

BILATERAL CEREBELLAR STROKE WITH GOOD FUNCTIONAL RECOVERY: A CASE REPORT

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ABSTRACT

Bilateral simultaneous infarction in the territories of the posterior inferior cerebellar arteries (PICAs) is rare. The cause of these infarcts is believed to be atherosclerotic or embolic occlusion of a dominant PICA, which perfuse the territories of the medial branches of both PICAs. We report a case of an elderly hypertensive male with bilateral cerebellar infarcts involving the territory of PICA. The clinical course, imaging results, and laboratory findings are presented.

INTRODUCTION

Ischemic strokes in the posterior circulation are caused by atherosclerosis or embolism. Cerebellar infarction accounts for 2% of acute strokes and in order of frequency are posterior inferior cerebellar artery (PICA) 40%, superior cerebellar artery (SCA) 35%, border zone infarcts 20% and anterior inferior cerebellar artery (AICA) 5%.^{1,2} Strokes in the distribution of posterior inferior cerebellar artery (PICA) are rare. PICA arises from the intracranial vertebral artery and supplies the lateral medullary tegmentum, inferior cerebellar peduncle, the ipsilateral portion of the inferior vermis and the inferior surface of the cerebellar hemispheres. The medial branch of PICA supplies the medial cerebellum and the dorsal medulla oblongata, and the lateral branch supplies the inferoposterolateral aspect of the cerebellum.

The clinical presentation of acute cerebellar infarcts depend upon the arterial territory involved and on the presence or absence of fourth ventricular and brainstem compression. The patient usually presents with sudden onset of occipital headache, severe vertigo, nausea, vomiting, ataxia of gait and trunk, ipsilateral axial lateropulsion, dysarthria and impairment of consciousness. Brainstem compression results in increasing headache, decreased level of alertness, head tilt and tonsillar herniation through the foramen magnum. These infarcts usually involve PICA, AICA or both. Embolism (cardiac or arterial) and thrombosis (local

atherosclerotic disease) account for most cerebellar infarction.

CASE HISTORY

We report a case of 65 years old gentleman, a known case of hypertension, who presented with sudden onset of headache and vomiting for three days and unresponsiveness for one day. Headache was generalized, radiating to the back of neck, severe in intensity, throbbing in nature and associated with 5-6 episodes of vomiting. There was no diplopia, seizures, tinnitus or loss of consciousness. Initially he was taken to a nearby hospital where he was treated as a case of hypertensive encephalopathy. During the hospital stay, he developed slurring of speech, difficulty in walking and his level of consciousness deteriorated so he was shifted to our hospital for further management.

On examination, he was drowsy, obeying single step command and had a left gaze preference. Pupils were 2mm and reactive to light, and he was moving all four limbs with bilateral extensor plantar response. Rest of the systemic examination was unremarkable.

His initial labs including complete blood count, electrolytes, urea, creatinine, blood sugar, coagulation profile and ECG were normal. Urgent MRI and MRA brain was done that showed acute ischemic infarction involving

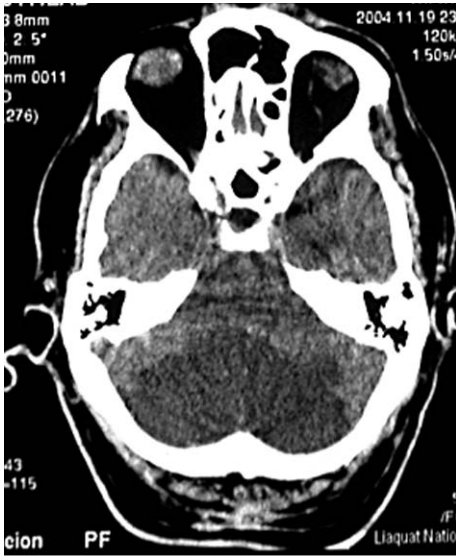


Figure 1: CT scan axial image showing hypodense area involving lower half of both cerebellar hemispheres representing acute ischemic infarction.



Figure 2: MRI T2W sagittal view showing abnormal signal intensity area involving lower cerebellar hemisphere



Figure 3: T2W axial view of the same patient



Figure 4: MRA showing segmental areas of narrowing in posterior circulation. The posterior inferior cerebellar arteries are not visualized

bilateral cerebellar hemisphere extending up to cerebellar vermis, associated with mild cerebellar tonsillar herniation.

He was shifted to ICU where he was managed conservatively with intravenous fluids and started on intravenous heparin. Next day, heparin infusion was stopped as he developed hematuria and was given Aggrenox (aspirin/extended release dipyridamole).

Transthoracic echocardiogram was performed that showed an ejection fraction of 50 - 55%, with normal LV size and mild septal hypertrophy. He was found to be dyslipidemic and hence was started on lipid lowering agent. The patient made a gradual recovery and his conscious level improved. Physiotherapy and occupational therapy was started subsequently. He was discharged and was advised a regular follow up.

DISCUSSION

Cerebellar infarction in the territory of posterior inferior cerebellar artery (PICA) is usually unilateral, as the origin of PICA arises from a single vertebral artery (VA). Very few patients with acute bilateral cerebellar infarcts in the territory of PICA have been described in literature to date. Different hypotheses have been put forth to explain the pathogenesis of bilateral cerebellar infarcts in the PICA territory:

1. Both PICAs arising from an occluded basilar artery
2. Branches to both PICA regions arising from one side
3. Pressure effect caused by a large PICA infarct
4. Hemodynamic mechanism with hypoperfusion in the most peripheral branches; and
5. Double, simultaneous embolic stroke.

PICA is the most variable cerebellar artery.³ It is absent in 20% of VA angiogram; in the majority of these instances, the AICA supplies the PICA territory. In cases when both PICAs are asymmetrical, branches of one PICA partially feed the territory of the other. Furthermore, an "extensive" PICA may supply the cerebellum bilaterally.

In our patient, the cardiac source of embolism was absent and the second hypothesis seems to be the most probable mechanism of bilateral PICA infarcts. The clinical features and the favorable outcome seen in our case were also similar to the series presented before.⁴⁻⁶

CONCLUSION

Bilateral cerebellar infarction is a life threatening condition. However, few patients survive with a good functional recovery. We believe that our patient had a dominant PICA which supplied medial branches of both PICAs and hence caused bilateral stroke

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