DIFFUSE CONTRAST ENHANCEMENT ON MR IMAGES IN BRAIN INFARCTION: “PSEUDOTUMOR SIGN”

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ABSTRACT

The purpose of this study was to describe the pattern of diffuse enhancement seen on contrast-enhanced MR images in patients with subacute infarction. A retrospective study of 104 patients with the diagnosis of stroke who had undergone contrast-enhanced MR scanning within 2 weeks of the inciting neurological event revealed 66 patients who demonstrated different patterns of contrast-enhancement in the region of infarction. Diffuse enhancement was seen in the cerebellum and occipital regions in 12 patients. As this diffuse enhancement could be confused with enhancement seen in primary or metastatic tumors of the brain, the term “pseudotumor sign” was used for this type of enhancement. We concluded that subacute infarct should be included in the differential diagnosis of tumors when this imaging pattern is observed.

Keywords: Infarction, pseudotumor, diffuse enhancement.


INTRODUCTION

The role of MR imaging in the evaluation of cerebral infarction has been thoroughly studied.1-10 Although gadopentetate dimeglumine (Gd-DTPA) has been used to evaluate disruption of the blood-brain barrier, and the different patterns of enhancement, such as early parenchymal, gyriform and intra-arterial, associated with cerebral infarction have been described,11,12,20,21 diffuse enhancement especially in the cerebellar hemisphere has not been reported. The purpose of our study was to describe the pattern of diffuse enhancement associated with subacute cerebral infarction, which can be confused with diffuse enhancement of tumors.

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Fig. 1. A 21 year old man presented with ataxia. Two weeks prior to MRI he underwent four-vessel cerebral angiography to rule out dissection. Axial (A) and coronal (B) MR images obtained after administration of contrast material show diffuse enhancement of left cerebellar hemisphere that follows the PICA vascular territory. Diagnosis was left cerebellar infarction.

Fig. 2. 74 year old woman with left homonymous hemianopsia of 10 days' duration. Axial MR images (A, B) obtained after administration of contrast material one week after occipital infarction show diffuse enhancement of the right occipital lobe. Sagittal MR image (C) obtained after administration of contrast material, 4 weeks later, shows an area of encephalomalacia with peripheral enhancement, confirming the diagnosis of infarction.
Fig. 3. 35 year old woman who had a gradual onset of right homonymous hemianopsia of two weeks. Contrast-enhanced T1-weighted MR images obtained in axial (A) and coronal (B) planes show diffuse enhancement of the left occipital lobe.

Postcontrast, as well as multi-echo T2-weighted in the axial plane, using the standard spin-echo technique. Postcontrast images were obtained immediately after infusion of contrast. Coronal images were available in some cases. Two neuroradiologists independently reviewed the MR images. Diffuse enhancement was called regardless of its homogeneity or enhancement. We included four patients whose MR images showed diffusely enhancing lesions of other etiologies such as meningioma and metastasis, for the purpose of comparison.

RESULTS

Diffuse enhancement was seen in the cerebellar hemisphere in seven patients (Fig. 1), and in the occipital lobe in 5 patients (Figs. 2-3). The time interval between clinical ictus and imaging of both groups ranged between 8 to 14 days. No diffuse enhancement was seen within the first week of ictus. The diffuse enhancement was nonhomogenous with irregular borders in four cases, and homogenous with smooth borders in eight cases. All showed high signal intensity in proton density and T2-weighted images. The diffuse enhancement patterns mimic the enhancement seen in some of the brain tumors such as meningioma (Fig. 4) and metastasis (Fig. 5).

DISCUSSION

The complex circulatory abnormalities associated with cerebral infarction have been the subject of many clinical and experimental investigations.\textsuperscript{9,11,14,17,18} Interruption of blood flow due to thromboembolism causes distal microstasis within 2 minutes of occlusion. This is followed by arteriolar-capillary block, which causes slow emptying of arterial channels. These factors cause decreased regional cerebral blood flow. Subsequently, hypoxia, hypercapnia, and acidosis cause loss of autoregulation of the leptomeningeal vessels and a regional compensatory vasodilatation develops rapidly and increases regional cerebral blood volume. Approximately 1 week after infarction an increase in cerebral blood flow occurs which is due to formation of collateral circulation.
Contrast-enhancement has been attributed to damage of the blood-brain barrier and to dislodgment of clot, collateral circulation, and compression of the capillary bed caused by vasogenic edema.\textsuperscript{14}

The appearance of infarction on contrast-enhanced MR imaging as a function of the time since the inciting neurologic event has previously been well established in the literature.\textsuperscript{2,10-16,19,21} In our study we experienced similar findings in regard to arterial enhancement and early and late parenchymal enhancement which was nicely described by Yuh, et al.\textsuperscript{3} and Elster, et al.\textsuperscript{12} However, our main focus was on diffusely enhancing infarcts which can be confused with diffuse enhancement seen in tumors. In fact, a patient was scheduled for operation in our institution with the diagnosis of meningioma which turned out to be an infarction on a follow-up study. The fact that subacute infarction can mimic neoplasm, particularly within two weeks of infarction, and can demonstrate both mass effect and contrast-enhancement is well known, and contrast-enhanced CT scans of these tumor-like infarcts have been described in the literature.\textsuperscript{22}

Fig. 4. 48 year old patient who presented with headache and gradual onset of left-sided hemianopsia. Contrast enhanced MR in axial plane demonstrates diffusely enhancing lesion involving right occipital lobe. At surgery, diagnosis was meningioma.

Contrast-enhancement has been attributed to damage of the blood-brain barrier and to dislodgment of clot, collateral circulation, and compression of the capillary bed caused by vasogenic edema.\textsuperscript{14}

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Fig. 5. 57 year old man with lung cancer who presented with headache and visual disturbances. Axial (A) and sagittal (B) MR images obtained after administration of contrast material show nonhomogenous but diffuse enhancing lesion involving left occipital lobe. Pathologic specimen obtained at surgery proved to be a metastasis from lung carcinoma.
However, the contrast-enhanced MR of these infarcts, which demonstrate more dramatic enhancement than that of CT and can be more easily confused with tumor, have not been published. In our study the tumor-like enhancing infarcts occurred mainly in the cerebellar hemispheres and occipital lobes.

Differences in the mechanisms of diffuse enhancement in the cerebellar cortex versus gyriform enhancement in cerebral cortex are related to anatomic differences. In the cerebrum, a number of convoluted eminencies, called gyri, are separated from each other by depressions (fissures or sulci) of various depths. The outer surface of each convolution, as well as the sides and bottom of the sulci between them, are composed of gray matter. The inferior portion of each convolution is composed of white matter. When cortical infarcts occur in the cerebral hemispheres, contrast enhancement follows the gyriform pattern of cortical sulci. In the occipital lobe, because of the close approximation of cortex, the gyriform enhancement can present as diffuse, or sometimes irregular, enhancement (Figs. 2-3). These areas of enhancement can be mistaken for tumors such as meningioma (Fig. 4) or brain metastasis (Fig. 5). On the other hand, the surface of the cerebellum is not convoluted like the cerebrum. In the cerebellar hemispheres, the cortex is extensively folded and is traversed by numerous thin, curved furrows or sulci. The cortex occupies the surface, and white matter occupies the interior of the cerebellum. This difference in anatomy and the lack of convolutions in the cerebellum may be the reason for diffuse enhancement (Fig. 1). The following well-established criteria should be used to differentiate diffuse enhancement associated with infarction from that associated with tumor: (1) tumors usually have a gradual clinical presentation while infarction presents with acute onset, (2) infarcts tend to predominate in gray matter, and tumors in white matter, and (3) tumors do not respect vascular boundaries; infarction follows vascular distribution. However, in some cases despite using the above criteria, it can be very difficult to differentiate tumors from subacute infarction, which therefore necessitates follow-up imaging in 1-2 months (Fig. 2c).

In conclusion, brain infarction especially in the cerebellar hemisphere and occipital lobes can show diffuse enhancement on contrast-enhanced MR images, and should be included in the differential diagnosis of diffusely enhancing lesions such as brain tumors.

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