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IMAGES

Post-traumatic total deafness with normal CT scan

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Case report

A 22-year-old man consulted 8 days after cranial trauma with initial loss of consciousness sustained in a fight. He complained of right hearing loss and loss of balance. Clinical examination found right vestibular impairment. Otoscopy was normal; pure-tone and vocal audiometry

found right total deafness with normal hearing in the left ear. Videonystagmography with caloric test found total right vestibular areflexia. CT brain scan at emergency admission was normal, without visible fracture line. Assessment was completed with CT scan centered on either temporal bone and MRI brain scan (Figs. 1–3).

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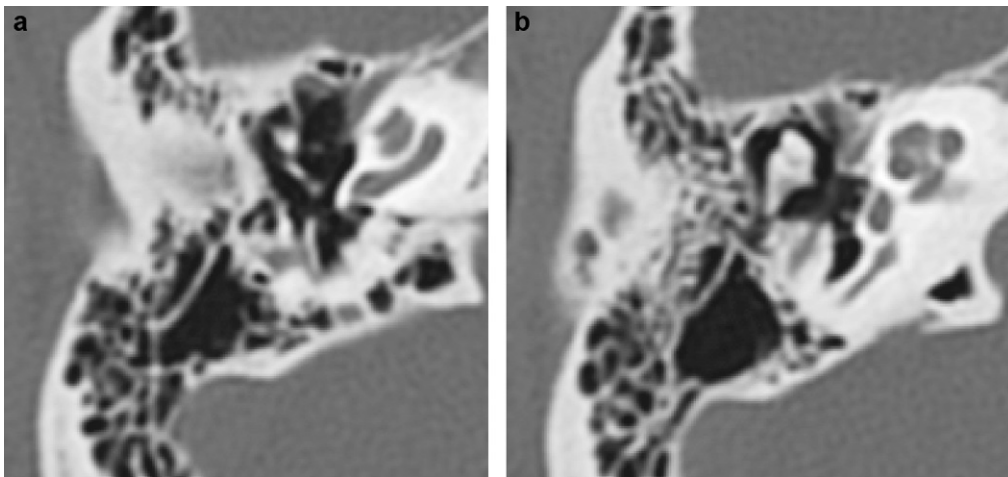


Figure 1

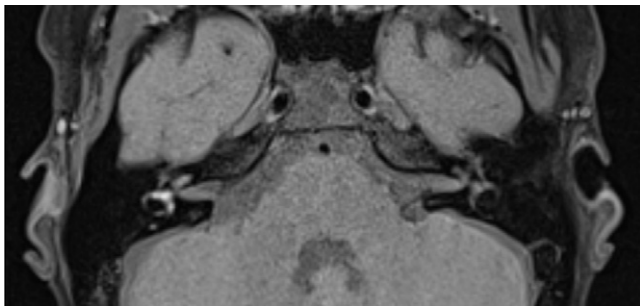


Figure 2

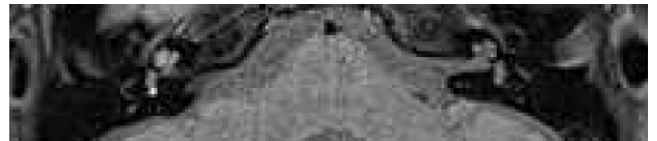


Figure 3

What is your diagnosis?

Post-traumatic right labyrinthine hemorrhage without visible associated fracture

Fig. 1 shows two transverse CT slices centered on the right temporal bone, one (a) through the round window and the other (b) through the stapes crura. No fracture line, pneumolabyrinth or mastoid cavity effusion is visible. Figs. 2 and 3 are axial slice T1-weighted MRI brain scans without contrast enhancement, centered on the labyrinths. They show right labyrinthine hemorrhage, in hypersignal on T1-weighted sequence without contrast enhancement.

Labyrinthine hemorrhage is a rare etiology for sensorineural hearing loss and vertigo. Some 20 cases have been reported, often associated with anticoagulant overdose or pathologies such as sickle-cell disease or leukemia [1]. Mechanisms involve labyrinthine venous micro-occlusion secondary to vasospasm, thrombosis or embolism. The resultant cochleovestibular hemorrhage induces labyrinthine membrane lesion by endo- and peri-lymphatic hyperpressure and osmotic and biochemical variation in inner-ear fluids.

Clinical symptoms comprise sudden hearing loss, usually profound to total, associated with peripheral vestibular syndrome. Diagnosis is founded on an MRI aspect of spontaneous hypersignal in non-enhanced T1-weighted sequences [2], suggesting spontaneous labyrinthine membrane lesion caused by variation in inner-ear fluids secondary to presence of methemoglobin following hemorrhage. The T2 signal varies progressively from initial hyposignal to hypersignal during the hemorrhage. T2 FLAIR brain sequences delete the cerebrospinal fluid and inner-ear fluid signals to reveal, at the acute and subacute phases, blood in the form of hypersignal localized in the inner ear [3]. The normal labyrinth shows in intermediate non-enhanced T1 signal, T2 hypersignal, and T1 and T2 isosignal with respect to the cerebrospinal fluid [4].

Various differential diagnoses are to be considered. In the present case, hypersignal preceded gadolinium injection,

thus ruling out intralabyrinthine schwannoma. Likewise, fat-sat sequences did not delete the hypersignal, eliminating the possibility of an exceptional intralabyrinthine lipoma. Spontaneous hemorrhage has also been reported secondary to heavy lifting inducing venous bleeding; the hypersignal may persist for several months. Hemorrhage may also be secondary to endolymphatic sac tumor, whether in a context of Von Hippel-Lindau disease or not; these fragile tumors may be microscopic and thus hidden by the hemorrhage [5].

Surgical exploration should not be undertaken until the clinical and radiological data have been analyzed, including MRI and temporal bone CT. Both ENT specialists and radiologists should be alerted to the need to perform T1-weighted sequences without gadolinium injection in case of hearing loss associated with normal temporal bone CT.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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