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Successful Staged Treatment for Acute Cholecystitis Complicated by Portal Vein Thrombosis
The American Surgeon

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Successful Staged Treatment for Acute Cholecystitis Complicated by Portal Vein Thrombosis

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Successful Staged Treatment for Acute Cholecystitis Complicated by Portal Vein Thrombosis

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Acute septic portomesenteric venous thrombosis is a rare disease and associated with high mortality and morbidity. This condition can be caused by cholecystitis, cholangitis, pancreatitis, appendicitis, and intra-abdominal abscess.[1,2] The optimal strategy for the treatment of acute cholecystitis complicated by portal vein thrombosis remains to be investigated.[2] Herein, we describe a patient who was diagnosed with acute cholecystitis complicated by acute portal vein thrombosis, who recovered well after "staged" treatment.

A 27-year-old male with no past history of systemic disease except a history of appendectomy and chronic hepatitis B infection suffered with progressive right upper quadrant abdominal pain and poor appetite for 2 weeks without seeking medical treatment. At our ER, the patient's general condition was fair, and vital signs were all within normal limits except for the presence of fever. The patient presented local tenderness over the right upper quadrant area without any peritoneal signs. Abdominal CT showed gallstones with acute cholecystitis combined with partial right portal vein thrombosis (Figure 1). There was no superior mesenteric venous thrombosis, ischemic bowel changes, ascites, or collateral venous return. The laboratory data revealed impaired liver function and hyper-bilirubinemia (ALT: 482 IU/L; total bilirubin: 2.18 mg/dl) as well as increased fibrinogen and D-dimer levels (642.4 mg/dL and 1773.34 ng/ml, respectively); the remaining parameters, including anti-thrombin III, protein C and protein S, were all within normal limits. The patient underwent early computed tomography (CT)-guided drainage of the gall bladder along with a large amount of parenteral hydration, broad-spectrum antibiotics were administered initially and the appropriate antibiotics were initiated after the susceptibility results from the bile and blood cultures were available. In addition, we administered heparin continuously to maintain the aPTT at approximately 60-80 seconds. Ten days later, the second

abdominal CT showed complete resolution of right portal vein thrombosis (Figure 2), and liver function recovered to the normal range. The patient was discharged on oral antibiotics and warfarin without any sequelae. One month later, he underwent an uneventful laparoscopic cholecystectomy and was discharged without any complications. Two months after the first hospitalization, we performed the third abdominal CT, which did not show recurrent portomesenteric venous thrombosis.

Acute portal vein thrombosis is an unusual clinical condition. It results from a combination of local and systemic risk factors. Local risk factors include abdominal infectious or inflammatory disease, such as cholecystitis, cholangitis, intra-abdominal abscess, appendicitis and pancreatitis.[1-4] Systemic risk factors include laparoscopic surgery-associated capnoperitoneum, portal hypertension-associated venous stasis, inherited or acquired hypercoagulable status and hypovolemia-associated increased intravascular hyperviscosity.[3,4] The established treatment for portomesenteric venous thrombosis include control of the underlying infectious disease, hydration, broad-spectrum antibiotics, anticoagulant therapy, and sometimes thrombolytic therapy or surgical embolectomy.[1] However, thrombolytic therapy is controversial for increased risk of hematoma and internal bleeding. In addition, the success of recanalization depends on the time interval between thrombosis formation and the initiation of the anticoagulation treatment.[1] To prevent capnoperitoneum-associated hypercoagulable status and mesenteric venoconstriction in laparoscopic surgery, the patient received percutaneous transhepatic gallbladder drainage (PTGBD) instead of urgent laparoscopic cholecystectomy. To decrease the likelihood of dehydration and increased intravascular hyper-viscosity, we hydrated the patient with a large amount of parenteral crystalloid. Using a “staged” strategy, which consisted of initial adequate drainage of the gall bladder, sufficient parenteral hydration, treatment with antibiotics

and anticoagulants, followed by delayed laparoscopic cholecystectomy, we helped the patient recover from the partial portal vein thrombosis within 10 days and facilitated his undergoing mini-invasive surgery for the treatment of gall stone-associated cholecystitis.

Laparoscopic cholecystectomy has been the gold standard for the treatment of benign gall bladder disease. However, capnoperitoneum in laparoscopic surgeries would induce decreased visceral venous return and mesenteric venous vasoconstriction, which theoretically could cause the propagation of partial portal vein thrombosis.[3] In our experience, sepsis in combination with dehydration and capnoperitoneum in acute surgical abdomen cases would cause the formation of portal vein thrombosis after uneventful laparoscopic surgeries.[4] Open cholecystectomy could be an effective alternative in this patient, but the patient would be subjected to a larger surgical wound and more severe postoperative pain. The use of PTGBD for adequate drainage of infected gall bladder combined with sufficient fluid hydration, early anticoagulants and antibiotics without any thrombolytic treatment allowed our patient to recover well from partial portal vein thrombosis with rapid recanalization of the portal vein, without any sequelae, within a brief period of time. CT-guided gall bladder drainage not only provided adequate drainage of the infected gall bladder but also prevented the capnoperitoneum-associated propagation of portal vein thrombosis, which may occur in laparoscopic surgeries.

In conclusion, the “staged” strategy could be feasible and safe in the management of acute cholecystitis that is complicated by partial portomesenteric venous thrombosis.

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Figure Legends:

Figure 1:

Upper panel shows acute cholecystitis with peri-gall bladder edematous change (arrow); lower panel shows partial right portal vein thrombosis (arrow) _without small bowel or omentum infarction. There was no ascites and no collateral venous return.

Figure 2:

After the 10-day treatment, the second abdominal CT showed complete resolution of the portal vein thrombosis.

Figure 1

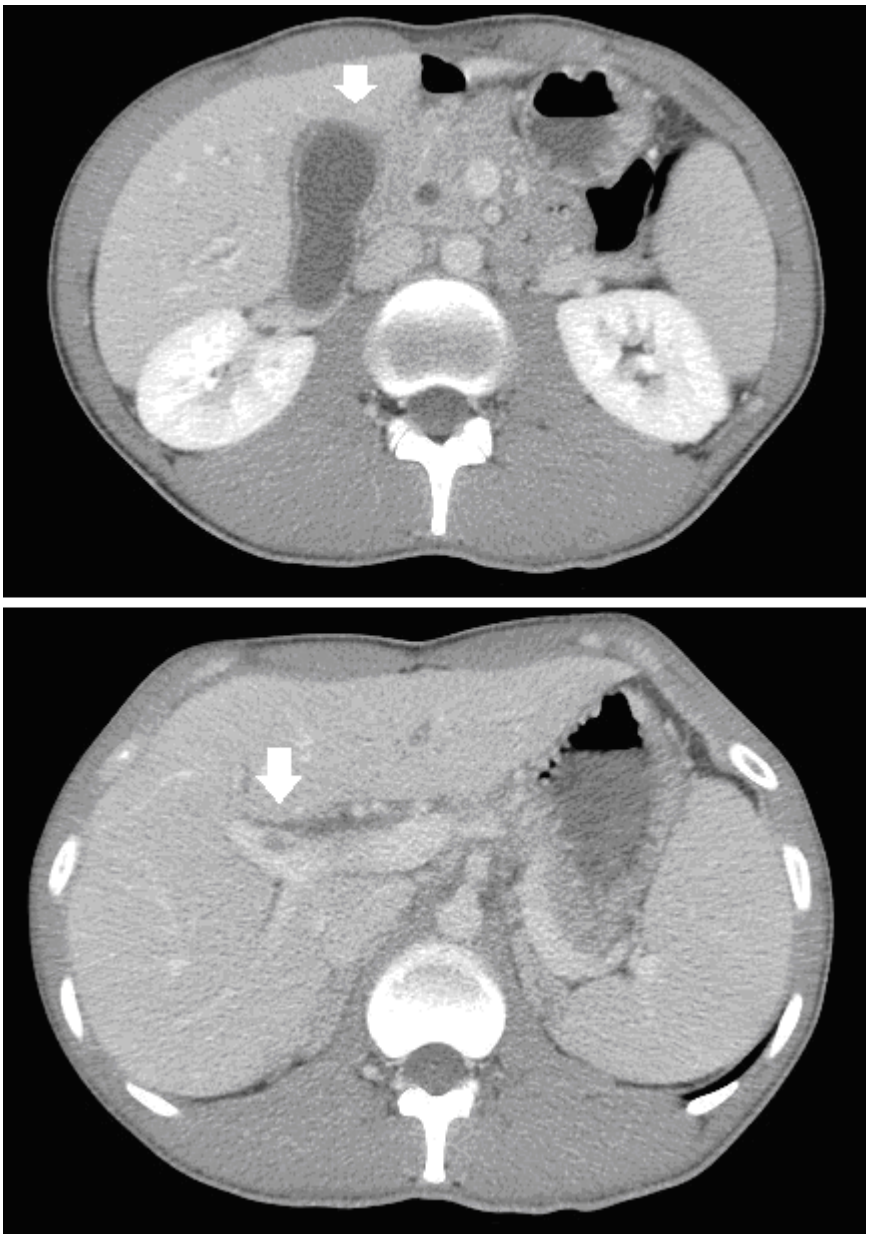

Figure 2