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Thoracic epidural anaesthesia in valvular cardiac surgery

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EDITOR:

The use of thoracic epidural anaesthesia (TEA) in patients undergoing cardiac surgery, although increasing in popularity, remains controversial [1,2]. Moreover, while relatively large series of patients undergoing coronary revascularization under TEA have been studied, a small number of cases has been reported in which TEA has been used in valvular cardiac surgery [3]. We report the successful use of TEA in a challenging case of mitral surgery, in a patient with severe co-morbidities and allergies contra-indicating the administration of non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. The patient was previously rejected by other cardiosurgical centres.

A 49-yr-old (157 cm, 59 kg) female with severe mitral regurgitation was scheduled for valve replacement. Co-morbidities included hypertension, a history of congestive heart failure and transient acute renal failure, pectus excavatum with severe respiratory deficit, multiple allergies (including various antibiotics, paracetamol and NSAIDs), previous transfusion reaction, glucose-6-phosphate dehydrogenase (G-6-PD) deficiency, post-thyroidectomy hypothyroidism and hypoparathyroidism. The patient was receiving amlodipine, clonidine, spironolactone, budesonide, calcium fumarate, salbutamol, beclomethasone, oxygen, l-thyroxine and calcium carbonate. She complained of fatigue and dyspnoea on minimum exertion and was orthopnoeic, with reduced chest expansion, reduced dipharagmatic mobility and abnormal sounds in the inferior pulmonary fields. Electrocardiography showed sinus rhythm and signs of left ventricular hypertrophy. Chest X-ray was consistent with the patient's cardiac and pulmonary diagnosis. Transthoracic echocardiography revealed moderate-to-severe mitral regurgitation caused by functional tethering and fibrosis of the posterior leaflet, moderate concentric left ventricular hypertrophy, hypokinetic cardiopathy with no geometric remodelling and left ventricular ejection fraction of 40%. It also showed increased pulmonary artery pressure (50 mmHg systolic), with moderate tricuspid regurgitation and mild right ventricular failure. No preoperative transoesophageal examination was performed because the patient did not tolerate the procedure. Spirometry confirmed the severe obstructive–restrictive pattern with forced expiratory volume in 1 s (FEV_1) 31% and forced vital capacity (FVC) 43% of predicted. Partial pressure of O_2 (PaO_2) and CO_2 ($PaCO_2$) were 69 and 40 mmHg, respectively, with saturation of O_2 (SaO_2) 92% at arterial blood gases analysis on room air. Other biochemical and haematological parameters were within normal limits.

In view of her preoperative status, it was believed that the patient would benefit from TEA. The risks and benefits were explained to her and she gave written informed consent. Epidural catheterization was performed on the day prior to surgery. Her pre-operative platelet count (390×10^9 mL^{-1}) and coagulation (international normalized ratio (INR) 0.87, partial thromboplastin time (PTT) ratio 0.87) were within the normal range. Catheter placement was accomplished by an experienced anaesthesiologist in a high-dependency area. Monitoring included electrocardiogram, non-invasive blood pressure and pulse oximetry. The patient was positioned sitting upright and an 18-G epidural catheter was inserted at T5–T6, using the loss-of-resistance technique and a midline approach, in a single attempt. The catheter was advanced 6 cm cephalic

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into the epidural space. No bleeding was noted and a test dose of 2 mL of 2% lidocaine was injected.

On the day of operation, the patient received her usual medication, followed by lorazepam 3 mg by mouth. Standard monitoring plus pulmonary artery catheter were applied. General anaesthesia consisted of fentanyl, midazolam, propofol, rocuronium and sevoflurane. After induction, a bolus dose of 5 mL of 1% lidocaine and fentanyl 5 $\mu g\,mL^{-1}$ was administered by epidural catheter, with supplemental boluses of 3–5 mL of the same mixture throughout the operation (20 mL total amount) according to arterial pressure stability. A mitral valve replacement with mechanical prosthetic valve using moderately hypothermia (32°C) and cardiopulmonary bypass was performed. The patient was given 300 $UI\,kg^{-1}$ of heparin and intermittent bolus doses to maintain the activated coagulation time (ACT) >480 s. At termination of bypass, normal coagulation was restored using protamine sulphate in the ratio of 1 $mg\,kg^{-1}$ for every mg of heparin administered according to the ACT. Surgery was uneventful and no difficulties in haemostasis were noted. Weaning required inotropic support with epinephrine 0.05 $\mu g^{-1}kg\,min^{-1}$ and an enoximone 30 mg bolus plus 3 $\mu g\,kg^{-1}\,min^{-1}$ continuous infusion. After the completion of the procedure, the patient was transferred to the intensive care unit. An epidural infusion of 0.2% ropivacaine and fentanyl 0.4 $\mu g\,mL^{-1}$, at a rate of 4–7 $mL\,h^{-1}$, was given as postoperative analgesia for the first 72 h.

Inotropic support was progressively reduced and stopped on the first postoperative day. Also tracheal extubation was performed on the same day with good post-extubation gas exchanges in comparison with preoperative values. Good analgesia was obtained, with a pain score of 1 at rest and 3 during coughing. No parenteral opioids were required postoperatively. Neither paresthesiae nor heaviness were reported, nor any motor weakness was detected. Low-molecular-weight heparin (LMWH) 100 $UI\,kg^{-1}$ twice daily was started on the second postoperative day as anticoagulation therapy. No oral anticoagulants were administered. The patient experienced a transient episode of atrial fibrillation that regressed after a 300 mg bolus dose of amiodarone on the third postoperative day. A pyrexia developed on the second and third days despite antibiotic prophylaxis with cefazoline. We removed the epidural catheter 12 h after a dose of LMWH and discharged the patient from ICU on the fourth postoperative day when starting oral anticoagulants.

The risk–benefit ratio of applying TEA to patients undergoing cardiac surgery is a lively debated topic [1]. Many potential clinical benefits have been suggested and investigated, but effects on clinical outcomes remain to be determined and compared with the risk of epidural haematoma [1,4,5]. The only clear benefit of using TEA in cardiac surgical patients is enhanced postoperative analgesia [6].

Respiratory therapy and aggressive control of postoperative pain were considered key components in the management of this high-risk patient, and TEA played an important role in view of the long list of patient co-morbidietes. A cautious application of TEA [7] provided excellent postoperative analgesia without parenteral opioids and NSAIDs, and allowed a rapid recovery of consciousness, a reduced time to tracheal extubation and an early active respiratory physiotherapy and mobilization, thus simplifying postoperative management in this complex patient.

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