



## UNUSUAL SUBCAPSULAR HEPATIC COLLECTION OF PANCREATIC ENZYMES FOLLOWING LAPAROSCOPIC CHOLECYSTECTOMY FOR BILIARY PANCREATITIS: A RARE CASE REPORT

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## UNUSUAL SUBCAPSULAR HEPATIC COLLECTION OF PANCREATIC ENZYMES FOLLOWING LAPAROSCOPIC CHOLECYSTECTOMY FOR BILIARY PANCREATITIS: A RARE CASE REPORT. [ENGLISH]

### SUMMARY

Background: Hepatic subcapsular collections are uncommon after laparoscopic cholecystectomy, and when they occur, they are almost always biliary in origin. Collections rich in pancreatic enzymes without biliary leakage are extremely rare.

Case Presentation: We report the case of a 54-year-old man with diabetes mellitus and hypertension who presented with acute biliary pancreatitis. After clinical stabilization, he underwent an uneventful laparoscopic cholecystectomy. On postoperative day 4, he developed abdominal pain, with recurrent elevation of serum amylase and lipase. Contrast-enhanced CT revealed large right and left hepatic subcapsular collections. Percutaneous drainage showed fluid with markedly elevated pancreatic enzyme levels but normal bilirubin, pointing to a pancreatic origin. MRCP and surgical review did not demonstrate any fistulous tract between the pancreas, biliary tree, or liver. The patient was treated conservatively with percutaneous drainage and supportive care; the collections regressed gradually, and he was discharged in good condition.

Conclusion: This case underscores a rare and unexpected postoperative complication in a patient with biliary pancreatitis undergoing cholecystectomy. Clinicians should consider pancreatic enzyme leakage as a possible etiology of hepatic subcapsular collections, even without demonstrable fistula. Early recognition, fluid analysis, and drainage may suffice without requiring surgical intervention.

### KEYWORDS

Biliary pancreatitis, Hepatic subcapsular collection, pancreatic enzyme leak, laparoscopic cholecystectomy, percutaneous drainage

### INTRODUCTION

Laparoscopic cholecystectomy is also established as the optimal technique for the management of symptomatic gallstone disease and its complications, such as biliary pancreatitis, owing to its association with reduced morbidity, brief durations of admission, and fast recovery versus open cholecystectomy [1, 2]. Despite the advantages and precautions, the procedure is not risk-free,

Postoperative complications that arise include injury of the bile duct, leakage of the bile, bleeding, abscesses of the intra-abdominal type, and infections of the operated site [1, 2].

Hepatic subcapsular fluid collections are referred to as a rare postoperative occurrence. When these collections come to consideration for identification, they are most frequently related to a biliary source, most notably occurring as bilomas secondary to leakage related to the cystic duct stump, auxiliary ducts, or iatrogenic trauma [3, 4]. Conversely, collections located beneath the hepatic capsule that are abundant in pancreatic enzymes, when bile leakage is not present, are exceedingly uncommon and have been minimally documented in existing scholarly works. The detection of pancreatic enzymes within the hepatic area is atypical from a pathophysiological perspective, since pancreatic secretions generally stay restricted to the retroperitoneal space or the peritoneal cavity, except in instances of compromised ductal integrity or direct release of enzymes. Pancreatic fluid collections are well-characterized complications of acute and chronic pancreatitis as well as of post-pancreatic operations.

Some of the complications include acute peripancreatic fluid collections, pseudocysts, pancreatic fistulas, and internal development of fistulas communicating with adjacent anatomical sites like the pleural cavity, mediastinum, peritoneum, or gastrointestinal tract [5, 6]. The underlying mechanism usually involves damage to the pancreatic duct, leading to leakage of secretions containing enzymes that follow tissue pathways. Formation of intrahepatic or subcapsular hepatic pancreatic pseudocysts is an extremely rare presentation of the process. Such collections have only been documented by isolated case reports and are believed to occur secondary to extravasation of enzymes dissecting into the hepatic parenchyma or capsule [7–10]. These rare presentations are of differential diagnostic challenge because they could present like more typical postoperative collections of biliary or infective origins.

This is the report of a case of a patient who developed a pancreatic enzyme-enriched hepatic subcapsular collection following laparoscopic cholecystectomy for biliary pancreatitis. This case report highlights the diagnostic challenge, treatment strategies, and clinical implications of this extremely rare complication.

### CASE PRESENTATION

A 54-year-old male with a history of type 2 diabetes mellitus and hypertension complained of acute epigastric pain that radiated towards the back. The initial laboratory evaluation confirmed greatly increased levels of serum amylase and lipase, but normal liver function tests and bilirubin levels that suggested biliary obstruction. Abdominal ultrasonography confirmed the existence of gallstones with no evidence of intrahepatic or extrahepatic biliary dilation. The patient was hospitalized and managed conservatively by bowel rest, intravenous fluid replacement, and analgesic treatment. The levels of pancreatic enzymes normalized over the next five days. Post two weeks of admission the patient underwent laparoscopic cholecystectomy. The procedure was uneventful with no perioperative bile leak (Figure 1). A surgical drain was placed and removed on postoperative day (POD) 2. But on POD 4, the recurrent epigastric and right upper quadrant pain reappeared. Laboratory tests done again revealed new elevations of serum amylase and lipase, but liver enzymes and bilirubin stayed normal. The chest X-ray on POD 4 demonstrates elevation of the right hemidiaphragm, raising suspicion for underlying hepatic or subphrenic pathology. This finding contrasts with the baseline, as the chest X-ray obtained two weeks prior to laparoscopic cholecystectomy was normal (Figure 1A&B). Contrast-enhanced CT scan revealed a large subcapsular hepatic fluid collection extending across both lobes, conforming to the hepatic contour and causing scalloping of the liver margins (Figure 3A&B). The collection measured roughly 9.6 × 3.2 × 9.3 cm in the left lobe with ring enhancement on post-contrast imaging (Figure 2B), leading to a suspicion of abscess formation. Other features included a tiny focus of intrahepatic pneumobilia, presumably postoperative; an enlarged pancreas with scarce peripancreatic fluid and fat stranding, characteristic of acute interstitial edematous pancreatitis without evidence of necrosis or organized collection; moderate ascites with predominance; right pleural effusion associated with lower lobe collapse-consolidation; and mild subdiaphragmatic and subhepatic free fluid. Normal spleen, kidneys, adrenals, and bowel loops. Percutaneous catheter drainage of the hepatic subcapsular collection was done, which produced clear fluid. Biochemical analysis of the fluid showed extremely high amylase and lipase (>1000 U/L) but very minimal bilirubin, indicative of a biliary rather than a pancreatic source. The glucose of the fluid was high (7.5 mmol/L), and the LDH was extremely high (7487 U/L). Cytology, as well as microbiological cultures after 48 hours of aerobic and anaerobic incubation, were negative.

Magnetic Resonance Cholangiopancreatography (MRCP) established the absence of biliary leak, ductal dilatation, or pancreas-biliary or gastrointestinal fistulous communication.

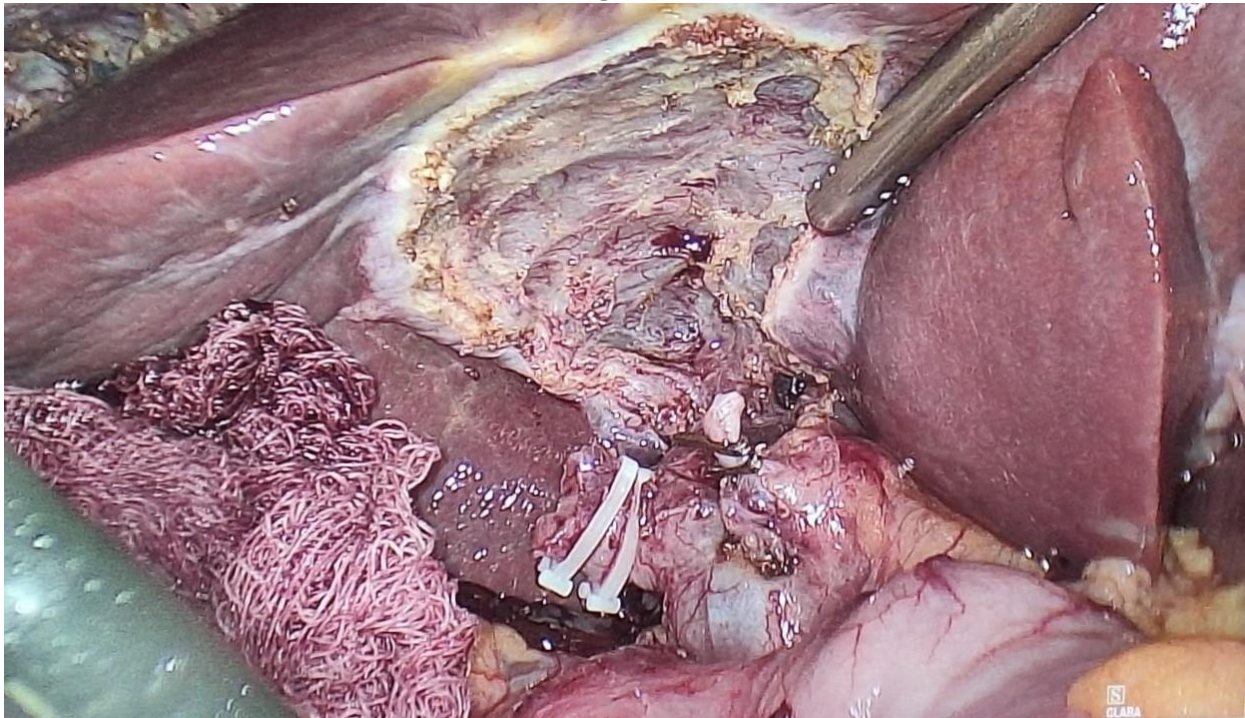
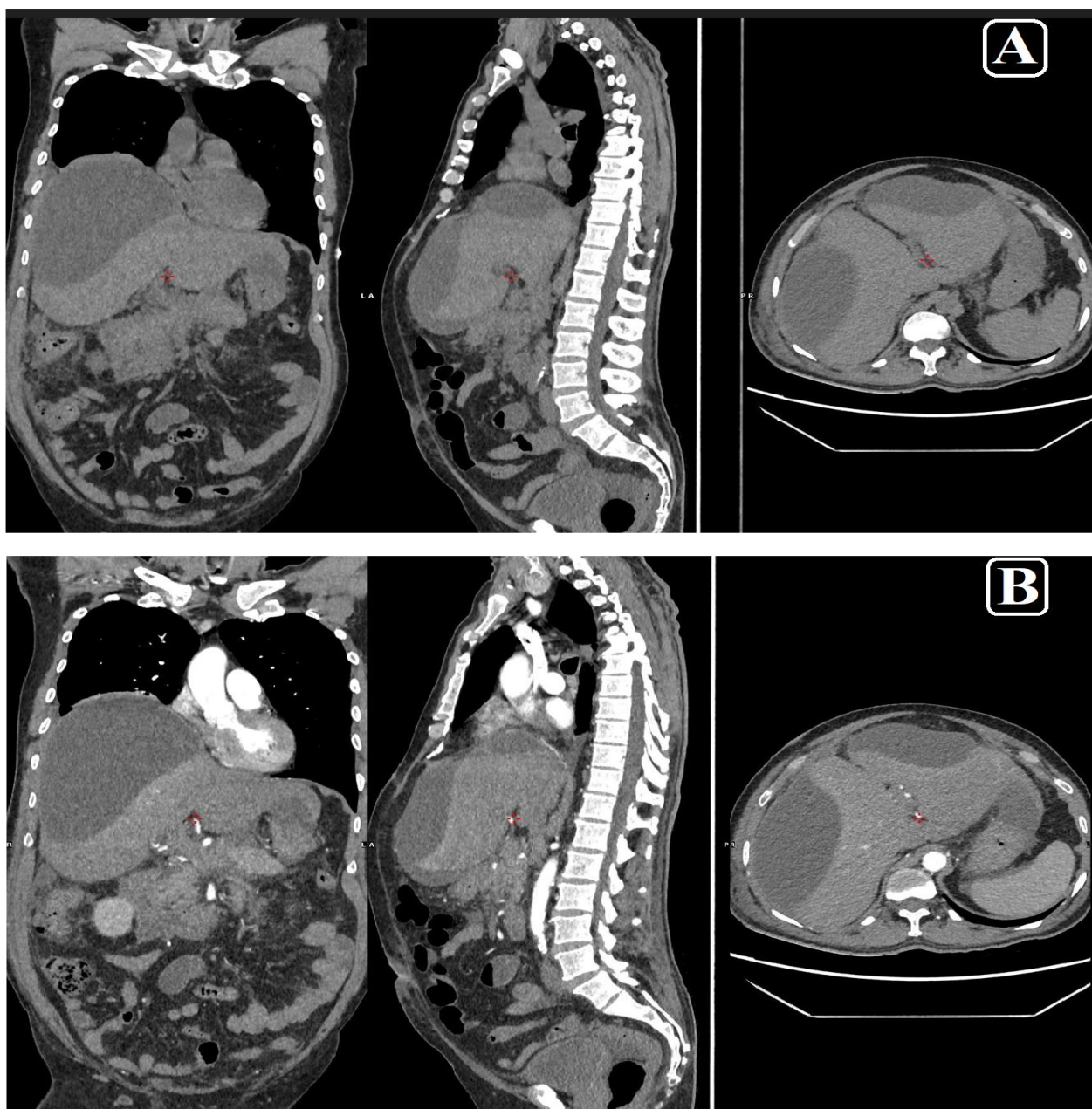


Figure 1. Intraoperative photograph during laparoscopic cholecystectomy.



Figure 2: Serial Chest X-Rays: (A) Two Weeks Pre-Laparoscopic Cholecystectomy, (B) Four Days Post-Laparoscopic Cholecystectomy, and (C) Three Weeks Post-Laparoscopic Cholecystectomy Prior to Hospital Discharge.

Serial chest X-Rays imaging also established progressive subcapsular collection regression with resolving drain output. The noted earlier right pleural effusion and ascites improved over the interval (Figure 2). The patient was treated conservatively with a catheter drain, supportive care, and observation. Fluid output substantially dwindled over two weeks. The drain was removed, and the patient remained clinically well, allowing discharge three weeks following laparoscopic cholecystectomy.



*Figure 3. Contrast-enhanced CT of the abdomen. (A) Pre-contrast images showing a hypodense lesion in the liver. (B) Post-contrast images demonstrating the enhancement pattern of the lesion.*

## DISCUSSION

Hepatic subcapsular collections after abdominal operations are rarely seen and, when noted, are almost entirely of biliary genesis (bilomas) [3, 4,11]. Bilomas occur secondary to leakage of bile into subcapsular or intrahepatic spaces, most often after cholecystectomy or hepatic resection [11, 12]. In comparison, collections of pancreatic juice are usually confined to peri-pancreatic areas or spread to adjacent retroperitoneal/intraperitoneal sites [5, 6,13]. Only a few instances have been reported delineating hepatic subcapsular pancreatic pseudocysts [7–10]. Topno et al. reported a case that underwent successful treatment with percutaneous drainage [7], while Sakhy et al. discuss them as a rare complication secondary to pancreatitis [10]. Minhas et al. documented an intrahepatic pancreatic pseudocyst (IHPP) after acute pancreatitis by exploring mechanisms of enzyme tracking and hepatic erosion [8]. Sowka et al.

reported the simultaneous occurrence of a pancreatic pseudocyst with a hepatic pseudocyst with probable fluid tracking by ligamentous tracts [14]. Other investigators have referred to the development of infected intrahepatic pseudocysts complicating chronic pancreas disease [18]. Because of their rarity, there is no standardized protocol presently available for their diagnosis or treatment.

The pathogenesis of our case is speculative. Possible mechanisms are retroperitoneal tracking of secretions of the pancreas along perihepatic fascial planes, microscopic leaks/ductal fistulae that are below the resolution of imaging evaluations, early extravasation of enzymes after an acute pancreatitis with secondary accumulation, or at operation disruption of perihepatic adhesions with diffusion of enzymes. Similar mechanisms exist for leaks of the pancreatic duct and internal fistulae of the pancreas, such that enzyme-rich fluid tracks by pathways of least resistance to peritoneum, pleura, or mediastinum [5, 13, 17].

Differential characterization of pancreatic from biliary collections is paramount. Fluid analysis is mandatory: high amylase and lipase with low bilirubin confirm a pancreatic source, while high bilirubin indicates bile leakage [13, 18]. Drainage fluid amylase is best known for diagnosing postoperative pancreatic leaks [5, 13, 15, 16], although systematic reviews raise skepticism about cutoff values and predictive validity in certain situations [13].

Diagnosis needs to be systematic. Contrast-enhanced CT or MRI/MRCP serves to clarify collections, exclude biliary leakage, and identify tracts of fistula. Secretin-enhanced MRCP enhances sensitivity for subtle disruptions of the ducts [17]. ERCP retains a place if there is doubt by confirming duct integrity and allowing stenting therapeutically [5, 17]. Regular follow-up is essential for detecting infection, hemorrhage, or rupture. Though our case was not after pancreas surgery, such consensus paradigms like the ISGPF leak/fistula grading system offer an excellent conceptual roadmap [5, 7, 13].

The therapeutic approach is governed by accessibility, clinical stability, and frequency of ductal disruption. Percutaneous drainage is the typical initial treatment and has been successful in multiple case reports [7–10, 14, 18]. Supportive conservative therapy (nil per os, analgesia, and fluid resuscitation) is essential. Persistent leaks may require endoscopic therapy, most commonly ERCP with stenting of the pancreatic duct [5, 17]. Refractory disease or serious complications are managed by surgery. In our case, percutaneous drainage with supportive care alone was sufficient, and no additional intervention was required.

This case offers multiple clinical lessons. Subcapsular collections after cholecystectomy should never be assumed biliary; pancreatic enzymatic leakage needs consideration, particularly after pancreatitis. Fluid analysis is the point of diagnostic pivoting. Conservative management with potential for drainage often suffices in the majority of cases, obviating invasive surgery. Follow-up over the long term is essential for the identification of recurrence or complications. Ultimately, such uncommon presentations need to be reported for broadening the evidence base informing future approaches for diagnostic as well as therapeutic strategies.

## CONCLUSION

Pancreatic enzyme-rich hepatic subcapsular collections following laparoscopic cholecystectomy are exceedingly rare but possible, particularly in the setting of biliary pancreatitis. Recognizing this atypical complication, performing fluid analysis, and managing with percutaneous drainage and supportive care can lead to favorable outcomes without extensive surgical intervention. Further case reports and possible aggregated reviews may help refine optimal diagnosis and management strategies.

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**Conflict of interest:**

The authors have no conflict of interest to declare.

**Patient Consent:**

Patient's written consent for the publication of this case and images was taken.

**Availability of supporting data:**

Supporting data are available from the corresponding author upon reasonable request.

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