

Bedside ultrasound diagnosis of portal vein thrombosis following cholecystectomy: a case report

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ABSTRACT

Portal vein thrombosis is a condition when the thrombus is blocking or narrowing blood flow of the portal vein. The initial approach in diagnosis of portal vein thrombosis for a non-transportable critically ill patient is a colour Doppler ultrasonography. We present a case of an 82-year-old female with partial portal vein thrombosis following urgent cholecystectomy and choledochotomy. The clinical deterioration of the patient with hemodynamic and respiratory instability, acute renal failure, liver damage and metabolic acidosis, prevented the patient's transport for computed tomography diagnostics. A bedside abdominal ultrasonography was performed and revealed a partial obstruction of the left branch of the portal vein, while a confluent part of the portal vein showed a complete absence of flow. Therapy with low molecular weight heparin was immediately started. Definitive confirmation of portal vein thrombosis with the abdominal computed tomography imaging was possible almost 24 hours after clinical and laboratory deterioration. This case illustrates the importance of rapid bedside ultrasonography in diagnosis of thromboembolic events in the abdomen.

Key words: bedside ultrasonography, portal vein thrombosis, liver dysfunction

INTRODUCTION

Portal vein thrombosis (PVT) is a condition when the thrombus in a portal vein causes a partial or total obstruction to the blood flow. It is a relatively uncommon sur-

gical complication, reported in literature mostly after laparoscopic procedures, liver transplantation or splenectomy. (1, 2) Other causes include cirrhosis, neoplasms, infections, and myeloproliferative disorders. (3) Although a diagnosis of PVT or thrombosis of other abdominal vessels is obtained by an abdominal computed tomography (CT) scan, the initial approach should be a Doppler ultrasonography. (4) In detecting PVT, colour Doppler sensitivity and specificity ranges from 89% to 93% and 92% to 99% respectively. CT scan shows similar results with the advantage of more accurate display of the portal vein anatomy. (5)

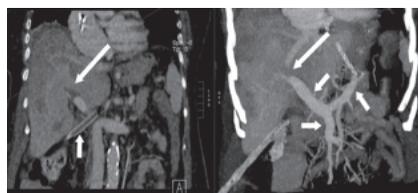


Figure 1. Computed tomography scan in portal venous phase. Long arrow is showing portal vein thrombosis. Short arrows are showing regular flow of splenic vein and superior mesenteric vein up to bifurcation of portal vein and T drainage after cholecystectomy and choledochotomy. Unfortunately, in the medical history of the patient there is only written ultrasound description and not the image itself.

CASE REPORT

An 82-year-old female was admitted to the Intensive Care Unit (ICU) following urgent cholecystectomy and choledochotomy, due to acute cholecystitis and choledocholithiasis. Her past medical

history included stable angina, hypertension, three coronary artery bypass grafts and one stent. Her usual therapy included bisoprolol, isosorbide mononitrate, trimetazidine dihydrochloride, perindopril, acetylsalicylic acid and simvastatin. At the time of admission in the ICU she was hemodynamically stable. Eight hours later she became hypotensive, anuric and hypoglycemic. Laboratory tests revealed metabolic acidosis, elevated liver function tests and a significant increase of lactates and d-dimers. Therapy was supportive and aimed towards the correction of single parameters. Since there was no improvement to volume resuscitation, a continuous administration of noradrenaline was started. On the chest x-ray, slight left pleural effusion was noticed. In the 12 hours interval, laboratory tests showed significant increase of alanine aminotransferase (ALT) 4568 U/L, aspartate aminotransferase (AST) 12070 U/L, alkaline phosphatase 664 U/L, lactate dehydrogenase (LDH) 7390 U/L and bilirubin 140 $\mu\text{mol/L}$. The platelet count was reduced ($52 \times 10^9/\text{L}$), the prothrombin time (0.20%) and activated partial thromboplastin time (49.6s) prolonged and d-dimers increased ($>10 \text{ mg/L}$). Severe metabolic acidosis (pH 7.18, BE -15.4, HCO_3^- 11.7 mmol/L, lactate 10.49 mmol/L) was present as well. We suspected the patient's sudden clinical deterioration (with laboratory signs of liver damage) was caused by hepatic blood flow obstruction. A radiologist was consulted and bedside ultrasound imaging was made immediately. A colour Doppler detected flow in the left branch of the portal vein, while the confluent part of the portal vein showed a complete absence of flow. The presence of some flow downstream from the thrombus was in-

dicative to the partial PVT. At that time, the patient was too unstable for transport and a definitive diagnosis by CT angiography. A therapeutic dose of low molecular weight heparin (LMWH) was immediately started. During the second postoperative day, her laboratory tests revealed aggravated liver damage (ALT 8320 U/L, AST 24100 U/L, LDH 13170 U/L, bilirubin 159 µmol/L). Despite generous volume therapy and diuretic stimulation, she was anuric, and continuous haemodialysis was necessary. At that moment, her hemodynamic status improved and a CT angiography was performed. A partial thrombosis of the portal vein, complete occlusion of the left portal branch and partial thrombosis of intrahepatic branches were found (figure 1). Further diagnostic procedures showed low levels of antithrombin III (AT III) (29%, range 75.0 - 125.0%), protein C (26.4%, range 70.0 - 140.0%) and protein S (45.2%, range 48.0 - 120.0%). In consultation with a haematologist, AT III replacement therapy was started and therapy with LMWH was continued. Soon, the patient's hemodynamic, respiratory and renal functions recovered, as well as coagulation and liver function tests. A control CT showed good conductivity in the main branch of the portal vein and hepatic veins. The patient was discharged to another ICU, where after few days biliary fistula and obstruction of choledochus were detected. Her clinical condition eventually deteriorated again, and she died of multiple organ failure.

DISCUSSION

Bedside ultrasonography in the ICU is an extremely valuable and sometimes underrated tool in abdominal vascular occlusions. (6) Severe hemodynamic instability and respiratory insufficiency in this group of patients, often delay definitive diagnosis and initiation of adequate therapy. In our patient, a definitive confirmation of PVT with the abdominal CT imaging was possible almost 24 hours after clinical and laboratory deterioration. Delay in the LMWH administration in therapeutic doses could increase the thrombotic occlusion of portal vein and other abdominal vessels or initiate pulmonary embolism. Initial differential diagnosis included ischemic hepatitis, acute coronary syndrome, pulmonary embolism, septic shock, aortic dissection and abdominal bleeding. The different therapy approach in these conditions and the dramatic clinical deterioration of our patient emphasise the importance of early bedside ultrasound diagnosis. Protein C, S and AT III deficiencies, that were later detected, may represent inherited disorders associated with PVT. (7) However, in the presence of liver damage, these proteins may be reduced secondary to the liver dysfunction. (8) No apparent cause of PVT is identified in more than 25% of patients. (7) Before there is any evidence of ischemic liver injury, hemodynamic instability may be clinically apparent. (9) Such was the characteristic of our patient as well. A rapid rise in AST, ALT and LDH levels are typical findings in ischemic liver injury. (9) In our case, a severe elevation of liver func-

tion tests was reached within the interval of eight hours, after the acute PVT caused hemodynamic disturbance with hypotension. After hemodynamic stabilization, liver function tests levels subsequently declined within seven to ten days. A treatment option in PVT includes anticoagulation with heparin to prevent extension of the clot, to allow recanalization and to prevent portal hypertension or intestinal infarction. (4, 5) There is an option for surgical thrombectomy, but in comparison to anticoagulation therapy alone, this procedure is not more efficient. (4, 8) Limited data suggest that patients diagnosed with acute PVT are unlikely to spontaneously recanalize their portal vein. (4) However, retrospective studies have found that in patients who were treated with anticoagulants, at least a partial recanalization occurs in 63 - 93% of cases. Complete recanalization occurs in 34 - 45% of cases. (4) In our case, therapeutic doses of LMWH attained the desired outcome, which was confirmed by the control CT scan.

CONCLUSION

This case demonstrates that in a critically ill patient a prompt bedside ultrasound diagnosis of PVT or other abdominal thromboembolic events may initiate appropriate therapy at the right time. It is an excellent diagnostic choice that may improve clinical outcome and possibly shorten ICU and hospital stays.

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