Objective: To describe the clinical evaluation and operative management of a unique case of posterior semicircular canal dehiscence due to a high jugular bulb. Methods: Retrospective case report. Results: The patient had clinical and audiometric findings consistent with semicircular canal dehiscence and imaging demonstrating erosion of the posterior semicircular canal by a high jugular bulb. Decompression of the jugular bulb with plugging and resurfacing of the eroded canal provided resolution of vestibular symptoms without significant loss of hearing. Conclusions: Dehiscence of the posterior semicircular canal can produce a spectrum of clinical findings similar to those of superior semicircular canal dehiscence syndrome. Jugular bulb decompression with plugging and resurfacing of the area of dehiscence can be performed to successfully relieve the vestibular symptoms in such cases by directly addressing the area of dehiscence.

INTRODUCTION

Minor and colleagues first described the spectrum of symptoms and clinical findings associated with bony dehiscence of the superior semicircular canal which include hearing loss, autophony, chronic imbalance and pressure or noise induced vertigo. Bony dehiscence of the posterior semicircular canal (PSCC) due to a high jugular bulb has been reported by a number of authors and can present with clinical findings similar to those seen with superior canal dehiscence. In the operative management of PSCCD due to a high jugular bulb has been described once in the literature, however the area of dehiscence was addressed indirectly through plugging of the uninvolved area of the canal. We report a case of PSCCD that was addressed through reduction of the jugular bulb and direct plugging and resurfacing of the area of dehiscence.

Case Presentation

A 20 year old man presented to our tertiary care center with complaints of noise-induced vertigo and right-sided pulsatile tinnitus starting four months earlier after a ground-level fall where he sustained a left-sided, otic capsule-sparing temporal bone fracture isolated to the external auditory canal (EAC). The patient had experienced one month of headaches, left-sided hearing loss and bloody otorrhea after the fall which had resolved spontaneously. A detailed neurotological examination on presentation, including fistula testing, was unremarkable aside from evidence of a healed fracture of the left external auditory canal.

Audiometry & vestibular testing summary

- Routine audiometry demonstrated a mild, symmetric sensorineural hearing loss (PTA 27 dB HL AD and 28 dB HL AS) with thresholds in the higher frequencies in the moderate to moderate-severe range. His speech discrimination scores were 96% bilaterally.
- Vestibular evoked myogenic potential (VEMP) testing (100μsec broadband click at 97 dB HL with 5 clicks) thresholds were present at 92 dB HL AS and 67 dB HL AD (Fig. 1). Interaural amplitude ratio was not significantly different.
- Upbeating, vertical nystagmus upon application of positive pressure in the right EAC (Fig. 2).
- Upbeating, vertical nystagmus after delivery of a 500 Hz tone at 110 dB HL to the right EAC (Fig. 3) occurring in a delayed fashion in noise presentation.
- A complete rotary chair testing battery produced no significant abnormalities.

Imaging:

High-resolution temporal bone computed tomography (CT) in oblique planes (Figure 4) delineated a broad area of dehiscence of the otic capsule bone due to a large, high-riding jugular bulb on the right side. The posterior semicircular canal was found to be widely dehiscent including the ampulla of the canal.

Operative management:

The patient actively sought definitive management of his vestibular symptoms as they were interfering with his work performance. Accordingly, operative management was undertaken through a transmastoid approach where the large, high jugular bulb was found to be apposed to the otic capsule. The bulb was decompressed using bipolar cautery, surgical packing and bone wax. After decompression a temporalis fascial graft was applied into the area of dehiscence then supported by placement of a layer of bone paste. A cortical bone graft was then placed beneath the bone paste to complete the composite repair.

Postoperative Course:

The patient has an uneventful recovery from surgery. His symptoms of noise-induced vertigo and pulsatile tinnitus resolved completely. Audiometry performed at 20 months postoperatively demonstrated unchanged discrimination scores and pure-tone threshold aside from a mild worsening (10-25 dB) of his pre-existing high frequency loss at 4, 5, 9 kHz only. The patient did complain of mild disoequilibrium at 20 months after surgery which improved with vestibular rehabilitation therapy.

DISCUSSION

Prevalence and Clinical Findings:

Dehiscence of the posterior semicircular canal from a high jugular bulb was first described radiographically in 19939. In 2003, a review of 507 temporal bone computed tomography scans from patients with inner ear symptoms found a 4.5% incidence of dehiscence of the posterior semicircular canal, over half of whom had evidence on CT with 41% and 36% of whom had vertiginous symptoms. Recent reports have better characterized the spectrum of clinical symptoms seen in this disorder which include hearing loss (generally conductive), pulsatile tinnitus, autophony, disesquilibrium, vertigo and noise or pressure induced vertigo. Most of these studies have found a great deal of variability of the clinical findings, which can potentially complicate the evaluation and treatment of patients with PSCCD.

The patient presented herein was found to have many of the clinical findings known to characterize PSCCD including hearing loss (generally severe), vertigo due to bone dehiscence of the superior semicircular canal and imaging demonstrating erosion of the posterior semicircular canal by a high jugular bulb. Decompression of the jugular bulb with plugging and resurfacing of the eroded canal provided resolution of vestibular symptoms without significant loss of hearing.

REFERENCES