Thoracic combined spinal epidural anesthesia in patient of dilated cardiomyopathy undergoing laparoscopic cholecystectomy

Sir,

A 54-year-old female (55 kg, 170 cm) was scheduled for laparoscopic cholecystectomy. She was a diagnosed case of dilated cardiomyopathy (DCM) with severe systolic dysfunction, left ventricular ejection fraction (LVEF) 20%, on cardiac resynchronization therapy (biventricular pacing) and hypertension, type 2 diabetes mellitus and interstitial lung disease. She had history of dyspnea (NYHA class II). On examination she looked ill, lying supine in bed. Blood pressure was 120/78 mmHg and heart rate was 76/min. Bilateral wheeze was heard on auscultation of chest. Her hemoglobin was 11.3 g/dl and normal coagulation profile. There were no signs of volume overload on chest radiograph. Echocardiography revealed global hypokinesia with LVEF 20%, dilated left atrium (LA), severe mitral regurgitation (MR), and moderate tricuspid regurgitation (TR). The patient was on Digoxin, Metoprolol, Ramipril, Aldactone, metered dose inhaler and insulin. She was not on any anticoagulant. Ramipril and diuretic were withheld 1 day prior to surgery.

After considering the options, and discussing them with both the patient and surgeon, informed consent was taken to administer thoracic combined spinal epidural anesthesia. No premedication was given. The patient was shifted to operation theatre, and radial arterial cannulation was done under local anesthesia for invasive blood pressure monitoring. Right internal jugular vein was cannulated under local anesthesia for central venous pressure monitoring. Other routine monitoring, namely pulse oximetry (SpO₂), ETCO₂ and electrocardiogram, was initiated. CSE was performed with the patient in the sitting position and block was performed at the 10th thoracic interspace using a midline approach. The epidural space was identified using the “loss of resistance” to air method. After entering the epidural space, a 27-gauge pencil point Whitacre spinal needle was advanced through the Tuohy needle. After free and clear flow of cerebrospinal fluid (CSF) was achieved, 2 ml isobaric Levobupivacaine 0.5% and 25 µg fentanyl was injected and then the spinal needle was removed. The epidural catheter was then threaded into place, and fixed at 4 cm within the epidural space. Immediately the patient was turned to the supine position for the operation. Oxygen at 3 to 4 L/min was given to the patient by the face mask. Within 5 min a segmental sensory (pinprick) block, extending between the fourth thoracic and first lumbar dermatomes was obtained. There was no motor weakness in the legs and no signs of respiratory distress.

Laparoscopic cholecystectomy was commenced after creating pneumoperitoneum with CO₂, flow rate of 1.8 L/min and intraabdominal pressure of 8 mmHg. Empyemic gall bladder was removed uneventfully (surgical time about 30 min). Short intervals of bipolar cautery were used. The patient remained hemodynamically stable throughout the procedure. However, she complained of shoulder tip pain intraoperatively which was relieved by 25 µg of fentanyl i.v. and shoulder massage. She received 1 L of i.v. crystalloid fluid perioperatively and central venous pressure (CVP) was maintained between 6 and 10 cmH₂O. Phentylephrine
was planned to be used in case of hypotension. In case of respiratory distress conversion to general anesthesia (GA) was planned.

Postoperatively she remained hemodynamically stable and required epidural analgesia (8 ml of 0.125% Levobupivacaine) three times in the next 24 hours. She was shifted to ward 24 hours later after removal of invasive lines. Patient was followed to discharge wherein she remained stable.

An enlarged left ventricle with decreased systolic function as measured by LVEF characterizes DCM. Systolic failure is more marked than the frequently accompanying diastolic dysfunction although the latter may be functionally severe in the setting of marked volume overload. Anesthetic management of patients with cardiomyopathies is associated with high morbidity and mortality, thus require careful planning. The goals for anesthetic management are maintenance of cardiac output, avoidance of drug-induced myocardial depression, maintenance of normovolemia, and prevention of increased ventricular afterload.

Theoretically neuraxial blocks may decrease systemic vascular resistance secondary to sympathetic blockade thereby increasing the vascular capacitance which results in a lowering of LV filling pressure, a reduction in mitral regurgitation, and improved forward cardiac output without increasing heart rate or causing arrhythmias; however, the decreased systemic vascular resistance produced by epidural or spinal anesthesia is not always predictable or easy to control, but if segmental blockade is achieved the sympathetic block is restricted to fewer dermatomes thus decreasing the chances of severe preload reduction.

El-Dawlatly et al. reported uneventful anesthetic management of a patient with DCM who underwent laparoscopic cholecystectomy under thoracic epidural anesthesia. Van Zundert et al. reported successful management of a patient posted for laparoscopic cholecystectomy having severe lung disease under segmental spinal anesthesia. Daabiss et al. reported successful management of a patient with severe DCM undergoing a long surgical procedure using combined thoracic epidural analgesia and general anesthesia. Hashimoto et al. reported that high-dose epidural fentanyl anesthesia is anesthetist method of choice for patients with DCM.

In conclusion, a patient with limited cardiac reserve like DCM presents challenge to anesthesiologists. Such patients should be thoroughly assessed preoperatively, all precipitating factors for heart failure should be sought and aggressively treated before proceeding with elective surgery. The presence of heart failure has been described as the single most important risk factor for predicting perioperative cardiac morbidity and mortality. Regional anesthesia may be an alternative to general anesthesia in selected patients with DCM, as it produces changes in the preload and afterload that mimic pharmacological goals in the treatment of this disease.

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