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Case Report

Subcapsular hematoma resulting in hepatic ischemia as a complication of necrotizing pancreatitis

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ABSTRACT

This report presents a case of necrotizing pancreatitis resulting in a large hepatic subcapsular hematoma that led to development of hepatic ischemia and early stages of liver failure. Following surgical decompression, liver function dramatically improved, but large areas of peripheral hepatic infarction had developed. This case demonstrates the risks of a rapidly expanding hepatic subcapsular hematoma, emphasizes the importance of recognizing and aggressively treating active bleeding, and cautions against administering anticoagulation and tissue-plasminogen activator in this clinical scenario.

Introduction

Hepatic subcapsular hematoma is a common entity typically associated with trauma, HELLP syndrome, pancreatitis, and as a complication following endoscopic retrograde cholangiopancreatography or laparoscopic cholecystectomy [1–4]. Life-threatening blood loss is generally considered the greatest concern. Depending on the associated mechanism and severity, management ranges from conservative therapy to surgical decompression and primary hepatic repair. This report presents a case of necrotizing pancreatitis resulting in a large subcapsular hematoma that led to development of hepatic ischemia and early stages of liver failure, a scenario not previously reported in the literature.

Case report

A 54-year-old female with rheumatoid arthritis, uncontrolled diabetes mellitus type II with neuropathy, and hypothyroidism

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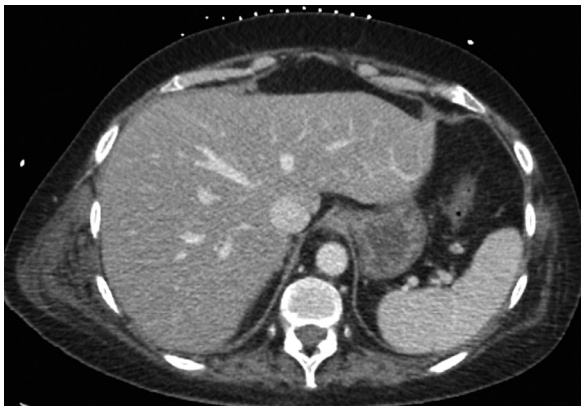

Fig. 1 – Axial portal venous phase CT image obtained at the outside hospital prior to transfer shows normal hepatic parenchymal enhancement and no subcapsular hematoma.

was transferred to the tertiary care referral center from a nearby community hospital where she was admitted for acute idiopathic necrotizing pancreatitis. She had multiple prior episodes of pancreatitis at the outside hospital, including sepsis from an infected pseudocyst requiring pressor support, acute respiratory distress syndrome, acute kidney injury, and deep vein thrombosis with pulmonary embolism requiring anticoagulation. Prior to transfer, a retroperitoneal drainage catheter was placed into the right paracolic gutter due to an enlarging abscess, there was no subcapsular hepatic hematoma, and the liver enhanced normally (Fig. 1).

The patient was transferred to the surgical intensive care unit in critical, but stable, condition. Anticoagulation therapy was continued. The day following admission, the patient had progressive abdominal pain and low drain output. Imaging demonstrated appropriate drain position within a persistent collection. Six mg of tissue-plasminogen activator (t-PA, Alteplase, Diapharma, West Chester, Ohio, United States) in 10 mL of sterile water was injected through the drainage catheter in attempt to improve drainage. The following day, fresh blood was noted in the drainage catheter. The patient subsequently became hypotensive, and laboratories demonstrated decreased hemoglobin from an already anemic baseline of 7.6 gm/dL to 6.2 gm/dL. Her platelet count, coagulation panel, and liver enzymes were within normal limits. Resuscitation was initially successful using crystalloid solution and blood transfusions. Pressors were not initiated. An unenhanced CT of the abdomen and pelvis demonstrated heterogeneously hyperdense subcapsular and perihepatic collections concerning for hemoperitoneum and hematoma. A subsequent contrast-enhanced multiphase CT of the abdomen and pelvis confirmed active hemorrhage in the gallbladder fossa and into the perihepatic hematoma (Fig. 2). Percutaneous endovascular embolization of the cystic artery to stasis was achieved with gelatin foam (Gelfoam, Upjohn, Kalamazoo, MI) and a detachable coil (POD detachable coil, Penumbra, Alameda, CA). During the procedure, the patient received transfusion of 5 units of packed red blood cells, 2 units of fresh frozen plasma and 400 mL of albumin. Blood pressure and hemoglobin stabilized, but she remained in critical condition and bedbound. Anticoagulation was not restarted.

Table 1 – Laboratory results at transfer, peak 6 days after transfer (correlating with Fig. 3), and immediately post-operatively.

	Transfer	Peak	4 Hours post-operative
Alkaline phosphatase (U/L)	166	1051	132
ALT (SGPT) (U/L)	21	2462	124
AST (SGOT) (U/L)	11	>7000	413
Total Bilirubin (mg/dL)	0.20	3.29	2.1
Direct Bilirubin (mg/dL)	<0.2	2.1	1.81

Two days following embolization, her serum transaminases, alkaline phosphatase, and bilirubin markedly increased, and her hemoglobin decreased from 8.4 gm/dL to 6.8 gm/dL, prompting a follow-up contrast-enhanced multiphase CT of the abdomen and pelvis. This demonstrated increased size of the hepatic subcapsular hematoma resulting in marked mass effect on the liver and new ill-defined hypoattenuation of the liver parenchyma, worse in the periphery, which did not change between the arterial and portal venous phases, suggesting hepatic ischemia (Fig. 3). Additionally, there were multiple punctate areas of contrast extravasation consistent with continued active bleeding along the periphery of the liver into the hematoma. The patient returned to the angiography suite, where hepatic angiography demonstrated a few punctate areas of contrast blush on arterial phase indicating bleeding along the liver edge. The cystic artery remained occluded. No embolization was performed as treating the punctate areas of bleeding along the liver edge would have required embolization of a large volume of liver. Anticoagulation remained discontinued. Blood products and crystalloid resuscitation were administered. Laboratory indicators continued to worsen (Table 1).

Due to concern that developing liver failure may be in part due to the mass effect of the hematoma on the liver, the patient was taken to the operating room for surgical evaluation 2 days later. Laparoscopy demonstrated extensive inflammatory adhesions in the right upper quadrant and a large organized hematoma along the right hepatic lobe that could not be efficiently suctioned nor safely morcellated despite utilizing a hand port. Diffuse oozing from the liver parenchyma was also noted. Therefore, a right subcostal incision was made and the large hepatic subcapsular hematoma spanning nearly the entire right hemi-liver and extending to the falciform ligament was evacuated. Numerous packs were placed along the right hepatic edge to ensure hemostasis. Once hemostasis was achieved at the subcapsular hematoma location, the subhepatic hematoma was evacuated. Cholecystectomy and retroperitoneal necrosectomy were performed. A feeding jejunostomy tube was placed. Primary closure was performed at the surgical incision. The patient returned to the intensive care unit for continued resuscitation and close monitoring. Repeat laboratories were drawn 4 hours following the operation that showed marked improvements in alkaline phosphatase, ALT, AST, and bilirubin (Table 1).

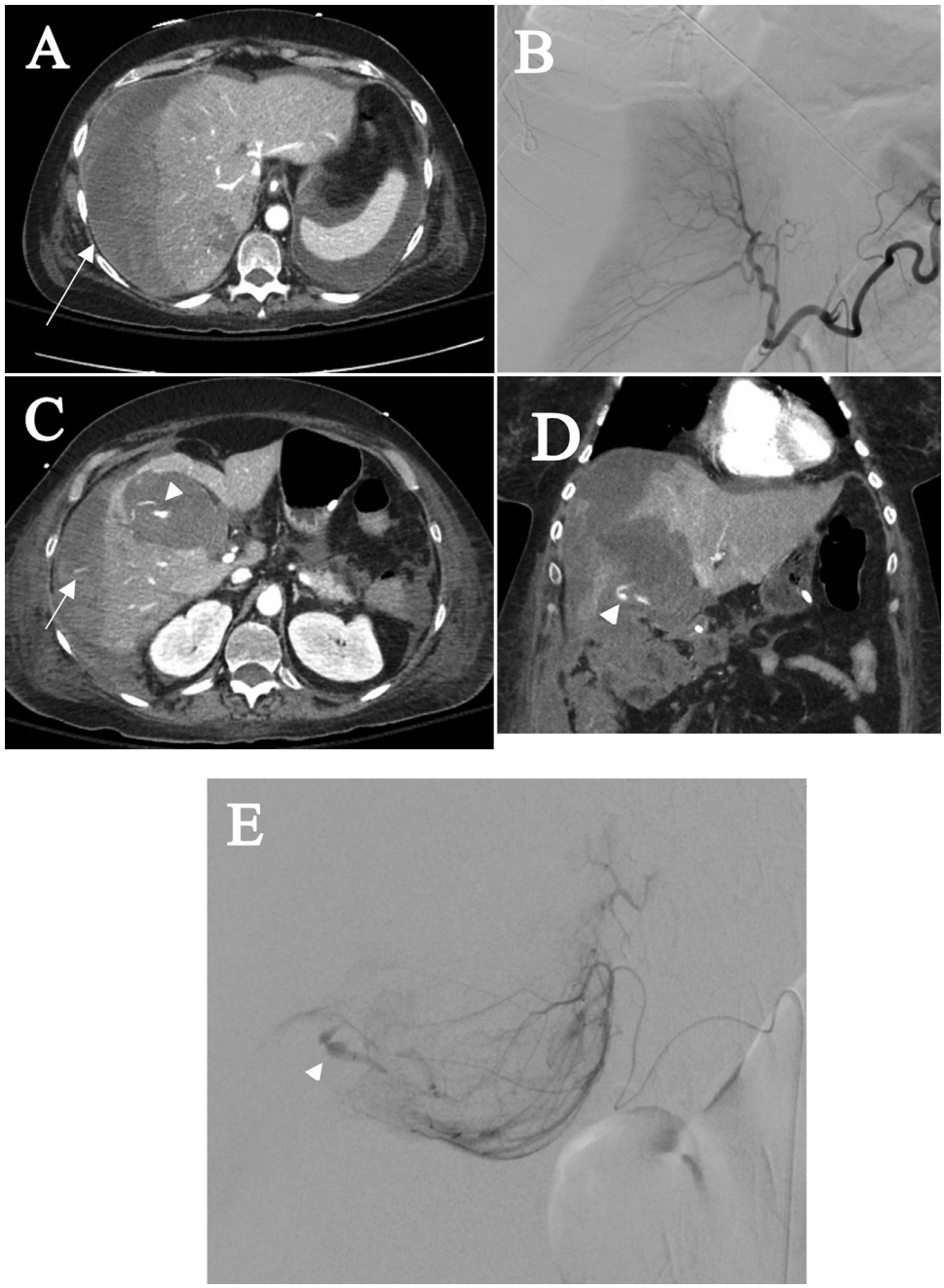

Fig. 2 – (A) Axial arterial phase CT and (B) fluoroscopic angiographic images 2 days after transfer demonstrate a large perihepatic subcapsular hematoma (arrow) resulting in marked mass effect upon the hepatic parenchyma and medial displacement of the right lobe of the liver. (C) Axial and coronal (D) arterial phase CT images demonstrate a focus of active arterial extravasation at the periphery of the liver within the hematoma (arrow, C) and active arterial extravasation (arrowhead) in the gallbladder fossa, which was confirmed at angiography (E).

The patient experienced no further bleeding events and her liver enzymes remained stable despite a protracted admission due to the severity of the pancreatitis. Follow-up imaging 32 days after perihepatic subcapsular hematoma evacuation demonstrated persistent areas of infarction of large portions of the right hepatic lobe (Fig. 4). These infarctions persisted on 5-month follow-up imaging (not shown).

Discussion

Subcapsular hematoma is a common entity typically resulting from trauma and hepatic laceration, though it has been associated with other nontraumatic conditions such as HELLP syndrome and pancreatitis [1,2]. It also can be seen as a complication following endoscopic retrograde

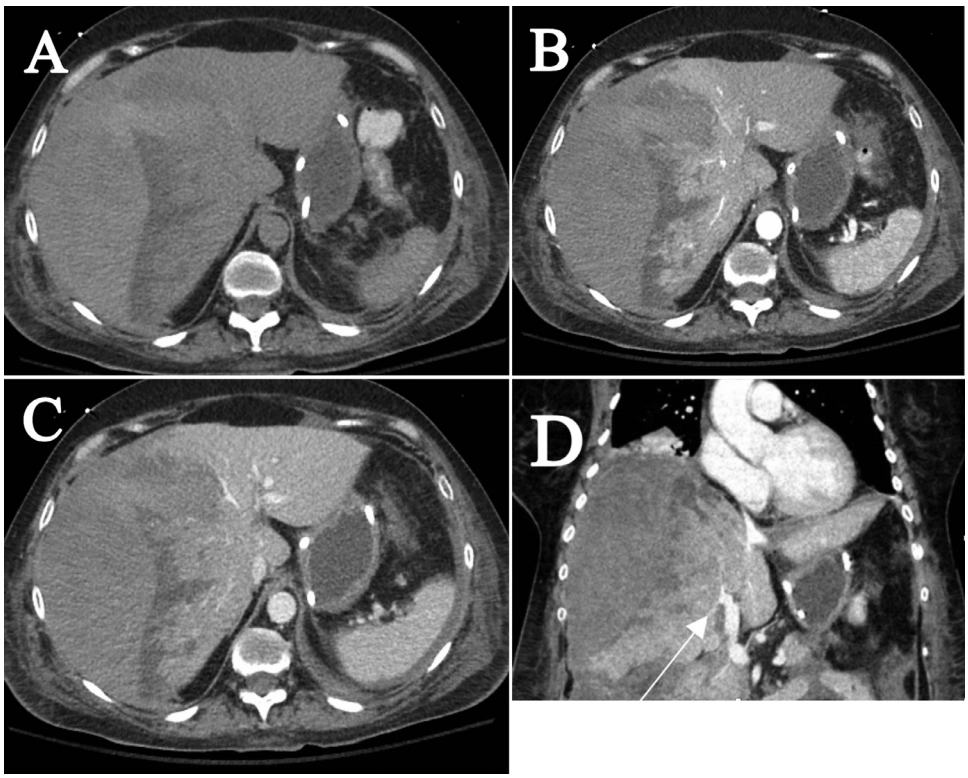

Fig. 3 – Multiphase CT examination performed 4 days after transfer and 2 days after embolization. Precontrast (A), hepatic arterial (B), and portal venous axial images through the liver (C) demonstrate peripheral-predominant, wedge-shaped hypoattenuating right hepatic regions that do not enhance significantly. Coronal portal venous phase CT image (D) demonstrates the marked mass effect of the perihepatic and subcapsular hematoma resulting in medialization of the liver parenchyma and compression of the right portal vein (arrow).

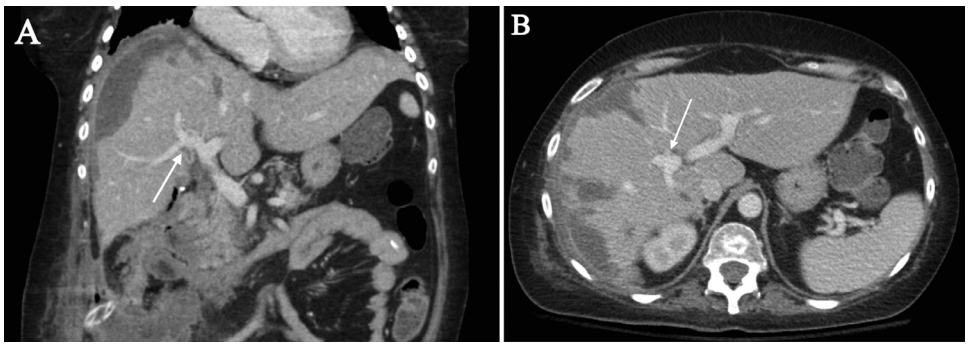

Fig. 4 – Coronal (A) and axial (B) contrast-enhanced CT images obtained 32 days after subcapsular hematoma evacuation demonstrate regions of persistent wedge-shaped nonenhancement in the periphery of the right hepatic lobe, consistent with peripheral hepatic infarcts. Note that the caliber of the right portal vein has been restored to normal (arrows) following hematoma evacuation and resolution of mass effect upon the liver.

cholangiopancreatography and laparoscopic cholecystectomy [3,4]. Pancreatitis-associated subcapsular hematomas are uncommon. The presenting symptoms are nonspecific but typically include right upper quadrant pain, hypotension, and tachycardia. The diagnosis relies heavily on imaging studies.

The size of the hematoma and hemodynamic status of the patient determine the course of treatment. Conservative management starts with serial labs, fluid resuscitation, blood transfusions if indicated, and strict bed rest [4]. If imaging shows active arterial bleeding, selective transcatheter embolization may be performed [1,4]. Surgical control of bleeding through electrocauterization, hemostatic devices, and packing may also be undertaken, particularly when a patient's condition deteriorates rapidly following treatment failure by other interventions [1,4]. Both laparoscopy and laparotomy approaches have been described [4].

In this case, severe pancreatitis caused extravasation from the cystic artery, which was successfully embolized. The underlying pancreatitis may have unleashed destructive pancreatic enzymes that may have weakened Glisson's capsule, irritating the hepatic surface and pericapsular small arteries, resulting in hematoma formation. This may have been exacerbated by anticoagulants and the administration of thrombolytics via the intra-abdominal drainage catheter. The CT and conventional angiographic images showed active bleeding into the subcapsular hematoma, but due to the peripheral location, embolization could not be safely performed. Presumably these persistent contrast blushes explain the continued expansion of the hematoma despite the discontinuation of anticoagulation medications.

As the hematoma grew, the mass effect upon the liver worsened. This led to ischemia and eventually infarction of portions of the peripheral hepatic parenchyma. The mass effect appeared to have resulted in narrowing of the right portal vein. This, in conjunction with possible diminished arterial supply from known pancreatitis-induced vasculopathy, could result in decreased vascular inflow from both hepatic arterial and portal venous supply to the liver.

Alternatively, the mass effect of the subcapsular hematoma may have a mechanism similar to a Page kidney [5,6]. As the mass effect increased, the pressure within the capsule may have increased to the point where arterial inflow was reduced. Also, since pathological correlation was not available, it cannot be completely excluded that the peripheral nonenhancing portions of the liver were the result of direct coagulative necrosis due to pancreatic enzymes. However, if this were the case, we would not expect to see wedge-shaped infarction, rather a more irregular and superficial distribution would be expected. Iatrogenic off-target embolization was considered as a possibility for causing the peripheral ischemia and wedge defects, but the amount of embolic material used and the location of the injections were deemed insufficient to account for the findings.

The trend of the laboratories also supports the hypothesis that mass effect from the hematoma contributed significantly to hepatic ischemia, infarction, and early stages of failure. At presentation, the subhepatic hematoma was relatively small and the transaminases were within normal limits. However, as the subhepatic hematoma increased in size and the mass effect worsened, serum transaminases and bilirubin also increased. Most importantly, within hours of hematoma evacuation, laboratories changed rapidly, trending to near normalization.

In summary, this case shows the importance of aggressively treating active bleeding and the risks of a rapidly expanding hepatic subcapsular hematoma while also cautioning against administering t-PA and anticoagulants to a patient with a hematoma. Although nonoperative management may be preferred and sufficient for many subcapsular hematomas, operative management may be necessary particularly if serial exams, laboratories, and imaging findings suggest hepatic ischemia, persistent peri-hepatic bleeding, and hepatic failure.

REFERENCES

[1] Kim DS, Lee SG, Sung GB, Ko GY, Park KM, Kim KH, et al. Management of subcapsular hematoma of the graft after liver donor liver transplantation. Liver Transpl 2006;12(7):1124–8. PMID: 16799940 https://doi.org/10.1002/lt.20791.

[2] Lin CK, Chen CH, Yeh CH, Lin SL, Tsang YM, Sheu JC. Intrahepatic hemorrhage and subcapsular hematoma developing in acute pancreatitis. Hepatogastroenterology 2003;50(50):571–3 PMID: 12749275.

[3] Liu QF, Bian LL, Sun MQ, Zhang RH, Wang WB, Li YN, et al. A rare intrahepatic subcapsular hematoma (ISH) after laparoscopic cholecystectomy: a case report and literature review. BMC Surg 2019;19(1):3. PMID: 30616574 http://doi.org/10.1186/s12893-018-0453-9 .

[4] Zappa MA, Aiolfi A, Antonini I, Musolino CD, Porta A. Subcapsular hepatic haematoma of the right lobe following endoscopic retrograde cholangiopancreatography: case report and literature review. World J Gastroenterology 2016;22(17):4411–15 PMID: 27158211. doi:10.3748/wjg.v22.i17.4411.

[5] Page IH. The production of persistent arterial hypertension by cellophane perinephritis. JAMA 1939;113(23):2046–8. https://doi:10.1001/jama.1939.02800480032008.

[6] Oliveira GHM, Schirger A. Page Kidney. N Engl J Med 2003;348:129. https://doi.org/10.1056/NEJMicm020037.