Response to Letter to the Editor of Kohui and Ghazavi
Giuseppe Magliulo
Otolaryngology -- Head and Neck Surgery 2013 148: 889
DOI: 10.1177/0194599813480482

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>> Version of Record - Apr 30, 2013

What is This?
Pneumolabyrinth following Eustachian Tube Insufflation

DOI: 10.1177/0194599813480481

Magliulo et al recently reported an interesting case of “pneumolabyrinth following Eustachian tube insufflation” in Otolaryngology—Head and Neck Surgery.¹ Pneumolabyrinth, as it has been described in the article, is a condition of vestibule and/or cochlea dysfunction due to the presence of air within the labyrinth. It is caused by a perilymphatic fistula that can be the result of temporal bone trauma, barometric pressure changes, or otologic surgery. The authors’ recommended treatment is “subtotal petrosectomy with blind sac closure of the external auditory canal and obliteration of the Eustachian tube.” This treatment strategy is an invasive approach, and it is not the most cost-effective treatment for solving the vestibular symptoms.

Although Eustachian tube insufflation is a described method to treat otitis media, the history of stapes surgery is a contraindication. Large pressure changes during this procedure almost always induce inner ear damage, and it should have been emphasized in the article as an important point to notice.

The vestibular symptoms in the patient seem to be related to massive inner ear damage, and it is not clear why petrosectomy and canal wall down procedure should be done in the patient who is deaf. The vestibular symptoms would be diminished with central compensation, and we believe that the petrosectomy does not have a dramatic effect, as pneumolabyrinth is a self-limiting symptom in such cases.

There are mixed reports of the advantages to surgical exploration versus conservative management of pneumolabyrinth, and there is not any standard practice in this case, although reported outcomes of conservative management with bed rest, antibiotics, nasal decongestant, and corticosteroids in several studies are encouraging for its use as initial treatment. Follow-up of these reports shows a complete closure of the air-bone gap with no residual dizziness and vertigo, while the air disappeared after 1 year.²³ Thus, surgical exploration should be limited, especially in progressive sensorineural hearing loss and persistent vertigo without any improvement after supportive management.

Fat and perichondrium, temporalis fascia, muscle, and tragal perichondrium bolstered with Gelfoam have been described for fistula repair, but the selected procedure in the presented case after one try for fistula repair without any supportive treatments is not the best approach. If hearing preservation is not a goal in this case, central compensation would have decreased vestibular symptoms anyway.

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Disclosures
Competing interests: None.
Sponsorships: None.
Funding source: None.

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DOI: 10.1177/0194599813480482

First, we thank Kohui and Ghazavi for the observations that gave us the opportunity to clarify the treatment adopted for our patient.
As you know, the section in which the article has been published limits the words to 700. Thus, our aim was essentially devoted to introducing another possible etiology (Eustachian tube insufflation) for the pneumolabyrinth related with perilymphatic fistula (PLF), and our recommendation was to “exercise caution when recommending this treatment option or other particular process of cannulation of ET for patients with history of stapes surgery.”

We have never recommended, as you stated, the subtotal petrosectomy as a first choice of treatment strategy. The patient had been initially treated by other physicians with conservative management, and the results were unsuccessful with a persistent profound hearing loss and vestibular symptoms. We performed 2 exploratory tympanotomies with no resolution of the pneumolabyrinth, and at this time, we decided to perform subtotal petrosectomy combined with Eustachian tube, middle ear, and mastoid obliteration. This strategy induced an initial improvement of the vestibular symptoms, but it is obvious that the following complete recovery of vestibular symptoms is attributable to adaptive mechanisms and that surgery was inconsistent at improving sensorineural hearing loss. In our case, we considered the subtotal petrosectomy as the best approach for the fistula repair, also taking into consideration the 2 months of persistence of the pneumolabyrinth, leaving the patient at risk for otogenic meningitis via the Eustachian tube. Note that one of the indications given by Fisch1,2 for the subtotal petrosectomy technique was to prevent the risk of meningitis in lesions violating the otic capsule. The first goal in patients with persistence of pneumolabyrinth suffering from profound hearing loss is to repair, as soon as possible, this potentially risky condition.

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Disclosures
Competing interests: None.
Sponsorships: None.
Funding source: None.

References

Letter to the Editor on “Cerebellar Infarctions Mimicking Acute Peripheral Vertigo: How to Avoid Misdiagnosis?”
DOI: 10.1177/0194599813481566

We read with interest the article by Casani et al1 on cerebellar infarctions mimicking peripheral vertigo. We agree that vertigo is a common presentation seen at the emergency department, and a method of differentiation between “peripheral” and “central” lesions would be useful.

Cerebellar strokes are easily distinguishable as they exhibit focal neurological features such as dysmetria; however, isolated cerebellar nodular infarcts are difficult to separate from vestibulopathy, as they present with vertigo without accompanying focal neurological deficits.

The nodulus is a small area of the cerebellum and, together with the flocculus, constitutes the vestibulocerebellum. They moderate the inputs from the vestibular system to regulate eye movements and modulate proprioception of the body and head in space.2 The flocculus derives its blood supply from the anterior inferior cerebellar artery, whereas the nodulus is perfused by the posterior inferior cerebellar artery; thus, the nodulus can be infarcted while the flocculus is spared.

We have a series of 8 patients diagnosed on magnetic resonance imaging with isolated nodular infarctions (Figure 1). All 8 cases presented with acute giddiness and demonstrated nystagmus at rest that worsened on head shaking. None of the cases demonstrated dysmetria or other cerebellar signs. Several cases

Figure 1. Left isolated nodular infarct seen on diffusion-weighted imaging (A). Important anatomical cerebellar structures (B). CH, cerebellar hemisphere.