Delayed Haemothorax Resulting from Indwelling Right Internal Jugular Central Venous Catheter: A Rare Complication

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Abstract

Haemothorax is an uncommon and serious complication, occurring most often during or immediately after percutaneous internal jugular and subclavian vein catheterizations. Delayed haemothorax is a rare complication, especially following right-sided catheterization. We report a case of acute yellow phosphorus poisoning with acute liver failure (resulting from rat killer paste ingestion) in a 28-year-old male who developed right-sided haemothorax eight days after placement of right internal jugular central venous catheter. The proposed pathogenesis involves vascular wall erosion by the indwelling catheter tip. Awareness of this complication perhaps avoids unnecessary investigations for other causes of haemothorax such as pulmonary embolism.

Introduction

Haemothorax is an uncommon and serious complication, occurring most often during or immediately after percutaneous internal jugular and subclavian central venous catheter (CVC) placement.¹ Delayed haemothorax, occurring several days after CVC placement, is a rare complication and most often reported following left-sided catheterizations.² We report a rare case of delayed right-sided haemothorax, resulting from indwelling right internal jugular CVC, occurring eight days after CVC placement, in a patient of acute yellow phosphorus poisoning with toxic hepatitis and acute liver failure. Pathogenesis, management and measures to reduce this complication are discussed.

Case Report

A 28-year-old male was admitted to our emergency department following suicidal ingestion of about 10 g of rat killer paste (RATOL), containing 3% yellow phosphorus. He had abdominal pain and vomiting initially. On day-3 post-ingestion, he developed icterus, confusion, irritability and asterixis. Laboratory evaluation revealed deranged liver function- serum bilirubin: 3.9 mg/dl [direct fraction: 3 mg/dl], aspartate transaminase: 240 IU/l, alanine transaminase: 365 IU/l, alkaline phosphatase: 196 IU/l, deranged coagulation tests- prothrombin time: 46 sec, partial thromboplastin time: 42 sec, hemoglobin: 14.6 g/dl, total leucocyte count: 12.6×10⁹/l, platelets: 44×10⁹/l, normal renal function, serum creatine phosphokinase and electrolytes. A diagnosis of acute toxic hepatitis, with acute liver failure was made and he was transferred to intensive care unit. He was started on N-acetyl cysteine, vitamin-K, lactulose, rifaximin and nasogastric tube feeds.

On day-5, he developed hypotension, with decreased urine output. Electrocardiogram and echocardiography were unremarkable and cardiac troponins were negative. For monitoring of central venous pressure and anticipating the requirement for hemofiltration, percutaneous right sided internal jugular double lumen CVC [12 Fr caliber, 16 cm length] was placed under aseptic conditions in single attempt [by blind anterior approach], after prophylactic administration of fresh frozen plasma. Post-procedure chest X-ray [CXR] had ruled out complications (Figure 1a). Hypotension and urine-output improved after fluid administration and vasopressor support. Over the next seven days, there was gradual improvement of encephalopathy, liver function, coagulation parameters and thrombocytopenia.

On day-13, patient complained of breathlessness and discomfort on right side of the chest. He was tachypnoeic and chest radiograph revealed moderate right-sided pleural effusion. Pleurocentesis revealed haemorrhagic fluid with a hematocrit of 28.3 %, confirming haemothorax. Electrocardiogram and

Fig. 1: Chest X-ray taken on day-5 (a) after uneventful right internal jugular venous catheter placement and repeat CXR done on day-13 (b), after placement of intercostal tube to drain right sided haemothorax

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two-dimensional echocardiography were normal. Ultrasound with color Doppler study did not reveal internal jugular or subclavian vein thrombosis on the right side. The CVC was removed immediately. Tube thoracostomy (Figure 1b) drained 1000 ml of hemorrhagic fluid, with prompt relief of breathlessness and chest discomfort. Contrast CT of the chest ruled out pulmonary thromboembolism. Patient received two units of packed red blood cells. Intercostal tube drain was removed on day-16 and he was discharged later.

Discussion

5-19% of patients have been reported to suffer mechanical complications such as pneumothorax, vascular injuries, CVC malposition, cardiac tamponade, hydrothorax and haemothorax, during or after CVC placement.1,2 Haemothorax complicates approximately 1% of CVC insertions,3 usually develops early- either during or immediately after CVC placement and is mostly due to inadvertent arterial injury or innominate vein/ superior vena cava perforation during internal jugular and subclavian vein catheterization.1 Delayed haemothorax, occurring several days after CVC placement, is rare and thought to result from superior vena cava erosion and perforation by the indwelling CVC tip.

Depending on the site of vascular perforation, cardiac tamponade from hydrothorax/ haemothorax may develop when the perforation occurs below the pericardial reflection or hydrothorax/ haemothorax can result- when the perforation occurs above the pericardial reflection.1 Perforation occurring above the pericardial reflection has been reported following 0.4 to 1% of CVC insertions1 and hydrothorax is more common than haemothorax in this situation. Left-sided catheterizations result in more horizontal position of CVC shaft, with perpendicular impingement, steady pressure and friction of the indwelling CVC tip on the vena cava wall and hence carry higher risk of vascular wall erosion and perforation.1,2 Chemical damage to vessel wall from the infused solutions or medications may predispose to erosion and perforation. Although right-sided CVC placement is much safer in this regard due to more parallel position of the catheter shaft to vessel wall, the index patient developed haemothorax from indwelling right internal jugular CVC. Subclavian and internal jugular CVC tips can move by up to 2 cm after insertion in adults,5 with head, neck and respiratory movements. CVC tip migration, coupled with chemical vessel wall damage might have contributed to vascular perforation and haemothorax in the index case. Management includes prompt CVC removal and drainage of the haemothorax.2

Manifestations of acute yellow phosphorus poisoning include hepatitis, acute kidney injury, encephalopathy, coagulopathy, hypotension/shock and other manifestations of multi-organ dysfunction. There was no clinical or radiologic evidence of pleural effusion in the index patient till eight days after CVC placement. Haemothorax developed after improvement of liver function, with normalization of the coagulation parameters and platelet count. Hence it was unrelated to coagulopathy and thrombocytopenia.

Using longer CVCs with a length of 20 cm for left sided catheterizations in adults, as against 16 cm CVCs for right sided catheterizations ensures more horizontal position of the distal CVC shaft to vessel wall, thereby reducing risk of vessel wall perforation.1 The finding of a curled-up tip of a CVC that does not normally have a curvature or abutment of the CVC tip on venous wall in frontal or lateral CXR films predicts impending vascular perforation1 and should prompt immediate repositioning of the CVC. However, the CXR done in present case after CVC placement (Figure 1a) had not shown curling-up of the CVC tip. “Pig-tail” tipped CVCs may reduce the risk of vascular erosion and perforation.1 Radiographic contrast injection to investigate suspected vascular erosion by the catheter tip and to demonstrate contrast extravasation could not be carried out in our patient.

Conclusion

Haemothorax, a complication most often encountered early following internal jugular and subclavian CVC placements, can rarely develop several days later. Pathogenesis involves vascular wall erosion by the indwelling CVC tip. Although reported most often following left-sided catheterizations, delayed haemothorax can rarely follow right-sided CVC placements. Awareness about this complication ensures proper management and avoids unnecessary investigations for other causes of haemothorax such as pulmonary embolism and malignancy of the lung or pleura.

References