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CASE REPORT

Case Of Bilateral Cerebellar Infarction In A Middle Aged Male Patient

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ABSTRACT

Cerebellar infarctions are relatively rare and account for ≈2% of all ischemic strokes. Possible pathogeneses include cardiac emboli, atherosclerosis of the great vessels, dissection of the vertebral artery, local arterial disease and, less commonly, hypercoagulable states, vasculitis, venous sinus thrombosis, and drug use.Cerebellar infarcts require notable observation because of the risk of cerebral edema in the posterior fossa. The initial symptoms of cerebellar infarction or cerebellar hemorrhage may be nonspecific, such as headache, dizziness, nausea, vomiting, and vertigo. > Fifty percent of cerebellar infarctions are associated with nausea and vomiting, and ≈75% of these are associated with dizziness. The clinical and imaging features of patients with cerebellar infarction may be helpful in clinical decision-making.

KEYWORDS: cerebellar infarction; cerebrovascular accident; coagulopathy; ischemic stroke

BACKGROUND

Cerebellar infarction (or cerebellar stroke) is a cerebrovascular event that primarily affects the cerebellum and the posterior fossa. Cerebellar strokes are responsible for a disproportionate amount of morbidity and mortality, despite accounting for a minute proportion of all strokes. 1-4% of all cerebral strokes are cerebellar strokes. [1] In a study of nearly 2,000 consecutive stroke patients, the mortality rate for cerebellar infarcts was nearly twice as high as for the more common cerebral strokes (12.5%), with brainstem infarcts falling in the middle at 17 percent. [2]

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The primary determinant of neurologic deficits is the physiologic function of the affected vascular territories. In addition to precipitating and attenuating factors, the timing of symptom onset and progression may help identify cerebellar strokes and differential diagnoses. Approximately 75% of patients report "vertigo" in some form, characterized by dizziness or falling to one side. Due to ataxia or focal or systemic "weakness," numerous patients complain that they cannot walk or have difficulty walking. Over fifty percent report nausea or vomiting. In most instances, the severity of the symptoms exceeds that of the examination findings. These neurologic findings may include localization signs like a trunk or limb ataxia, cranial nerve defects like diplopia or nystagmus (often directional or caused by eye movements, more concerning if vertical or torsional), and dysarthria. Unlike cerebral infarcts, these conditions are typically ipsilateral (on the same side of the patient as the cerebellar stroke).
In some cases, a cerebellar stroke is not diagnosed due to the nonspecific nature of its symptoms. The lesion's location and size primarily determine its appearance. Cerebellar lesions must be diagnosed with a comprehensive history and neurologic examination. [3]

CASE PRESENTATION

A 52-year-old man was brought to the emergency room of a hospital connected with Santosh Medical College after experiencing sudden-onset headaches, vomiting, and vertigo over the course of two days.He had a five-year history of hypertension and was receiving rough treatment. During the admission examination, the patient was found to have focal neurological deficits, such as dysarthria and ataxia. Biochemistry and hematology were unremarkable, with no evidence of infection. The patient's initial systolic blood pressure was greater than 160 mm Hg. He felt nauseous due to his persistent vertigo. To rule out hemorrhage, an NCCT was performed, which revealed a well-defined lobulated hypodense area with surrounding mild hyperdensity in the cerebellum, along with mass effect and small lacunar infarcts in the left periventricular region and left corona radiate (Figures A-E). He was administered dual antiplatelet medication and transferred to the Intensive Care Unit for observation. The next day, aspirin and clopidogrel were withheld in favor of LMWH in case neurosurgical intervention was required. His echocardiogram was normal, with an EF of 55% and no RWMA in the left ventricle. Homocysteine levels were normal at 22 mcg/L; BT, CT, and PT/INR were also normal. His fundus was examined to rule out elevated ICP. Later, a contrast-enhanced MRI was performed, which revealed multiple large bilateral (Right>Left) cerebellar infarcts involving multiple cerebellar territories and vermis. MR spectroscopy reveals a restriction in diffusion and a significant lactate peak, indicating an acute cerebellar infarct with chronic ischemic changes and a small chronic lesion in the periventricular region. The neurosurgery review recommended intravenous mannitol and monitoring for signs of an elevated ICP. After seven days of observation, there were no indications of cerebellar edema, which typically manifests as a

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decreased level of consciousness and naturally occurs within 1 to 7 days, with an average peak of 3 days. He was prescribed a dual antiplatelet agent. His condition was diagnosed as an idiopathic cerebellar stroke, and he underwent community-based rehabilitation.

DISCUSSION

Although cerebellar infarctions are uncommon, they carry a high morbidity risk for patients. A young person's stroke can have devastating effects in terms of lost productive years and diminished quality of life. To improve the diagnosis of cerebellar infarction, clinicians should conduct a comprehensive neurologic examination and be well-versed in all relevant brain imaging techniques. A multifaceted approach is required to improve the management and outcomes of young adult stroke. [4] Aspirin (81 mg) and ticagrelor (180 mg under stress followed by 90 mg twice daily) are used daily for 30 days for the treatment of acute stroke, after which ticagrelor is discontinued, and aspirin (81 mg) is continued. [5]

CONCLUSION

Cerebellar infarcts are treated similarly to other ischemic cerebrovascular accidents; the clinical and imaging characteristics of patients with cerebellar stroke may aid clinical decision-making. Clinically significant cerebellar edema, typically manifested by a decreased level of consciousness, typically develops within 1 to 7 days, with a peak occurring on average after three days. A patient is eligible for thrombolysis with recombinant tissue plasminogen activator if the onset of an acute event occurred within 4.5 hours (rtPA). However, this is frequently not possible, given the difficulty in diagnosing posterior wall infarction. Patients should undergo a diagnostic evaluation to determine the infarction's cause. This includes an echocardiogram, screening for stroke risk factors such as diabetes mellitus, hypertension, hyperlipidemia, and antiplatelet or anticoagulant therapy. Magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) of the brain is the gold standard for evaluating cerebellar infarction. This allows for visualizing poor blood flow and tissue damage indicators. MRA can also localize vasoconstriction and guide endovascular treatment in the event of a large vessel occlusion, which is especially helpful in basilar artery occlusion.

Competing interests None declared.

Patient consent for publication was obtained.

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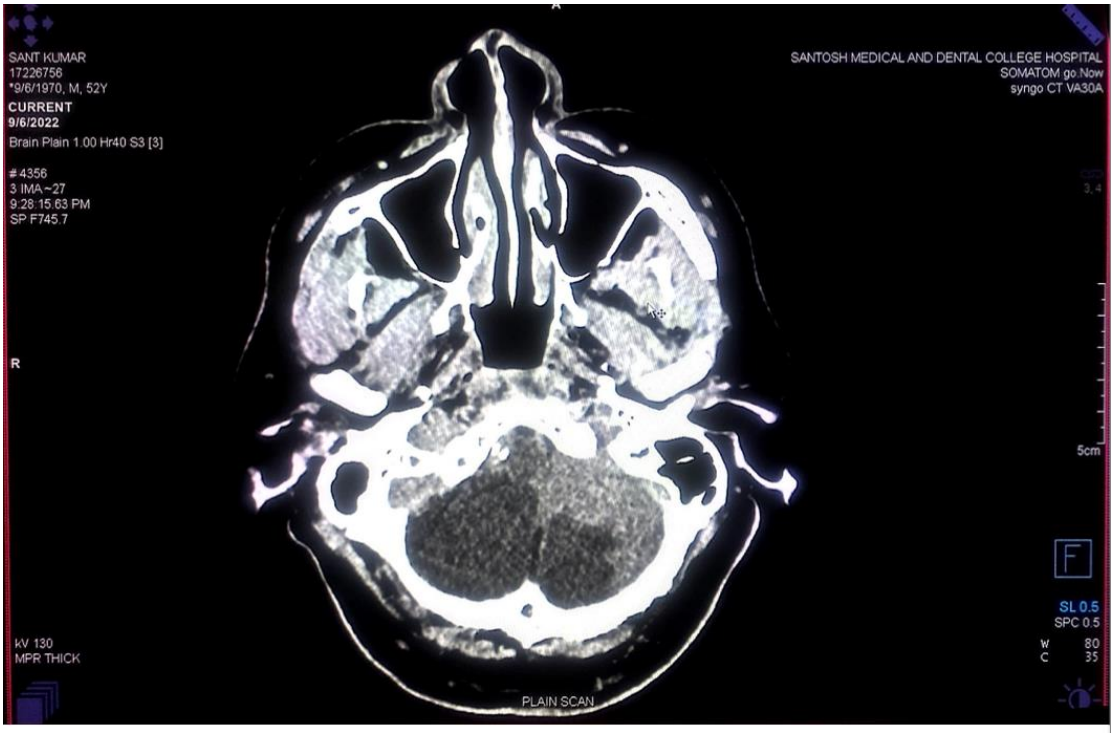
KEY POINTS

- The mortality rate for cerebellar infarcts was nearly twice as high as for the more common cerebral strokes.
- Cerebellar infarcts are rare in young adults with bilateral and multiple cerebellar involvements.
- Increased awareness of the symptoms of cerebellar infarction and knowledge of appropriate imaging techniques aid in the early diagnosis and treatment of cerebellar infarctions.



(A) NCCT head image showing acute intracranial pathology-a well-defined lobulated hypodense area with surrounding mild hyperdensity in the cerebellum with mass effect with small lacunar infarct in the left periventricular region and left corona radiata.

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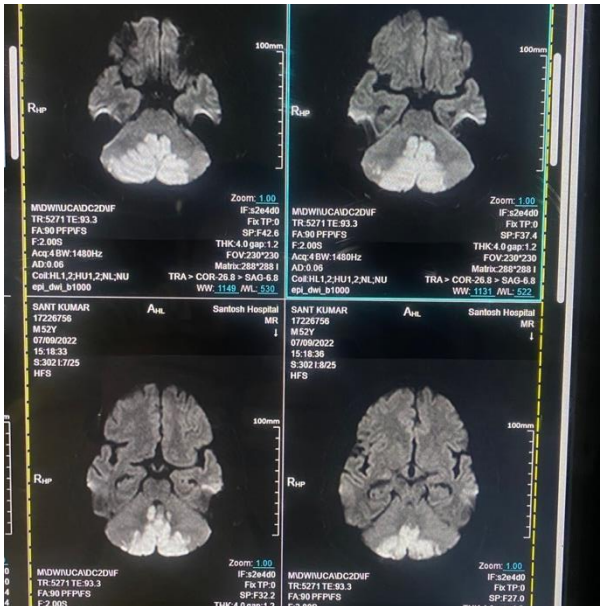
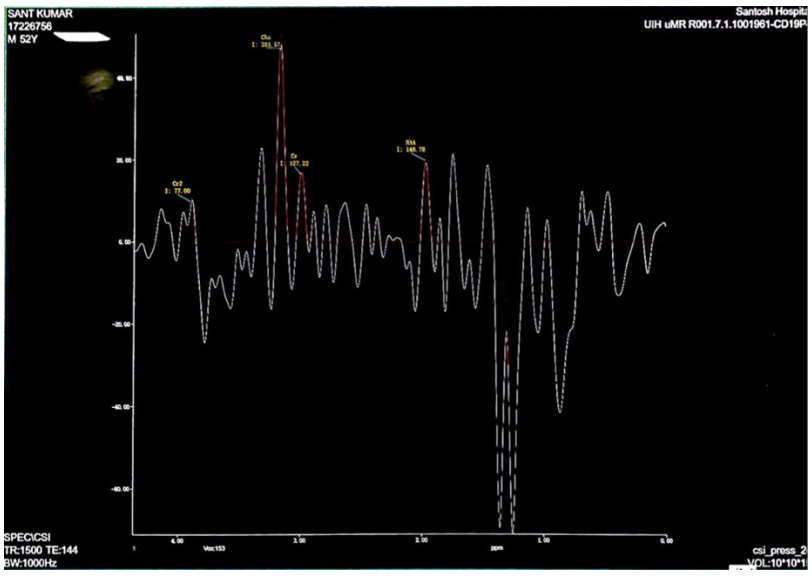


(B)The MRI image showed multiple large areas of restricted diffusion on both sides of the cerebellar hemispheres, suggestive of acute cerebellar infarction.



(C) significant lactate peak on MR spectroscopy

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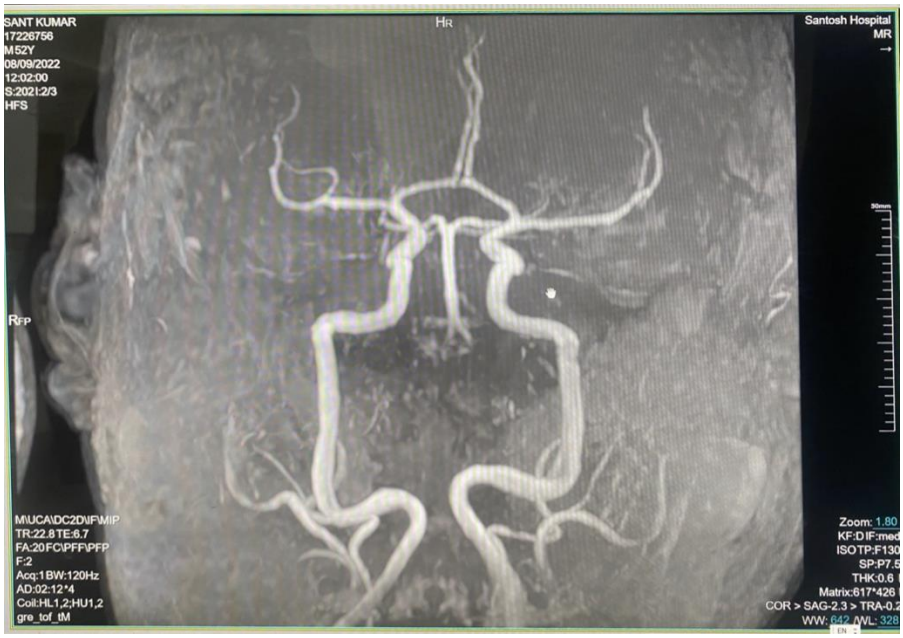
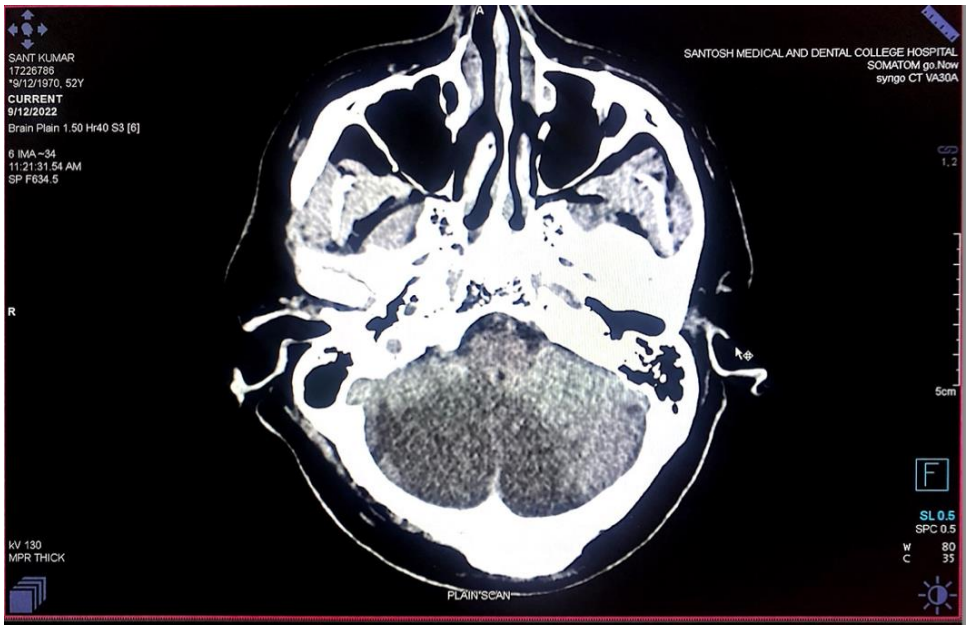


(D) Magnetic resonance arteriography demonstrated regular appearances of intracranial arteries with stenosis of basilar arteries.



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(E) Well-defined lobulated hypodense area surrounding mild hyperdensity in the cerebellum with mass effect. No significant time interval change was seen compared to the previous CT dated 06/09/2022.

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