Posterior cerebral stroke: An unusual cause of postoperative bilateral sensorineural hearing loss after laparoscopic surgery

Hearing impairment has been reported in patients following both general and regional anesthesia.\textsuperscript{[1]} Sensorineural hearing loss (SNHL), which occurs in the postoperative period, can be conductive or sensorineural, unilateral or bilateral, and transient or permanent.\textsuperscript{[2-6]} There are various causes attributed to it.\textsuperscript{[1]} Very rarely an impending circulation stroke can be attributed as a cause of SNHL.\textsuperscript{[7-10]}

Here, we describe such a case under general anesthesia and discuss its possible etiologies, diagnosis, possible prevention and treatment and suggest including it as a differential diagnosis of postoperative SNHL.

A 68-year-old, 70 kg, American Society of Anesthesiologists II female patient was scheduled to undergo laparoscopic surgery for cholelithiasias along with common bile duct exploration. There was no previous known history of atrial fibrillation, arteriovenous malformation or aneurysm. Preoperative baseline blood investigations were within the normal limits (WNL) except a serum glutamic oxaloacetic transaminase/PT (aspartate transaminase alanine transaminase) of 123/135 IU/L (N = 6-40 IU/L). Viral markers and preoperative ultrasound abdomen were nonsignificant. Chest X-ray showed patchy infiltrates in left lower zone, electrocardiograph revealed occasional ventricular ectopic, echocardiography revealed grade 1 diastolic dysfunction with ejection fraction of 60-62% with no intra-atrial thrombus and no patent foramen ovale. Pulmonary function tests revealed mild restrictive airway disease with mild airflow obstruction. Preoperative vital signs included pulse rate (PR) of 82/min and noninvasive blood pressure (NIBP) of 130/90 mmHg.

In the operation theater, standard monitors were attached, which revealed a preoperative NIBP of 170/94 mmHg and PR of 86/min. General anesthesia was induced by standard methods with maintenance with isoflurane 1.5% in nitrous oxide and oxygen (60:40). Surgery was performed laparoscopically in reverse trendelenberg position. Intra-operative two episodes of hypertension (>30% of baseline mean arterial pressure) were managed by intravenous boluses of diltiazem 2 mg. Surgery lasted for 3 h. Intra-operative ventilatory parameters were adjusted to keep the EtCO\textsubscript{2} WNL. After completion of surgery, neuromuscular blockade was reversed with glycopyrrolate and neostigmine and trachea was extubated. Her immediate postoperative recovery period was uneventful.

In the surgical ward, 4 h after surgery, she complained of severe vertigo and bilateral hearing impairment. An otolaryngology examination revealed bilateral dull tympanic membrane with significant nystagmus. Audiometric analysis confirmed bilateral profound sensorineural hypoaucosis on pure tone audiometry [Figure 1] and brainstem evoked response telemetry [Figure 2]. The impedance audiometry showed a type A curve showing lack of fluid or negative pressure in the middle ear or a prior Eustachian tube dysfunction or perilymph leak. An immediate computed tomography (CT) did not reveal any significant pathology intracranially or in the auditory system. Tablet prednisolone 1 mg/kg OD and tablet aspirin 150 mg were prescribed. However within next 24 h, she developed slurring of speech, weakness of all 4 limbs and became unconscious (E2M2V2). This time a magnetic resonance imaging (MRI) brain was performed which revealed diffuse cerebral abnormality involving deep white matter, corpus colossum, dentate nucleus of the cerebellum revealing ischemic changes [Figure 3a and b] with no dissection of cerebral arteries. Intravenous heparin was added to her treatment regime and was continued for 3 days along with subcutaneous injection enoxaparin 40 mg OD. Further biochemical investigations, lower limb Doppler and echocardiography revealed no abnormality except thyroid function tests with mild decrease in thyroid-stimulating hormone (TSH) ([TSH = 0.26 ug/dL; N = 0.39-4.6 ug/dL], T3 and T4 WNL). She subsequently improved to E4M4V2 status in 21 days and was discharged with advice to follow-up in neurology and surgery out-patient department. She has further improved to E4M4V3 in the last 2 years.

Sensorineural hearing loss, which is a rare entity, may be evident either immediately after emergence from anesthesia or noted several days later. It is most commonly associated with cardiopulmonary bypass (CPB) surgeries and is uncommon following non-CPB surgery.\textsuperscript{[1]}

Unilateral hearing loss occurs commonly due to embolism and subsequent ischemic injury to areas of the Organ of Corti and almost uniformly results in some permanent hearing deficit.\textsuperscript{[1]} Causes of bilateral hearing loss can be attributed to various causes in literature like changes in middle ear pressure (mainly with N\textsubscript{2}O anesthesia), vascular pathology (inner ear has no collateral supply), cerebrospinal fluid pressure changes, embolism, ototoxic drugs (diuretics, antineoplastics, antiinflammatory), and other miscellaneous causes.\textsuperscript{[1]}

An increased intra-abdominal pressure that accompanies laparoscopic surgeries, can redistribute blood to vital organs
Deviating the blood from the internal ear, which is particularly susceptible as it lacks collateral circulation.

The symptoms in our patient were initially attributed to the complication of prolonged laparoscopic surgery in reverse trendelenberg position with N\textsubscript{2}O anesthesia, and a CT scan was sought. However, she soon developed a posterior cerebral stroke.

Posterior circulation strokes can be hemorrhagic or thrombotic and can present with a wide variety of symptoms, including 5 Ds (dizziness, diplopia, dysarthria, dysphagia, and dystaxia).[^11]

Some authors have emphasized that even brief episodes of audio vestibular symptoms (minutes) can be a warning of an impending brainstem stroke.[^12] The main cause of this is that the inner ear is particularly sensitive to transient ischemia because of its high-energy requirements, and the labyrinthine artery is an end artery without collateral circulation.[^1]

The limitations of CT scanning for imaging the posterior fossa results from interference from the closely surrounding bone, which produces artifact and limits resolution. Thus, MRI is a better diagnostic tool than a CT scan in first 24-48 h of a stroke with higher success rate (it pick up 5 times more cases).[^13]

Traditionally heparin has been used in the treatment of posterior circulation strokes. However, there is wide variation in treatment regimens, including intravenous and intra-arterial thrombolysis, stenting of the basilar artery and surgical evacuation of the hematoma.[^14] Further studies are needed to define the safest and most efficacious treatment modalities for posterior circulation stroke.

The prognosis for hearing loss occurring postanesthesia is variable-poor for CPB and relatively good for nonbypass surgery. Patients at high risk for developing otoxicity are patients with impaired renal function; preexisting ototoxic drug serum levels; preexisting SNHL; and who could receive a synergistic combination of ototoxic drugs[^1].

An awareness of the potential for and the causes of hearing loss during anesthesia may permit the anesthesiologist to prevent or minimize the risk of significant hearing. Intra-operatively avoidance of nitrous oxide and use of calf pumps maybe helpful in preventing a peri-operative stroke.

By this case report we would like to highlight that though a rare entity sudden vertigo followed by bilateral sensorineural deafness in the immediate postoperative period could be a warning sign of an impending brainstem or cerebellar infarction/stroke and thus should be kept in the initial differential diagnosis of acute SNHL.

**Financial support and sponsorship**

Nil.
Letters to Editor

Conflicts of interest
There are no conflicts of interest.

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Access this article online

Quick Response Code:  
Website: www.joacp.org
DOI: 10.4103/0970-9185.168206

How to cite this article: Punj J, Nagaraj, Divya, Preetam P, Darlong V, Pandey R. Posterior cerebral stroke: An unusual cause of postoperative bilateral sensorineural hearing loss after laparoscopic surgery. J Anaesthesiol Clin Pharmacol 2016;32:528-30.

Source of Support: Nil, Conflicts of Interest: None declared.