ORIGINAL SCIENTIFIC ARTICLES

Evolution of a Lateral Medullary Syndrome to a Bilateral Medial Medullary Infarct: A Case Report

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ABSTRACT

Medial medullary infarction is a rare type of stroke and is usually secondary to a large artery atherothrombosis and small penetrating artery disease. Bilateral medial medullary infarction is even less frequent with a much poorer outcome. We present the case of a 55-year-old Filipino male, hypertensive, who initially complained of vertiginous dizziness with signs and symptoms of right lateral medullary infarction. Within 24 hours of hospitalization, patient's symptoms evolved into a bilateral medullary infarct. Up to this date, this is the first case report of a Wallenberg syndrome that evolved into a bilateral medial medullary infarct.

BACKGROUND

Medial medullary infarction is an uncommon type of stroke accounting for only about 0.5 – 1.5% of all strokes.[15] Bilateral medial medullary infarction (MMI) is even rarer with a poorer prognosis.[3,15] Classical triad of MMI include contralateral hemiparesis, contralateral deep sensory impairment, and ipsilateral tongue weakness.[3,4] Patient with bilateral MMI syndrome usually present with quadriplegia, bilateral loss of proprioception, hypoglossal palsy, dysphagia and speech difficulties.[3,15] Both stroke syndromes are often secondary to a large artery atherosclerosis and branch occlusion disease.[15] Wallenberg syndrome on the other hand, is a wedge like shaped infarction on the lateral medullary posterior to the inferior olivary nucleus. It is mostly secondary to atherothrombotic occlusion of penetrating branches associated with distal vertebral artery disease.[9,14] Through the advent of neuroimaging, particularly the Magnetic Resonance Imaging has proven to be useful in arriving at the correct neuroanatomical diagnosis.[3,5,7]

We report here a case of a middle-aged man who was initially admitted as a case of lateral medullary syndrome whose symptoms evolved into a bilateral medial medullary infarction within 24 hours of hospitalization based on clinical and MRI features.

OBJECTIVE

This paper aims to present a case of a lateral medullary infarct which evolved into a bilateral medial medullary infarct in a hypertensive adult male.

CASE PRESENTATION

We present the case of a 55-year old, hypertensive, male who came in at the emergency room (ER) with a one-day history of vertiginous dizziness and non-projectile vomiting after alighting from a jeepney with associated veering to the right while walking. Patient self-medicated with one tablet of amlodipine 10mg, which provided no relief of symptoms. Upon waking up in the morning, patient still had vertiginous dizziness not aggravated by head movement. He then began

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to complain of hoarseness of voice and dysphagia. Patient was ambulatory but with gait imbalance and decided to seek consult in our institution. Neurological examination at the ER revealed pseudoptosis on the right, a 50% sensory deficit in pinprick and temperature modalities on the right side of the face, unequal palatal elevation with uvula deviated to the left, a weak gag reflex with a 50% sensory deficit to pain and temperature on the left extremities, and limb ataxia on the right. Patient also had horizontal nystagmus to the left. There was no lateralizing weakness or Babinski sign, and vibratory and position sense were intact. Plain cranial CT scan was unremarkable; however his clinical presentation pointed to a lateral medullary infarction on the right. Hence patient was subsequently admitted and was started on double antiplatelets.

However, during the first hospital day, there was progression of his neurologic findings. The patient presented with right peripheral facial palsy, had absent gag reflex, and difficulty of tongue protrusion with weak movements on all sides. Patient became quadriplegic with a Manual Muscle Test (MMT) grading of 2/5 on all extremities. He also presented with hyporeflexia, bilateral Babinski, and bilaterally impaired vibratory and position sense.

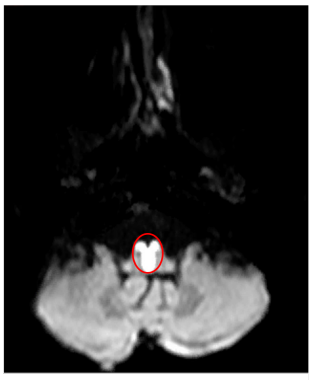
Accurate neuroanatomical localization of the lesion is crucial because appropriate and timely management of posterior circulation strokes direct neurological outcomes, thus the adage "Time is Brain". Cranial MRI was subsequently done as shown in Figure 1.

During his entire hospital stay, patient was awake but with persistent quadriplegia. The patient was eventually intubated due to dyspnea and desaturation and he was subsequently subjected to have a tracheostomy tube placed due to the prolonged need of oxygen support via endotracheal tube. After about a month of hospitalization, patient was discharged with improvement of motor functioning, though still bedridden.

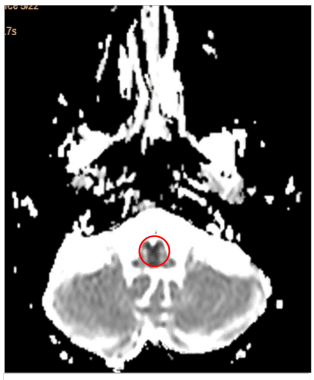
DISCUSSION

Posterior circulation infarction accounts for about 20-25% of all ischemic strokes in the general adult population.[1,2] Medial medullary infarction (MMI) is reported to be less than 1% of vertebrobasilar artery strokes, with only about 18% of such cases occurring bilaterally to involve both medial side of the medulla.[14] Patients with MMI commonly present with bilateral motor weakness (78.4%)[3,14] and has a poor functional outcome. High suspicion and vigilance are necessary otherwise, the patient

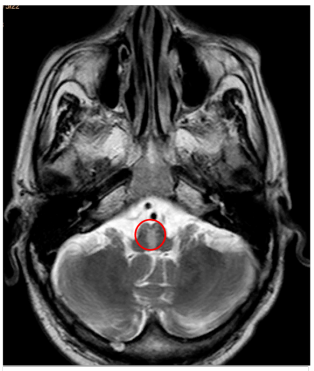
Figure 1. The red circle shows the focal area of restricted diffusion in the bilateral ventro-medial aspect of the medulla with corresponding hyperintense signal on T2 Flair pulse sequence.



Diffusion Weighted Imaging

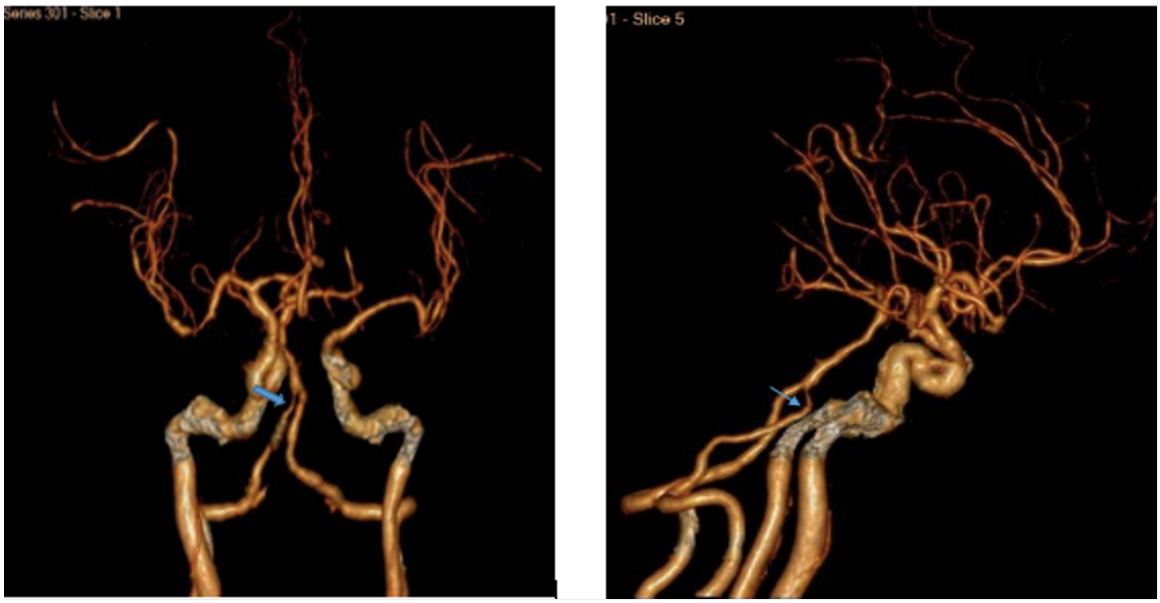


ADC Map



T2 Flair

Figure 2. CT Angiographic results revealed "Distal segment of the right Vertebral Artery is stenotic, with dominant left Vertebral Artery. The right PICA is not visualized". The blue arrow points to the right vertebral artery narrowing.

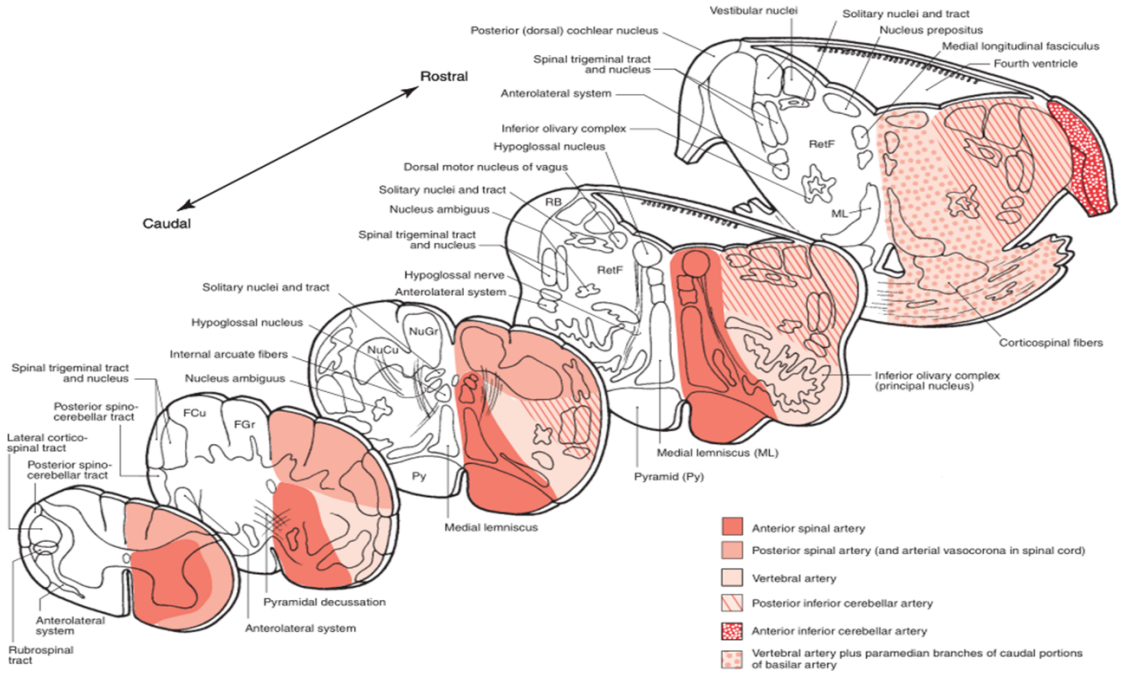


may be misdiagnosed with Guillain-Barre Syndrome during its early course.[3,4]

Blood supply to the medulla oblongata arises mainly from the distal vertebral artery and its penetrating arteries. The dorsal area also receives blood supply from the branches arising from posterior inferior cerebellar artery (PICA).[14] The most rostral part may also receive blood supply from branches arising from basilar artery and anterior inferior cerebellar artery (AICA).[14] The penetrating arteries from anterior spinal artery (ASA) supply the most caudal part of the anterior medulla.[14]

In a large comparative analysis in Japan comparing lateral medullary infarct from medial medullary infarct, LMI accounts

Figure 3. Transverse section of the different levels of the medulla oblongata with the corresponding blood supplies[17]



for 78% of cases while only 19% represents MMI[13]. Lateral medullary infarct is a wedge-like shaped infarction on the lateral medulla posterior to the inferior olivary nucleus.[9] The most common etiology of Wallenberg's syndrome is atherothrombotic occlusion of penetrating branches associated with distal vertebral artery disease (63%).[14] In medial medullary syndrome, it is composed of a triad of symptoms: Contralateral hemiplegia with facial sparing, contralateral loss of position and vibration sense and ipsilateral paralysis of the tongue.[3,15] Large artery atherothrombosis and small penetrating disease remains to be the most common mechanism of infarction.[15]

In bilateral MMI, common presentations include quadriplegia, bilateral loss of proprioception, hypoglossal palsy, dysphagia and speech difficulties. Patients with bilateral MMI can also present with ipsilateral peripheral palsy, and this attributed to the involvement of the uncrossed aberrant corticofacial fibers which descend to the upper-middle medulla prior to their decussation to the contralateral facial nucleus.[7, 16]

Seventy-six percent (76%) of bilateral MMIs involve the rostral part of the medulla.[7] Interestingly, among the different possible vascular territories, the ventromedio-dorsal type of infarction predominates in 41% of cases.[7] A vascular event remains to be the main etiology leading to neurological deficits.[7, 14] Research shows that an atheromatous occlusion of the distal vertebral artery (VA) was incited as the main pathology underlying bilateral medial medullary infarctions.[7] Hence, a distal vertebral artery disease tends to produce a deeper and wider lesion which occasionally results to a bilateral MMI, perhaps due to either occlusion of multiple perforator or more extensive medial medullary hypoperfusion, compared with those of single perforator disease.[7,14]

Bilateral MMI as a poor clinical outcome with dependency rate of 61.9% and mortality rate of about 23.8%. This is due to the presence of a severe motor dysfunction, which is the main predictor for poor outcome.[14]

Currently, the gold standard in diagnosing posterior circulation infarction is Magnetic Resonance Imaging–Diffusion Weighted Coefficient. Within minutes from the onset of symptoms, DWI detects infarcted areas and is seen as hyperintense signals with corresponding hypointensities on Apparent Diffusion Co-efficient (ADC) map compatible with restricted diffusion.[3]

In our case, MR-DWI with corresponding ADC map enabled timely and accurate diagnosis of bilateral MMI. It showed the typical "heart shaped" appearance with accompanying hyperintensity on DWI and T2 sequences on the right lateral part of the medulla. The patient's last follow up was in November 2019, more than a year from his initial symptoms. His best motor improvement was being able to sit with support. Though recovery was slow, modest improvement with his speech was also noted. Eventually, his tracheostomy was closed with no episodes of dyspnea or desaturation.

CONCLUSION

Bilateral medial medullary infarction is a rare stroke which has a poor clinical outcome. Though it occurs due to an atheromatous occlusion of a distal Vertebral Artery, it can also be preceded or simultaneously occur with Wallenberg syndrome. Early diagnosis is of utmost importance to help not only the patient but also the family to better understand the illness given its impact in their quality of life. Appropriate care and support for the patient can be better provided if the quality of life of the family has also been addressed. Hence, diagnosis of such combination warrants vigilance, thorough neurologic examination follow-up and a high index of suspicion together with the aid of cranial MRI to arrive with an accurate neurologic diagnosis.

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