

Unilateral Paresthesia after Isolated Infarct of the Splenium: Case Report

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Abstract-

Purpose: We describe a patient who presented with unilateral paresthesia after acute isolated infarct of the splenium.

Case report: A 74-year-old woman presented with acute onset of right sided numbness and tingling. MR imaging of the brain showed hypointensity on T1-weighted images and on apparent diffusion coefficient maps, and hyperintensity on T2-weighted and on diffusion-weighted images in the splenium, suggestive of acute infarction. MR angiography showed narrowing of left posterior cerebral artery with decreased branches. On detailed high cortical function assessment, she did not have frontal lobe dysfunction, alien hand syndrome, apraxia, optic ataxia, cortical sensation dysfunction, alexia, agraphia, visual field defect, nor color agnosia.

Conclusion: Infarcts of the splenium are not common. Splenial lesion may be associated with altered mental status, ataxia, recent seizure, hemispheric disconnection, and dysarthria. The presentation of isolated unilateral paresthesia makes the patient different from those previously described. The paresthesia could be caused by selective lacunar infarcts in the diencephalic and mesencephalic regions or could be caused by the diaschisis in the parietal cortex. Single photon emission CT may be indicated to provide further information of central nervous system dysfunction in splenial lesion.

Key Words: paresthesia, splenium, diaschisis

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INTRODUCTION

Splenium originates in the Latin spleni-um, from the Greek splenion meaning a bandage, patch or compress⁽¹⁾. The splenium is the posterior fifth of the corpus callosum, and it consists of an upper and a lower part. The

human corpus callosum is a crucial structure providing interhemispheric communication between hemispheres of the brain. In most lesion confined to the splenium, only the visual part of the disconnection syndrome occurs.

Infarcts of the splenium are not common⁽²⁾. The vas-

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cular supply to the splenium is the posterior pericallosal artery (also known as splenial artery). The infrequent involvement of the splenium is possibly due to the posterior pericallosal artery (branch of the parieto-occipital, calcarine, or medial posterior choroidal artery of the posterior cerebral artery) forming interarterial anastomoses with branches of the pericallosal artery (branch of the anterior cerebral artery)⁽³⁾. Clinically, splenial infarcts may present as an acute process or with gradually worsening non-localizing neurological signs.

We describe a patient who presented with right-sided paresthesia after acute isolated infarct of the splenium and try to elucidate the possible mechanism of the paresthesia.

CASE REPORT

A 74-year-old non-vegetarian woman, with past medical history significant for hypertension and diabetes mellitus, presented with acute onset of right face, arm and leg numbness and tingling. The unilateral paresthesia persisted for one day before she sought medical consultation. On admission, she was alert and oriented. She had no fever, meningismus, headache, or signs of head injury. Blood pressure was 154/86 mmHg. Neurological examination and the results of basic laboratory tests were unremarkable. Initial non-contrast brain CT performed upon admission showed no obvious lesion. MR

imaging of the brain performed one day later showed hyperintensity on T2-weighted (Fig. 1A) and on diffusion-weighted images (Fig. 1B) in the splenium, and hypointensity on T1-weighted images and on apparent diffusion coefficient map (Fig. 1C), suggestive of acute infarction. There is an old infarct at pons. MR angiography showed irregularity and narrowing of left posterior cerebral artery with decreased branches (Fig. 2). On detailed high cortical function assessment, she did not have frontal lobe dysfunction, alien hand syndrome, apraxia, optic ataxia, cortical sensation dysfunction, alexia, agraphia, visual field defect, nor color agnosia.

DISCUSSION

The exact underlying pathophysiology of splenium lesions is not fully understood. In human, the splenium of the corpus callosum receives fibers from the caudal two-thirds of the temporal lobe, including the temporal pole, superior and inferior temporal gyri and parahippocampal gyrus, and fibers from the occipital cortex pass through the inferior portion of the splenium⁽⁴⁾. As the fibers emerge from the splenium, they trace out several different pathways within the white matter and diverge around the lateral ventricles. The principle destinations of the inferomedial projections connect the left and right vertical meridian representations of the visual field maps (V1/V2). The large set of dorsal projections

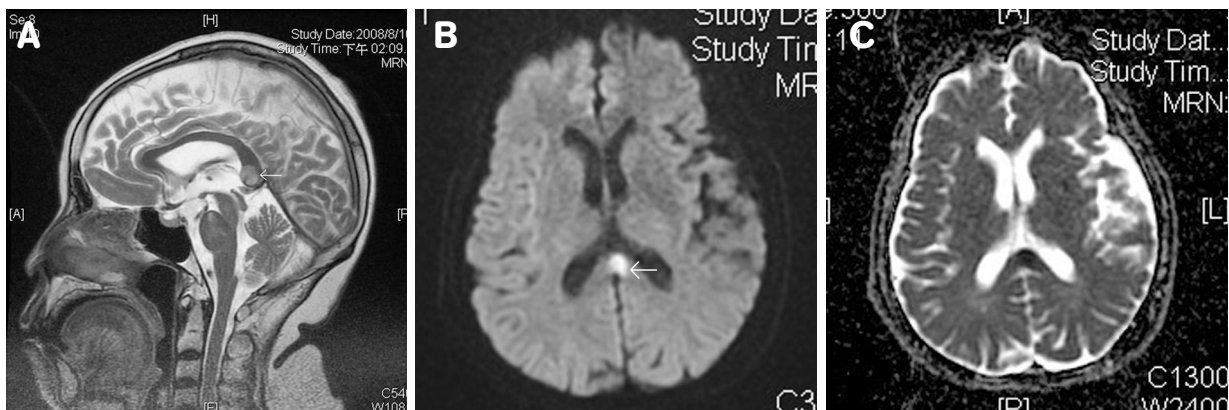


Figure 1. Sagittal T2-weighted MR image (A) showing hyperintensity in the splenium of the corpus callosum (arrow). Axial diffusion-weighted MR image (B) showing hyperintensity in the splenium (arrow) with hypointensity on apparent diffusion coefficient map (C), which corresponds to the lesion on T2-weighted image, suggestive of acute infarction.



Figure 2. MR angiography showing irregularity and narrowing of left posterior cerebral artery with decreased branches (arrow).

spans much of the intraparietal sulcus⁽⁴⁾. In the splenium, fibers conveying information about letters, colors and visuo-spatial localization are following a ventro-dorsal lamination⁽⁵⁾.

The splenium is a clinically silent area. In the literature review, splenial lesion may be associated with altered mental status, ataxia, recent seizure, hemispheric disconnection (apraxia of the left hand, pseudoneglect, alien left hand, astereognosis, agraphia, alexia, visual apraxia, and hemianopsia), dysarthria, anisocoria and hemiparesis⁽⁶⁾. The presentation of isolated unilateral paresthesia makes the patient different from those previously described.

Multiple causes can result in the splenium changes seen on MRI diffusion-weighted imaging (DWI). The possible causes include glucose and electrolytes abnormalities, renal failure, alcohol and malnutrition, seizure, ongoing infection or parainfectious process, brain tumor, chemotherapy, stroke, altitude sickness, demyelinating disease, substance or antiepileptics exposure, and head trauma⁽⁶⁾. The DWI signal changes suggest restricted movement of free water and the reduced ADC values suggest cytotoxic edema. In this patient, the hypersignal intensity on DWI images with the reduced ADC values in the splenium suggests acute infarction.

The sensory disturbance in this patient could be easily explained by selective infarction in the diencephalic and mesencephalic regions which are fed by the proximal thalamic branches of the posterior cerebral artery even without other detectable MR imaging abnormalities except the splenial lesion. However, sensitivity of DWI for ischemic stroke detection is reported up to 94% ~ 100%⁷⁻⁸. The possibility of this assumption is low.

We propose that the functional depression of metabolism in the parietal cortex caused by a decreased neural input in acute splenial infarction as a result of disconnection (diaschisis) may be another possible explanation of the paresthesia. The diaschisis is interpreted as functional disruption without morphological changes in regions remote from the site of damage. With the advent and development of functional neuroimaging, the functional specialization is not such a fixed property of brain lesions as previously supposed⁽⁹⁾. Thus, this concept of diaschisis might provide a possible explanation of diverse clinical manifestations of the splenial lesion except the merited disconnection syndrome. One recent study by Noboru et al.⁽¹⁰⁾ demonstrated splenial lesion may have generate more widespread cerebellar and frontal dysfunction without corresponding MRI abnormalities in a patient with central nervous system (CNS) Legionnaires' disease. Although the pathogenesis differs from our patient, single photon emission CT (SPECT) may be indicated to provide further information of CNS dysfunction in splenial lesion.

The case demonstrated unilateral paresthesia after acute isolated infarct of the splenium. The paresthesia could be caused by selective lacunar infarcts in the diencephalic and mesencephalic regions or could be caused by the diaschisis in the parietal cortex. SPECT may be indicated to provide further information of CNS dysfunction in splenial lesion.

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