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CASE REPORT **OLGU SUNUMU**

BILATERAL ANTERIOR INFERIOR CEREBELLAR INFARCTION PRESENTING SEVERE VERTIGO AND HEARING LOSS

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ABSTRACT

Anterior inferior cerebellar (AICA) infarctions are rarely seen, and bilateral AICA infarctions are mostly seen in the literature as case reports. Herein, a rare case with bilateral AICA infarction with severe vertigo, nausea and vomiting is presented. A sixty-nine-year-old woman was seen with the complaints of vertigo, tinnitus,nause, vomiting and hearing loss. In neurological examination, a dysarthric speech, horizontal nystagmus, bilateral peripheral facial palsy, right trigeminal hypoesthesia and mild ataxia were also noted. Mild sensorineural hearing loss (%30) was detected in the left ear through audiogram examination. In cranial MR imaging, a bilateral AICA infarction was reported. In addition, there were also bilateral hypoplastic posterior cerebral artery (PCA) and basilar artery stenosis in cranial MR angiography. The sympomts were partially disappeared following anti-platelet treatment. As in this case, in a patient with vertigo, AICA infarct should be kept in mind and detailed neurological examination should be performed.
Key Words: Vertigo, hearing loss, bilateral AICA infarction.

CİDDİ VERTİGO VE İŞİTME KAYBI İLE SEYREDEN BİLATERAL ANTERİOR İNFERİOR SEREBELLAR ARTER ENFARKTI

ÖZET

Anterior inferior serebellar enfarktlar nadir olarak görülür ve bilateral AİCA enfarktları literatürde çoğunlukla olgu bildirimi şeklinde görülür. Burada, ciddi vertigo, bulantı ve kusma ile prezente olan nadir görülen bilateral AİCA olgusu sunulmuştur. 89 yaşında hadın hasta vertigo, tinnitus, bulantı, kusma ve işitme kaybı şikayeti ile başvurdu. Nörolojik muayenesinde, dizartrik konuşma, horizontal nistagmus, bilateral periferik fasiyal paralizi, sağ trigeminal hipoestezi ve hafif ataksi tespit edildi.Sol kulak odiyogram incelemesinde hafif sensorinöral işitme kaybı (%30)tespit edildi. Kraniyal MR görüntülemesinde bilateral AİCA enfarktı saptandı. Ek olarak kraniyal MR anjiografisinde bilateral posterior serebellar arter hipoplazisi ve basiller arter stenozu mevcuttu. Semptomlar antiplatelet tedavi ile kısmen düzeldi. Bu vakadaki gibi vertigo şikayeti olan olgularda, AİCA enfarktları akılda tutulmalı ve detaylı nörolojik muayene yapılmalıdır.
Anahtar Sözcükler: Vertigo, işitme kaybı, bilateral AİCA enfarktı.

INTRODUCTION

Basilar artery plays a crucial role in the nutrition of the brain stem and cerebellum. (1) AICA is the first major branch of the basilar artery. It continues to the internal auditory canal through posterolateral course inside of the cerebellopontine cistern. It nourishes VI, VII, VIII nerves, inferolateral pons, middle cerebellar peduncle, flocculus, and anterolateral surface of the cerebellar hemispheres (1,2). AICA infarctions are so rare in general, however, bilateral forms are much more uncommon. (3). The most common clinical findings are vertigo, nausea, vomiting,

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tinnitus, nystagmus and dysarthria. In some cases, clinical picture may be accompanied with facial paralysis (ipsilateral in unilateral infarctions), trigeminal sensation disorder, Horner's syndrome, hearing loss, ataxia, and ache in body and extremity, loss of temperature (4). As stated, isolated bilateral AICA infarctions are very rare. In this article, a case with bilateral AICA infarction was presented with clinical and radiological findings.

CASE

Sixty-nine-year-old woman applied to hospital with vertigo ongoing for ten days. She stated her vertigo increased with sudden movements and sometimes accompanied by nausea and vomiting. In addition, tinnitus and hearing loss were present in the left ear. Her consciousness was clear, cooperated and oriented during neurological examination. Her understanding was deemed as natural, and her speech was assessed as dysarthric. Bilateral pupils were in normal sizes, and direct and indirect light reflexes were positive. Nystagmus were detected in horizontal gaze to the left, and hypoesthesia was detected in the trigeminal territory in the right. Finally, bilateral peripheral facial palsy was obvious. No pathological findings were observed in walking examination except for mild ataxic, hesitant walk. Nothing was remarkable except for hypertension in her history. Cranial tomography (CT) and magnetic resonance imaging (MRI) examinations were performed. An infarction that is compatible with bilateral AICA in diffusion weighted cranial MR imaging was reported. (Figure 1). No additional pathology was found in routine biochemistry and hemogram tests except for the increase in LDL. Carotid-vertebral doppler and echocardiography were normal. A mild sensorineural hearing loss (30%) was detected in the left ear through audiogram examination. Bilateral hypoplastic PCA was found in cranial MR angiography and stenosis was observed in the central section of the basilar artery (Figure 2). The patient were given anti-platelet and symptomatic treatment, which worked well. Following partial recovery and stabilization, she has no vertigo, ataxia and nistagmus but hearing loss, trigeminal hypoesthesia remained while facial palsy recovered partiallly, so the patient was discharged in the 10th day of hospitalization.

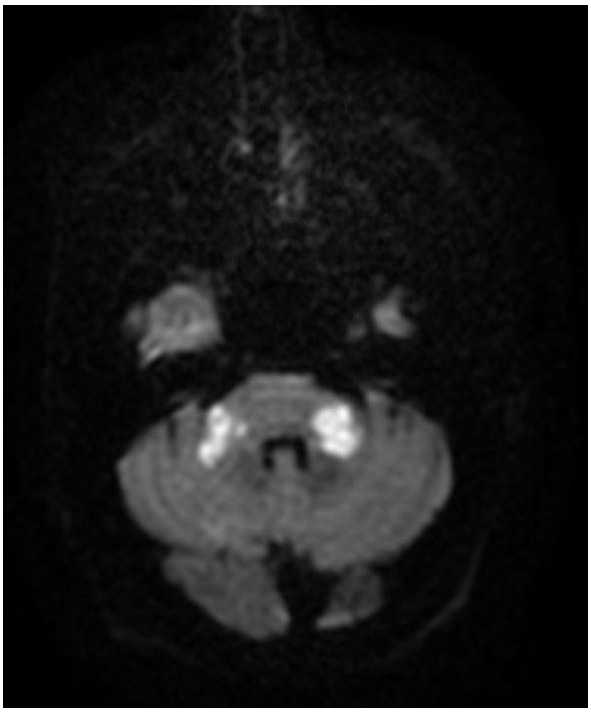

Figure 1. Diffusion–weighted MRI images shows hyperintense foci in the bilateral middle cerebellar peduncle and lateral pons.

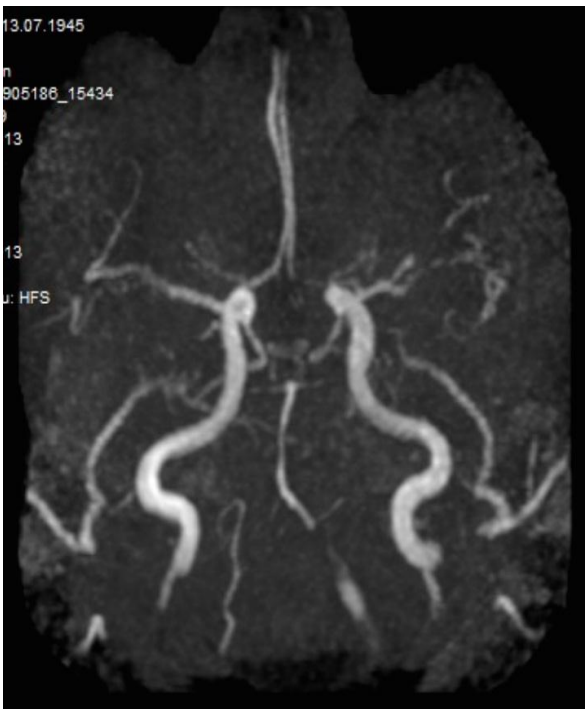

Figure 2. Magnetic resonance angiography images showes right hypoplastic vertebral artery and stenosis of the left vertebral artery and the central section of the basilar artery.

DISCUSSION

Isolated AICA infarctions are rarely seen. Only six cases were reported in a study which documented 1350 cases with posterior system infarction (5). In another study it was reported that AICA formed 0.9% of all the cerebrovascular events and 5.2% of vertebrobasillar infarctions (6). Bilateral AICA infarctions are present only in the form of case reports in the literature. In a study where thirteen AICA infarctions were presented, only three of them were bilateral (7). Lee et al (8) examined the relationship between AICA infarction and hearing loss in a study including 82 AICA infarction cases, and only 3 of them were bilateral. It is stated that AICA infarctions can be developed as a result of thrombotic stenosis in AICA due to atherosclerosis or obstruction of the plaque in basilar artery at the entrance of the AICA. Clinical pictures vary according to the infarction localization in the infarctions compatible with AICA irrigation area. Whereas symptoms such as isolated vertigo mimicking labyrinthitis or ataxia are present in partial AICA, bilateral AICA may follow a severe clinical course which ultimately can cause coma (6,7,8). In our case, although bilateral involvement was present, it was not that severe. However, as seen in partial involvement, severe nausea, vomiting and vertigo were present. The clinical picture was accompanied with cranial nerve involvement and ataxia. In literature, there were few reports about bilateral AICA infarctions. Tsukamoto et al. reported a case with bilateral cerebellar pedincule infarction, which is known to be supplied mostly by anterior inferior cerebellar artery. They presented their case with severe headache, vertigo, nause, nistagmus difficulity in walking, limb ataxia and disatrhria as similar in our case (9). Kattah et al. also reported a case of bilateral AICA infarction with progressive truncal ataxia, frequent falls, dysarthria and episodic vomiting but no vertigo. Therein, the medical treatment was unsuccessful, and ataxia continued. Furtherly they found multifocal basiller artery strenosis in MR angiogram and performed basiller artery stent succesfully (10). In another report, Algin et. al presented a case with acute bilateral hearing loss, vertigo, dysarthria, dsyphagia, hemihypoesthesia, peripheral facial paralisy and ataxia caused by infarction of bilateral AICA. They found basiller artery occlusion in MR angiogram as in our case (11). When compared to Algin's presentation, while severe vertigo, nause, difficulity in walking and hearing loss, partially mimicking isole peripheral vertigo were common, ipsilateal trigeminal and bilateral facial cranial nerve involvement were seen in our case. AICA infarction's clinical picture is often accompanied by hearing loss and it is stated the incidence varies from 30 to 100%. Sudden hearing loss is claimed to occur due to ischemia in the inner ear (12). In our case, similarly, 30% sensorineural hearing loss was detected in the right. It was thought that bilateral AICA infarction in this case was seen as a result of bilateral hypoplastic PCA (confirmed in cranial MR angiography) and stenosis in the central section of the basilar artery due to atherosclerosis. AICA infarction may give clinical symptoms in a wide spectrum, from isolated vertigo caused by a labyrinthitis, to coma findings accompanied by tetraplegia. (13)

Detailed neuorological examination is paramount in patients with vertigo, nausea, vomiting, tinnitus, and hearing loss. Advanced imagining attempts will help in differential diagnosis.

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