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A Case Report of Bilateral MCP Infarction due to Ipsilateral Vertebral Artery Occlusion

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Abstract

We report a 62-year-old man with the presentation of progressive vertigo, diplopia and then dysarthria, nausea and lower limbs paresis. On evaluations, he was found to have acute infarction of bilateral middle cerebellar peduncles. Diagnostic workups for the underlying cause revealed ipsilateral occlusion of vertebral artery that is a rare presentation. We discuss this rare stepwise presentation of bilateral MCP infarction and possible underlying pathophysiology and workups that we performed to rule out underlying causes.

Keywords: Vertigo; Middle cerebellar peduncle; MCP; Infarction; Stroke

Case presentation

A 62-year-old gentleman with known hypertension and diabetes mellitus presented with progressive vertigo, diplopia and vomiting from 30 days before admission, which had been intensified in the last ten days and had been accompanied with dysarthria, nausea and sudden lower limbs paresis leading to walking disturbance. Neurological examination on admission was notable for dysarthria, slurred speech, nystagmus, left sided facial paresthesia and ataxia. Due to our high suspension to cerebrovascular accident (CVA), a brain computed tomography (CT-scan) without contrast was performed, which revealed bilateral hypodense areas at middle cerebellar peduncle (MCP) region. At the time, Brain magnetic resonance imaging (MRI) revealed multiple lesions with restriction in bilateral pre-pontine regions and bilateral cerebellar hemispheres and a few of the mentioned regions showed post-contrast enhancement which were suggestive of ischemic lesions.

His brain and cervical CT-angiogram revealed thrombosis of distal section of the V2, originating from the left vertebral artery, at the site of foramen neural and the artery has been occluded thoroughly at V3 and there is vivid evidence of acute thrombosis in distal vertebral artery. However, they have reported that right side basilar and vertebral arteries are intact, which is a quite rare presentation in this context. Magnetic resonance spectroscopy (MRS) test reported no neoplastic or inflammatory activities as well.

With ischemic stroke being confirmed, anticoagulation and aggressive risk factor control was started.

Since bilateral MCP ischemic infarction is so rare, comprehensive investigations for the underlying cause of this phenomenon had been performed, having cardiac, infectious, inflammatory and paraneoplastic etiologies in mind.

For investigation of paraneoplastic causes, peripheral blood smear (PBS), spiral lung and mediastinum CT scan and abdominal and pelvic sonogram was performed and showed no abnormality.

In order to evaluate infectious and inflammatory activities, which might have made him prone to thrombosis formation and vasculitis, we ran a couple of laboratory tests including hepatitis B and C evaluations, CSF Coombs Wright, blood culture, HIV

Ag/Ab, aerobic bactalert, urine culture and other routine tests that they were all reported in their normal range.

Finally, for evaluating cardiac abnormalities, we carried out transesophageal echocardiogram, resulting no vegetation or clot, and his ejection fraction (EF) was estimated 50-55%. Also ECG and 24-hour cardiac holter monitoring showed no abnormality. Hence, all underlying reasons were ruled out and our patient was discharged with oral anticoagulant and improved condition. His diplopia was recovered completely, however he still had a slight ataxia on discharge.

Discussion

The results of imaging studies, alongside with clinical symptoms, revealed an infarction corresponded to the middle cerebellar peduncles (MCP). Isolated MCP infarction is an extremely rare phenomenon, comprising about 0.12% of all strokes (1). Other etiologies for bilateral MCP involvement include acute osmotic demyelination syndrome, early stages of Wilson's disease, acute toxic leukoencephalopathy (e.g.,heroin intoxication) or wallerian degeneration of MCP after 3-4 weeks of acute pontine insult, all of which have been ruled out (2).

MCP is predominantly supplied by the AICA, but it also gets a contribution from the SCA which anastomoses the terminal branches of the AICA, hence, creating a watershed area (1). It can explain the development of MCP infarction by decreased flow in AICA artery, while the SCAs flow preserves through collateral arteries from the anterior circulation (1).

Isolated bilateral MCP infarction have been described in only a few case reports (1-5). Based on these reports, hypoperfusion in AICAs with preserved flow of SCA due to collaterals from anterior circulation is considered the main etiology, considering the MCP being a watershed zone between AICA and SCA (1, 2). It also have been proposed that MCP infarction could be resultant of superimposed thrombosis over previously atheromatous stenosis, which is located on AICA or basilar artery (5).Our case was affected by ipsilateral vertebral artery, which is a quite rare accident in this context and make our case even more interesting. The treatment mainly comprises of antiplatelet therapy and anticoagulation. More recently, stent implantation in the occluded artery has become an essential therapeutic option however, there is still lack of information on this field and needs further researches.

Abbreviations: MCP: middle cerebellar peduncle, CSF: Cerebrospinal fluid, AICA: anterior inferior cerebellar artery, SCA; Superior Cerebellar artery

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