CASE REPORT

Bilateral sudden sensorineural hearing loss in *Staphylococcus aureus* endocarditis

Joanne Wai Ling Lau, Borka Ceranic, Robert Harris, Elwina Timehin

St George's University
Hospitals NHS Foundation
Trust, London, UK

Correspondence to
Dr Joanne Wai Ling Lau,
joanne.lau@doctors.org.uk

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SUMMARY

This case highlights the diagnostic challenges in patients presenting with bilateral sudden sensorineural hearing loss (SNHL). The aetiology of bilateral sudden SNHL may span several medical disciplines. Therefore, clinicians should be mindful of such presentations, and consider aetiologies beyond otological and neurological causes. We present a case of a previously healthy 51-year-old woman who presented with coryzal symptoms and sudden audiovestibular failure. Examination revealed fever, tachycardia, bilateral profound hearing loss and nystagmus. Following investigations, an initial working diagnosis of vasculitis was made. Later, blood cultures revealed methicillin-sensitive *Staphylococcus aureus* (MSSA) and a transoesophageal echocardiogram confirmed endocarditis. The patient made a good recovery, but the hearing loss was permanent and managed with a cochlear implant.

BACKGROUND

To the best of our knowledge, this is the first reported case of hearing loss and dizziness being the initial presenting complaint of a patient with methicillin-sensitive *Staphylococcus aureus* (MSSA) endocarditis. This case highlights the importance of urgent detailed systemic investigations for bilateral sudden sensorineural hearing loss SNHL.

CASE PRESENTATION

A previously healthy 51-year-old woman presented to the local emergency department with bilateral tinnitus, 'muffled' hearing and rotational vertigo on getting up that morning, preceded by 3 days of coryzal symptoms. While the acute vertigo had settled, the other symptoms remained. Systemic review was unremarkable. On examination, the patient was febrile and tachycardic. Nystagmus was noted by the clerking physician, but not described. There were no other focal neurological or physical signs.

INVESTIGATIONS

Investigations showed neutrophilia ($9.7 \times 10^9/L$) with elevated C reactive protein (316.8 mg/L) and erythrocyte sedimentation rate (62 mm/h). ECG demonstrated widespread T-wave depression, with consistently raised troponin I. A bedside transthoracic echocardiogram found no abnormality. MRI of the head and internal acoustic meati showed multiple acute cortical infarcts and a microhaemorrhage in the left cerebellum, while the vestibulocochlear complexes were normal and the antero-inferior cerebellar artery flow voids

preserved. The autoimmune screen was negative, apart from a slightly raised rheumatoid factor (44.0 IU/mL). Lumbar puncture revealed a raised white cell count (predominantly lymphocytes) but no organisms. Bacterial and tuberculosis cultures of the cerebrospinal fluid were negative, as was the viral PCR.

TREATMENT

The initial working diagnosis from the multidisciplinary team was vasculitis; pulse methylprednisolone therapy was started. Pure tone audiometry (PTA) was first performed during steroid treatment (day 4 of admission), and showed right moderate-to-profound and left profound SNHL. Otoacoustic emissions were absent bilaterally, suggesting cochlear cell damage. The patient reported an improvement in hearing during steroid therapy and a minor improvement in right low-frequency thresholds was noted on the second PTA (figure 1).

On the second day of steroid treatment, a pansystolic murmur was identified. Blood culture grew MSSA. An urgent transoesophageal echocardiogram showed a vegetation causing destruction of the posterior mitral valve with severe mitral regurgitation. A diagnosis of definite MSSA infective endocarditis was made with the modified Duke criteria (one major, three minor). Steroid therapy was discontinued; the patient underwent an urgent valve replacement and intravenous antibiotic therapy.

OUTCOME AND FOLLOW-UP

Approximately a week after stopping steroid therapy, the patient reported a gradual deterioration in hearing. PTA confirmed deterioration in right low-frequency thresholds. The right ear was treated with intra-tympanic steroid therapy (dexamethasone, 4 mg/mL), but there was only minor right low-frequency threshold improvement, which was not sustainable. Therefore, this treatment was discontinued. The patient has otherwise made a good recovery and received a left cochlear implant, in accordance with the current National Institute for Health and Care Excellence (NICE) guideline for cochlear implants for children and adults with severe-to-profound deafness.

DISCUSSION

The pathogenesis of sudden SNHL is poorly understood and different underlying mechanisms, including vascular, infective, toxic and autoimmune, have been proposed.¹ The blood supply to the inner ear is derived from the internal auditory artery, an end

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Figure 1 Pure tone audiograms. The right audiograms on days 4, 14 and 30 after the onset of bilateral sensorineural hearing loss are shown. Intravenous methylprednisolone was given on days 3 and 4. The left audiogram on day 4 is shown. The left hearing loss remained profound.

artery. Along with a high-energy metabolism, the inner ear is thus vulnerable to ischaemia. Infections through the inner ear, from haematogenous spread or other routes such as from the cerebrospinal fluid space or from the middle ear, may cause ear cell damage. Infections may cause hearing loss indirectly by triggering an antibody response that cross-reacts with an inner ear antigen.²

While unilateral sudden SNHL is more common, bilateral sudden SNHL is rare (<5% of all reported cases of sudden SNHL). It is mostly associated with systemic pathology³ and more likely to be severe; it has a poorer prognosis than unilateral hearing loss.⁴ Corticosteroid therapy, both systemic and/or intra-tympanic, is used to treat sudden SNHL in the hope of an improvement of the hearing thresholds and/or to prevent

further deterioration, although its effectiveness is uncertain given the conflicting results of clinical trials.^{1,5} Prior to initiating corticosteroid therapy, infective causes must be ruled out. In situations of uncertainty, use of intra-tympanic corticosteroids may be a more suitable option than intravenous therapy, to manage the risks of immunosuppression.

In the context of MSSA infective endocarditis and cerebrovascular/embolic changes, plausible underlying pathophysiology for the bilateral sudden SNHL in our patient may include infective labyrinthitis (involving auditory and vestibular sensory epithelium), hypoxia due to a compromised vascular supply to the audiovestibular labyrinths, or a staphylococcal toxic effect.

Contributors All the authors looked after the patient. JWLL prepared the manuscript. BC, ET and RH revised the manuscript.

Competing interests None declared.

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REFERENCES

- Schreiber BE, Agrup C, Haskard DO, *et al.* Sudden sensorineural hearing loss. *Lancet* 2010;375:1203–11.
- Greco A, Fusconi M, Gallo A, *et al.* Sudden sensorineural hearing loss: an autoimmune disease? *Autoimmun Rev* 2011;10:756–61.
- Sara SA, Teh BM, Friedland P. Bilateral sudden sensorineural hearing loss: review. *J Laryngol Otol* 2014;128(Suppl 1):S8–15.
- Oh JH, Park K, Lee SJ, *et al.* Bilateral versus unilateral sudden sensorineural hearing loss. *Otolaryngol Head Neck Surg* 2007;136:87–91.
- Stachler RJ, Chandrasekhar SS, Archer SM, *et al.* Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg* 2012;146:S1–35.

Learning points

- Bilateral hearing loss may be the only initial presentation of a significant systemic pathology and it must be thoroughly investigated.
- Transthoracic echo is insufficient to rule out endocarditis.
- Systemic and/or intra-tympanic steroids are used to treat sudden sensorineural hearing loss, but the effectiveness is uncertain. In this case, it did not result in recovery of the patient's hearing.

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