Migraine-Associated Hearing Loss After General Anesthesia

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Abstract

Objectives:
1. Propose migraine associated hearing loss after general anesthesia as a distinct entity. 2. Suggest vasospasm as a possible etiologic mechanism for migraine associated hearing loss after general anesthesia. 3. Discuss management strategies.
Methods:
A retrospective review from our tertiary medical center from September 2003 to January 2007 identified three patients with a history of migraine headache who developed sudden hearing loss after general anesthesia for non-cardiac, non-otologic surgery. Patient charts were reviewed for relevant medical history, audiometric data, anesthesia records, management strategy, and outcome.
Results:
All patients met IHS diagnostic criteria for migraine headache. Unilateral hearing loss developed either immediately postoperatively (n = 3) or within one week postoperatively (n = 1). The hearing loss was purely sensorineural in three patients and mixed (with a slight conductive component) in one patient. PTA at peak hearing loss ranged from 62 to 75dB. One patient, identified immediately after surgery with sudden SNHL, was treated with high dose steroids and fully recovered. Another patient saw an outside otolaryngologist one month after sudden SNHL developed and received intratympanic dexamethasone without improvement. The third patient presented to our clinic six months after undergoing general anesthesia for bone marrow donation at another institution and did not recover hearing. The fourth patient underwent distal pancreatectomy and experienced unilateral sensorineural hearing loss and was subsequently lost to follow up.
Conclusions:
We have identified three cases of sudden hearing loss after general anesthesia in patients with migraine headache. Hearing loss may occur as a result of cochlear damage from migraine related vasospasm. Corticosteroids may have a role for improving hearing, especially if initiated quickly after the onset of hearing loss.

Introduction

Migraine is a common disorder, affecting nearly 20% of women and 6% of men[1]. Migraine has specific diagnostic criteria outlined by the International Headache Society (Figure 1). For the majority of patients the cause is unknown but most have a strong family history of migraine. There are currently two theories regarding the pathophysiology of migraine headache. Migraine with aura (classic migraine) is associated with spreading cortical oligemia and neuronal inhibition[2]. Migraine without aura (common migraine) is thought to be related to dysregulation in the limbic system and hypothalamus and may be mediated by serotonin[3].

The spreading oligemia and vasoconstriction associated with migraine with aura can cause migrainous infarction. The International Headache Society classification of migraine describes migrainous infarction as cerebral infarction occurring during a typical migranous attack[2]. These patients will demonstrate focal neurologic deficits and typical radiographic signs of ischemic stroke in addition to their typical migraine symptoms. Fortunately, this devastating complication of migraine is rare. However, vasospasm occurring within the inner ear may be more common and result in many of the neurotologic symptoms experienced by migraineurs[4]. Vertigo is more common than auditory complaints; as many as one quarter of migraine patients will experience vertigo[4]. Auditory complaints range from phonophobia or fluctuating hearing levels to permanent hearing loss[5,6].

There are a small number of reports in the literature regarding migraine associated hearing loss [5,7]. The authors of these reports speculated that the sensorineural hearing loss affecting these patients may be caused by vasospasm of the cochlear blood vessels. Some patients regained hearing after administration of ergot alkaloids[5].
Migraine frequently occurs after anesthesia in those with a history of migraine[8,9,10]. There are however, no reports of hearing loss associated with general anesthesia in the migraineur. We report four cases of sensorineural hearing loss associated with general anesthesia in migraine patients.

Migraine without aura	A. At least 5 attacks1 fulfilling criteria B–D B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated) C. Headache has at least two of the following characteristics: 1. unilateral location 2. pulsating quality 3. moderate or severe pain intensity 4. aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs) D. During headache at least one of the following: 1. nausea and/or vomiting 2. photophobia and phonophobia E. Not attributed to another disorder
Migraine with aura	A. At least 2 attacks fulfilling criteria B–D B. Aura consisting of at least one of the following, but no motor weakness: 1. fully reversible visual symptoms including positive features (eg, flickering lights, spots or lines) and/or negative features (ie, loss of vision) 2. fully reversible sensory symptoms including positive features (ie, pins and needles) and/or negative features (ie, numbness) 3. fully reversible dysphasic speech disturbance C. At least two of the following: 1. homonymous visual symptoms1 and/or unilateral sensory symptoms 2. at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes 3. each symptom lasts ≥5 and <60 minutes D. Headache fulfilling criteria B–D for 1.1 Migraine without aura begins during the aura or follows aura within 60 minutes E. Not attributed to another disorder

Figure 1. International Headache Classification of Headache disorders. Taken from Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia, 2004, 24 Suppl 1, 9-160.

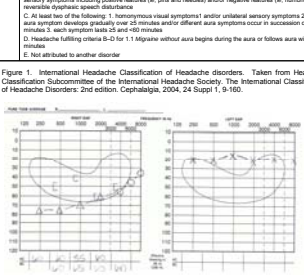

Figure 2. Patient 1 Audiogram – The left ear demonstrates normal hearing levels through all frequencies. The right ear demonstrates an upsloping sensorineural hearing loss (SNHL). The low frequencies (250-1000 Hz) demonstrate severe SNHL from 70-80 dB HL with improvement in the PTAs at 8000Hz at 35dBHL. The word recognition scores for the right ear were 12% and 96% for the left ear.

Table 1. Patient hearing profile and treatment regimen

Patient	PTA in affected ear (dB HL)	PTA in normal ear (dB HL)	WRS in affected ear (% correct)	WRS in normal ear (% correct)	Treatment
1	60	20	12	96	None
2 (pre treatment)	60	30	36	100	intratympanic dexamethasone
2 (after treatment)	65	25	28	100	N/A
3 (on presentation)	70	60	DNT	DNT	IV dexamethasone
3 (after treatment)	35	40	50	70	N/A
4	55	20	64	92	none

Table 2. Patient surgery profile

Patient	Surgery	Anesthetic Agents	Length of Surgery (hours)
1	Bone marrow harvest	Propofol, sevoflurane, rocuronium, Fentanyl, nitrous oxide	2.5
2	Bilateral knee replacement	N/A	N/A
3	Open prostatectomy and lymph node dissection	Propofol, desflurane, rocuronium, fentanyl	2.25
4	Laproscopic distal pancreatectomy and splenectomy	Propofol, sevoflurane, rocuronium, fentanyl	4.75

Case Reports

Case 1
A 53 year old female underwent general anesthesia for bone marrow harvest for an unrelated patient in need of a stem cell transplant. Immediately post-operatively she noted right aural fullness, tinnitus and diminished hearing in her right ear. This hearing loss was not addressed until several days later when she presented to an urgent care facility for evaluation. She did not have associated vertigo. She had a history of migraine headache, which had been more frequent during pregnancy many years earlier. She did not have a family history of migraine. Her outpatient medications prior to the procedure where synthetic estrogen/progesterone, paroxitine, lisinopril and loratidine. Her hearing did not improve over the subsequent months.

Prior to the procedure, the patient received midazolam 2mg intravenously. On induction of anesthesia, the patient received propofol 160mg, rocuronium 50 mg, fentanyl 250 mg and lidocaine 1% 80 mg. She was maintained in anesthesia on nitrous oxide and sevoflourane (Table 2). She received 2800 cc of crystalloid and the blood loss was 1500 cc. Her vital signs were stable throughout the procedure. The procedure lasted 155 minutes. In the recovery room there is no record of any nausea, vomiting or headache. She did receive 2 units of packed red cells post-operatively due to anemia secondary to the procedure.

The patient's audiogram on presentation to our tertiary referral center demonstrated an upsloping severe to moderate sensorineural hearing loss (SNHL) in the right ear with normal pure tone averages (PTA) in the left ear (Figure 2). Her word recognition score on the right was 12% and 96% on the left (Table 1). Her tympanograms were type A. A contrast enhanced MRI of the brain and temporal bones were unremarkable. There was no evidence of stroke. Electronystagmography (ENG) and vestibuloocular reflexes (VOR) were unremarkable.

Case 2
A 64 year old female underwent bilateral total knee replacements for osteoarthritis approximately two months prior to presenting to a neurootologist. Approximately 48 hours after emerging from anesthesia she reported a significant decrease in hearing in her left ear. She reported a "vibration" sensation in the left ear along with left sided constant pulsatile tinnitus. She denied vertigo, otalgia, or otorrhea. She had a history of migraine headaches but no family history of migraine. Her preoperative medications were Tavist, Wellbutrin, Ambien, Nadolol, Percocet, acetaminophen and Amerge.

The anesthetic records for this patient are not available.

She had a preoperative audiogram that demonstrated bilateral mild hearing loss (PTA of 25-30 across all frequencies) (Table 1). At the time of presentation she was noted to have a flat moderate SNHL in the left ear with mildly decreased PTA in the right ear (Figure 3A,B). Word recognition scores were 52% in the left ear and 100% in the right. The patient underwent intratympanic dexamethasone (40mg/mL) injection. Two weeks following this injection her low frequency thresholds had worsened by 10dB and her word recognition had decreased to 28% (Figure 3C). She also underwent a contrast enhanced MRI of the temporal bones that was normal.

Case 3
A 61 year old male underwent an open radical prostatectomy and bilateral pelvic lymph node dissection for prostate carcinoma. Immediately postoperatively he noted bilateral aural fullness and his wife noted his hearing had diminished. An urgent audiogram was obtained and the patient was started on high dose dexamethasone. After the induction of the steroids the patient noted improvement in his hearing. The patient reported a history of sudden SNHL and vertigo 20 year previously; three months later his hearing had improved but he did have residual left tinnitus. The patient also reported a history of classic migraine with visual aura (scintillating scotoma). He denied photophobia or phonophobia. He did not have a family history of migraine and his only preoperative medication was aspirin which he had discontinued seven days prior to the operation

Case Reports cont'd

Case 3 continued
Prior to the operating room, the patient did not receive midazolam. On induction of anesthesia, the patient was given propofol 200 mg, rocuronium 50mg, fentanyl 200 mg, and lidocaine 1%. Anesthesia was maintained with desflurane. No nitrous oxide was used (Table 2). During the procedure he received an additional 70 mg of rocuronium, toradol 30 mg, pseudoephedrine one dose, and ephedrine 2 doses. At emergence he was given glycopyrolate, Anzamet 12.5 mg, neostigmine 5 mg, and morphine sulfate 5 mg. The procedure lasted 135 minutes. During the case, the patient experienced mild hypotension, 85 systolic, for which he received the above mentioned pressors. He was infused 3300 cc of crystalloid and the estimated blood loss was 1100 cc. In the recovery room, the patient's vital signs were stable and there is no mention of nausea, vomiting or headache.

The patient's initial audiogram demonstrated moderate to profound sloping SNHL in the right ear and mild to severe sloping SNHL in the left ear (Figure 4A / Table 1). During his inpatient audiogram word recognition scores were not obtained. One month later the patient's PTA thresholds had significantly improved (Figure 4B). His right ear demonstrated only a bilateral moderate high frequency SNHL with 100% discrimination scores bilaterally. The patient had ENG and VOR testing that was normal. A MRI was not obtained.

Case 4
A 67 year old female was noted to have an incidental complex mass in the tail of the pancreas. She underwent a distal pancreatectomy and splenectomy for pancreatic adenocarcinoma. Her post operative course was complicated by hemorrhage that required transfusion and revision laparoscopy with ligation of a bleeding vessel. At her post operative follow up 2 weeks after surgery, she reported left sided hearing loss, aural fullness and constant "running water" tinnitus. She denied any vestibular symptoms. She was referred to a neurotologist and was seen approximately 3 weeks after her initial surgery. She did not receive steroids. Her preoperative medications were verapamil, Fosamax, glucosamine/chondrotin sulfate, and an iron supplement. She has stopped taking aspirin 1 week prior to the operation.

An audiogram was obtained upon her presentation to the neurotologist (Table 1). There was moderate to mild upsloping SNHL in the left ear and mild high frequency SNHL on the right (Figure 5). Word discrimination scores demonstrated 64% in the left ear and 92% in the right ear. Tympanograms were type A bilaterally. ENG and VOR testing was ordered but was not completed and the patient was lost to follow up.

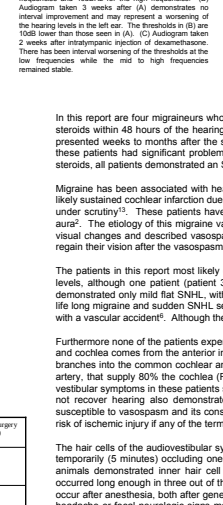

Figure 3. Patient 2 Audiograms – (A) Audiogram taken approximately 2 weeks after bilateral knee replacement surgery. The right ear demonstrates a mid to high frequency sloping SHNL. The left ear demonstrates an upsloping sensorineural loss from 65dBHL for the low frequencies and 40dBHL for the high frequencies. (B) Audiogram taken 3 weeks after (A) demonstrates no interval improvement and may represent a worsening of the hearing levels in the left ear. The thresholds in (B) are 10dB lower than those seen in (A). (C) Audiogram taken 2 weeks after intratympanic injection of dexamethasone. There has been interval worsening of the thresholds at the low frequencies while the mid to high frequencies remained stable.

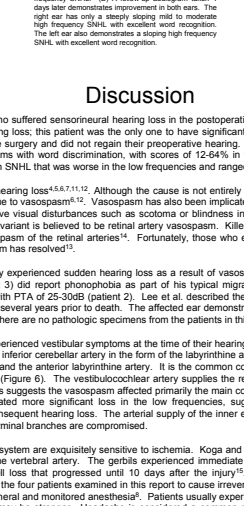

Figure 4. Patient 3 Audiograms – (A) Inpatient urgent audiogram taken within 24 hours of the operation. The right ear demonstrates low frequency moderate SNHL with a steeply moderate to profound sloping high frequency loss. The left ear demonstrates low frequency mild SNHL with a steeply sloping high frequency SNHL. (B) A follow-up audiogram taken 4 days later demonstrates improvement in both ears. The right ear has only a steeply sloping mild to moderate high frequency SNHL with excellent word recognition. The left ear also demonstrates a sloping high frequency SNHL with excellent word recognition.

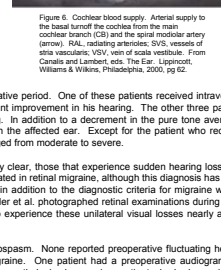

Figure 5. Patient 4 Audiogram – The right ear demonstrates a slight high frequency SNHL and excellent word recognition. The left ear shows a severe to mild upsloping SNHL. The left ear demonstrates only marginal word recognition scores (64%).

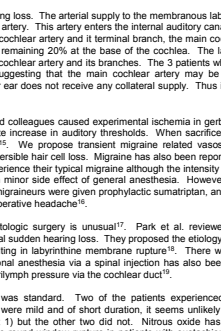

Figure 6. Cochlear blood supply. Arterial supply to the basal turnoff the cochlea from the main cochlear branch (CB) and the spiral modiolar artery (arrow). RAL, radiating arterioles; SVS, vessels of stria vascularis; VSV, vein of scala vestibule. From Canalis and Lambert, eds. The Ear. Lippincott, Williams & Wilkins, Philadelphia, 2000, pg 62.

Discussion

In this report are four migraineurs who suffered sensorineural hearing loss in the postoperative period. One of these patients received intravenous steroids within 48 hours of the hearing loss; this patient was the only one to have significant improvement in his hearing. The other three patients presented weeks to months after the surgery and did not regain their preoperative hearing. In addition to a decrement in the pure tone averages, these patients had significant problems with word discrimination, with scores of 12-64% in the affected ear. Except for the patient who received steroids, all patients demonstrated an SNHL that was worse in the low frequencies and ranged from moderate to severe.

Migraine has been associated with hearing loss[4,5,6,7,11,12]. Although the cause is not entirely clear, those that experience sudden hearing loss have likely sustained cochlear infarction due to vasospasm[5,12]. Vasospasm has also been implicated in retinal migraine, although this diagnosis has come under scrutiny[13]. These patients have visual disturbances such as scotoma or blindness in addition to the diagnostic criteria for migraine without aura[2]. The etiology of this migraine variant is believed to be retinal artery vasospasm. Killer et al. photographed retinal examinations during these visual changes and described vasospasm of the retinal arteries[14]. Fortunately, those who experience these unilateral visual losses nearly always regain their vision after the vasospasm has resolved[13].

The patients in this report most likely experienced sudden hearing loss as a result of vasospasm. None reported preoperative fluctuating hearing levels, although one patient (patient 3) did report phonophobia as part of his typical migraine. One patient had a preoperative audiogram that demonstrated only mild flat SNHL, with PTA of 25-30dB (patient 2). Lee et al. described the pathologic changes in a patient who had experienced life long migraine and sudden SNHL several years prior to death. The affected ear demonstrated loss of hair cells and significant fibrosis consistent with a vascular accident[6]. Although there are no pathologic specimens from the patients in this report, they experienced a similar clinical course.

Furthermore none of the patients experienced vestibular symptoms at the time of their hearing loss. The arterial supply to the membranous labyrinth and cochlea comes from the anterior inferior cerebellar artery in the form of the labyrinthine artery. This artery enters the internal auditory canal and branches into the common cochlear and the anterior labyrinthine artery. It is the common cochlear artery and it terminal branch, the main cochlear artery, that supply 80% the cochlea (Figure 6). The vestibulocochlear artery supplies the remaining 20% at the base of the cochlea. The lack of vestibular symptoms in these patients suggests the vasospasm affected primarily the main cochlear artery and its branches. The 3 patients who did not recover hearing also demonstrated more significant loss in the low frequencies, suggesting that the main cochlear artery may be more susceptible to vasospasm and its consequent hearing loss. The arterial supply of the inner ear does not receive any collateral supply. Thus it is at risk of ischemic injury if any of the terminal branches are compromised.

The hair cells of the audiovestibular system are exquisitely sensitive to ischemia. Koga and colleagues caused experimental ischemia in gerbils by temporarily (5 minutes) occluding one vertebral artery. The gerbils experienced immediate increase in auditory thresholds. When sacrificed, the animals demonstrated inner hair cell loss that progressed until 10 days after the injury[15]. We propose transient migraine related vasospasm occurred long enough in three out of the four patients examined in this report to cause irreversible hair cell loss. Migraine has also been reported to occur after anesthesia, both after general and monitored anesthesia[8]. Patients usually experience their typical migraine although the intensity of the headache or focal neurologic signs may be stronger. Headache is considered a common minor side effect of general anesthesia. However, this side effect may be more common in patients with a history of migraine[10]. In case reports, migraineurs were given prophylactic sumatriptan, an ergot alkaloid, in anticipation of post operative migraine. These patients reported little to no perioperative headache[16].

Although migraine is not unusual after anesthesia, hearing loss after non-cardiac/non-otologic surgery is unusual[17]. Park et al. reviewed the literature and reported on 7 patients who underwent spine surgery and experienced unilateral sudden hearing loss. They proposed the etiology to be either perilymphatic fistula due to excessive Valsalva or accumulation of nitric oxide resulting in labyrinthine membrane rupture[18]. There was no mention in this report if the patients also suffered from migraine. Hearing loss after regional anesthesia via a spinal injection has also been well documented and appears to be related to cerebrospinal fluid losses causing a change in perilymph pressure via the cochlear duct[19].

For the three patients with anesthesia records available, the administered anesthesia was standard. Two of the patients experienced mild hypotension with systolic pressures reaching the 85-90 mm Hg level. As these episodes were mild and of short duration, it seems unlikely to be causally related to the hearing loss. One of the patients received nitrous oxide (patient 1) but the other two did not. Nitrous oxide has been proposed as a cause of perioperative hearing loss by causing a perilymphatic fistula due to round window rupture in patients with eustachion tube dysfunction[17]. None of the patients were reported to have nausea, vomiting or headache after their operation; while nausea and vomiting are common after anesthesia, they are also a symptom of vestibular dysfunction and vertigo. There is no evidence they experienced vestibular dysfunction after the operation. Thus, perilymphatic fistula is unlikely. There is no common thread in the anesthetic agents that point to a cause of the hearing loss; it is unlikely that anesthesia is to blame for the hearing loss.

The patients in this report all had a history of migraine. Given the evidence that migraine can cause hearing loss, we propose migraine related vasospasm as the likely cause of their hearing loss. The patient who received steroids in this report did fare better with significantly improved PTA and WRS. Therefore, we recommend that patients who present with migraine associated hearing loss after anesthesia be started on high dose steroids, as their condition permits. It remains unclear if prophylactic ergot alkaloids or other vasodilatory medications in patients with a migraine history would prevent this complication. Furthermore, these patients should be allowed to avoid their trigger mechanisms on the day of surgery, if possible[9]. Further research in the area is needed.

Conclusions

- Hearing loss after general anesthesia in migraine patients is a separate clinical entity.
- Cochlear vasospasm is the likely cause of hearing loss in these patients.
- Patients may benefit from immediate treatment with steroids.

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