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A Clinical Case Of Bilateral Medial Medullary Infarction Presenting With "Airpod"Sign

Abdellatif Bekraoui, Yassine Lamrini, Fatima Akkaoui, Omar Ghomari Khayat, Zahra Ferjouchia, Mariam Jilla, Rachid Belfkih.
Department Of Neurology At The University Hospital Mohammed Vi Of Tangier, Morocco

Abstract:
Bilateral medial medullary infarction (MMI) is an exceedingly uncommon cerebrovascular event, accounting for only 1% of confirmed ischemic strokes. This particular stroke subtype elicits profound neurological deficits, including alterations in consciousness, tetraplegia, and cranial nerve involvement, frequently leading to dysphagia and respiratory distress, thereby posing a life-threatening risk.. While advancements in brain MRI technology have enabled the diagnosis of bilateral MMI in recent years, identifying it remains exceptionally challenging.
Herein, we present a clinical case outlining a patient who exhibited a severe and rapidly progressing condition over a 72-hour period. The patient demonstrated a range of symptoms, including tetraplegia, dysarthria, respiratory distress and dysphagia.
Key words: *Stroke, medial medullary infarction, brain MRI.*

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I. Introduction:

Medial medullary infarction (MMI) constitutes less than 1% of all cases of cerebral infarctions and bilateral occurrences are exceptionally rare. MMI typically manifests acutely or subacutely, characterized by dysarthria, dysphagia, and quadriplegia, occasionally accompanied by respiratory complications, leading to a grim prognosis[1]. While brain MRI findings have facilitated the diagnosis of bilateral MMI in recent years, it remains challenging to identify.

The medial structures of the medulla oblongata receive vascular supply from the paramedian branches of the anterior spinal artery, originating from the vertebral artery, a branch of the subclavian artery. Medial medullary syndrome, also referred to as Dejerine syndrome, arises from an infarction affecting the medial portion of the medulla. This syndrome was first described by Joseph Jules Dejerine in 1915 (1).

In this report, we describe a clinical case detailing a patient who presented with a severe and rapidly deteriorating condition within a three days period. The patient manifested various symptoms, including tetraplegia, dysarthria, respiratory distress, and dysphagia.

II. Clinical Case:

A 64-year-old woman, with a medical history of hypertension and obesity, presented to the emergency department with the sudden onset of right hemiplegia. Upon arrival, she was conscious, hypertensive with a blood pressure of 160/90 mmHg, afebrile, and displayed right hemiparesis and dysarthria on neurological examination. Laboratory investigations yielded normal results, and electrocardiogram (ECG) showed a regular sinus rhythm. Brain magnetic resonance imaging (MRI) revealed a lesion characterized by hyperintensity on diffusion-weighted imaging and FLAIR sequences, alongside reduced apparent diffusion coefficient (ADC) values, consistent with a distinctive "AirPod sign" appearance suggestive of bilateral medial medullary infarction (MMI). Time-of-flight (TOF) magnetic resonance angiography demonstrated a diminutive caliber of the right vertebral artery with absence of visualization of the anterior spinal artery. Antiplatelet therapy was promptly initiated.

However, after 48 hours, there was a deterioration in neurological status with the onset of tetraparesis and respiratory distress. Consequently, the patient was transferred to the intensive care unit and underwent tracheostomy placement. Unfortunately, she succumbed to infectious pneumopathy two weeks later.

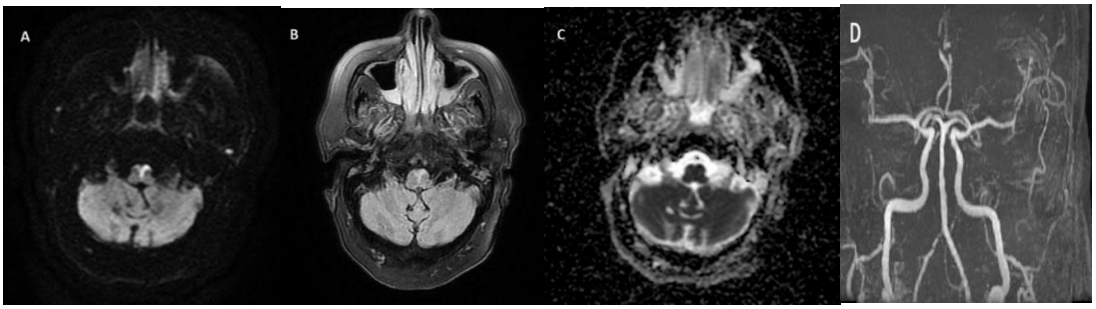

Figure 1: Diffusion-Weighted Imaging (A) And FLAIR (B) Sequences Showing Bilateral Hyperintense Signal In The Medullary Pyramids Associated With ADC Restriction (C) Consistent With A Characteristic "Airpod Sign". Time-Of-Flight (TOF) Magnetic Resonance Angiography Demonstrated A Diminutive Caliber Of The Right Vertebral Artery With Absence Of Visualization Of The Anterior Spinal Artery.

III. Discussion:

Bilateral medial medullary infarction (BMMI) typically results from occlusion or stenosis of the vertebral arteries or the paramedian branches of the anterior spinal artery. This vascular insult leads to ischemia and infarction of the bilateral medial medullary structures, including the pyramidal tracts, medial lemnisci, and hypoglossal nuclei. The intricate vascular anatomy and watershed zones within the medulla predispose certain regions to ischemic injury, resulting in the characteristic clinical features observed in BMMI (1).

The clinical presentation of BMMI varies depending on the extent and localization of the infarcted areas within the bilateral medial medulla. Common neurological deficits include quardriparesis/quardriplegia due to involvement of the corticospinal tracts, impaired sensation of touch, vibration, and proprioception from disruption of the medial lemnisci, and dysarthria, dysphagia, and tongue weakness resulting from hypoglossal nerve dysfunction. Additionally, patients may exhibit signs of brainstem dysfunction such as impaired consciousness, respiratory distress, and autonomic instability (2).

The imaging features of bilateral medial medullary infarction (BMMI) typically involve distinct findings on magnetic resonance imaging (MRI). These include the presence of infarcts in the medial medulla of the brainstem, often visualized as a "heart appearance" sign on diffusion-weighted imaging (DWI) (3). Additionally, high-resolution MRI may reveal thromboembolism in the vertebral artery, particularly in cases where BMMI is associated with vascular pathology. The identification of these imaging features is crucial for the accurate diagnosis and management of these patients. The predominant mechanisms commonly observed include large-artery atherosclerosis and small penetrating artery disease, notably branch disease. It was noted that a significant portion, comprising over one-third (38%), of the patients exhibited no detectable abnormal vascular findings. Bilateral medial medullary infarctions may be associated with either extensive thrombosis at the vertebrobasilar junction or variations in the anatomy of perforator branches supplying the medulla. Additionally, variations in the anterior spinal artery (ASA) originating from the vertebral artery (VA) may contribute to the occurrence of simultaneous bilateral MMI (4).

When comparing unilateral medial medullary infarctions MMI to bilateral MMI, the latter manifest more commonly with bilateral weakness, bilateral hypoglossal palsy, and respiratory failure. Large-artery atherosclerosis and branch disease remain the predominant stroke mechanisms observed in both unilateral and bilateral MMIs. However, in contrast to unilateral MMIs, the clinical prognosis of bilateral MMIs is generally unfavorable. The prognosis is contingent upon factors such as the velocity, site, and size of the thrombus, the degree of cerebral edema, the presence or absence of occlusive vascular disease in other areas of the posterior circulation, and the collateral circulation. Acute respiratory failure and quadriplegic muscle strength graded as are strong indicators of a poor prognosis (5).

Unfortunately, there is a limited number of reports about intravenous thrombolytic therapy being administered for bilateral medial medullary infarction (BMMI). The reasons behind the absence of thrombolytic therapy administration were analyzed to be associated with delayed treatment, failure to promptly diagnose the condition, and missing the critical time window for thrombolytic intervention (1).

IV. Conclusion:

The clinical presentation of this condition is intricate, featuring early symptoms that are often atypical and a notable incidence of respiratory dysfunction. Simple CT-scan carries a risk of misdiagnosis or overlooking the condition. Hence, further magnetic resonance imaging (MRI), particularly with diffusion-weighted imaging (DWI) sequence, is imperative, as it can reveal high signal intensity in the lesion area during the earliest stages of infarction. For a more comprehensive etiological diagnosis, computer tomography angiography (CTA) or magnetic resonance angiography (MRA) are viable options to delineate the affected artery. High-resolution MRI

(HR-MRI) advancements offer a novel approach to vascular assessment. Moreover, this disease typically carries a poor prognosis, with outcomes influenced by the rate of disease progression, infarct location, size, and collateral circulation. Early diagnosis and intravenous thrombolytic therapy may enhance patient prognosis.

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