

International Journal of Information Research and Review Vol. 2, Issue, 04, pp. 631-632, April, 2015



# Full Length Research Paper

# ANAESTHETIC MANAGEMENT OF A PATIENT WITH PANCREATIC PSEUDOCYST WITH THROMBOSIS OF MULTIPLE LARGE VESSELS AND INTRAVENTRICULAR SEPTUM OF THE HEART FOR OPEN CYSTOGASTROSTOMY

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Received 19th April 2015; Accepted 30th April 2015

#### Abstract

Pancreatic pseudocyst associated with multiple large vessels in a patient suffering from pancreatitis is rarely described in literature. Earlier case reports have mentioned involvement of inferior vena caval (IVC) thrombosis. However additional involvement of bilateral common iliac vein, left main pulmonary artery and intraventricular septum was seen in our case. The anaesthetic purview of this rare case being an increased risk of intra operative embolism, the choice of anaesthetic technique was focused on minimizing hemodynamic fluctuations and providing adequate post-operative analgesia. Therefore thoracic epidural analgesia with general anaesthesia, combined with invasive monitoring was deemed as a safe option with successful outcome in this case. Pathophysiology of thrombus formation in pancreatic patients, preoperative optimisation, role of intraoperative invasive monitoring and postoperative management has been discussed.

Keywords: Anaesthetic Management, IVC Thrombosis, Pancreatic Pseudocyst.

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To cite this paper: Dr. Deepa, Y. 2015. Anaesthetic management of a patient with Pancreatic Pseudocyst with Thrombosis of multiple large vessels and Intraventricular septum of the heart for open Cystogastrostomy. International Journal of Information Research and Review. Vol. 2, Issue, 04, pp. 631-632.

## INTRODUCTION

Pancreatic pseudocyst may lead to a variety of vascular complications commonly involving thrombosis of splenic vein, portal vein, superior mesenteric vein (Butler et al., 2011). Isolated inferior vena cava (IVC) thrombosis has been previously described in a few studies, (Ma et al., 2002). We describe here a patient of pancreatic pseudocyst with thrombosis of IVC, bilateral common iliac vein, left main pulmonary artery and intraventricular septum of heart. Anaesthetic considerations included preoperative optimisation, intraoperative invasive monitoring (due to increased risk of embolization) and postoperative management. Case report: 32 year old man, chronic alcoholic with previous history of acute pancreatitis, presented with history of abdominal pain for one week. On examination, he was afebrile, hemodynamically stable. Abdominal examination revealed palpable tender mass in the left upper quadrant measuring 8X8cm. On auscultation of the chest there was reduced air entry bilaterally, more on left side compared to right. Pre anaesthetic evaluation revealed hyponatremia, prothrombin time of 16.3 (control 13). His CT scan of the abdomen and pelvis revealed pseudopancreatic cyst measuring 10x4cm in the superior recess of lesser sac compressing suprahepatic Inferior vena cava (IVC) with thrombi noted in IVC and both common iliac veins. Sections obtained through the thorax showed partial occluding thrombus

in the left main pulmonary artery. Moderate ascites and bilateral pleural effusion (left more than right) with subsegmental collapse of the underlying lung parenchyma was also noted. Venous Doppler showed no evidence of deep vein thrombosis. There was generalised ST-T flattening on electrocardiography. In his echocardiography, ejection fraction of 60%, mild Tricuspid regurgitation, pulmonary artery hypertension with pulmonary artery systemic pressure of 40mmHg and a large clot was seen on the intraventricular septum. According to a cardiology consultation, prophylactic treatment with low molecular heparin (LMWH, Enoxaparin 40mg/BD/SC) was begun five days before the operation. Chest radiography showed blunting of cardiophrenic angle and chest ultrasound confirmed bilateral pleural effusion. Patient was premedicated with diazepam (2mg/PO) and ranitidine (150mg/PO) on the operation night. LMWH was omitted 12hours prior to surgery and prothrombin time was monitored. General anaesthesia (GA) along with epidural analgesia (postoperative pain relief) was planned. Epidural catheter passed in thoracic 11-12 interspace under strict asepsis. General anaesthesia was induced with IV fentanyl 2 mcg/kg, midazolam 1mg, IV thiopentone 1.25% till loss of eye lash reflex and tracheal intubation with 8.0mm ID Endotracheal Tube was facilitated with atracurium 0.5mg/kg without gross hemodynamic instability. Anaesthesia maintained with 66% nitrous oxide in oxygen and sevoflurane 1-2%, with

intermittent positive pressure ventilation. Neuromuscular blocking was supported with repeated atracurium bolus doses if necessary. After anaesthesia induction, arterial and central venous catheters were placed for continuous monitoring of systemic blood pressure (IBP) and central venous pressure (CVP) intraoperatively and the lower extremites were wrapped with elastic bandages for the prophylaxis of deep venous thrombosis. Intraoperatively, analgesia maintained with epidural sensorcaine 0.25% infusion of 5cc/hr. Patient was hemodynamically stable and fluids maintained as per Central venous pressure. Neuromuscular blockade was reversed with standard doses of neostigmine and glycopyrrolate followed by extubation. The patient was monitored overnight in the intensive care unit (ICU).

### **DISCUSSION**

Nearly one fourth of patients having pancreatitis may develop vascular complications. Hemorrhage being the most common, (Vujic et al., 1989). Splenic vein thrombosis is more common and may occur in about 10 - 40% of patients (Bernades et al., 1992). IVC thrombosis is rarely reported, (Ma et al., 2002). Pulmonary thromboembolism is also a known complication following venous thrombosis after pancreatitis, (Jorge Cerrudo et al., 2012). Our patient had thrombosis of IVC, bilateral common iliac vein, left main pulmonary artery and intraventricular septum of heart. The patient did not have lower limb edema or abdominal collaterals despite having a large IVC thrombus. The pathogenesis of IVC thrombosis in pancreatitis is not well understood but the factors contributing to the formation of IVC thrombus are likely to be similar as of spleno-portal thrombosis. The possible factors inflammation, edema and cellular infiltration adjacent to IVC; mass effect and compression due to pseudocyst or bulky/calcified pancreas; intimal injury caused by acute pancreatitis or recurrent episodes of pancreatitis. Strict vigilance is required for this condition as it may further lead to pulmonary embolism. Standard treatment for DVT and IVC thrombosis is systemic anticoagulation, however mechanical interruption of the pathway may be offered to patients who have contraindication for anticoagulation by placing IVC filters. In our case anticoagulation was started 5 days prior to surgery. Invasive monitoring in the form of arterial blood pressure, CVP and urine output should be established. Left internal/external jugular cannulation with catheter tip not going beyond the superior vena cava under ultrasound guidance should be preferred (Malhotra et al., 2005).

CVP in these cases may not be accurately reflected because of impaired venous return. This impaired venous return also predisposes the patient to hypotension during induction of anaesthesia. Hence, the patient needs to be adequately hydrated to prevent hypotension from IVC compression during positioning. Venous obstruction leads to dilation of epidural veins and hence caution should be exercised if epidural catheterisation is attempted. Cardiopulmonary bypass and hypothermic circulatory arrest are the ideal modalities for intraoperative management of extensive caval thrombosis, (Staehler et al., 2000). In conclusion, we reiterate that thrombosis of inferior vena cava is a rare complication of pseudocyst of pancreas and may be associated with increased risk of intraoperative pulmonary embolism. A good preoperative preparation, discussion of surgical plan with the surgeon, intraoperative strict vigilance and postoperative pain relief is the key to a successful anaesthetic management.

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