MENA Journal of Case Reports
Doi: 10.64913/mmrmjcr.v1i2.26
Volume No.1, 2026

Case Report

Psoas Abscess in a Post-Nephrectomy Patient with Decompensated Liver Cirrhosis: A Case Report Illustrating the Intersection of Surgical Alteration and Immune Dysfunction

Hassan Aziz[1*], Muzeer Ahmed[1], Syed Muhammad Omar[1], Fivzia Farooq Herekar[1]

[1] Department of Internal Medicine, Indus Hospital and Health Network, Karachi, Pakistan.

*Corresponding Author: Hassan.aziz@tih.org.pk

Received: 17/02/2026; Accepted: 16/06/2026; Published: 13/07/2026

Abstract

Introduction

Psoas abscess (PA) is an uncommon condition, particularly in patients with altered retroperitoneal anatomy or compromised immunity. We report a case of a delayed psoas abscess in a patient with a prior right nephrectomy and decompensated liver cirrhosis (DCLD). While psoas abscesses following nephrectomy or in the setting of cirrhosis are documented separately, this case illustrates a unique intersection of these factors. We propose the concept of "Chronic Latent Deep Nidus Formation" (CLDNF) as a descriptive framework for discussing how dormant surgical site infections might reactivate in the context of systemic immune dysfunction.

Case Presentation

A 60-year-old male presented with abdominal distension and low-grade fever. Initially managed for spontaneous bacterial peritonitis (SBP), the ascitic fluid analysis was non-revealing. Cross-sectional imaging revealed a large right psoas abscess extending into the ipsilateral nephrectomy bed, and microbiological culture identified *Klebsiella pneumoniae*. The patient improved following percutaneous drainage and targeted antibiotic therapy.

Discussion

This case describes a rare and diagnostically challenging condition, Psoas abscesses that developed years after nephrectomy in a patient with decompensated chronic liver disease (DCLD). The case provides additional clinical insight into the diagnostic complexity and management of atypical infections in cirrhotic patients. The microbiological etiology of psoas abscesses varies according to geographic region and host-related factors. In our patient, Klebsiella pneumoniae, exclusively isolated from the abscess fluid, supports the possibility of a localized infectious process rather than hematogenous dissemination. It's plausible that delayed post-nephrectomy psoas abscesses could be related to residual infective foci, altered retroperitoneal anatomy, or delayed bacterial colonization.

Conclusion

This case provides clinical insight into how altered anatomical spaces may harbor subclinical infection that reactivates within the permissive environment of cirrhosis-associated immune dysfunction (CAID).

Keywords: Psoas abscess; Decompensated Liver Disease; Immunosuppression

1. Introduction

Psoas abscess (PA) is a rare and diagnostically challenging condition characterized by the accumulation of pus within the iliopsoas muscle compartment. Diagnosis in PA is frequently delayed due to nonspecific clinical presentations, such as abdominal pain, fever, or malaise. Psoas abscesses are broadly classified as primary or secondary. Primary PA, accounting for approximately 21.8% of cases, typically arises via hematogenous spread from occult or distant infections without a clear local

source. In contrast, secondary PA, which accounts for around 78.2% of cases, results from direct extension of infection from adjacent structures. The most associated sources include the gastrointestinal tract (24.7%), such as appendicitis, colorectal malignancies, and inflammatory bowel diseases; the urinary tract (17.5%), especially pyelonephritis; and the spine (50.5%), notably vertebral osteomyelitis [1].

We present a case report of psoas abscess in a post-nephrectomy patient with decompensated liver cirrhosis (DCLD). Although several reports of psoas abscesses after nephrectomy exist, to our knowledge, this is the first case of its kind to be reported from Pakistan. This case reports an intersection of prior surgical alteration and cirrhosis-induced immune dysfunction, making this case a unique diagnostic dilemma. We hypothesize that, in this case, the altered retroperitoneal anatomy resulting from nephrectomy served as a locus minoris resistentiae, while cirrhosis-associated immune dysfunction (CAID) further predisposed the patient to atypical and delayed infection. Reporting this rare case and its management may improve understanding of such diagnostically challenging cases, helping management and outcomes for these patients.

This case highlights the critical importance of maintaining a high index of suspicion and utilizing early cross-sectional imaging in cirrhotic patients with non-specific febrile illness, especially when there is a history of prior urologic surgery.

2. Case Presentation

2.1. History and Initial Presentation

A 60-year-old male with a history of right nephrectomy for pyonephrosis (performed five years prior) and DCLD presented with a one-week history of worsening abdominal distension, persistent low-grade fever, and thrombocytopenia **(Table 1)**.

Table 1: A summary of the clinical timeline from diagnosis to last follow-up.

Year	Event
2021	Right nephrectomy performed for pyonephrosis
2023–2025	Diagnosis and progressive decompensation of liver cirrhosis
February 2026	The patient presented with a one-week history of fever and abdominal distension. A psoas abscess was diagnosed on contrast-enhanced CT and managed with CT-guided percutaneous drainage. Intravenous meropenem was administered at a dose of 1 g three times daily (TDS) for 21 days.

2.2. Diagnostic workup

Initial evaluation focused on spontaneous bacterial peritonitis (SBP). Paracentesis revealed a serum-ascites albumin gradient (SAAG) of 1.7, consistent with portal hypertension, but a low neutrophil count ruled out SBP. An abdominal ultrasound confirmed features of chronic liver disease, including coarse echotexture and splenomegaly, but failed to identify the abscess in the renal bed. This diagnostic limitation was likely due to the collection's deep retroperitoneal location and the presence of overlying bowel gas and tense ascites, which often obscure the nephrectomy bed on transabdominal sonography.

Due to clinical deterioration, including hypotension and hypoglycemia, a contrast-enhanced CT was performed. The CT revealed a large right-sided psoas abscess that demonstrated clear anatomical continuity with the prior nephrectomy bed, suggesting the surgical site as the primary origin of the collection **(Figure 1).**

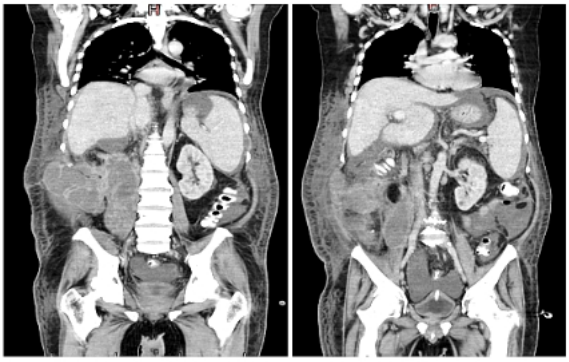

Figure 1: *Contrast CT scan showing the abscess in the right psoas muscle on the same side as the nephrectomy*

2.3. Treatment and treatment response

Ultrasound-guided percutaneous drainage was performed, and the abscess culture grew Klebsiella pneumoniae. Based on the culture and sensitivity results, intravenous meropenem (1 g three times daily) was administered for 21 days. The patient remained hospitalized for continued clinical observation, and an ultrasound performed before discharge demonstrated complete resolution of the abscess with no residual collection. The patient was discharged in stable condition on hospital day 27 with no evidence of recurrence.

2.4. Follow-up

The patient completed a 21-day course of intravenous meropenem (1 g three times daily) following ultrasound-guided percutaneous drainage. Serial laboratory investigations demonstrated progressive normalization of inflammatory markers, consistent with a favorable response to treatment **(Table 2).** An ultrasound performed before discharge on hospital day 26 showed complete resolution of the psoas abscess with no residual fluid collection. The patient was discharged in stable condition and remained asymptomatic, with no clinical evidence of recurrence at follow-up.

Table 2: Serial laboratory parameters demonstrating treatment response

Parameter	At Presentation	Before Discharge
Hemoglobin (g/dL)	10	11
Total leukocyte count ($\times 10^9$/L)	20	7
C-reactive protein (mg/L)	125	3
Procalcitonin (ng/mL)	12	0.01

3. Discussion

PA is a rare, diagnostically challenging condition, particularly when it develops years after nephrectomy in patients with decompensated chronic liver disease (DCLD). In our case, the patient initially presented with nonspecific symptoms including fever, abdominal distension, and thrombocytopenia, prompting evaluation for spontaneous bacterial peritonitis (SBP), which was excluded through ascitic fluid analysis. Persistent fever necessitated further investigation, and contrast-enhanced computed tomography (CT) ultimately revealed a right-sided psoas abscess extending into the previous nephrectomy bed. This case provides additional clinical insight into the diagnostic complexity and management of atypical infections in cirrhotic patients. The microbiological etiology of psoas abscesses varies according to geographic region and host-related factors. While *Staphylococcus aureus* and *Escherichia coli* are among the most commonly implicated organisms, psoas abscesses in developing countries such as Pakistan and India are frequently associated with *Mycobacterium tuberculosis* [1]. In contrast, *Klebsiella pneumoniae* has been increasingly reported in East Asian populations, particularly among diabetic patients with poor glycemic control [2]. In our patient, the previous history of nephrectomy for pyonephrosis five years earlier represents a potential predisposing anatomical factor. Delayed post-nephrectomy psoas abscesses, although rare, have previously been described as late complications related to residual infective foci, altered retroperitoneal anatomy, or delayed bacterial colonization [3, 4]. Tez *et al.* and Di Marco *et al.* reported cases of psoas abscess formation occurring 10–21 years after nephrectomy, both associated with *Proteus mirabilis* infection and successfully managed with drainage and antibiotics **(Table 3).** While our patient's coexisting DCLD may have plausibly contributed to increased susceptibility to infection, a direct causal relationship cannot be established from a single case report.

Table 3: Comparison of clinical profiles of patients with post-nephrectomy psoas abscess

Age	Gender	Abscess formation after nephrectomy	Diagnostic tool	Treatment	Organism	Year (Ref)
66	Male	21 years	CT scan	U/S guided percutaneous abscess drainage and antibiotic therapy	Proteus Mirabilis	2002 [3]
77	Male	10 years	CT scan	Surgery and Antibiotic therapy	Proteus Mirabilis	2007 [5]
60	Male	5 years	Ct scan	U/S guided percutaneous abscess drainage and antibiotic therapy	Klebsiella Pneumonia	Our case

Patients with DCLD are particularly susceptible to infections due to cirrhosis-associated immune dysfunction (CAID), which involves impairment of both innate and adaptive immune pathways. Beyond the classical description by Bonnel *et al.* [5], more recent studies have demonstrated that CAID is characterized by reduced complement activity, impaired neutrophil chemotaxis and phagocytosis, intestinal bacterial translocation, monocyte dysfunction, reticuloendothelial impairment, and altered gut microbiota, collectively predisposing patients to severe and atypical infections [6, 7]. These mechanisms have been implicated not only in spontaneous bacterial peritonitis but also in deep-seated and extra-peritoneal infections, including muscle abscesses, retroperitoneal infections, and occult bacteremia in cirrhotic patients [8]. In our case, the absence of bacteremia or urinary tract infection, combined with isolation of *Klebsiella pneumoniae* exclusively from abscess fluid, supports the possibility of a localized infectious process rather than overt hematogenous dissemination.

Rather than proposing a novel pathological entity, we describe "Chronic Latent Deep Nidus Formation" (CLDNF) as a descriptive and hypothesis-generating conceptual framework. This terminology aims to contextualize previously recognized mechanisms whereby surgically altered anatomical spaces may harbor clinically silent infective foci that later manifest under conditions of immune dysfunction. Similar concepts have previously been discussed under theories of delayed deep surgical site infection, residual infective foci, and locus minoris resistentiae. Importantly, we acknowledge that the proposed nidus mechanism in our case remains speculative because longitudinal microbiological correlation with the original pyonephrosis is unavailable. Therefore, while reactivation of a latent infective focus is biologically plausible, alternative explanations—including delayed colonization of the post-nephrectomy retroperitoneal space or transient subclinical hematogenous seeding facilitated by CAID—must also be considered.

To our knowledge, few reports have discussed the possible interplay between cirrhosis-associated immune dysfunction and delayed abscess formation at prior surgical sites. Accordingly, this case highlights the importance of maintaining a high index of suspicion for atypical and deep-seated infections in immunocompromised cirrhotic patients, particularly in the presence of altered postoperative anatomy.

4. Conclusion

This case illustrates the intersection of prior surgical alteration and the permissive environment created by CAID. While the individual components of this presentation—surgical site latency and cirrhosis-related vulnerability are recognized, their combination underscores the need for a high index of suspicion. Early cross-sectional imaging remains essential when standard therapies for febrile cirrhotic patients fail, as ultrasound has limited sensitivity for retroperitoneal infections.

5. Declarations

Ethics Approval and Consent to Participate

Ethical approval and written informed consent were obtained from the patient for participation and publication of this case report.

Consent for Publication

Written informed consent for publication was obtained from the patient.

Data Availability Statement

Not applicable.

Conflicts of Interest

The authors declare no competing interests.

Funding

No funding was received for the preparation of this manuscript.

Author Contributions

HA contributed to the conceptualization of the study, development of the study idea, literature review, drafting and writing of the original manuscript, and supervision. MA AND SMO contributed to the literature review, scientific editing, and critical revision of the manuscript. FFH provided expert supervision and critically reviewed the entire manuscript for scientific accuracy and intellectual content, and approved the final version submitted for publication.

Acknowledgments

All medical content, clinical decisions, and interpretations are the sole responsibility of the authors.

AI Declaration

The authors confirm that no content in this manuscript was generated using artificial intelligence (AI) tools and the authors take full responsibility for the accuracy and integrity of the work.

6. Abbreviations

The following abbreviations are used in this manuscript:

PA	Psoas Abscess
DCLD	Decompensated Chronic Liver Disease
CAID	Cirrhosis-Associated Immune Dysfunction
SBP	Spontaneous Bacterial Peritonitis
SAAG	Serum-Ascites Albumin Gradient
CT	Computed Tomography
CLDNF	Chronic Latent Deep Nidus Formation
U/S	Ultrasound
CRP	C-Reactive Protein
IRB	Institutional Review Board

References

[1] López, V.N.; Ramos, J.M.; Meseguer, V.; Pérez Arellano, J.L.; Serrano, R.; Ordóñez, M.A.G.; et al. Microbiology and outcome of iliopsoas abscess in 124 patients. *Medicine (Baltimore)* **2009**, 88(2), 120–130. [CrossRef]

[2] Chang, C.M.; Ko, W.C.; Lee, H.C.; Chen, Y.M.; Chuang, Y.C. Klebsiella pneumoniae psoas abscess: predominance in diabetic patients and grave prognosis in gas-forming cases. *J. Microbiol. Immunol. Infect.* **2001**, 34(3), 201–206. [PubMed]

[3] Tez, S.; Dilmen, G.; Unsal, A.; Koktener, A.; Cimentepe, E.; Saglam, R. Psoas abscess twenty-one years after ipsilateral nephrectomy. *Int. Urol. Nephrol.* **2002**, 34(3), 311–313. [CrossRef]

[4] Guillaume, M.P.; Allé, J.L.; Cogan, E. Secondary psoas abscess twenty-seven years after nephrectomy. *Eur. Urol.* **1994**, 25(2), 171–173. [CrossRef]

[5] Di Marco, L.; Sciascia, V.; Salmi, R.; Manfredini, A.; Cocuzza, C.; Berghenti, M. Psoas abscess ten years after ipsilateral nephrectomy for pyonephrosis. *G. Chir.* **2007**, 28(4), 139–141. [PubMed]

[6] Bonnel, A.R.; Bunchorntavakul, C.; Reddy, K.R. Immune dysfunction and infections in patients with cirrhosis. *Clin. Gastroenterol. Hepatol.* **2011**, 9(9), 727–738. [CrossRef]

[7] Ali, S.; Sohail, A.; Brown, K. A Case of a Large Intraabdominal Abscess in a Patient with Cirrhosis Misdiagnosed as Spontaneous Bacterial Peritonitis. *Case Reports Hepatol.* **2022**, 5951115. [CrossRef]

[8] de Jesus Lopes Filho, G.; Matone, J.; Arasaki, C.H.; Kim, S.B.; Mansur, N.S. Psoas abscess: diagnostic and therapeutic considerations in six patients. *Int. Surg.* **2000**, 85(4), 339–343. [PubMed]