Anterior Inferior Cerebellar Artery Infarct with Unilateral Deafness

R Rajesh*, M Rafeequ**, AS Girija***

Abstract
We report the case of a young man with anterior inferior cerebellar artery infarct causing unilateral deafness. Clinical features and audiometry suggested cochlear localization for deafness. MRI brain showed an infarct in the right AICA territory with involvement of pons. Involvement of the internal auditory artery explains the cochlear deafness.

INTRODUCTION
Unilateral, sudden deafness in a young patient is usually idiopathic and is often thought to be of viral or autoimmune in origin. On the other hand, sudden deafness in an older patient with cerebrovascular occlusive disease suggests the likelihood of ischaemia in the distribution of the internal auditory artery, ordinarily a branch of the anterior inferior cerebeller artery (AICA).1 We report a case of AICA infarct with unilateral deafness in a young man.

CASE REPORT
Thirty nine year old man was admitted in our ward with sudden onset vertigo, deafness of right side with unsteadiness of gait. He was a heavy smoker. He was conscious with normal blood pressure and pulse. There was lower motor neuron type of facial palsy on the right side, sensori-neural deafness on the right side with right-sided cerebellar signs. There were no other neurological deficits. MRI brain showed an infarct in the right AICA territory with involvement of pons (Figs. 1, 2). MR angiogram of both the extra and intracranial vessels was normal. His haemogram, blood coagulation and clotting profile, blood sugar, lipid profile and, renal and liver functions were normal. HIV-ELISA, VDRL, ANA, RA factor, sickling test and urine homocysteine were negative. Chest X-Ray, ECG and trans-oesophageal echocardiography were normal. Audiometry showed moderate sensori-neural deafness with good speech discrimination. He was given aspirin. After 1 month he made good recovery in facial paralysis and ataxia; but the deafness remained the same.

DISCUSSION
The AICA arises from the caudal third of the basilar artery in 75%, sometimes from the middle third, occasionally from its inferior limit, and is lacking in only 4% of individuals.2 Because of its usual small size, the AICA supplies a small area of the anterior and medial cerebellum. Proximal branches of the AICA usually supply the lateral portion of the pons, including the facial, trigeminal, vestibular and the cochlear nuclei, the root of the seventh and eighth cranial nerves and the spinothalamic tract.3

AICA occlusions are atherothrombotic in most cases.3 Aetiology of the stroke could not be detected in our case. There was no evidence for vertebrobasilar dissection in the MR angiogram. Trans-oesophageal echocardiography did not reveal any source of embolus. Only risk factor identified was smoking. Symptoms of AICA infarct include vertigo, vomiting, tinnitus and dysarthria. Signs include ipsilateral facial palsy,
hearing loss, trigeminal sensory loss, Horner’s syndrome, appendicular dysmetria, and contralateral temperature and pain sensory loss over the limbs and trunk. An AICA occlusion is often misdiagnosed as lateral medullary infarction. But features such as severe facial palsy, deafness, tinnitus and multimodal sensory impairment over the face favor the former.

Hearing impairment is not a frequent symptom of vertebrobasilar occlusive disease. Roquer et al described 15 patients with AICA stroke and six had deafness. In Ameranco’s series, of the 13 patients with cerebellar infarction involving only the anterior inferior cerebellar artery territory, three had deafness. There are several reports of even bilateral deafness with AICA infarcts. To our knowledge so far no case of AICA infarct with deafness has been reported from India.

AICA is termed the cerebellolabyrinthine artery because it gives rise to internal auditory artery (IAA) in 83% cases. The labyrinthine type syndrome that follows infarcts in the AICA territory could then be explained by several different lesions. 1. Occlusion of the IAA. 2. Involvement of the vestibular nuclei in the pontine tegmentum. 3. Involvement of the fibres of the eighth nerve in the lateral pontine area, and 4. Involvement of the floccular lobe or its connections. Deafness could be linked to the direct involvement of the cochlear nuclei at the pontomedullary junction, or the lateral lemniscus and the intraaxial fibres of the eighth nerve. Vertebrobasilar ischaemia may selectively damage the inner ear because of this structure’s high energy requirements and the lack of adequate collateral blood supply. With few exceptions, the IAA receives its sole blood supply from the AICA, and the inner ear receives its sole blood supply from the IAA.

Hearing loss for pure tone is unusual with central lesions, even in late stages. Our patient had persistent, unilateral deafness in pure tone audiometry, but the ataxia essentially resolved and the facial palsy also cleared significantly. The combination of these audiometric results and the improvement of the central signs localize the lesion causing deafness to the inner ear. Further more, cochlear localization was suggested by relatively preserved speech discrimination in audiometry.

References

Announcement
Institute of Haematology and Transfusion Medicine, Medical College, Kolkata, would be organising a National Workshop cum Symposium on Recent Development in Haematology: An Indian Perspective from June 4-6, 2004.
For the Workshop, 20 young scientist (Basic & Medical) would be given hands on training in different modern technologies including molecular biology and proteomic analysis. The symposium would be open to all interested medical and technical personnel.
For further details please write Dr. Prantar Chakrabarti, Organising Secretary, Assistant Professor, Institute of Haematology and Transfusion Medicine, 3rd Floor, MCH Building, Medical College, 88, College Street, Kolkata 700073.
Tel: 91-33 22198727, Fax: 91-33 22198727                 e-mail: prantar@ihtmindia.org, prantar@vsnl.net