Peripheral Digital Infarction as a Rare Vascular Complication of Pyogenic Liver Abscess: A Case Report

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Abstract

Pyogenic liver abscess (PLA) is a notable intra-abdominal infection that can result in serious systemic complications, especially in people with weakened immune systems, like those suffering from diabetes mellitus. Despite the growing recognition of PLA due to advancements in diagnostic imaging, its link to uncommon vascular complications is still not widely documented. We describe the case of a 54-year-old man with diabetes who developed multiple liver abscesses, complicated by sepsis, multi-organ dysfunction syndrome (MODS), and an uncommon vascular manifestation peripheral digital ischemia of the left hallux (big toe).

Even with patent major arterial and venous circulation, the patient showed ischemic **changes** of the toe, probably due to septic microembolism or suspected disseminated intravascular coagulation (DIC) based on thrombocytopenia, elevated D-dimer, and clinical context, though formal ISTH criteria were not fully met a dangerous thrombotic complication associated with severe infection. The management of the patient involved broad-spectrum antibiotics (adjusted according to culture results), anticoagulation therapy (for suspected DIC), and intensive organ support. Although his condition improved over time, digital ischemia resulted in partial-thickness tissue loss at the tip of the hallux, underscoring the irreversible effects of delayed recognition.

This case highlights the importance of suspecting microvascular thrombosis early in PLA-associated sepsis, even without large-vessel occlusion, and acknowledging diagnostic limitations including the absence of dedicated arterial imaging and clinical photographs. It is necessary to conduct additional research in order to elucidate the mechanisms that connect PLA, DIC, and peripheral gangrene, especially in high-risk groups such as individuals with diabetes.

Keywords: Pyogenic liver abscess, Disseminated intravascular coagulation, Unilateral acral ischemia, Microvascular thrombosis, Escherichia coli, Sepsis, Diabetes mellitus.

Introduction

Liver abscess (LA) refers to a localized accumulation of pus within the liver tissue resulting from infection. It can be categorized as either an amebic liver abscess (ALA), mainly caused by *Entamoeba histolytica*, or a pyogenic liver abscess (PLA), which generally has a bacterial origin. While ALA is more common in tropical and endemic regions, PLA can be found globally and is usually polymicrobial. The most frequently identified organisms in PLA include *Klebsiella pneumoniae*, *Escherichia coli*, and various anaerobic bacteria.[1] The clinical symptoms are often nonspecific, featuring fever, pain in the right upper quadrant of the abdomen, and occasionally jaundice, especially in high-risk groups such as individuals with diabetes mellitus, hepatobiliary disorders, or those who are immunocompromised.[2]

Even with progress in diagnostic imaging and the availability of broad-spectrum antibiotics, pyogenic liver abscess (PLA) still results in considerable morbidity and mortality. Most cases are identified promptly thanks to enhanced imaging techniques like ultrasonography and computed tomography (CT), which are very effective at spotting intrahepatic fluid collections.[3] Nevertheless, complications can still occur, especially in situations where diagnosis is delayed or in individuals with pre-existing health issues.

Known complications of PLA include rupture into the peritoneal or pleural cavities, septicemia, and secondary metastatic infections. More uncommon complications include thrombophlebitis, perihepatic abscesses, and systemic embolic events.[4] Among these, vascular complications such as peripheral digital infarction are extremely rare and sparsely reported in the literature. Pathogenesis is not well understood but may involve septic microembolism or disseminated intravascular coagulation (DIC).

It can be challenging to differentiate PLA and ALA at presentation since their clinical and laboratory characteristics sometimes coincide. Leukocytosis, increased liver enzymes, and high inflammatory markers including C-reactive protein (CRP) are typical findings. These are nonspecific, however, and imaging remains the principal diagnostic method.[5] In this report, we describe an uncommon PLA complication—peripheral digital ischemia—that occurred in a patient with diabetes mellitus who also had coagulopathy, bacteremia, and severe sepsis.

Case history/examination:

Day 0 (Presentation): A 54-year-old gentleman, known to have type 2 diabetes mellitus and hypertension, presented to Hamad Medical General Hospital (HMGH) with a chief complaint of right epigastric pain radiating to the back. He reported recurrent episodes of similar pain over the past weeks, with no associated gastrointestinal or systemic symptoms. Physical examination showed right upper quadrant tenderness without guarding or rebound. Vital signs: temperature 38.1°C, heart rate 102 bpm, blood pressure 138/86 mmHg, respiratory rate 18/min, oxygen saturation 96% on room air.

Initial Investigations: Abdominal ultrasound: No intra-abdominal collection; thickened, edematous gallbladder wall suggesting possible cholecystitis (Figure 1). Stool and serology work-up: All negative including Entamoeba histolytica, HIV, hepatitis panel, dengue PCR, malaria smear, and C. auris PCR. Urine culture: No growth.

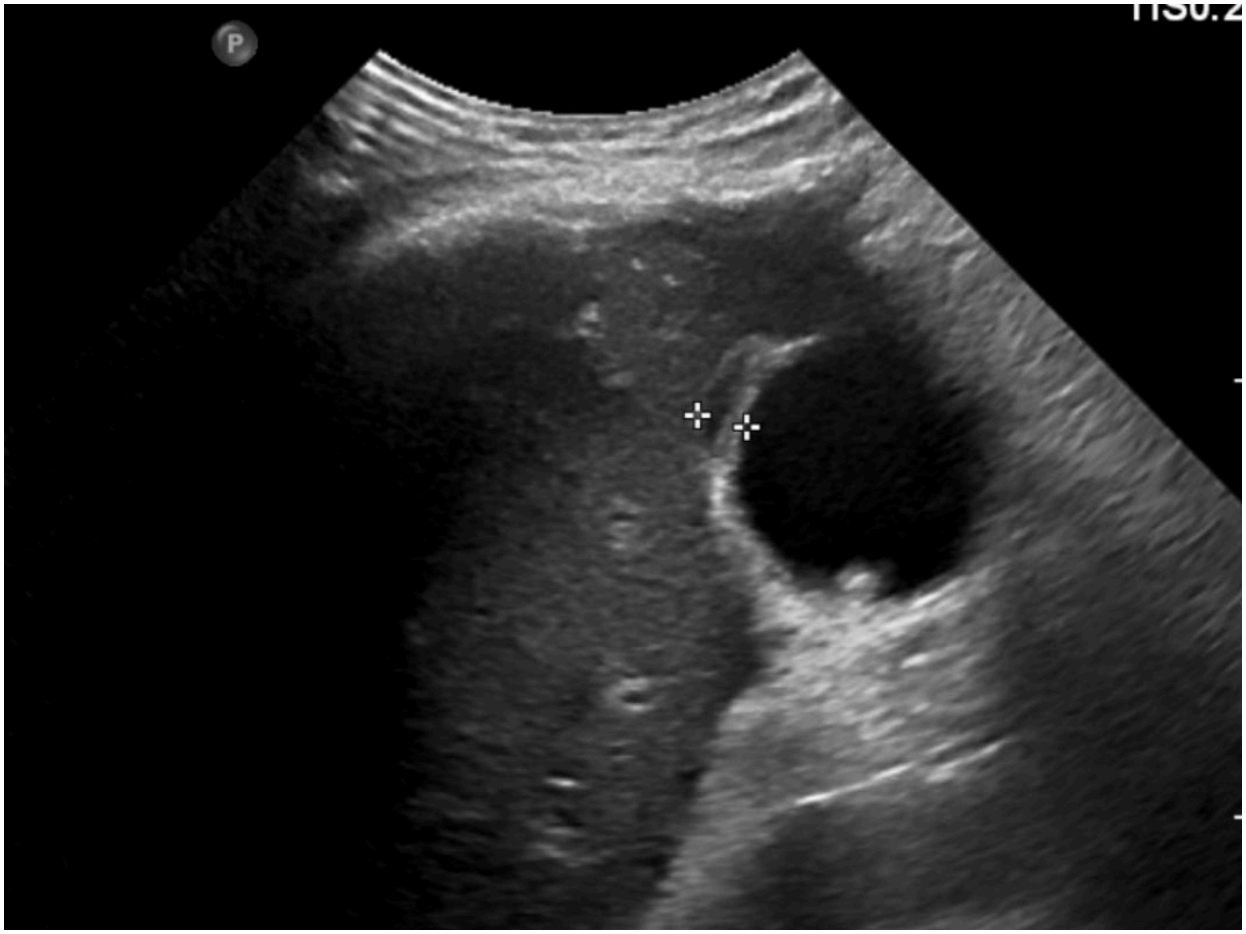

Figure 1: Ultrasound abdomen: Gallbladder: Gallbladder wall thickening noted, measuring 6.3 mm, with associated edema and trace pericholecystic fluid. A 5.5 mm non-mobile, hyperechoic, non-shadowing lesion is seen within the gallbladder. Common bile duct: Not dilated, measuring 3 mm. (No intra-abdominal collection. Oedematose thick gallbladder wall might indicate cholecystitis.)

Admission and Early Course: The patient was admitted and started empirically on doxycycline and piperacillin-tazobactam. Over the next 24 hours, he rapidly deteriorated with worsening hypotension, oliguria, and altered mental status.

ICU Day 1: The patient was transferred to the ICU with a diagnosis of suspected sepsis and multi-organ failure, including hepatic and renal dysfunction, suspected disseminated intravascular coagulation (DIC) based on platelet count 11 × 10^3/μL, D-dimer 5.47 μg/mL, INR 1.8, and fibrinogen 185 mg/dL (Table 1) (ISTH score 4, inconclusive for overt DIC**)**, and evidence of rhabdomyolysis (CK 1,240 U/L, myoglobin 420 ng/mL). Platelets (12 units) and fractionated plasma (2 bottles) were administered prophylactically due to severe thrombocytopenia and DIC-associated coagulopathy, with the goal of reducing spontaneous bleeding risk. No active bleeding or invasive procedure was present at the time of transfusion. Enoxaparin 40 mg once daily was initiated for DVT prophylaxis despite the low platelet count, as the patient had no active bleeding and was at high thromboembolic risk. Chest X-ray and CTPA ruled out pneumonia and pulmonary embolism. Echocardiography (TTE) revealed no valvular pathology. The CTPA was also reviewed for aortic atheroma or shaggy aorta, but only the visualized thoracic aorta was unremarkable; dedicated abdominal aortic imaging was not performed, a significant limitation.

ICU Day 2: Antibiotics were escalated to meropenem.

ICU Day 3: Blood cultures confirmed sensitive *E. coli* bacteremia. Sensitivity pattern: E. coli sensitive to piperacillin-tazobactam, meropenem, and ampicillin-sulbactam; resistant to ampicillin and ciprofloxacin. Antibiotics were then de-escalated to piperacillin-tazobactam (Tazo Cin) and continued doxycycline. The patient received further platelet support (additional 6 units on Day 3, 4 units on Day 4). The platelet count improved to $68 \times 10^3/\mu L$ by ICU Day 6 (Table 1), at which time enoxaparin was escalated to 80 mg daily in response to progressive toe ischemia (see below).

ICU Day 4-6: The patient's hemodynamics stabilized. A subsequent MRCP revealed multiple new hepatic lesions not previously seen, demonstrating restricted diffusion highly suggestive of multiple liver abscesses (Figure 2). A repeat ultrasound confirmed progressive enlargement of the lesions. Percutaneous drainage was deferred due to severe thrombocytopenia (platelet count $11 \times 10^3/\mu L$)(Table 1) and coagulopathy. The abscesses were managed conservatively with antibiotics.

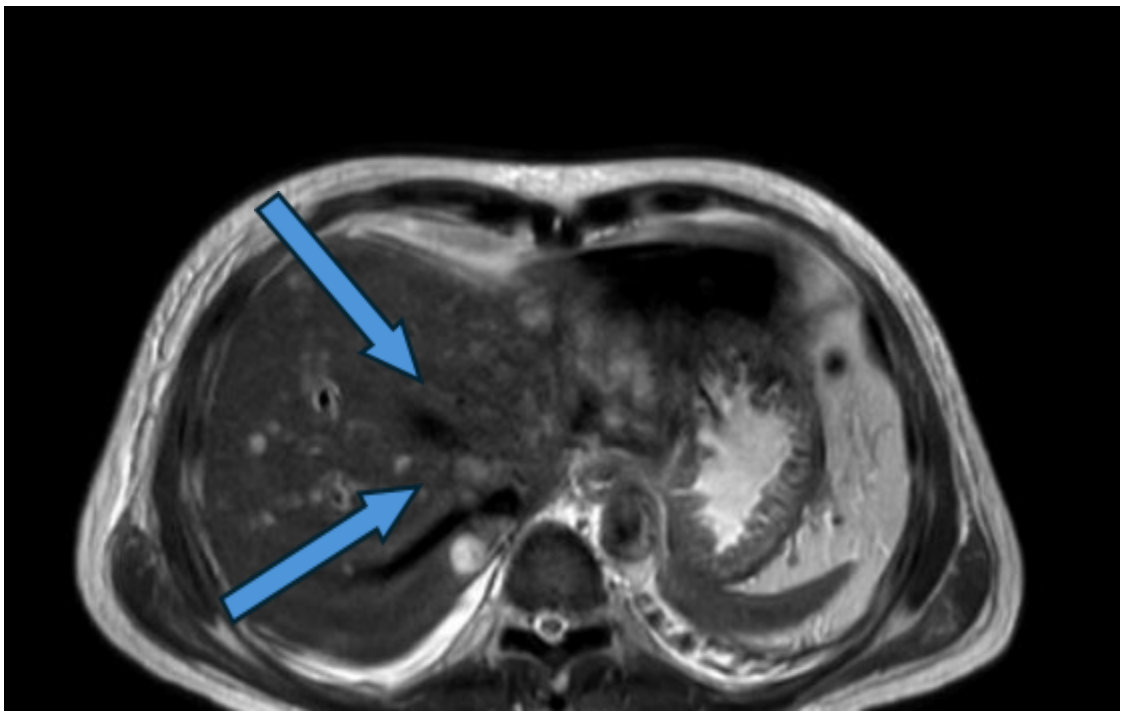

Figure 2: MRCP: Multiple liver lesions which have appeared over a very short span of time (previous recent ultrasound did not show a focal liver lesion). These show varying patterns of restricted diffusion and are highly suspicious of multiple liver abscesses. Features of calculus cholecystitis. Incidental intraspinal lesion. Complete MRI examination of the spine with contrast is suggested after the AKI settles. (Multiple liver lesions which have appeared over a very short span of time highly suspicious for multiple liver abscesses (Blue arrows).)

Step-down to Ward – Ward Day 1-2: The patient was stepped down from the ICU and continued ampicillin-sulbactam per infectious disease recommendations.

Ward Day 3: The patient developed new onset swelling and erythema of the left lower limb, with redness, pain, and tenderness localized to the left big toe. The left big toe initially showed well demarcated erythema and edema, progressing over 72 hours to violaceous discoloration with coolness to touch. No livedo reticularis, purpura, or involvement of other digits on either foot was observed. Unfortunately, clinical photographs were not obtained, which is a limitation of this case report. Pedal pulses were intact. Foot X-ray and lower limb Doppler ultrasound were all unremarkable. There was no evidence of livedo reticularis, purpura fulminans, or involvement of other digits.

Ward Day 4-12: Over the next 9 days, the toe ischemia progressed to partial-thickness tissue loss at the tip of the hallux, but the toe was not completely infarcted, and progressive improvement occurred with continued anticoagulation, resolved completely at the superficial level with anticoagulation, antibiotics, and control of blood glucose and blood pressure. A follow-up ultrasound at 4 weeks post-discharge demonstrated near complete resolution of the hepatic lesions.

Table 1: Laboratory investigations over the clinical course

Parameter	At Admission	ICU Day 1	ICU Day 3	Ward Day 5 (pre-discharge)	Reference Range
WBC ($\times 10^3/\mu L$)	8.4	6.2	9.1	7.8	4.0–11.0
Bands (%)	5	18	12	4	0–5
Hemoglobin (g/dL)	12.4	10.2	9.8	11.2	13.0–17.0
Platelets ($\times 10^3/\mu L$)	187	11	34	156	150–450
INR	1.1	1.8	1.6	1.1	0.9–1.2
Fibrinogen (mg/dL)	320	185	210	310	200–400
D-dimer (µg/mL)	0.5	5.47	4.2	0.8	<0.5
ALT (U/L)	42	756	620	95	10–40
AST (U/L)	38	475	390	55	10–40
Total bilirubin (mg/dL)	0.8	3.2	2.8	1.1	0.3–1.2
Creatinine (mg/dL)	0.9	2.8	2.1	1.0	0.6–1.3
CRP (mg/L)	45	189.9	156	22	<5
Lactate (mmol/L)	1.2	6.0	2.8	1.1	0.5–2.0
CK (U/L)	120	1,240	890	145	30–200
Myoglobin (ng/mL)	50	420	280	65	<90
Troponin-T (ng/mL)	0.02	0.89	0.45	0.03	<0.05

Discussion

The Systemic Consequences of Pyogenic Liver Abscess. The second most prevalent infectious illness of the liver, pyogenic liver abscess (PLA), poses serious risks to life, particularly for those who already have diabetes mellitus or underlying hepatobiliary abnormalities.[1] In Asian populations, Klebsiella pneumoniae remains the most common pathogen, while Escherichia coli accounts for only 6.6% of PLA cases.[2,3] It is important to note that the death rates and incidence of septic shock (38.6%) linked to PLA caused by E. coli are disproportionately greater than those linked to other causes.[4] With a prevalence of only 2.88% among PLA cases in one cohort, sepsis is still a rare consequence.[2] Recent data from the Oman Medical Journal have also highlighted that diabetic patients with PLA have a higher risk of complications, including sepsis and coagulopathy.[6,7]

A Rare Vascular Complication: Unilateral Acral Ischemia (Revised). Our patient's unilateral toe ischemia differs from classical symmetrical peripheral gangrene (SPG), which typically presents with symmetric distal necrosis. However, sepsis-induced microvascular thrombosis can occasionally present asymmetrically. Sepsis induced disseminated intravascular coagulation (DIC) is closely linked to acral ischemia, a rare condition characterized by distal necrosis without large vessel blockage or vasculitis.[5,8] Since acral ischemia in sepsis is so uncommon, it is difficult to determine its true occurrence; the majority of information is based on case reports and short case series.[5,8]

Severe sepsis with suspected DIC is demonstrated by the rapid progression of our patient's condition from right upper quadrant pain to multi-organ dysfunction (acute kidney injury, hepatic failure, type 2 myocardial injury), critical thrombocytopenia ($11 \times 10^3/\mu L$), and markedly elevated inflammatory markers (CRP 189.9 mg/L). The ISTH overt DIC scoring system yielded a score of 4 (thrombocytopenia + elevated D-dimer + clinical deterioration), which is inconclusive (score ≥5 required for overt DIC). This highlights the diagnostic uncertainty inherent in such cases. Laboratory data also showed elevated levels of D-dimer (5.47 µg/mL) and lactate (6 mmol/L). According to published research, DIC is present in 85–100% of SPG cases, underscoring its critical role in the onset of this illness.[9,10]

Thrombosis, Hepatic Dysfunction, and Diagnostic Uncertainty (Revised). The proposed mechanism of acute ischemic hepatitis (also known as "shock liver") was **suspected** by the patient's markedly raised liver enzymes. Notably, the pattern showed ALT > AST (756 vs 475 U/L), which is atypical for pure ischemic hepatitis (where AST > ALT is expected). This may reflect a mixed picture of sepsis related liver injury, possible **cholecystitis,** and early ischemic insult. E. coli septic shock, which caused suspected disseminated intravascular coagulation (DIC), most likely started the chain of events.

The hepatic damage then significantly worsened this prothrombotic condition. The liver's ability to produce natural anticoagulants, particularly protein C and antithrombin, is significantly reduced during ischemic hepatitis.[11,12] A vicious cycle was started by this inability to control coagulation, which significantly exacerbated coagulopathy and encouraged widespread microvascular thrombosis, which ultimately led to acral necrosis. The development of digital ischemia in spite of patent microvasculature can be explained by this pathophysiological cascade (Sepsis → DIC → Hepatic Dysfunction → Worsening Coagulopathy → acral ischemia). Warkentin et al. have described a similar "temporal gap" of 1–5 days between hepatic injury and limb ischemia; our patient developed toe changes after 6 days, consistent with this literature.[13]

Diagnostic Limitations and Alternative Diagnoses (New paragraph). A major limitation of this report is the absence of dedicated lower extremity or aortic arterial imaging (e.g., CTA or MRA). Therefore, blue toe syndrome secondary to cholesterol crystal embolization or an alternative embolic source cannot be completely excluded, although the clinical context of severe sepsis and coagulopathy favored DIC mediated microvascular thrombosis. Alternative mechanisms must also be considered. Septic microembolism originating from the liver abscess could theoretically embolize to the digital circulation, though the absence of echocardiographic evidence of endocarditis and the lack of other embolic phenomena make this less likely. Sepsis-induced vasculopathy, including direct endothelial injury from circulating cytokines and bacterial toxins, may also contribute to acral ischemia independent of overt DIC. The normal WBC count ($8.4 \times 10^3/\mu L$) in the setting of severe sepsis is atypical; however, a left shift was present with bands of 18%, suggesting a septic leukocyte response.

Diagnostic Journey and Challenges. The diagnostic journey highlights the challenges in detecting PLA: several liver abscesses were discovered by magnetic resonance cholangiopancreatography (MRCP) after initial abdominal ultrasonography failed to yield any answers. The pyogenic etiology was validated by the isolation of E. coli from blood cultures and negative Entamoeba histolytica serology results.

The Impact of Diabetes Mellitus. Because of endothelial dysfunction and hypercoagulability, diabetes mellitus raises the risk of DIC.[14] Our patient's concomitant diabetes likely made him more susceptible to PLA-related sepsis and eventual acral ischemia, which is in line with studies showing diabetes to be a risk factor for adverse outcomes in PLA.[15] A recent study in the Oman Medical Journal similarly reported worse clinical outcomes in diabetic patients with pyogenic liver abscess.[7]

Therapeutic Strategies and Clinical Dilemmas. Three key pillars served as the center of management[1]: Source control: In compliance with antimicrobial stewardship guidelines, empiric broad-spectrum antibiotics (piperacillin-tazobactam) were initiated and then de-escalated to ampicillin-sulbactam once E. coli was identified. Drainage was deferred due to coagulopathy, and antibiotic therapy alone was successful.[2] Anticoagulation: Enoxaparin was started for DVT prophylaxis and escalated once platelet count recovered, given progressive ischemia.[3] Supportive care: Multi-system dysfunction was lessened by intensive organ support. Anticoagulation's efficacy in sepsis associated acral ischemia is still up for question,[5] although new research suggests that it might be helpful in sepsis-associated coagulopathy.[16,17] This case highlights the need for tailored risk-benefit analyses in handling such complex circumstances.

Clinical Implications and Limitations Summary. This case offers several critical lessons: Unilateral acral ischemia can occur in PLA without large-vessel occlusion. Clinicians should maintain a high index of suspicion for microvascular thrombosis in septic patients with unexplained digit pain, even when pulses are intact and findings are unilateral. Early intervention with anticoagulation (after weighing bleeding risk) may help reduce tissue loss. Multidisciplinary cooperation is essential. Important limitations of this report include the lack of confirmatory DIC parameters, absence of dedicated arterial imaging, and lack of clinical photographs.

Conclusion

Pyogenic liver abscess remains a substantial infection risk, causing significant morbidity, particularly among those with underlying health issues like diabetes mellitus. While progress in early detection and treatment has improved patient outcomes, there is still a risk of rare and possibly life-threatening complications.

This instance exemplifies peripheral digital ischemia, an extremely uncommon vascular complication linked to liver abscesses, probably caused by sepsis-induced coagulopathy or microvascular thrombotic events in the setting of suspected DIC, though alternative diagnoses such as blue toe syndrome cannot be fully excluded. The emergence of ischemic changes without large vessel occlusion highlights the need for increased vigilance in patients whose condition is worsening, even if they do not exhibit typical symptoms and even when findings are unilateral. In this case, prompt recognition, appropriate care, fitting antimicrobial therapy, and thromboprophylaxis were essential for recovery. To prevent irreversible consequences, healthcare providers need to be highly vigilant for atypical manifestations of liver abscesses, especially in patients who are critically ill.

Disclosure

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