Sick sinus syndrome with Alzheimer’s disease for bilateral hernia repair

Sir,

There is paucity of literature on delivery of anesthesia to patients with coexisting sick sinus syndrome and Alzheimer’s disease. We recently administered anesthesia to a 79-year-old, 34 kg, 148 cm male patient of Alzheimer’s disease with thoracolumbar kyphosis for bilateral inguinal hernia repair. The patient was a known case of Sick Sinus syndrome and had a permanent pacemaker implanted 11 years back. He was diagnosed to have senile dementia with Alzheimer’s disease 7 years back and was taking oral quetiapine 6.25 mg once a day and rivastigmine 1.5 mg twice a week.

The patient was irritable and uncooperative. Preoperative blood investigations were normal. ECG showed occasional premature ventricular beats with evidence of pacemaker spikes. 2D echocardiography revealed left ventricular ejection fraction of 62%, and pacemaker electrodes were visible at the apex of right ventricle. X-ray chest confirmed the electrode position, and the pacemaker pulse generator was seen in the right thorax. The pacemaker type was Regency SC + 2402L VVI as per the patient records. The patient was on regular cardiac checkup, and functioning of pacemaker along with battery check was done by cardiologist prior to surgery.

The patient received the morning dose of 6.25 mg of quetiapine. Routine monitors were attached and intravenous access was established. Supplemental Oxygen was administered at the rate of 2 L/min. Emergency cardiac drug tray and defibrillator were kept ready in the operation theater. The right internal jugular vein was cannulated for central venous pressure (CVP) monitoring and emergency insertion of temporary pacemaker leads, if needed. An arterial line was established for beat-to-beat monitoring of blood pressure. Epidural anesthesia was planned. As the patient was irritable, anxious, and uncooperative, titrated doses of midazolam 1.25 mg and fentanyl 50 μg were administered to sedate the patient. Epidural catheter was placed via the L3–L4 intervertebral space in the left lateral position. An initial test dose of 2 ml of 2% lignocaine was given. Two more topups of 4 mL 1.5% lignocaine each were given after confirming no adverse reaction to the test dose. Sensory block up to T7 level was achieved in 20 min. After 30 min, an epidural infusion of 0.5% bupivacaine with fentanyl 2 μg per mL was started at the rate of 5 mL/h, which provided adequate level of anesthesia.

Additional supplemental doses of local anesthetic were not required. The surgery lasted for 2.5 h.

Hemodynamic parameters and oxygen saturation were maintained within normal limits. Hemostasis was achieved by conventional ligation of bleeders. A bipolar cautery was kept ready but its use was avoided. The patient was monitored in intensive care unit for 48 h and discharged on the 7th postoperative day.

Sinus node dysfunction in the elderly can have many causes but is usually idiopathic. The sick sinus syndrome refers to a combination of symptoms (dizziness, confusion, fatigue, syncope, and congestive cardiac failure) caused by sinus dysfunction and manifested by marked sinus bradycardia, sinoatrial block, or sinus arrest. Permanent pacemakers are the mainstay of therapy for patients with symptomatic sinus node dysfunction.[1] Our patient had a VVI type rate adaptive demand pacemaker which automatically adjusts ventricular pacing rates to a sensed indicator of exertion.

Demand pacemakers can sense electrocautery, and inhibit pacemaker firing, leading to asystole. Using a bipolar rather than unipolar electrocautery is recommended. The electrocautery grounding pad should be placed as far away from the pulse generator as possible. It should be used at the lowest effective current and in short bursts with long pauses. To monitor perfusion, a Doppler detector or intraarterial line may be used.[2]

Alzheimer’s disease is a common form of dementia, characterized by the degeneration of the basal forebrain cholinergic neurons innervating the cortex, amygdale, and hippocampus, with difficulty in sustaining attention and profound cognitive impairment, such as loss of memory and learning ability.[3] Most of the general anesthetic agents interact with both nicotinic and muscarinic receptors. They produce inhibition of central cholinergic transmission, which is already impaired because of age-related changes.[4] Anesthetists need to be aware of the possibility of unfavorable drug interactions, for example, cholinesterase inhibitor rivastigmine and its metabolites block acetylcholine hydrolysis and antagonize atracurium, whereas it accentuates the effects of succinyl choline.

We avoided General Anesthesia and planned regional anesthesia. Several studies have shown that analgesia levels obtained after subarachnoid block are approximately 3–4 spinal segments higher in elderly compared to young adults.[5] A paced heart cannot compensate for hypotension by tachycardia and hence spinal anesthesia should be cautiously used in such cases. The more gradual onset of sympathetic blockade after epidural...
anesthesia provides more time for compensatory function to take over. As our patient had thoracolumbar kyphosis, we avoided large bolus dose and gave titrated bolus doses local anesthetic and thereafter administered supplemental dose by infusion. This was sufficient to achieve the desired anesthetic level and we succeeded conducting this case uneventfully.

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