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UNEXPECTED PULMONARY EMBOLISM FOLLOWING SEQUENTIAL SURGERIES FOR CHOLECYSTOCOLONIC FISTULA AND TRANSVERSE COLON PERFORATION: A CASE REPORT

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Background: Pulmonary embolism is a fatal postoperative complication, particularly in patients undergoing major abdominal surgery complicated by intra-abdominal infection and immobilization. Although rare, cholecystocolonic fistulas with associated colonic perforation and sepsis markedly elevate the risk of venous thromboembolism and pulmonary embolization. **Case Presentation:** A 56-year-old male presented with a three-week history of right upper quadrant abdominal pain, nausea, and vomiting. Magnetic Resonance Cholangiopancreatography (MRCP) demonstrated gallbladder wall thickening, emphysematous changes, and cholelithiasis. The patient underwent two sequential abdominal surgeries: laparoscopic adhesiolysis with partial cholecystectomy and primary colonic repair, followed by an exploratory laparotomy with right hemicolectomy and ileostomy for colonic perforation. On postoperative day 11 after the second surgery, he developed acute respiratory distress, hypoxia, and hemodynamic instability. PE was suspected and subsequently confirmed by characteristic electrocardiographic changes and a markedly elevated D-dimer level (19.83 µg/mL). The patient responded well to anticoagulation and supportive care and was discharged on hospital day 29 without complications. **Discussion:** PE remains a significant cause of postoperative morbidity and mortality, particularly following major abdominal surgeries complicated by sepsis, inflammation, and immobility. This case highlights the multifactorial etiology of thromboembolism, including multiple surgical interventions for cholecystocolonic fistula and colonic perforation, ongoing septic burden, and surgical stress. Systemic inflammation and infection create a hypercoagulable state, while delayed mobilization further increases venous thromboembolism risk. **Conclusion:** Early recognition and prompt management of pulmonary embolism are essential in high-risk postoperative patients. A multidisciplinary care approach is critical to improve clinical outcomes and ensure a favorable prognosis.

Keywords: Pulmonary embolism; Cholecystocolonic fistula; Transverse colon perforation

INTRODUCTION

Cholecystocolonic fistula is a rare complication, occurring in about 1 in 10,000 cases, and accounts for 6.3%–26.5% of all cholecystoenteric fistulas (1,2). It usually arises from chronic gallbladder disease, particularly in patients with longstanding cholelithiasis and inflammation (3,4). Preoperative diagnosis is often difficult, and missing the diagnosis can complicate surgery (1). If undetected, Cholecystocolonic fistula may cause colonic perforation, leading to fecal peritonitis and intra-abdominal sepsis. Such secondary peritonitis carries high morbidity and mortality, especially in emergency surgeries for colonic perforation (1). The surgical complexity and inflammation also increase the risk of postoperative complications, including thromboembolism. Intra-abdominal infections may trigger systemic inflammatory responses, further raising the thromboembolic risk (5).

Venous thromboembolism (VTE), which include deep vein thrombosis (DVT) and pulmonary embolism (PE), is a serious condition with high morbidity and mortality (6). PE remains a major threat in hospitalized and postoperative patients, particularly after major abdominal surgeries (7). Risk factors such as prolonged surgery, older age, systemic inflammation, intra-abdominal sepsis, coagulopathy, and extended immobility contribute to a hypercoagulable state, increasing VTE risk (8,9). Herein, we report a rare case of acute PE following two abdominal surgeries for a cholecystocolonic fistula and transverse colon perforation. This case emphasizes how intra-abdominal infection and surgical stress can elevate thrombotic risk, highlighting the need for proactive VTE prevention and early diagnosis in high-risk postoperative patients.

CASE REPORT

A 56-year-old male, with no history of cholelithiasis or other medical condition, presented with a three-week history of recurrent right upper quadrant abdominal pain, multiple episodes of nausea and intermittent non-bilious vomiting, with no jaundice appearance and hemodynamically stable. Physical examination revealed localized tenderness in the right upper quadrant without peritonitis.

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Laboratory results showed within normal limits. Initial imaging from the referred hospital, including Magnetic Resonance Cholangiopancreatography (MRCP), demonstrated gallbladder wall thickening, intramural gas consistent with emphysematous changes, and multiple cholelithiases.

A laparoscopic cholecystectomy was planned for a preoperative diagnosis of acute cholecystitis and cholelithiasis. However, intraoperative findings revealed dense adhesions on the fundus of the gallbladder and approximately 200 cc of pus within the abdominal cavity. Adhesiolysis was performed to release the adhesions. During exploration, a cholecystocolonic fistula between the gallbladder and transverse colon was identified (**Figure 1.**), along with a perforation of the transverse colon. The colonic perforation was repaired using a continuous 3-0 V-Loc suture. Subsequently, a fundus-down or retrograde cholecystectomy was performed, during which necrotic tissue was evacuated. Due to extensive inflammation, a partial cholecystectomy was required. The proximal gallbladder stump was closed with a 3-0 V-Loc suture, and a surgical drain was placed. Intraoperative blood loss was estimated at 600 cc, requiring transfusion of one unit of packed red blood cells and fluid resuscitation. The patient was transferred to the ICU for postoperative monitoring, where he remained hemodynamically stable. Broad-spectrum parenteral antibiotics were initiated, targeting both aerobic and anaerobic pathogens.

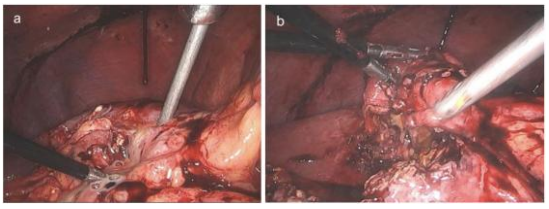

Figure 1. Intraoperative findings: (1a) accumulation of pus within the abdominal cavity. (1b) cholecystocolonic fistula identified.

On postoperative day four, the patient subsequently developed new-onset abdominal distension, left lower quadrant tenderness, inability to pass flatus, raising suspicion for paralytic ileus or localized peritonitis. Abdominal X-ray in three positions (**Figure 2.**) showed elongated, dilated bowel loops with gas extending to the rectum. Psoas shadows and renal outlines were obscured by gas, consistent with paralytic ileus. Ultrasound confirmed ileal wall thickening and bowel distension, with no pneumoperitoneum and minimal ascites. Inflammatory markers were markedly elevated, with a quantitative CRP of 285.11 mg/L and procalcitonin of 2.91 ng/ml leading to septic condition.

Based on these findings, a second exploratory laparotomy was performed. Intraoperative findings revealed feculent contamination of the abdominal cavity, a recurrent perforation of the transverse colon, and bile leakage. A right hemicolectomy was carried out, and fenestration of the gallbladder was performed to control the bile source. A double-barrel ileostomy was created, and additional drainage was established.

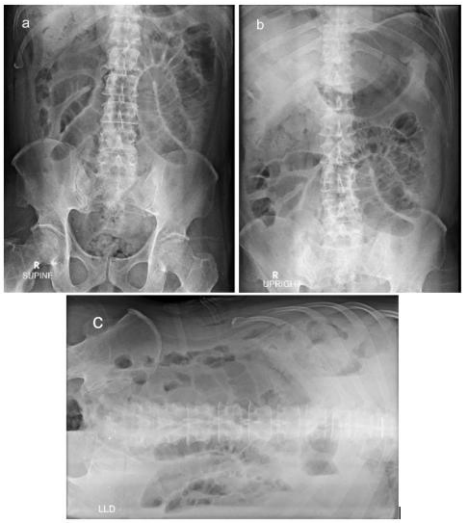

Figure 2. Abdominal X-ray in three position (2a, 2b, 2c) confirmed paralytic ileus

Postoperatively, the patient was stabilized in the ICU. Stoma output remained reddish and bilious, and drainage was closely monitored. Postoperative laboratory results showed leukocytosis (13,000/µL), elevated CRP (129 mg/L), procalcitonin (2.26ng/mL), D-dimer (5.2 µg/mL), and hypoalbuminemia (2.21 g/dL), consistent with sepsis and a hypercoagulable state. Chest X-ray showed increased bronchovascular marking, suggestive of pneumonia. Empiric antibiotics—tigecycline and metronidazole—were initiated while waiting blood culture results. Anticoagulation for VTE prophylaxis was initiated with intravenous heparin after correcting albumin to above 3.0 g/dL and ensuring bleeding risk was controlled. Compression stockings were also applied for DVT prophylaxis. Physiotherapy and gradual mobilization were initiated as the patient's condition progressively improved.

Eleven days following the second surgery, the patient developed sudden-onset dyspnea, chest pain, and oxygen desaturation to SpO_2 80% despite high-flow oxygen via non-rebreathing mask. A clinical suspicion of pulmonary embolism (PE) was raised, supported by an S1Q3T3 pattern on ECG (**Figure 3.**) and a markedly elevated D-dimer level (19.83 mg/L). CT pulmonary angiography, the definitive imaging for PE, could not be performed due to the patient's hemodynamic instability.

The patient was intubated and managed in the ICU with mechanical ventilation, vasopressor support (norepinephrine and vasopressin), and intravenous heparin anticoagulation. ECG evaluation later showed resolution of PE features. Echocardiography showed a hyperkinetic right ventricle with no active PE, suggestive of a previously resolved embolic event. Femoral vein Doppler ultrasound demonstrated

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venous stasis, indicating a thrombosis risk factor. Based on these findings, continuation of anticoagulation therapy for 3–6 months was recommended.

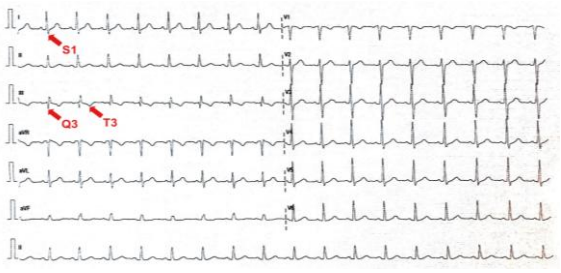

Figure 3. Electrocardiography on the day of acute PE onset: ECG showed an S1Q3T3 pattern.

After three days of intensive support, the patient was successfully extubated. Drain output remained minimal. In this case, anticoagulation was promptly initiated using intravenous unfractionated heparin (UFH). Following hemostatic stabilization, low-dose intravenous UFH was resumed despite minimal ongoing bleeding, in consideration of the high risk of thromboembolic complications. The nasogastric tube showed no output, allowing for the gradual reintroduction of oral intake, starting with clear liquids and progressing to a soft diet. Urine output remained adequate. The patient showed functional improvement and maintained stable respiratory function on room air. The postoperative wound appeared clean, and sutures were partially removed in a staggered manner and the stoma was functional. Serial D-dimer and aPTT levels showed a sharp rise preceding PE onset, followed by a marked decline with UFH therapy. Anticoagulation was later transitioned to a direct oral anticoagulant with rivaroxaban (**Figure 4.**). The patient was discharged on hospital day 29 without complications. The patient's recovery was supported by a multidisciplinary team, including surgical, internal medicine, cardiology, pulmonology, anesthesiology, and medical rehabilitation specialists.

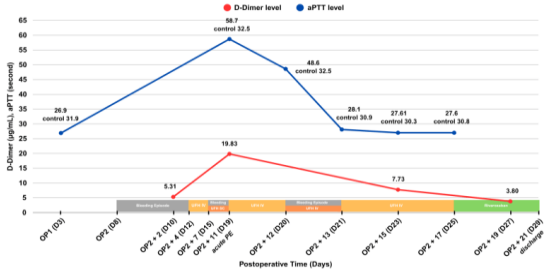

Figure 4. Summary of postoperative progress: rapid increase in D-dimer levels from the immediate postoperative period up to the day of pulmonary embolism (PE) onset, followed by a marked decrease in D-dimer with heparin therapy, monitored using activated partial thromboplastin time (aPTT) as an indicator. The anticoagulation treatment was transitioned to a direct oral anticoagulant (DOAC) 17 days after the second operation and continued until discharge. *OP1: first operation; OP2: second operation; D: day of hospitalization; UFH: unfractionated heparin.*

DISCUSSIONS

The patient initially presented with clinical features consistent with uncomplicated acute cholecystitis and cholelithiasis. However, intraoperative findings revealed an emphysematous gallbladder with a cholecystocolonic fistula, an exceedingly rare complication typically associated with chronic inflammation and gallstone disease, with an incidence of 0.13% [10]. Interestingly, this patient had no prior history of cholelithiasis or other predisposing conditions, making the presentation atypical. Cholecystocolonic fistula is often presented with non-specific symptoms that closely mimic common biliary pathologies, such as acute cholecystitis, making preoperative diagnosis challenging [11,12]. In fact, only 7.9% of cases are identified preoperatively, while the majority are discovered intraoperatively in 0.06–0.14% of patients undergoing cholecystectomy [12,13].The underlying mechanism likely begins with compression of the gallbladder wall by large gallstones, combined with recurrent episodes of cholecystitis, leading to chronic inflammation and dense adhesions. Over time, this process may lead to erosion into adjacent visceral structures, resulting in fistula formation [12]. The presence of colonic perforation intraoperatively in this patient necessitated primary repair and partial cholecystectomy due to extensive inflammation.

Despite initial surgical management, the patient progressed to a septic state, with clinical and radiological signs of paralytic ileus and elevated inflammatory markers. Re-exploration revealed bile leakage and a recurrent transverse colon perforation, prompting a right hemicolectomy and creation of a double-barrel ileostomy. The presence of fecal peritonitis confirmed the severity of intra-abdominal contamination and systemic inflammatory burden. These conditions are known to significantly increase the risk of PE due to a combination of endothelial injury, stasis, and a hypercoagulable state, collectively described as Virchow's triad. Virchow's triad comprises three principal factors that contribute to thrombus formation: endothelial (vessel wall) injury, alterations in normal blood flow such as stasis or turbulence, and a hypercoagulable state that predisposes the blood to clot more readily (14). Virchow's triad elucidates the fundamental physiological processes contributing to thrombus formation. The presence of intravascular clots significantly elevates the risk of thromboembolic events, including cerebrovascular accidents, pulmonary embolism (PE), and organ infarctions, which may result in ischemia and subsequent cellular death (15).

The annual incidence of acute PE is estimated to range between 39 and 115 cases per 100,000

individuals, while deep vein thrombosis (DVT) occurs slightly more frequently, with an incidence between 53 and 162 cases per 100,000 population (16). Epidemiological data indicate that acute PE is more commonly observed in males than in females (17). The subsequent development of PE in this patient is multifactorial. Key contributing factors include prolonged immobilization following two major abdominal operations. Sepsis-induced hypercoagulability, as evidenced by markedly elevated D-dimer level and procalcitonin. Surgical stress, with associated endothelial injury and systemic inflammation promoting endothelial activation and thrombin generation. Possible femoral vein thrombosis, supported by Doppler findings of venous stasis. PE is associated with significant mortality, however its true impact is difficult to determine, as many cases of sudden cardiac death are retrospectively suspected to be caused by undiagnosed thromboembolic events, including PE (18). PE results from a thrombus entering the pulmonary circulation. Large emboli can obstruct the main pulmonary artery, producing a saddle embolus that severely compromises cardiovascular function. Smaller emboli may lodge in peripheral branches, leading to pulmonary infarction characterized by intra-alveolar hemorrhage. PE increases pulmonary vascular resistance due to both mechanical obstruction and hypoxic vasoconstriction. This raises right ventricular (RV) afterload, leading to RV dilation and septal deviation, which in turn impairs left ventricular (LV) filling and reduces cardiac output. These changes may manifest as systemic hypotension and hemodynamic instability. Acute RV failure due to pressure overload is a leading cause of early (in-hospital or 30-day) mortality (19).

Although CT pulmonary angiography (CTPA) is the gold standard for diagnosing PE, it could not be performed in this case due to the patient's hemodynamic instability. In this case, an S1Q3T3 pattern on ECG, elevated D-dimer, and clinical signs of respiratory distress strongly supported the diagnosis of acute PE. The classic S1Q3T3 pattern on ECG, characterized by a deep S wave in lead I, a Q wave in lead III, and T wave inversion in lead III, reflects acute right ventricular strain and may be seen in conditions causing acute cor pulmonale, such as PE, pneumothorax, or bronchospasm. This pattern is present in approximately 15–25% of confirmed PE cases. Other ECG findings suggestive of PE include new right bundle branch block, right-axis deviation, ST elevation in V1/aVR, T wave inversion in V1–V4, sinus tachycardia, atrial arrhythmias, and low-voltage QRS complexes(20).

To estimate pretest probability, the Wells' score was applied. The Wells' score is a semi-quantitative clinical tool used to estimate the pre-test probability of PE. The original model categorizes patients into low, moderate, or high-risk groups based on clinical findings (21). With a total score of 6.0, our patient falls into the moderate-risk category for PE, warranting further investigation and consideration of empiric anticoagulation. In the postoperative setting, risk stratification for venous thromboembolism (VTE) is essential. The Caprini Risk Assessment Model is one of the most widely validated tools in surgical patients. It assigns weighted points based on risk factors such as age, surgical duration, comorbidities, and immobility, stratifying patients into five categories: very low (0 points), low (1–2 points), moderate (3–4 points), high (5–8 points), and very high (>9 points). In this patient, the Caprini score was 8, indicating high VTE risk, for which pharmacologic thromboprophylaxis is strongly recommended. It is known that a score of 5 or higher serves as the threshold for high risk, at which point pharmacological thromboprophylaxis, with or without mechanical methods, is recommended (22).

In patients who have undergone major surgical procedures, it is generally recommended to resume low molecular weight heparin (LMWH) or unfractionated heparin (UFH) 48 to 72 hours postoperatively, depending on the patient's hemostatic status and risk of bleeding. This timing allows for adequate initial wound healing while minimizing the risk of postoperative hemorrhage, particularly in high-risk surgeries (23). Current guidelines recommend direct oral anticoagulants (DOACs), including rivaroxaban, apixaban, dabigatran, and edoxaban, as the preferred agents for both initial and long-term management of PE. In certain cases, LMWH may be used as an alternative. For PE provoked by surgery, a three-month course of anticoagulation is generally sufficient (24). To prevent PE in hospitalized individuals, pharmacologic prophylaxis options include LMWH, low-dose UFH administered two to three times daily, or fondaparinux, depending on the patient's bleeding risk and clinical status (25). The patient in our case was promptly managed with intubation, vasopressors, and anticoagulation using intravenous UFH. UFH was preferred due to its short half-life and reversibility, especially in the setting of fluctuating bleeding risk (e.g., bloody stoma output). Notably, when minimal bleeding persisted, UFH was not withheld but continued at a reduced dose, in alignment with best practices. Serial echocardiography and laboratorium results supported resolution of PE, and the patient was later transitioned to oral anticoagulation (rivaroxaban) for a planned 3–6 month duration.

Early recognition and prompt management of pulmonary embolism (PE) are critical, particularly in patients with a history of multiple abdominal surgeries complicated by intra-abdominal sepsis. In high-risk cases such as the one presented, pharmacologic anticoagulant prophylaxis should be initiated as soon as hemostasis is achieved and the risk of bleeding is adequately controlled. The complexity of the postoperative course, marked by delayed mobilization, bleeding episodes during observation, and ongoing sepsis—highlights the

significant challenges in both the prevention and management of PE in complicated surgical settings. This case illustrates how timely clinical judgment and decisive management can be life-saving in the face of acute PE following major surgery. A multidisciplinary approach played a pivotal role in the patient's recovery. Close coordination among surgical, critical care, cardiology, pulmonology, and rehabilitation teams facilitated the early detection and effective management of complications, ensuring comprehensive care throughout the recovery period.

CONCLUSION

Our case highlights the risk of pulmonary embolism following surgery for rare complications of gallbladder disease, such as cholecystocolonic fistula along with transverse colon perforation, particularly in the patient with prolonged bed rest and septic burden. Careful postoperative monitoring should be carried out to reach a comprehensive clinical decision. Early recognition and prompt management of pulmonary embolism are essential in high-risk postoperative patients. A multidisciplinary care approach is critical to improve clinical outcomes and ensure a favorable prognosis.

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