Case study: An unusual cause of clotted blood in epidural catheter

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Keywords: epidural catheter; blood; IVC thrombus; epidural veins

Abstract

The failure of an epidural catheter after initially functioning well may be due to kinking, knotting or epidural catheter lumen blockage. The presence of blood in the epidural catheter is usually due to the catheter's traumatic placement or to intravascular migration. We describe an unusual cause of blood in the epidural catheter.

Introduction

Epidural analgesia is a well-established technique for postoperative pain relief in patients undergoing major thoracoabdominal surgery. However, failure to inject through the epidural catheter after it initially functioned well may be due to catheter blockage caused by kinking or knotting.[1] Defective connection assembly or failure to insert the catheter properly into the connection assembly can also result in the failure of a well-placed epidural.[2] We report a case in which epidural analgesia was planned to provide intra- and postoperative pain relief. After the initial successful administration of a drug for intraoperative analgesia, further administrations could not be injected. Catheter removal after the surgical procedure revealed clotted blood in the epidural catheter lumen.

Case description

A 67-year-old ASA II patient was scheduled for a right-sided radical nephrectomy for renal cell carcinoma. Preoperatively the patient was detected to have chronic obstructive airway disease and was stabilised on inhaled and oral bronchodilators. His blood gas analysis on room air revealed a PaO_2 of 74 mmHg, a $PaCO_2$ of 50 mmHg, and his pH was 7.4. The patient also had a partially occluding level I tumour thrombus in the inferior vena cava, for which he was on low molecular weight heparin that was stopped 12 hours prior to surgery. Written informed consent was obtained and the patient was advised to continue the bronchodilators. Sedative premedication was withheld in view of his respiratory disease. In the operation theatre, the epidural catheter was placed atraumatically with the first attempt at the L_{3-4} epidural space with the patient placed in the lateral position. After placing the patient supine, a test dose of 3 ml 2% lignocaine with adrenaline was injected through the catheter after negative aspiration. Two boluses of 5 ml 0.125% bupivacaine with 25 µg of fentanyl were injected into the epidural space with a 10 min interval. General anaesthesia was then induced with 100 mg of propofol, and endotracheal intubation was facilitated using 8 mg vecuronium bromide. Incremental doses of fentanyl were used for intraoperative analgesia. Intraoperative monitoring was done with electrocardiography, pulse oximetry, end-tidal carbon dioxide measuring, a naso-pharyngeal temperature probe, intra-arterial blood pressure monitoring and a central venous pressure catheter. Three hours after the commencement of surgery we failed to inject a second bolus via the epidural catheter, as marked resistance to the injection was encountered. The initial patent catheter was now dysfunctional due to luminal obstruction. No further boluses could be administered through the epidural catheter and surgery proceeded uneventfully. The dressing covering the epidural catheter was removed at

the end of surgery and clotted blood was noticed in the catheter lumen up to the 4 cm mark (from the tip). The patient was subsequently extubated. The International Normalised Ratio (INR) was determined to be normal at 1 and the epidural catheter was removed. No kinking of the catheter was found upon removal. Patient-controlled analgesia was used for postoperative pain relief and the patient was discharged home ten days after surgery.

Discussion

The benefits of epidural analgesia are limited by technical failure in up to one in seven patients. McLeod et al[3] and Ballantyne et al[4] reported failure of epidural analgesia despite a functioning catheter in 1.4% and 3.6% of cases respectively. In our patient, epidural failure was due to the presence of clotted blood in the epidural catheter. Blood in an epidural catheter can be due to blood vessel trauma while placing the catheter, accidental intravenous placement or migration and/or a deranged coagulation profile. In our patient the epidural catheter was apparently placed atraumatically, with no evidence of intravascular catheter placement/migration after the first epidural bolus. Our patient had a normal coagulation profile preoperatively. A possible reason for the presence of blood in the epidural catheter before the second bolus could be that once the inferior vena cava (IVC) was clamped, the vertebral azygos system acted as an alternative pathway for the venous return, resulting in distension of the epidural veins, with increased flow. We postulate that fragile epidural veins in this elderly patient might have sustained (partial) trauma at the time of catheter placement, and then were not able to bear the stress of the increased flow after clamping of the IVC. Blood subsequently entered the epidural catheter and eventually clotted. The patient was not moved after the first bolus of drug, making intravascular migration of the catheter less likely.

The epidural veins form a network of valveless veins that are situated mainly in the anterolateral aspect of the epidural space. Venous return from the pelvis passes through the epidural venous plexus to the azygos vein, bypassing the IVC. Thus, any obstruction to the IVC flow redirects venous return through the vertebral venous plexus. Not only are the epidural veins then distended, but flow is also markedly increased and probably channelled. Any accidentally injected agent will go as a bolus directly to the right atrium, arriving at the heart in a concentration many times greater than under conditions of free, unobstructed caval flow. The dilatation of the epidural veins decreases the epidural space volume, distributing the local anaesthetics drug more widely and increasing the extent of the block. Exposure of the local anaesthetic agent volume to a greater vascular surface area increases the risk of local anaesthetic agent toxicity owing to greater absorption from the epidural space.[5]

In brief, in a patient with IVC obstruction dilated epidural veins possibly sustain injury at the time of epidural catheter placement or later, resulting in accidental intravascular placement or migration of the catheter. Therefore extreme caution must be applied when utilising a constant infusion of drug(s) through an epidural catheter after IVC clamping. Epidural bleeding can occur subsequent to IVC clamping, blocking an initially well-functioning catheter. In addition, less drug is needed to achieve a desired block level secondary to the epidural venous engorgement. If such a patient is receiving a continuous epidural infusion, catheter patency should be assessed frequently and the haemodynamics monitored closely for signs of intravascular drug administration. It is also paramount that the practitioner be on the lookout for a symptomatic epidural haematoma.

References

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