

Sitting-position Craniotomy in a Patient with Permanent Pacemaker

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ABSTRACT

Posterior fossa tumor surgeries are challenging from the point of view of both surgeons and anesthetists owing to the proximity of the tumor to vital centers, the difficulty faced in approaching the tumor, and complications associated with the positioning. The challenge in this patient undergoing a sitting-position craniotomy for a left-sided acoustic schwannoma was further augmented by the presence of a complete heart block in our patient (for which a pacemaker had been inserted) and the co-existence of a moderate aortic regurgitation.

Keywords: Aortic regurgitation, Pacemaker, Sitting-position craniotomy.

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INTRODUCTION

Conducting a surgery for posterior fossa tumors in a sitting-position is a challenge owing to the proximity of vital structures, occurrence of arrhythmias on tumor manipulation, hemodynamic changes due to the sitting-position, and increased possibility of air embolism.¹ Here we present a unique case featuring the successful management of a patient with a permanent pacemaker, co-existing aortic regurgitation, and posted for excision of a left-sided acoustic schwannoma in a sitting position. While there are case reports of patients undergoing other surgeries with pacemakers *in situ*, none are available for a patient undergoing a sitting-position craniotomy.²⁻⁴

CASE DESCRIPTION

A 68-year-old female was diagnosed with a complete heart block after a history of giddiness followed by a loss of consciousness seven months back. A permanent pacemaker (Medtronic) was inserted in the left pectoral region, with a VDD (i.e., ventricle-paced, dual-sensed, dual response) setting for an intrinsic ventricular rate of 30/minutes. She had no further episodes of loss of consciousness but continued to complain about giddiness. Left-sided acoustic schwannoma was found on further evaluation. Her preoperative echocardiography showed a moderate aortic regurgitation, concentric left ventricular hypertrophy, and left ventricular ejection fraction of 50%.

On the day of surgery, the pacemaker mode was changed to asynchronous fixed [i.e., ventricle-paced, none sensed, no response (VOO)], and the baseline heart rate was increased from 60 to 80 beats per minute. An intra-arterial cannula was secured before induction for invasive blood pressure monitoring. Transthoracic cutaneous defibrillator pads were attached to the patient in an anteroposterior position. Emergency drugs were available. Central venous access was secured with a peripherally inserted central catheter through the right cubital vein. A transesophageal echocardiography probe could not be used on account of unavailability. Preloading was done with 500 mL of Ringers lactate infused over 20 minutes. Sitting-position was given slowly with a careful watch on blood pressures. Bipolar cautery was used. Fluids were administered judiciously to target adequate pulse pressure variability and urine output.

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The patient maintained her hemodynamics in the intraoperative period. After extubation, the pacemaker was changed back to VDD with a baseline rate of 60 per minute. In the immediate postoperative period, we witnessed three episodes where the patient had no intrinsic rhythm and became pacemaker-dependent. During these periods, each lasting 1-2 minutes, her cardiac output dropped with mean arterial pressure (MAP) of less than 60 mm Hg and was followed by a spontaneous recovery. The baseline heart rate was then changed to 80 beats/minute. While the patient continued to remain pacemaker-dependent intermittently, her blood pressure remained normal.

The patient was shifted to the intensive care unit for monitoring. She was shifted to the ward 48 hours after the surgical procedure and was discharged 3 days later.

DISCUSSION

Cerebellopontine angle (CPA) tumors are the most common posterior fossa neoplasms, accounting for 5-10% of intracranial tumors.⁵ Surgery for these is a challenge because of the small space with poor compliance, presence of important structures (including the brainstem, cerebellum, and lower cranial nerves), presence of large venous sinuses, and a narrow pathway for the cerebrospinal fluid.¹

Surgical stimulation and tumor manipulation in the posterior fossa can lead to a wide range of arrhythmias ranging from bradycardia, tachycardia, and ventricular dysrhythmias to asystole,

accompanied by blood pressure changes. These hemodynamic changes warn about the impending damage to the adjacent cranial nerve nuclei and respiratory centers.⁶ A fixed heart rate setting for the pacemaker masks bradyarrhythmias, and supraventricular tachycardias are not conducted from the atrioventricular node, thus obfuscating the signs of brainstem handling.

Sitting-position increases the likelihood of hypotension owing to an increased systemic vascular resistance, heart rate, and pulmonary vascular resistance, as well as decreased venous return, arterial pressure, and cardiac output.⁷ Venous air embolism (VAE) is also a common complication with an incidence between 25 and 75% depending on the monitors used for diagnosis.¹ In spite of the risk of hypotension and VAE, the surgeons continued with the sitting position, as it improves surgical access, reduces the requirement of surgical retraction, and decreases intracranial pressure. Since the treatment for VAE involves aspiration of air, insertion of a central venous catheter (CVC) is inevitable. The guidewire of a CVC is arrhythmogenic, may dislodge recently inserted endocardial leads, may short the proximal lead with the distal lead, and may also activate the anti-tachycardia mechanism in intracardiac defibrillators. To avoid guidewire insertion, we inserted a peripherally inserted central catheter from the side opposite to the pacemaker.

The anesthesia management for a patient with moderate aortic regurgitation requires maintaining a slightly higher heart rate and lower systemic vascular resistance while maintaining a good preload and contractility. The increase in systemic vascular resistance resulting from the sitting-position was counteracted by maintaining a deep plane of anesthesia. The resting rate of the pacemaker was increased to reduce the amount of backflow into the left ventricle and the patient was given crystalloids to build pre-load.⁸

Electromagnetic interference (EMI) can temporarily or permanently damage the pacemaker, hence external defibrillators should be available. The anteroposterior pad placement, with the anterior pad placed away from the pulse generator, reduces EMI by creating an electrical field perpendicular to the intracardiac ventricular lead electrodes, which may thus prevent damage during cardioversion.⁹

HRS/ASA recommends a preoperative evaluation of all pacemakers once in 12 months. Postoperative interrogation is recommended when the patient undergoes a hemodynamically challenging surgery.¹⁰ This proved particularly useful in our patient, where we were able to detect the decreased cardiac output. This leads us to the possibility that the symptoms that lead to the diagnosis of the acoustic schwannoma could have resulted from decreased cardiac output in pacemaker-dependent episodes.

CONCLUSION

The required position for surgery and the co-existing aortic regurgitation had conflicting demands for the anesthetic management for the patient. Both the acoustic schwannoma and complete heart block had similar symptoms, which possibly lead to a failure to diagnose an insufficient intrinsic pacemaker rate. However, with careful planning, and vigilance throughout the procedure, we were able to safely anesthetize the patient.

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