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Sudden deafness of vascular origin

Nagła gluchota pochodzenia naczyniowego

Etiology of sudden deafness remains still an open issue. The majority of authors think that the factors causing sudden deafness are the following: vascular disorders, viral infections or allergic–immunological background. The explanation for vascular origin is the structure of labyrinthial artery (long and narrow lumen) creating extremely good conditions for being closed due to contraction resulting from stressing situation or creation of intravascular clots in it.

The aim of the study was to present sudden deafness of vascular etiology after stressful situation.

METHODS

In the diagnosis of the described case the following audiological tests were performed: pure tone audiometry and impedance audiometry, brain stem responses, otoacoustic emission, and radiological tests: CT and MRI of the head.

CASE HISTORY

A boy M. D., 14 years old was admitted to the hospital due to acute left ear hearing loss. The interview revealed that 6 days earlier after a stressful situation (his father had a car accident) he had felt tinnitus in his left ear, nausea, vertigo and hearing loss. Paediatrician prescribed him Cocarboxylase, Torecan, Cinnarizine and Doxacicline. In spite of the treatment no improvement was achieved and the boy was immediately admitted for hospitalisation. During the laryngological test no deviations from the standard values were noticed. There were performed the following tests: a pure tone audiometry and impedance audiometry (tympanometric curves of type "A" on both sides were the same, ipsilateral stapedial reflexes were present in the right ear and contralateral in the left ear (Fig. 1).

CT of the head was performed. On the brain base in hippocamp area on the right side a small ischaemic focus was visible, the brain and pyramids of temporal bone were normal (Fig. 2).

After neurological examination (we find drifting of the tongue to the left) the treatment with Dexamethazone, Cocarboxylase, Vit. B12, Adavin, Asparagin, Vit. of B group was extended by administering Sermion and Nootropil intravenously and Betaserc. After the treatment there was a subjective and objective improvement of hearing (Fig. 3).

Then after 10 months MRI test was performed and it revealed: fluid space 3.5 cm x 2.5 cm x 2 cm at the middle part of the bottom of the skull, which demonstrates a signal similar to cerebrospinal fluid and hypoplasia of the adjoining structures of the right temporal lobus. The MRI image suggests communicative arachnoidea cyst (Fig. 4).

The subsequent control examination after 22 months confirmed the following:

1. Pure tone audiometry – the result was the same as the result on the discharge from the hospital; 2. Impedance audiometry: tympanometric curves of type "A" on both sides, ipsilateral stapedial reflexes in the right ear and in the left ear were present for 500 and 1000 Hz; 3. ABR: latency, interpeak latency and morphology for the right ear were normal. In the left ear – wave V with latency 8.2 ms was hardly seen; 4. Otoacoustic emission: TOAE and DPOAE were present in the right ear, without responses in the left ear; 5. Psychological examination: no deviations from the standard; 6. Logopedic examination: dyslalia – incorrect realisation of glosso–dental phonemes (t, d, n) and, cleft ones, (s, z); 7. MRI test: the result identical with the previous one. (Fig. 5).

The patient remains under constant audiological control of the hospital.

DISCUSSION

Hearing loss of vascular origin was described comprehensively in the literature. Sensorineural hearing loss was confirmed in patients with atherosclerosis and circulatory cerebral insufficiency basing mainly on the speech audiometry (6). Also Ohnaka and others report a weak discrimination of speech after bleeding into the left temporal lobus (5).

Hariri and co–workers and Fromby describe hearing loss after the brain stroke. (3, 4). Cohen after the ABR tests in patients with haemorrhage to the ponds confirmed the elongation of V wave latency and the presence of cross stapedial reflex (2). The authors suggest the failure of medial superior olivary nuclei and trapezoid body involving both afferent and efferent fibres.

Most of the authors agree that magnetic resonance imaging is a good method confirming the vascular and postischaemic origin in sudden deafness (1).

Hearing loss of vascular origin concerns mainly the elderly. The described case seems to be interesting due to young age of the patient and efficiency of the therapy having been applied immediately.

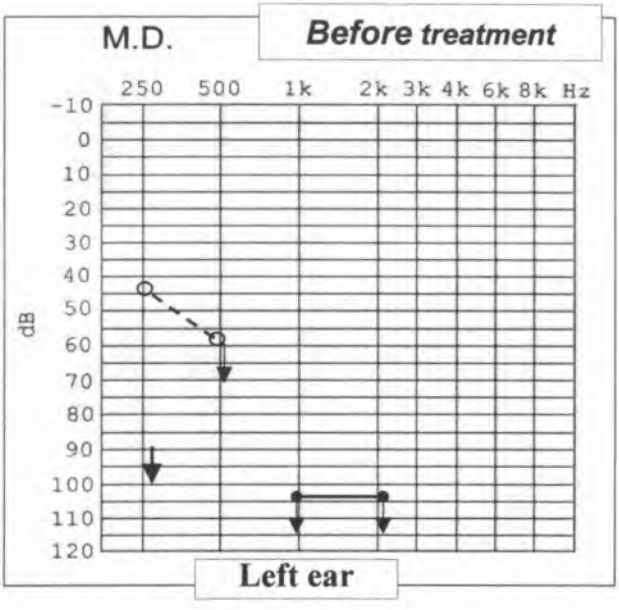

Fig. 1. Audiogram just after the stroke

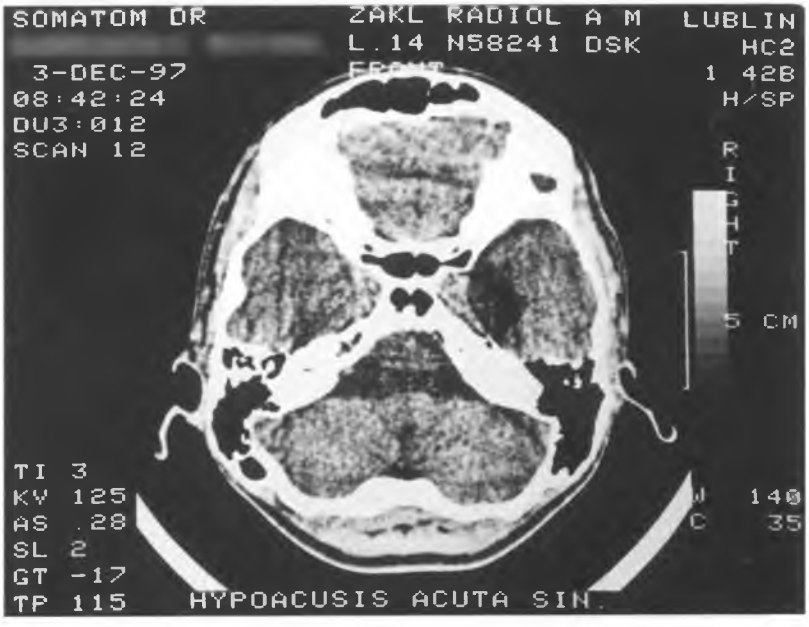

Fig. 2. CT of the head just after the stroke

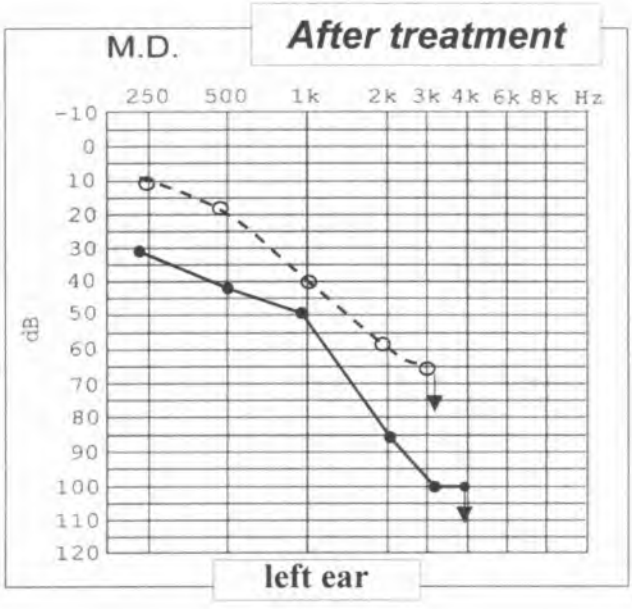

Fig. 3. Audiogram after the treatment

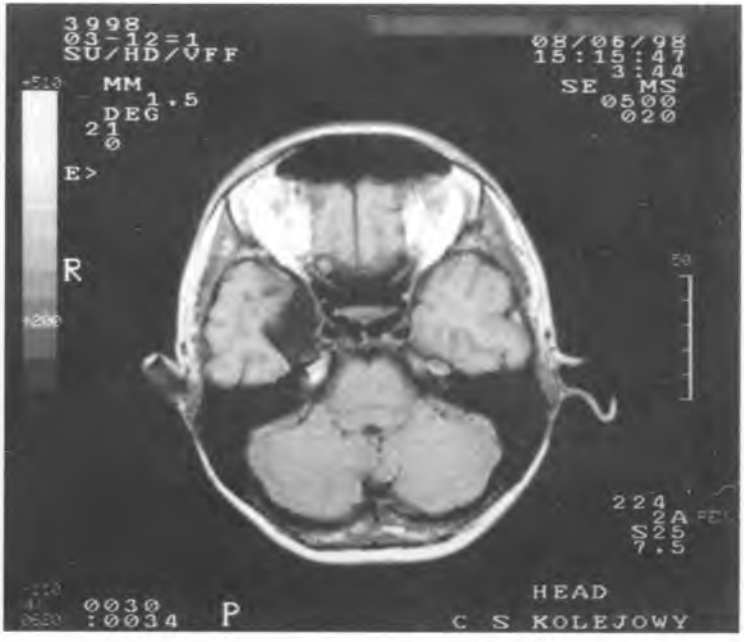

Fig. 4. MRI of the head 10 months later

Fig. 5. MRI of the head 22 months after the stroke

REFERENCES

1. Biavati M.J. et al.: Magnetic resonance imaging evidence of a focal pontine ischemia in sudden hearing loss and seventh nerve paralysis. Am. J. Otol. Mar. 15(2), 250, 1994.
2. Cohen M. et al.: Auditory deficits and hearing loss associated with focal brainstem haemorrhage. Scand. Audiol., 25(2), 133, 1996.
3. Fromby C. et al.: Hearing loss among stroke patients. Ear Hear. Dec., 8(6), 326, 1987.
4. Hariri M.A. et al.: Auditory problems in elderly patients with stroke. Age Ageing. Jul., 23(4), 312, 1994.
5. Ohnaka K. et al.: Pure word deafness after cerebral hemorrhage in the left temporal lobe: a case report. Rinsho Shinkeigaku. Mar., 35(3), 290, 1995.
6. Ryndina A.M. et al.: Alternating signal speech audiometry in the diagnosis of central lesions of the acoustic analyser. Vestn Otorhinolaryngol., (6), 13, 1998.

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STRESZCZENIE

Etiologia nagłych głuchot pozostaje nadal zagadnieniem otwartym. Większość autorów uważa, że czynnikami je wywołującymi są: zaburzenia naczyniowe, zakażenia wirusowe czy tło alergiczne. Wytłumaczeniem tła naczyniowego jest budowa tętnicy (długa i o wąskim świetle), stwarzająca wyjątkowo dobre warunki do jej zamknięcia, pod wpływem skurczu wywołanego stresem, czy do powstania w niej zakrzepów.

Celem pracy było przedstawienie nagłej głuchoty o etiologii naczyniowej, która była wynikiem zaistniałej sytuacji stresowej.

Na podstawie badań klinicznych stwierdzono głębokiego stopnia niedosłuch czuciowo–nerwowy. Badania CT i MRI wykazały ogniska niedokrwienne na podstawie mózgu. W trybie pilnym wdrożono leczenie, uzyskując dobry efekt terapeutyczny w postaci poprawy słyszenia. W obszarze niedokrwiennym mózgu w badaniu kontrolnym stwierdzono przestrzenie płynowe.

Przedstawiony przypadek jest potwierdzeniem teorii naczyniowego pochodzenia nagłej głuchoty oraz pozytywnego efektu leczniczego w przypadku, gdy leczenie wdraża się natychmiastowo.