Dexmedetomidine in anaesthesia for a high-risk case of pheochromocytoma with poor left ventricular function

Sir,

Anaesthesia for pheochromocytoma is challenging and stressful to the caregivers. Reports of a successful outcome in pheochromocytoma with limited cardiac reserve are few.[1] We present the management of one such case who underwent laparotomy and excision of pheochromocytoma.

A 52-year-old male (weight = 53 kg) presented with paroxysmal headache, sweating, palpitation, chest pain, tremors and abdominal pain since 2 months. He was a smoker and was hypertensive for the past 3 years. He suffered anterior wall myocardial infarction

3 years back (thrombolysed) and coronary angiogram done revealed mild left anterior descending artery disease and with NYHA grade 3 then.

His pulse rate (PR) was 120/min, regular, and blood pressure (BP) 214/140 mmHg. Haemoglobin was 11.8 g/dl, haematocrit 35.6, platelet count 471,000/mm^3, urea 31 mg/dl, creatinine 1.7 mg/dl, blood sugar 86 mg/dl, blood group "O" negative and urinary vanillylmandelic acid 66 mg/24 h. Electrocardiogram showed left bundle branch block and left axis deviation [Figure 1a]. Echocardiography showed thin akinetic interventricular septum and anterior-wall, dilated left ventricle with apical aneurysm and left ventricular ejection fraction of 30%. Computed tomography revealed a 5.2 cm × 6.8 cm × 6.1 cm heterogeneously enhancing mass in the right suprarenal region with multiple non-enhancing necrotic areas and calcifications [Figure 1b].

He was started on oral prazosin 5 mg, clinidipine 10 mg and torsemide 10 mg twice daily, aspirin-atorvastatin 75/10 mg once daily and discharged after 1 week. On readmission 3 weeks later, his PR was 98/min, and BP 152/100 mmHg in sitting position and 142/100 mmHg on standing. Tablet atenolol 50 mg once daily was started and aspirin was stopped a week before surgery.

He was fasted overnight and received oral diazepam 10 mg. A 14-gauge intravenous cannula was secured under local anaesthesia and intravenous fentanyl 50 μg was administered. A radial arterial cannula, epidural at T_{6-7} and central venous and pulmonary artery catheter, was secured via the right internal jugular vein. The mean pulmonary artery pressure (PAP) and wedge pressure were 29 mmHg and 24 mmHg, respectively. Initial cardiac output (CO) was 3.6 L/min. Bispectral index (BIS™) monitoring was commenced.

Dexmedetomidine 50 μg was administered over 10 min followed by 0.5 μg/kg/h infusion which was stopped 30 min before extubation. Nitroglycerine infusion was started (5 μg/min) and continued until extubation. Epidural analgesia was initiated with 10 ml of bupivacaine 0.125% with fentanyl 50 μg and maintained with bupivacaine 0.125% and fentanyl 2 μg/ml infused at 4 ml/h intra- and post-operatively.

Anaesthesia was induced with fentanyl 100 μg, propofol 40 mg and vecuronium 6 mg. Esmolol 20 mg was administered 90 s before endotracheal intubation. Anaesthesia was maintained (BIS = 35–50) with propofol, N_2O, O_2, vecuronium and dexmedetomidine infusion with controlled ventilation. Four boluses of phenylephrine 50 μg each of which three were administered 5–10 min post-intubation and one during tumour handling. Hypertension during tumour handling was managed with sodium nitroprusside, esmolol 100 mg and metoprolol 15 mg. Noradrenaline and dopamine infusions were started following ligation of the adrenal veins. The highest and lowest PRs were 130/min and 62/min and BPs were 219/94 mmHg and 75/40 mmHg respectively, during tumour handling. Mean PAP and CO showed fluctuations similar to the systemic BP and heart rate [Figure 2]. Arterial blood gas analysis performed intra-operatively showed no significant metabolic acidosis or electrolyte imbalance.

A total of 2.5 L crystalloid and 0.5 L starch was infused. Blood loss was 250 ml, urine output 750 ml and duration of anaesthesia was 4½ h. Trachea was extubated on the table. In the intensive-care-unit, invasive pressures and blood sugar were monitored, inotropes were tapered off, and he recovered well.

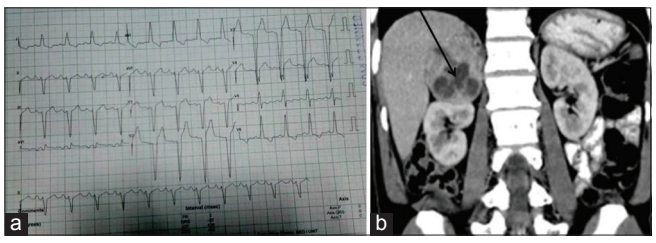

Figure 1: (a) Electrocardiogram showing left bundle branch block and left axis deviation, (b) Computed tomography showing right suprarenal mass (5.2 cm × 6.8 cm × 6.1 cm) with multiple non-enhancing necrotic areas and small calcifications within the mass

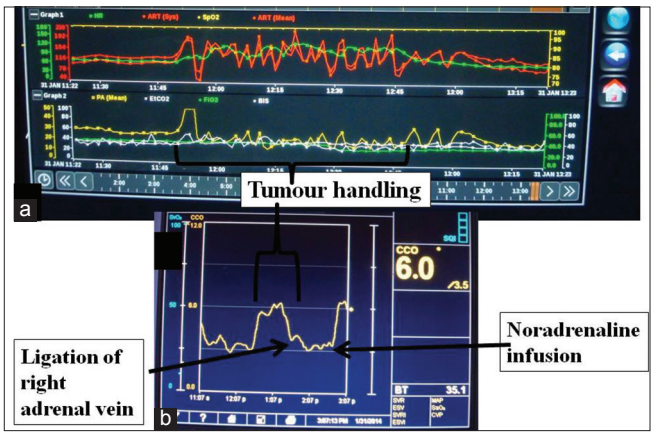

Figure 2: Records of (a) intra-operative vital parameters showing fluctuations in haemodynamics and (b) cardiac output

Excessive catecholamine release, increased O_2 demand, toxic effects and coronary spasm may be the cause of myocardial injury and infarction in pheochromocytoma. There is a correlation between intra-operative haemodynamic instability and plasma catecholamine levels.[2] There are a few reports of the use of dexmedetomidine as anaesthetic adjuvant for resection of pheochromocytoma.[3,4] Dexmedetomidine, a shorter-acting highly selective central α2-adrenoceptor agonist, attenuates the sympathetic response to intubation and emergence from anaesthesia,[5] reduces anaesthetic requirements and enhances post-operative analgesia. Considering the usual haemodynamic instability during pheochromocytoma resection, we think that dexmedetomidine contributed towards haemodynamic stability (systolic BP > 200 mmHg only once) in our patient by limiting the cardiac effects of catecholamines.

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Conflicts of interest

There are no conflicts of interest.

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