

NEAR FATAL ASYSTOLE DURING MIDDLE EAR MICROSURGERY - CASE REPORT

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Introduction:

Middle ear microsurgery is one of the several types of surgeries where bloodless operative field is desired, and is usually achieved through deliberate hypotension. Various anaesthetic agents and adjuvants have been used for this purpose such as inhalation anaesthetics, beta blockers and α_2 agonists^{1,2,3}. Bradycardia has been reported as one of the adverse effects produced by these agents. But often, bradycardia is a manifestation of the vasovagal reflex, and anaesthetic agents merely act as potentiating agents. Ocular surgeries are the most common surgical procedures that evoke this reflex bradycardia^{4,5}. We report the occurrence of near fatal asystole due to vasovagal reflex during middle ear microsurgery.

Case report:

A 33-year old man (ASA I, 67kg, 160cm) was posted for excision of a recurrent right ear polyp. He had presented with foul smelling and non blood-stained discharge from the right ear with associated hearing loss for 10 years. He was operated 11 years ago for similar complaints. Apart from history of smoking (12 pack years) he gave no other significant history.

His preoperative laboratory results were normal. High resolution computed tomogram of the right temporal bone was suggestive of a cholesteatoma or an epidermoid in the middle ear cavity. The day before the procedure, his heart rate was 80/min and arterial blood pressure was 130/90 mmHg, and the remainder of the physical examination was also unremarkable.

He was premedicated with 3µg/kg of oral clonidine, two hours prior to procedure. Morphine 6mg, fentanyl 40µg, midazolam 1mg and thiopentone 200mg were used for induction and vecuronium 7mg for neuromuscular blockade. Trachea was secured with an 8.5mm cuffed orotracheal tube. Air, oxygen and halothane were used for maintenance. Preinduction heart rate and arterial blood pressure were 58/min and 118/80 mmHg respectively.

Fifteen minutes after the post aural incision, and immediately after the mastoid retractor was applied, there was a sudden drop in heart rate to less than 40/min which progressed to asystole with a simultaneous fall in arterial blood pressure. When the retractor was released, the asystole reverted to sinus rhythm spontaneously, but recurred when the retractor was reapplied. Atropine sulphate 0.2 mg was given intravenously immediately after the second asystole, which restored sinus rhythm. No bradycardia was encountered during the rest of the procedure. Once the surgery was completed, neuromuscular blockade was reversed with 0.05mg/kg of neostigmine and 0.02mg/kg of atropine sulphate, and the tracheal tube was removed when the patient was awake. He was shifted to postanaesthesia care unit and observed for two hours. Postoperative stay was uneventful and patient was discharged two days later.

Discussion:

Bradycardia and asystole occur most commonly during ocular surgeries and sometimes during non-ocular maxillofacial surgeries^{4,5,6}. Reports of bradycardia in middle ear surgeries are lacking.

Factors known to cause bradycardia during the intraoperative period include hypoxemia, light plane of anaesthesia, young age, type of stimuli and pharmacological agents like beta blockers, α_2 agonists and potent narcotics^{2,6,7,8}. Reflex bradycardia which is augmented by several pharmacological agents used in anaesthesia is also recognized

as an important cause. While oculocardiac reflex is an established phenomenon during ocular surgeries, variants of this reflex termed trigeminocardiac reflexes have been proposed to cause reflex bradycardia in non-ocular maxillofacial surgeries⁶. In our patient, bradycardia and asystole were encountered in response to surgical manipulation in the post auricular region, which indicates a vasovagal reflex, since this region receives afferent fibers from the auricular branch of the vagus (Arnold's nerve)⁹.

Other reported manifestations of this vagal reflex mediated through the Arnolds nerve are syncope and cough⁸.

Several factors that potentiate reflex bradycardia exist during anaesthesia. Under general anaesthesia the basal metabolic rate decreases, therefore, the cardiac work will also decrease resulting in bradycardia. The concomitant use of halothane further decreases the chronotropicity of the heart primarily by direct myocardial depression. Also, the use of clonidine could further aggravate this bradycardia by blocking central sympathetic output. The use of clonidine as premedication and the intraoperative use of narcotics, halothane and vecuronium could have contributed in potentiating this vasovagal reflex in our patient^{2,6,7,10}.

Reflex bradycardia during surgical procedures could be managed effectively by cessation of the surgical manipulation and administering anticholinergic agents like atropine or glycopyrrolate intravenously. If the reflex persists, infiltration of the nerve responsible for the afferent stimuli with local anaesthetic will attenuate this reflex.

Awareness of the existence of this reflex and its augmentation by the anaesthetic agents is central for early recognition and management of this potentially serious cardiac event. Continuous monitoring and anticipation during surgical manipulation are essential to prevent serious consequences.

Conclusion:

The vasovagal reflex can be triggered during mastoid surgery and can lead to cardiac asystole. A general awareness of this phenomenon is important to prevent catastrophe during an otherwise simple surgical procedure.

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