). The patient had no complications. A repeat MR image 1 day after the procedure revealed some diminution in the left hemispheric vascular enhancement. The patient’s signs and symptoms abated and she was discharged 3 days after the procedure on anticoagulation medication. A follow-up MR image 1 month later showed resolution of the vascular enhancement (Fig 1D). Given the clinical circumstances and related time course, the enhancement was thought to most likely represent a transient phenomenon related to regional ischemia.

Discussion

Soon after its introduction, contrast-enhanced MR imaging began to be used in the assessment of ischemia and infarction. Initial studies with this technique by such authors as Crain et al.(1), Elster and Moody (2), and Yuh et al. (3) demonstrated parenchymal enhancement with gadopentetate dimeglumine in ischemic regions usually by about day 7 in the evolution of an infarction. The enhancement was shown to be most intense between the 8th and 18th days by Virapongse and colleagues (4). The mechanism for this parenchymal enhancement was proposed to be related to the leakage of macromolecules from a disrupted blood-brain barrier following reperfusion of an affected area, and this theory was supported by work with acute experimental cerebral ischemia in cats (5).

A more intriguing finding in this area of study was that of vascular enhancement, which could be observed within the first 2 hours after the onset of ischemia (1). This was shown to occur in large vessels supplying cortical distributions and not in terminal branches and vessels supplying noncortical lesions, most likely because of their small size. The mechanism of this enhancement is postulated by...
many authors to be slow flow within arteries distal to an occlusion that are filled by collateral supply and enhance in much the same way as do some cortical veins and the cavernous sinuses (6). This arterial enhancement has been touted as a potentially valuable aid in the early diagnosis of ischemia, often preceding even the development of T2 signal abnormalities (1).

Although initially studied in the setting of ischemia usually progressing to complete infarction, the vascular enhancement sign, as illustrated in the above case, promises to be a useful adjunct to other radiologic and clinical signs both in verifying ongoing ischemia and in determining its course and response to therapy. This is especially so given the advent of cerebral revascularization techniques, such as thrombolysis and angioplasty.

References