EDITORIAL

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Microbiological aspects of peritonitis associated with peritoneal dialysis

Ulises Rodríguez-Medina,* Ulises Rodríguez-Wong**

** Doctor en Ciencias Sociales y Administrativas. Médico Residente de Medicina Interna, University of New Mexico Hospital, USA.*
*** Cirujano Gastroenterólogo y Coloproctólogo. Hospital Ángeles Health System. Maestro en Ciencias de la Salud. Doctor en Ciencias Sociales y Administrativas.*

Aspectos microbiológicos de la peritonitis asociada a diálisis peritoneal

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Peritonitis is one of the most common and serious infectious complications in patients undergoing peritoneal dialysis (PD). Its microbiological etiology is diverse and directly influences the choice of antimicrobial therapy, clinical prognosis, and the feasibility of dialysis treatment. This article reviews the main etiologic agents, mechanisms of infection, and antimicrobial resistance patterns associated with peritonitis in PD.

Peritoneal dialysis is an effective form of renal replacement therapy, but its success can be compromised by recurrent episodes of peritonitis. This complication represents a frequent cause of hospitalization, technique failure, and mortality in patients with chronic kidney disease (CKD) on PD.[1] Up-to-date knowledge of microbiological aspects is essential to optimize the prevention and treatment of peritonitis in this setting. Approximately 60-80% of PD peritonitis cases are caused by gram-positive bacteria, with coagulase-negative staphylococci (such as *Staphylococcus epidermidis)* and *Staphylococcus aureus* being the main culprits.[2] These microorganisms usually originate from the skin flora of the patient or healthcare personnel, and their transmission is related to catheter manipulation or errors in aseptic technique.

Gram-negative bacteria, primarily *Escherichia coli, Klebsiella spp.,* and *Pseudomonas aeruginosa,* are responsible for 15-30% of cases and are commonly associated with enteric infections or retrograde catheter contamination.[3] Gram-negative peritonitis tends to have a more aggressive course and a worse prognosis.

To a lesser extent, fungal infections (usually *Candida spp.),* non-tuberculous mycobacteria, and other unusual pathogens have been identified, particularly in immunocompromised patients or after multiple courses of antibiotics.[4]

The main mechanisms of infection include:

a) Intraluminal contamination during dialysate exchange.
b) Transmural migration from the gastrointestinal tract, especially in patients with constipation or diarrhea.
c) Hematogenous spread from distant foci of infection.
d) Contamination by tunneling or catheter exit, which can progress to peritonitis if not treated promptly.[5]

Diagnosis is established by clinical evaluation, peritoneal fluid analysis (cell count and cytology), and culture. The International Society for Peritoneal Dialysis (ISPD) recommends the use of direct inoculation methods of fluid into blood culture bottles to increase diagnostic yield.[6] Despite technical improvements, 10-20% of episodes are classified as "culture-negative" peritonitis, which represents a significant therapeutic challenge.

Antimicrobial resistance to the pathogens that cause peritonitis in PD is a growing concern. An increase in the prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA) and extended-spectrum beta-lact-

Correspondencia:

Dr. Ulises Rodríguez-Medina
Río Bamba Núm. 639-330. Col. Magdalena de Las Salinas. C.P. 07760, Alcaldía Gustavo A. Madero, CDMX, México
Tel.: 55 5754-8504. Correo electrónico: som514_ulises@hotmail.com

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amase-producing gram-negative bacilli has been observed.[7] This requires adjusting empirical regimens and emphasizes the need for local microbiological monitoring.

Initial empirical treatment should cover both gram-positive and gram-negative bacteria and be adjusted according to the antibiogram. The ISPD suggests the use of vancomycin or a first-generation cephalosporin plus a third-generation cephalosporin or an aminoglycoside as initial treatment.[6]

Accurate microbiological identification allows for tailoring therapy, reducing toxicity, and improving clinical prognosis.

A detailed understanding of the microbiological aspects of peritoneal dialysis-associated peritonitis is essential for its proper management. Continuous monitoring of etiological patterns and local sensitivity is key to optimizing empirical therapy and preventing long-term complications.

REFERENCES

1. Li PK, Chow KM, Cho Y, Fan S, Figueiredo AE, Harris T*, et al.* ISPD peritonitis guideline recommendations: 2022 update on prevention and treatment. *Perit Dial Int* 2022; 42(2): 110-53. doi: 10.1177/08968608221080586
2. Brown MC, Simpson K, Kerssens JJ, Mactier RA; Scottish Renal Registry. Peritoneal dialysis-associated peritonitis rates and outcomes in a national cohort are not improving in the post-millennium (2000-2007). *Perit Dial Int* 2011; 31(6): 639-50. doi: 10.3747/pdi.2010.00185
3. Boudville N, Kemp A, Clayton P, Lim W, Badve SV, Hawley CM, et al. Recent peritonitis associates with mortality among patients treated with peritoneal dialysis. *J Am Soc Nephrol* 2012; 23(8): 1398-405. doi: 10.1681/ASN.2011121135
4. Prasad N, Gupta A. Fungal peritonitis in peritoneal dialysis patients. *Perit Dial Int* 2005; 25(3): 207-22.
5. dos Santos ACML, Hernandes RT, Montelli AC. *et al.* Clinical and microbiological factors predicting outcomes of nonfermenting gram-negative bacilli peritonitis in peritoneal dialysis. *Sci Rep* 2021; 11: 12248. https://doi.org/10.1038/s41598-021-91410-0
6. Li PK, Szeto CC, Piraino B, de Arteaga J, Fan S, Figueiredo AE*, et al.* ISPD Peritonitis Recommendations: 2016 Update on Prevention and Treatment. *Perit Dial Int* 2016; 36(5): 481-508. doi: 10.3747/pdi.2016.00078
7. Al Sahlawi M, Bargman JM, Perl J. Peritoneal Dialysis-Associated Peritonitis: Suggestions for Management and Mistakes to Avoid. *Kidney Med* 2020; 2(4): 467-75. doi: 10.1016/j.xkme.2020.04.010