Case Report

Central neuraxial blockade for splenectomy in myeloproliferative disease: A word of caution

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ABSTRACT

We describe management of portal vein thrombosis (PVT) in a patient with myeloproliferative disease after splenectomy. This case posed a unique therapeutic challenge in maintaining a fine balance between life-saving thrombolysis and the risk of neuraxial complications due to bleeding. The incidence of PVT after splenectomy in patients with myeloproliferative disorders is high (40%). Anaesthesiologists should be aware of this and avoid central neuraxial blockade in such cases. If post-operative emergency thrombolysis is required in a patient having an epidural catheter *in situ*, it should be done under close monitoring, weighing the risks and benefits. Fibrinogen levels should be monitored to evaluate the presence of residual thrombolytic effects and to time the catheter removal.

Key words: Epidural, myelofibrosis, portal vein thrombosis, splenectomy, thrombolysis, venous thrombosis

INTRODUCTION

Myelofibrosis is a myeloproliferative disease (MPD) in which bone marrow is replaced with collagen connective tissue fibres due to the proliferation of an abnormal clone of haematopoietic progenitor cells. Splenectomy is sometimes performed before bone marrow transplant (BMT), especially if the patient has splenomegaly with hypersplenism. We describe a case of portal vein thrombosis (PVT) after splenectomy for MPD with an epidural catheter *in situ* which posed a therapeutic challenge. We wish to highlight the need for anaesthesiologists to be aware of the high risk of PVT after splenectomy for MPD.

CASE REPORT

A 51-year-old male patient with myelofibrosis presented with gross splenomegaly with left-sided obstructive uropathy and no co-morbidities. Other than anaemia and slightly deranged renal parameters (blood urea: 57 mg/dl, serum creatinine: 1.6 mg/dl), other investigations were normal. Prior to BMT, he underwent splenectomy under a combination of epidural and general anaesthesia. Intraoperative course was uneventful. On post-operative day (POD) 1, the patient developed fever, disproportionately severe abdominal pain in spite of adequate epidural analgesia and rise in liver enzymes (serum glutamic oxaloacetic transaminase: 1947 IU/L, serum glutamic pyruvic transaminase: 2798 IU/L). Ultrasonography of abdomen showed mild ascites. A contrast-enhanced computed tomography scan revealed right and main PVT extending up to proximal superior mesenteric vein, infarction of right lobe of liver (segment V, VI, VII), and oedematous wall of gastric and small bowel loops, moderate ascites and splenic bed collection [Figure 1]. Systemic heparinisation was immediately started to target an activated partial thromboplastin time of 2–3 times the control.

The intensivist, anaesthesiologist, surgical oncologist, intervention radiologist and medical oncologist jointly

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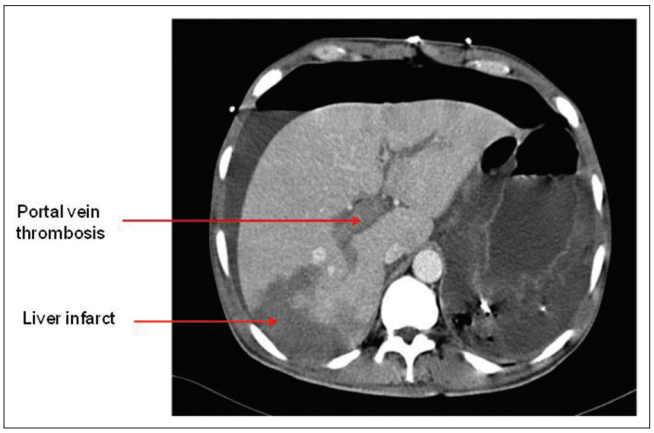

Figure 1: Computed tomography scan of patient showing portal vein thrombosis and infarction of liver

discussed the pros and cons of removing the epidural catheter and waiting before starting local thrombolysis in an already anticoagulated patient. Removing the catheter before thrombolysis would have significantly delayed thrombolysis with the risk of extension of liver infarction. Hence, arterial thrombolysis keeping the catheter *in situ* with close neurological monitoring was planned. Epidural local anaesthetic infusion was stopped, and intravenous patient-controlled analgesia with morphine was started. Indirect thrombolysis of mesenteric and portal vein was started with injection alteplase (6 mg bolus + 1 mg/h) through the micro-catheter placed in the superior mesenteric artery. Systemic heparinisation was continued. Patient was clinically assessed for lower limb neuromotor deficit at close intervals and haemoglobin and lactate levels were monitored.

On POD 4, there was reduction in haemoglobin levels with significant bleeding from epidural site with soakage of bed linen (approximately 100–200 ml) and without any neurodeficit. Repeat angiography demonstrated partial recanalisation of portal vein and hence the micro-catheter was removed, alteplase was stopped and systemic heparinisation continued.

On POD 7, heparin was stopped, and the epidural catheter was removed under fresh frozen plasma cover. Patient was subsequently discharged without any neurological deficit on a therapeutic dose of low molecular weight heparin.

DISCUSSION

PVT can be a serious and potentially life-threatening complication of splenectomy. The patient had emergent indications for thrombolysis posing a unique management challenge. On one hand, there was an urgent need for thrombolysis for the PVT, while on the other hand there was a risk of neurological complications due to recent epidural catheter insertion.[1] Thrombolysis was considered to be more effective, despite the increased risk of bleeding, and inability to monitor, titrate or reverse its effect, as compared to anticoagulation alone.

The American Society of Regional Anesthesia and Pain Medicine[2] (ASRA) suggests avoidance of thrombolytic therapy for 10 days after neuraxial puncture (Grade 1A). However, this was not considered feasible in our patient. ASRA recommends neurological monitoring with interval ≤2 h and avoidance of epidural infusion causing sensory and motor block to facilitate neurological assessment. There is no definitive recommendation for removal of neuraxial catheters in patients who unexpectedly receive thrombolytic therapy during a neuraxial catheter infusion. They suggest measurement of fibrinogen levels to evaluate the presence of residual thrombolytic effect and to time catheter removal accordingly.

Patients undergoing splenectomy have an increased risk of splenic/PVT (SPVT). With improved quality and frequency of radiological imaging, it is clear that this occurs more frequently than was earlier thought.[3,4] In a systematic review, the overall risk of symptomatic SPVT was found to be 3.3%. The incidence of PVT varies depending on the indication for splenectomy, being high in MPDs and hereditary haemolytic anaemias and low in trauma and autoimmune thrombocytopaenia.[5] In a prospective study of 101 patients who underwent splenectomy, 40% patients with MPD and 25% patients with haemolytic anaemia developed SPVT. Three of 4 patients (75%) with MPD and splenic weight >3000 g developed PVT.[6] Our patient had the splenic weight of 3270 g. Unfortunately, the weight of the spleen cannot be accurately determined before surgery, but the presence of massive splenomegaly must lead to a presumption of increased risk of PVT. Thrombosis of the splenoportal axis is being increasingly recognised even after laparoscopic splenectomy,[7] being reported as high as 55% in one study.[8] This high probability of SPVT should make us reconsider the use of epidural anaesthesia and analgesia in such cases. Though the median interval between splenectomy and symptomatic SPVT is 8–12 days, it may present as early as 2 days as in our case, when an epidural catheter is likely to be in place.[3,9,10]

This case highlights the fact that despite the well-known benefits of epidural analgesia in upper abdominal surgery, the high risk of PVT requiring anticoagulation or thrombolysis in patients undergoing splenectomy for MPD may well preclude the use of central neuraxial blockade.

CONCLUSION

Anaesthesiologists should be aware that the risk of PVT after splenectomy varies with the indication for splenectomy and is extremely high in patients with MPD, especially with massive splenomegaly. Considering this high likelihood of PVT requiring anticoagulation or thrombolysis, the central neuraxial blockade should be avoided. If post-operative thrombolysis is required in a patient already having an epidural catheter *in situ*, it should be done under close neurological monitoring, weighing the risks and benefits. Fibrinogen levels should be monitored to evaluate the presence of residual thrombolytic effect to determine the appropriate time for catheter removal.

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Conflicts of interest

There are no conflicts of interest.

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