Peritoneal Tuberculosis In A Hemodialysis Patient With Longstanding Ascites: A Diagnostic Challenge

Dyah Retno Wulandari[1,2*], Widodo[3,4]

[1]Internal Medicine Subspecialist Study Program, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.
[2]Internal Medicine Subspecialist Study Program, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia.
[3]Division of Nephrology, Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia.
[4]Division of Nephrology, Department of Internal Medicine, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia.
Corresponding author:
Dyah Retno Wulandari
dyahretnosppd@gmail.com

ABSTRACT

Introduction: Tuberculous peritonitis is an uncommon form of extrapulmonary tuberculosis that is difficult to diagnose in patients receiving maintenance dialysis because its clinical manifestations are often nonspecific and overlap with nephrogenic ascites or fluid overload. Delayed diagnosis may postpone appropriate treatment and worsen clinical outcomes.
Case: We report the case of a 44-year-old man with end-stage renal disease on maintenance hemodialysis who presented with persistent ascites. The condition was initially considered nephrogenic ascites, but the subsequent development of constitutional symptoms prompted further evaluation. Ascitic fluid analysis and additional investigations confirmed the diagnosis of tuberculous peritonitis, and antituberculous therapy was initiated.
Discussion: This case illustrates the diagnostic challenge of distinguishing tuberculous peritonitis from other causes of ascites in dialysis patients. Persistent ascites that does not improve with optimized dialysis should prompt consideration of infectious etiologies, particularly tuberculosis in endemic settings. Early diagnostic workup is crucial for timely treatment.
Conclusion: Tuberculous peritonitis should be included in the differential diagnosis of refractory ascites in patients undergoing maintenance dialysis. Early recognition and prompt investigation can facilitate timely treatment and improve patient outcomes.

Keywords: Peritoneal tuberculosis, Hemodialysis, Ascites, End-stage kidney disease.

INTRODUCTION

Tuberculosis (TB) remains a major global health challenge, and the World Health Organization (WHO) continues to emphasize early case detection and systematic screening among high-risk populations as key components of TB control (1,2). Patients with chronic kidney disease (CKD), particularly those receiving maintenance dialysis, are at substantially increased risk of developing TB because of impaired cell-mediated immunity (2,3). A meta-analysis reported that individuals undergoing dialysis have a 3.62-fold higher risk of TB than the general population (3). With the increasing global burden of CKD, particularly in TB-endemic regions, the coexistence of CKD and TB has become an important clinical concern (2,4,5).

Peritoneal tuberculosis is an uncommon manifestation of extrapulmonary TB, accounting for approximately 3.5% of all TB cases and 31–58% of abdominal TB cases (6). Although uncommon, peritoneal TB should be considered in the differential diagnosis of patients receiving hemodialysis who present with persistent ascites. Its clinical manifestations are often subtle and nonspecific, and chronic ascites may be misattributed to nephrogenic ascites rather than an infectious etiology (6,7). Nephrogenic ascites is a diagnosis of exclusion in patients with end-stage renal disease and is associated with poor prognosis (8). Consequently, failure to recognize peritoneal TB may result in delayed diagnosis and treatment, particularly in TB-endemic settings.

We report the case of a 44-year-old man receiving maintenance hemodialysis who presented with longstanding refractory ascites that was initially considered nephrogenic ascites but was ultimately diagnosed as tuberculous peritonitis. This case highlights the importance of maintaining a high index of suspicion for peritoneal TB in dialysis patients with persistent unexplained ascites, even in the absence of typical pulmonary manifestations.

CASE ILLUSTRATION

A 44-year-old man with end-stage renal disease (ESRD) receiving maintenance hemodialysis for four years presented with persistent ascites for two years, without constitutional symptoms or other clinical evidence of infection. Four months before admission, he developed intermittent mild abdominal pain, which progressively worsened during the week preceding hospitalization. Two months before admission, he was treated empirically

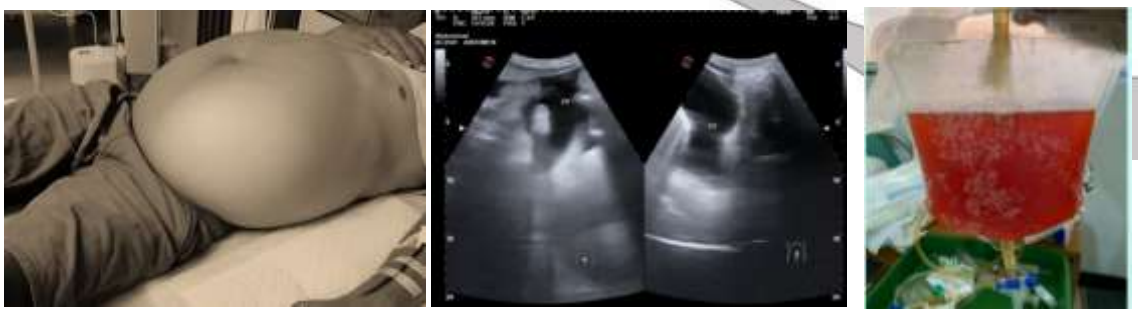
for presumed spontaneous bacterial peritonitis after ascitic fluid culture grew coagulase-negative Staphylococcus. Although his symptoms partially improved following antibiotic therapy, the ascites persisted and abdominal discomfort remained unresolved.

He had been receiving maintenance hemodialysis twice weekly for four years, with an ultrafiltration volume of 3–4 L per session, a blood flow rate of approximately 200 mL/min, and a dialysate flow rate of 500 mL/min. Residual urine output was negligible, measuring less than 100 mL/day. At the time of admission, he reported intermittent fever with chills, particularly following hemodialysis sessions, accompanied by an unintentional weight loss of 4 kg over the preceding four months. On physical examination, his temperature was 39.5°C, heart rate 115 beats/min, respiratory rate 20 breaths/min, blood pressure 140/98 mmHg, and oxygen saturation 92% on room air. Initial laboratory investigations revealed severe anemia (hemoglobin 7.2 g/dL). Serum tumor markers, including carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA 19-9), were within the normal range at 2.99 ng/mL and 13.39 U/mL, respectively. Serum adenosine deaminase (ADA) was mildly elevated at 25.8 U/L and increased to 52.4 U/L two weeks later. Representative clinical photographs, abdominal ultrasonography, and the gross appearance of the ascitic fluid are shown in Figure 1.

Diagnostic paracentesis yielded ascitic fluid with a pH of 7.32 and a total leukocyte count of 577 cells/µL. Differential cell count showed a predominance of mononuclear cells (63.7%; 344 cells/µL) over polymorphonuclear cells (36.3%; 196 cells/µL). The red blood cell count was 2 × 10^3/µL. Biochemical analysis showed elevated total protein (5.37 g/dL), glucose of 96 mg/dL, and LDH of 320 U/L. These findings were consistent with exudative ascitic fluid characterized by a predominance of lymphocytes/mononucleocytes. Gene Xpert MTB/RIF performed on the ascitic fluid detected Mycobacterium tuberculosis without rifampicin resistance. These findings established the diagnosis of tuberculous peritonitis. Relevant laboratory findings are summarized in Table 1.

Table 1. Serial Laboratory, Ascitic Fluid, and Microbiological Findings

Items	January 2025	14th April 2025	27th April 2025
Albumin (serum)	2.60 g/dL	2.14 g/dL	1.85 g/dL
ADA (serum)	25.8 U/L		52.4 U/L
WBC (ascites)	2.551 x10^3/µL	0.540 x10^3/µL	0.457 x10^3/µL
MN (ascites)	47.1 %	63.7%	54.2%
PMN (ascites)	52.9 %	36.3%	45.8%
Protein (ascites)	4.54 g/dL	5.37 g/dL	4.51 g/dL
Glucose (ascites)	129 mg/dL	96 mg/dL	90 mg/dL
LDH (ascites)	302 U/L	320 U/L	228 U/L
Fluid Culture (January 2025)	Staphylococcus coagulase-negative		
Gene Xpert MTB (April 2025)	MTB Detected RIF Sensitive		



(a) (b) (c)

Figure 1. (a). Clinically ascites, (b). Abdominal USG, (c). Ascitic Fluid

DISCUSSION

Persistent ascites in patients with end-stage kidney disease presents a significant diagnostic challenge because nephrogenic ascites is considerably more common than tuberculous peritonitis. Among patients with stage III or higher chronic kidney disease (CKD), ascites has been reported in approximately 12.9%, with nephrogenic ascites accounting for nearly 78% of cases, whereas abdominal tuberculosis represents only about 2% (8,9). Peritoneal tuberculosis is an uncommon form of extrapulmonary TB, accounting for approximately 3.5% of all TB cases and 31–58% of abdominal TB (6). Clinical manifestations are typically nonspecific, including chronic ascites, abdominal pain, fever, anorexia, and weight loss, often resulting in delayed diagnosis, particularly in patients receiving maintenance hemodialysis (6,10). Previous studies have reported symptom duration ranging from 2 weeks to 2 years before diagnosis, with an average exceeding 16 weeks (11,12). Similarly, our patient had persistent ascites for two years that was initially attributed to nephrogenic ascites, illustrating the considerable diagnostic challenge posed by this condition.

The diagnostic process was further complicated by a previous ascitic fluid culture that yielded coagulase-negative Staphylococcus (CoNS), leading to an initial diagnosis of spontaneous bacterial peritonitis. However, CoNS is a common constituent of normal skin flora and is among the microorganisms most frequently encountered as contaminants in clinical microbiology specimens. True CoNS peritonitis is uncommon in patients undergoing maintenance hemodialysis without peritoneal access and is more typically associated with peritoneal dialysis catheter-related infections because of its ability to form biofilms on indwelling devices (13). Current international guidelines define spontaneous bacterial peritonitis by an ascitic fluid polymorphonuclear leukocyte count of ≥ 250 cells/mm³ regardless of culture positivity, whereas culture-positive ascites with a lower PMN count (bacterascites) may represent transient colonization or specimen contamination rather than true infection (14). Therefore, microbiological findings should always be interpreted together with the patient's clinical presentation, ascitic fluid characteristics, and response to antimicrobial therapy. In our patient, persistent ascites despite appropriate antibiotic treatment raised concern for an alternative diagnosis (15).

The development of constitutional symptoms, including prolonged fever and weight loss, together with protein-rich ascites demonstrating mononuclear predominance, further increased the suspicion of tuberculous peritonitis. Although serum adenosine deaminase (ADA) is not recommended as a standalone diagnostic marker for peritoneal tuberculosis and has only moderate sensitivity (sensitivity approaches 58%) (16). Elevated serum ADA levels may reflect enhanced cell-mediated immune activation associated with tuberculosis when interpreted in the appropriate clinical context. In our patient, serum ADA increased from 25.8 U/L to 52.4 U/L over two weeks, supporting the clinical suspicion in conjunction with the overall presentation. Ultimately, the diagnosis was confirmed by Xpert MTB/RIF performed on ascitic fluid, a rapid molecular assay with excellent specificity approaching 100% but only moderate sensitivity (approximately 60–70%) for extrapulmonary tuberculosis, making a positive result highly reliable for establishing the diagnosis (17).

This case underscores the importance of avoiding diagnostic anchoring in patients receiving maintenance hemodialysis with persistent ascites. Although nephrogenic ascites remains the most common diagnosis after exclusion of cirrhosis, clinicians should maintain a high index of suspicion for tuberculous peritonitis in TB-endemic regions, particularly when ascites persists despite appropriate therapy, is characterized by mononuclear cell predominance and high protein content, and is accompanied by constitutional symptoms. Early consideration of tuberculosis and timely use of molecular diagnostic tests such as Xpert MTB/RIF may facilitate earlier diagnosis and treatment, thereby reducing diagnostic delay and improving patient outcomes.

CONCLUSION

Peritoneal TB should be considered in patients with ESRD and persistent or recurrent ascites, particularly when ascitic fluid shows mononuclear predominance and high protein content. Xpert examination can be used to confirm the diagnosis.

REFERENCES

1. WHO. Global tuberculosis report. In: World Health Organization; 2018.
2. Alemu A, Bitew ZW, Diriba G, Seid G, Eshetu K, Chekol MT, et al. Tuberculosis incidence in patients with chronic kidney disease: a systematic review and meta-analysis. International Journal of Infectious Diseases. 2022;122: 188–201. https://doi.org/10.1016/j.ijid.2022.05.046.
3. Al-Efraij K, Mota L, Lunny C, Schachter M, Cook V, Johnston J. Risk of active tuberculosis in chronic kidney disease: A systematic review and meta-analysis. International Journal of Tuberculosis and Lung Disease. 2015;19(12): 1493–1499. https://doi.org/10.5588/ijtld.15.0081.
4. Bikbov B, Purcell C, Levey AS, Smith M, Abdoli A, Abebe M, et al. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet. 2020;395(10225): 709–733. https://doi.org/10.1016/S0140-6736(20)30045-3.
5. Stanifer JW, Muiru A, Jafar TH, Patel UD. Chronic kidney disease in low- and middle-income countries. Nephrology Dialysis Transplantation. 2016;31(6): 868–874. https://doi.org/10.1093/ndt/gfv466.
6. Popa R, Georgescu CC, Caragea DC, Cana-Ruiu D, Ene C, Radu L, et al. Rare Onset of Tubercular Peritonitis Amidst Chronic Renal Dysfunction. Reports. 2023;6(4): 44. https://doi.org/10.3390/reports6040044.
7. Mehershahi S, Haider A, Fortuzi K, Shaikh DH, Patel H. Abdominal Ascites of Unknown Origin: Diagnostic Accuracy of Adenosine Deaminase for Tuberculous Peritonitis. Case Reports in Gastroenterology. 2021;15(1): 418–423. https://doi.org/10.1159/000514892.
8. Tasneem AA, Khan AA, Abbas Z, Luck NH, Hassan SM. Ascites in patients on maintenance hemodialysis: Causes, characteristics and predicting factors. Journal of the College of Physicians and Surgeons Pakistan. 2016;26(5): 413–419. https://doi.org/2326.
9. Roy A, Kulkarni A V. Ascites in patients with end-stage renal disease: Challenges and solutions from diagnosis to management. Hepatology Communications. 2025;9(4). https://doi.org/10.1097/HC9.0000000000000687.
10. Lauwers GY, Mino-Kenudson M, Kradin RL. Infections of The Gastrointestinal Tract. In: Diagnostic Pathology of Infectious Disease. 2018. p. 233–271.
11. Pramugaria ELIAN, Setiawan PB, Purbayu H, Sugihartono T, Maimunah U, Kholili U, et al. Problematic Diagnosis of a Patient with Tuberculosis Peritonitis. In: Proceedings of Surabaya International Physiology Seminar SIPS. 2017. p. 462–469.
12. Walter FM, Emery JD, Mendonca S, Hall N, Morris HC, Mills K, et al. Symptoms and patient factors associated with longer time to diagnosis for colorectal cancer: Results from a prospective cohort study. British Journal of Cancer. 2016;115(5): 533–541. https://doi.org/10.1038/bjc.2016.221.
13. Gruszecka J, Filip R. Epidemiological Study of Pathogens in Spontaneous Bacterial Peritonitis in 2017–2024—A Preliminary Report of the University Hospital in South-Eastern Poland. Microorganisms. 2024;12(5): 1–11. https://doi.org/10.3390/microorganisms12051008.
14. Dawit L, Lee V, Lehoang D, Furey C, Chowdhury A, Mai TA, et al. Clinical Significance of Ascitic Fluid Polymorphonuclear Leukocyte Percentage in Patients with Cirrhosis Without Spontaneous Bacterial Peritonitis. Clinical and Translational Gastroenterology. 2023;14(9): e00614. https://doi.org/10.14309/ctg.0000000000000614.
15. Oey RC, van Buuren HR, de Jong DM, Erler NS, de Man RA. Bacterascites: A study of clinical features, microbiological findings, and clinical significance. Liver International. 2018;38(12): 2199–2209. https://doi.org/10.1111/liv.13929.
16. Crossman L, Ronald Funk C, Kandiah S, Hemrajani R. Disseminated Peritoneal Tuberculosis Initially Misdiagnosed as Nephrogenic Ascites. Case Reports in Nephrology. 2023;2023. https://doi.org/10.1155/2023/4240423.
17. Dahale AS, Puri AS, Kumar A, Dalal A, Agarwal A, Sachdeva S. Tissue Xpert® MTB/RIF Assay in Peritoneal Tuberculosis: To be (Done) or Not to be (Done). Cureus. 2019;11(6). https://doi.org/10.7759/cureus.5009.