

Chyloperitoneum secondary to the use of calcium channel blockers in a peritoneal dialysis patient: Case report.

Received: January 8, 2026.

Accepted: February 7, 2026.

Published: February 7, 2026.

Editor: Dr. Franklin Mora.


How to cite:

Ortega H, Toral C, Ugalde A, Argudo D, Cordero A. Chyloperitoneous secondary to the use of calcium channel blockers in a peritoneal dialysis patient: Case report. REV SEN 2026; 14(1):120-129.

DOI: <http://doi.org/10.56867/162>






Sociedad Ecuatoriana de Nefrología, Diálisis y Trasplantes.

ISSN-L: 2953-6448

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Abstract

Introduction: Hypertension is one of the most prevalent diseases worldwide, especially in patients with advanced kidney disease receiving renal replacement therapy. Antihypertensive treatment in this patient group generally involves drug combinations that include calcium channel blockers, which, although effective, can be associated with adverse effects such as chyloperitoneum.

Case report: We present the case of a 45-year-old female patient with a history of hypertension and stage V chronic kidney disease who was receiving continuous ambulatory peritoneal dialysis for renal replacement therapy. Following modifications to her antihypertensive treatment, lercanidipine was added to her treatment in combination with valsartan. After this, she presented with cloudy, thick, low-volume drainage. The presence of triglycerides at elevated values was identified in the sample, and it was decided to discontinue the drug. She showed improvement in the following days.

Discussion: Chyloperitoneum is a rare clinical entity, with an estimated incidence of up to 1 in 20,000 cases, characterized by the accumulation of milky peritoneal fluid with triglyceride levels exceeding 200 mg/dL. Its pathogenesis is associated with lymphatic drainage dysfunction, a condition that can be exacerbated by calcium channel blockers, such as lercanidipine, which increase lymphatic pressure through vasodilation. Unlike infectious peritonitis, this condition lacks inflammatory criteria or positive cultures, as evidenced in our patient (triglycerides: 264 mg/dL). Fundamental management consists of the immediate discontinuation of the implicated drug, which usually allows for clinical and laboratory resolution within 24 to 72 hours after drug withdrawal.

Conclusion: Chyloperitoneum due to calcium channel blockers is an uncommon condition that is often mistaken for peritonitis. Definitive diagnosis through biochemical and cellular analysis of peritoneal fluid allows for resolution after the discontinuation of the drug. Correct identification avoids invasive procedures, unnecessary treatments, and additional costs, ensuring efficient and safe clinical management for the patient.

Keywords: Peritoneal dialysis, chyloperitoneum, arterial hypertension.

Introduction

Peritoneal dialysis is the second most commonly used renal replacement therapy after hemodialysis. It is a continuous process designed to remove solutes and excess fluid through bidirectional exchange between capillary blood and the dialysate, which is introduced into the peritoneal cavity via a permanent catheter [1-3]. This transport mechanism is made possible by the semipermeable properties of the peritoneal membrane [4]. However, volume management often presents a significant clinical challenge in this modality. Ultrafiltration depends directly on the osmotic gradient of the dialysate, typically using solutions with glucose concentrations of 1.5%, 2.5% or 4.25%, depending on the patient's needs and the functional status of the membrane. Chronic exposure to glucose—the main osmolar agent used—induces varying degrees of peritoneal fibrosis, which compromises transport capacity [5]. As a result, hypervolemia and high blood pressure are frequent complications in these patients [5, 6].

Hypertension (HTN) is defined as a persistent elevation of blood pressure, with diagnostic thresholds that vary according to international guidelines: >130/80 mmHg according to American guidelines and >140/90 mmHg according to the European consensus [7, 8]. In patients with stage V chronic kidney disease (CKD) on dialysis, the prevalence of HTN is high, driven primarily by fluid overload and sodium retention [8]. Other contributing factors include increased arterial stiffness, overactivation of the renin–angiotensin–aldosterone system (RAAS) and the sympathetic nervous system, and the use of erythropoiesis-stimulating agents [9]. Pharmacological management in this population aims not only to control blood pressure but also to minimize side effects and provide cardiovascular protection. The therapeutic arsenal is based mainly on RAAS inhibitors, β -adrenergic blockers and calcium channel blockers (CCBs) [9, 10]. Despite their effectiveness, CCBs have been associated with uncommon but significant adverse effects, such as chyloperitoneum, which is defined as the accumulation of lymph in the abdominal cavity [11, 12].

Chyloperitoneum associated with calcium channel blocker (CCB) use is a rare condition, with an estimated prevalence of 1 in 20,000 cases. Although the exact pathophysiology remains unclear, it is hypothesized that their lipophilic nature enables them to interfere with calcium channels in the smooth muscle cells of lymphatic vessels and the gastrointestinal sphincter, thereby altering lymphatic flow [7, 11]. Owing to effluent turbidity, this condition is often misdiagnosed as infectious peritonitis, leading to unnecessary antibiotic use. However, the diagnosis is confirmed by quantifying triglycerides in the peritoneal fluid and the absence of criteria for infection [6, 12]. We describe the case of a patient who developed chyloperitoneum after starting calcium channel blockers as part of her antihypertensive regimen.

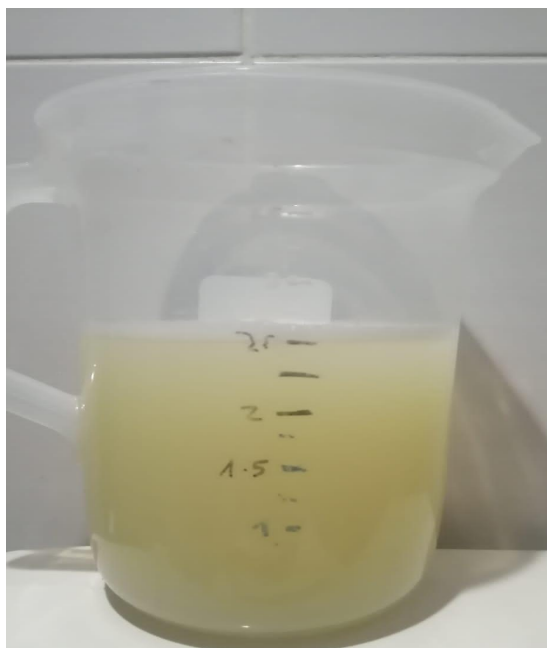
Case report

Medical records

This is a 45-year-old woman with a medical history of chronic hypertension and stage V chronic kidney disease since approximately age 28. She initially received renal replacement therapy via hemodialysis for 1 year and 4 months. Subsequently, at age 30, she received a kidney transplant from a deceased donor. Four years later, at age 35, she developed chronic allograft nephropathy, requiring progressively increasing immunosuppression, associated infections, and a renal abscess in the transplanted kidney. She then experienced a high-risk pregnancy, and as a result, she required renal replacement therapy via peritoneal dialysis, which she continued to receive.

She was treated for hypertension with 100 mg losartan orally once daily (7 a.m.), 10 mg amlodipine orally once daily (7 p.m.), and 12.5 mg carvedilol orally every 12 hours (7 a.m.–7 p.m.). During her clinical course, her treatment required adjustments, eventually reaching valsartan 160 mg, amlodipine 5 mg, and carvedilol 25 mg orally daily. In June 2025, the patient experienced a hypertensive crisis despite maintaining her regular treatment, so her antihypertensive therapy was modified again to valsartan 320 mg orally daily plus lercanidipine 10 mg orally daily. In the two days following the start of lercanidipine therapy, she presented with decreased peritoneal ultrafiltration, which was described as cloudy and thick ([Figure 1](#)).

Figure 1. Peritoneal fluid during the time the patient was on Lercanidipine-based treatment.



Diagnostic tests

In the absence of additional symptoms or abdominal pain, a peritoneal fluid study is performed for cytology, Gram and culture, with the following findings: leukocytes, 1 cell \times mm; peritoneal

fluid culture, negative; Gram, without the presence of germs; and triglyceride analysis, which is performed in the peritoneal fluid, where elevated values (264 mg/dL) are present.

Evolution

Owing to the absence of additional clinical findings, plus laboratory results and the patient's recent history (modifications in treatment), the lercanidipine dose was discontinued, and an amlodipine dose of 10 mg orally every night plus valsartan 320 mg orally at 7 am was started, resulting in an increase in the ultrafiltrate in the following days, with the presence of clear and transparent peritoneal fluid ([Figure 2](#)).

Figure 2. Peritoneal fluid sample after discontinuing Lercanidipine.



Discussion

Hypertension is a common comorbidity in patients with end-stage renal disease undergoing dialysis, with an estimated prevalence between 72% and 88%. The coexistence of both conditions significantly increases cardiovascular risk and mortality [13]. Many of these patients require polypharmacy to control hypertension when initial treatment is insufficient [5].

On the other hand, chyloperitoneum is a rare condition characterized by the presence of milky peritoneal fluid due to the accumulation of triglycerides in the peritoneal cavity [14, 15]. Its incidence is unknown, but it is estimated to occur in 1 in 20,000 to 187,000 cases [13, 16]. The mechanism by which triglycerides accumulate is still unknown; however, it is thought to be due to decreased lipid degradation in the peritoneum or reduced lymphatic drainage, primarily due to

peritoneal membrane dysfunction. Furthermore, the use of calcium channel blockers can exacerbate this condition by dilating lymphatic vessels and increasing internal pressure [17]. On average, this adverse effect appears approximately 38 days after starting treatment [18], although in the case presented here, the symptoms arose a few days after beginning treatment with lecanidipine.

For the diagnosis of chyloperitoneum, the triglyceride content of the peritoneal fluid should be analyzed; levels above 200 mg/dL are usually indicative of the presence of this condition. Other markers are also evaluated, such as a leukocyte count greater than 500, total protein between 2.5 and 7.0 g/dL [14], a serum-ascites albumin gradient less than 1.1 g/dL, LDH levels between 110 and 200 IU/L, positive cultures in some cases of tuberculosis, positive cytology in the presence of cancer, and high amylase in pancreatitis [14], although these are not always present, as in the study by Biais et al., where the peritoneal fluid showed 225.8 mg/dL triglycerides without other plasma alterations [19]. Similar to our findings, the presence of elevated triglycerides (264 mg/dl) and the absence of markers indicating a current infectious process led to the dismissal of a diagnosis of peritonitis, as the following clinical and laboratory criteria were not met: abdominal pain, cloudy fluid, leukocytosis in the effluent and a positive culture [5].

Bhardwaj suggested that, when chylous ascites associated with calcium channel blocker use is suspected, the initial management should be discontinuation of the medication. Joubran et al. support this recommendation, noting that chyloperitoneum induced by these drugs usually resolves within 24 hours of withdrawal [20]. This pattern was also observed in our patient, whose symptoms improved significantly between 24 and 72 hours after treatment was discontinued.

Conclusions

Chyloperitoneum associated with calcium channel blockers is considered an uncommon clinical condition and is frequently misdiagnosed as peritonitis. The clinical presentation and analysis of peritoneal fluid, including biochemical and cell count measurements, help establish a definitive diagnosis. The condition resolves upon discontinuation of treatment, thus avoiding unnecessary, costly, and invasive diagnostic procedures and treatments.

Abbreviations

LDH: lactate dehydrogenase.
HTA: high blood pressure.
CKD: chronic kidney disease.

Supplementary information

The supplementary materials have not been included.

Acknowledgments

Not declared.

Authors' contributions

Hugo Ortega Durán: Conceptualization, data curation, research, visualization, original draft writing.

César Toral Chacón: conceptualization, data curation, research, visualization and writing of the original draft.

Alejandro Ugalde Noritz: Conceptualization, formal analysis, methodology, project management, resources, software, supervision, validation, writing, review and editing.

Diego Argudo Sánchez: Conceptualization, data curation, research, visualization and writing of the original draft.

Adriana Cordero Neira: conceptualization, visualization and writing of the original draft

All the authors read and approved the final version of the manuscript.

Financing

The study was self-funded by the authors.

Availability of data or materials

Not applicable.

Statements

Ethics committee approval and consent to participate

Clinical cases are not needed.

Consent for publication

The authors have the patient's authorization to publish the images and the clinical case.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Use of generative AI

The authors declare that they did not use generative AI in this document.

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