■ CASE REPORTS

Sudden-Onset Bilateral Hearing Loss: Case Report

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An ischaemic infarction of the anterior inferior cerebellar artery (AICA) is usually associated with vertigo, hearing loss, facial palsy, nystagmus, or truncal ataxia; it is often accompanied by other brainstem or cerebellar signs. Sudden-onset bilateral hearing loss without associated neurological symptoms is infrequent in the literature. We report a case of sudden bilateral hearing loss, later diagnosed as AICA infarction without other symptoms.

Key words: AICA. Infarct. Sudden-onset bilateral hearing loss.

Sordera brusca bilateral: a propósito de un caso

El infarto isquémico en el territorio de la arteria cerebelosa anteroinferior (AICA) se asocia a vértigo, hipoacusia, parálisis facial, nistagmus o ataxia, suele acompañarse de síntomas neurológicos o cerebelosos y no es infrecuente encontrar afección auditiva concomitante. La hipoacusia bilateral como signo casi único de presentación de infarto cerebeloso es infrecuente y escasamente relatado en la literatura. El caso clínico presentado plantea el diagnóstico de un infarto en el territorio de la AICA, que se inicia como una sordera brusca bilateral.

Palabras clave: Infarto. AICA. Sordera brusca bilateral.

INTRODUCTION

Ischaemic infarction in the territory of the anterior inferior cerebellar artery (AICA) is associated with vertigo, hearing loss, facial palsies, nystagmus, or ataxia.[1]

In affected patients, the neuro-otological symptoms may appear as the first clinical manifestations or prodrome, hence the importance of differentiating them from other aetiologies.

Cerebellar infarctions represent from 2% to 4% of encephalic infarctions, in some cases presenting an aggressive behaviour, with a large oedema acting as a mass effect on the structures of the posterior fossa, and urgent decompression is necessary.[2] The cerebellum is irrigated by 3 pairs of arteries: posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery (AICA), and superior cerebellar artery (SUCA). The associated risk factors are high blood pressure, hypercholesterolaemia, and diabetes. The cause of the infarction depends on the artery affected, with the PICA often associated with embolisms, atherothrombosis, or dissection of the vertebral artery.

Sudden-onset hearing loss is defined as the onset of high-intensity sensorineural deafness appearing in a matter of minutes or days, without any prior history of ear disease and without any clear triggering cause. It is not associated with neurological symptoms and is usually unilateral. Its aetiology is variable and comprises: viral inflammations of the labyrinth, vascular causes, auto-immune causes, syphilis and, in many cases, idiopathic origin. Sudden-onset hearing loss associated with an infarction of the brainstem was described by Goodhart in 1936. AICA infarction is usually accompanied by central or cerebellar symptoms and it is not infrequent to find accompanying auditory involvement.[2]

Bilateral hearing loss, as almost the only presenting sign of cerebellar infarction, appears very rarely in the literature[3] and we have therefore considered the present case to be of interest.

The cause of hearing loss is related with damage in the inner ear.[4] Lee presented a review of 12 patients with infarction of the AICA and sudden-onset deafness, in which he observed that the site involved was the middle cerebellar peduncle and that the hearing loss in most patients was cochlear due to the ischaemia suffered by the inner ear. Adams[5] thought that the damage was to the cochlear nuclei or the auditory nerve.

Bilateral hearing loss with unilateral involvement of the cerebellum might be explained by a lesion to the radicular portion of the auditory nerve, which is irrigated through the auditory branch artery of the AICA, or by small pontine infarctions.[5] Other causes might be atheroma plaques on the basilar artery, which would affect both AICAs, either through a reduction in the flow or else by causing embolisms.

Neuroimaging techniques, such as computerized tomography (CT), magnetic resonance (MR), angiography, or angioresonance are of fundamental importance for the diagnosis and identification of cerebellar infarctions.

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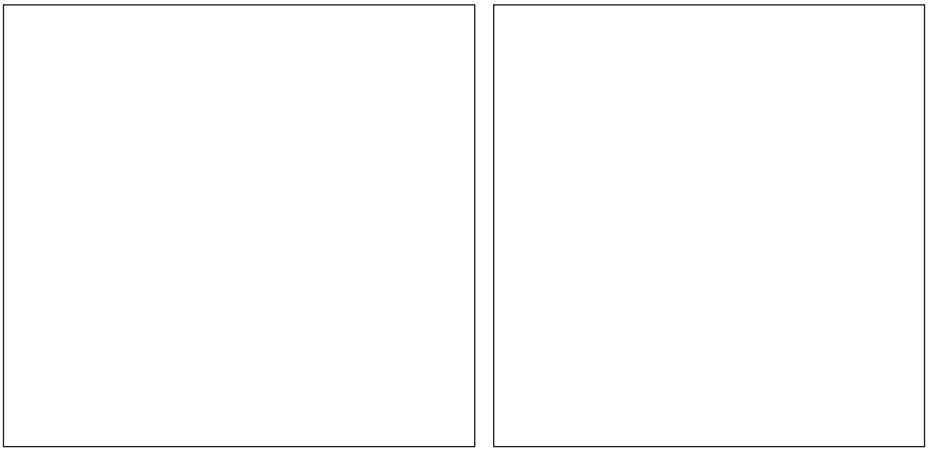

Figure 1. Magnetic resonance image showing the cerebellar ischaemic lesion with involvement of the protuberance.

Audiometry tests, evoked potentials or videonystagmography will help to find the cause of the hearing loss and any subsequent sequelae. Treatment is based on observation in a hospital setting and anti-coagulation therapy.

The prognosis is good as 70% of patients with this condition are able to carry out their daily activities after the procedure.

CASE REPORT

A female aged 69 attended the emergency room because of a 3 hour long situation of rotating vertigo accompanied by nausea without vomiting, associated with tinnitus in both ears, reported as an intense sound of high tonality, and sudden bilateral hearing loss.

Her personal history included: diabetes, high blood pressure, dyslipidaemia, and an acute myocardial infarction 12 years previously with occlusion of the anterior descending artery requiring thrombolysis. There was no prior history of surgery nor any otological involvement and she did not complain of prior hearing loss as she had had good hearing in both ears.

The emergency examination revealed severe bilateral hearing loss, spontaneous left-rotating horizontal nystagmus with the Romberg, and Barany tests showing deviation to the right. No dysmetria or adiadochokinesia was observed in the cerebellar tests and the rest of the cranial pairs and the strength and sensitivity tests were normal. The otoscopy was also normal.

Among the complementary tests performed, the emergency CT of the brain was reported as normal without haemorrhagic lesions or evidence of mass effects or any displacement of the midline. The tonal audiometry test was reported as cophosis of the right ear and profound hearing loss with thresholds of 100 dB HL in the left ear.

In view of the clinical situation, an MR scan was requested and it was reported to show an ischaemic lesion in the right hemisphere of the cerebellum (Figure 1) that also affected the protuberance, compatible with an ischaemic infarction in the territory of the right AICA.

The angioresonance of the supra-aortic trunks revealed virtually complete stenosis at the start of the origin of the right vertebral artery, albeit with distal flow (Figure 2), and a reduced calibre in comparison with the left side; the rest of the vascular structures are normal.

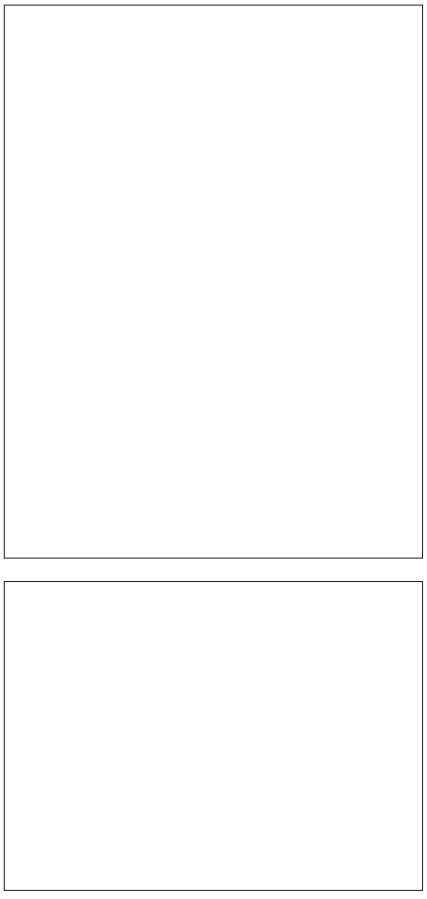

Figure 2. Angioresonance reported as stenosis of the origin of the right vertebral artery (arrow).

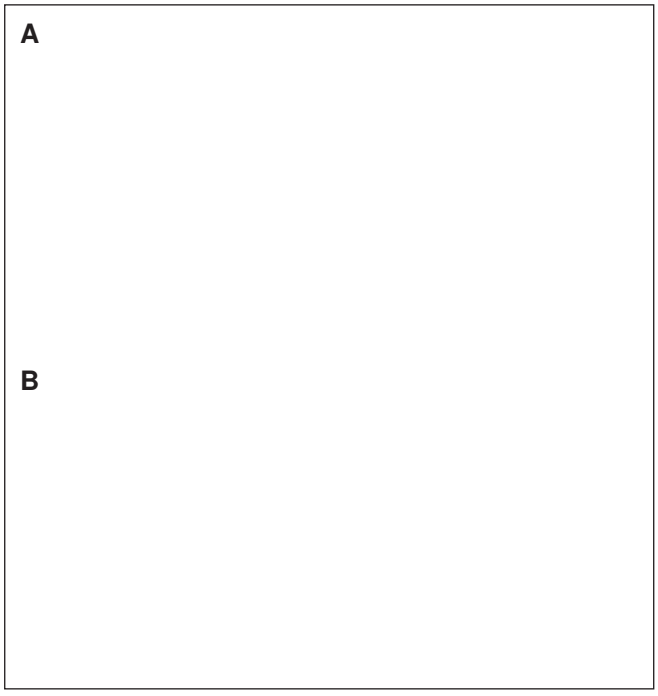

Figure 3. A: audiometry on admission. B: audiometry after 10 days of treatment (unblinded test, with lateralization phenomenon).

The analytical and serological tests for syphilis, ANA, and ANCA were normal, as was the ecocardiogram.

Following admission, the patient began treatment with double anti-aggregation therapy (clopidogrel 75 mg/24 h and acetylsalicylic acid 300 mg/24 h), anti-vertigo therapy (sulpiride 100 mg/8 h), methylprednisolone (60 mg/24 h), and piracetam (4.8 g/24 h). The patient presented good clinical progress, both objectively and subjectively, with a notable improvement in her stability during the vestibulospinal tests. In the tonal audiometry, an improvement was observed in the left ear, but not in the right where severe hearing loss persists (Figure 3B).

It was not possible to continue with the long-term assessment, as the patient did not attend subsequent check-ups.

DISCUSSION AND CONCLUSIONS

Sudden-onset hearing loss is generally a consequence of cochlear lesions. The appearance of bilateral hypoacusia, even if it presents no other associated neurological signs, must lead to the discarding of central involvement. The treatment of cerebellar infarction is based on admission to hospital and anti-coagulation therapy, with a good prognosis in this case.

REFERENCES

1. Lee H. Sudden deafness and anterior inferior cerebellar artery infarction. Stroke. 2002;33:2807-12.
2. Lee H. Sudden deafness with vertigo as a sole manifestation of anterior inferior cerebellar artery infarction. J Neurol Sci. 2004;228:99-104.
3. Deplanque D. Sudden bilateral deafness: lateral inferior pontine infarction. J Neurol Neurosurgery Psychiatry. 1998;64:817-8.
4. Son EJ. Anterior inferior cerebellar artery infarction presenting with sudden hearing loss and vertigo. Laryngoscope. 2007;117:556-8.
5. Adams R. Occlusion of the anterior inferior cerebellar artery. Arch Neurol. 1943;49:765-71.