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THERAPEUTIC APPROACH TO UREMIC PERICARDITIS

ABORDAGEM TERAPÊUTICA DA PERICARDITE URÊMICA

ABORDAJE TERAPÉUTICO DE LA PERICARDITIS URÉMICA

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ABSTRACT

Uremic pericarditis is an inflammatory condition affecting the pericardium in patients with severe renal dysfunction, particularly those with end-stage renal disease. Toxic metabolic byproducts accumulate in the bloodstream, triggering inflammation of this fibroelastic sac. Common clinical features include positional chest pain, a pericardial friction rub, and, in advanced cases, signs of cardiac tamponade. Uremic pericarditis is currently rare due to improved hemodialysis practices, but its decreasing frequency may contribute to delayed diagnosis. Early echocardiographic evaluation is essential in suspected cases to identify pericardial effusion or hemodynamic compromise. Although most patients improve with intensification of dialysis, some require surgical intervention, such as pericardiocentesis or the creation of a pericardial window, to relieve tamponade or obtain tissue for diagnostic purposes.

Keywords: Uremic Pericarditis. Hemodialysis. Chronic Kidney Disease. Cardiac Tamponade. Uremic Toxins. Pericardial Inflammation.

RESUMO

A pericardite urêmica é uma condição inflamatória que afeta o pericárdio em pacientes com disfunção renal grave, particularmente aqueles com doença renal terminal. Subprodutos metabólicos tóxicos se acumulam na corrente sanguínea, provocando inflamação desse saco fibroelástico. As características clínicas comuns incluem dor

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torácica posicional, atrito pericárdico e, em casos avançados, sinais de tamponamento cardíaco. A pericardite urêmica é rara atualmente devido às práticas aprimoradas de hemodiálise, mas sua frequência decrescente pode contribuir para o diagnóstico tardio. A avaliação ecocardiográfica precoce é fundamental em casos suspeitos para identificar derrame pericárdico ou comprometimento hemodinâmico. Embora a maioria dos pacientes melhore com a intensificação da diálise, alguns necessitam de intervenção cirúrgica, como pericardiocentese ou janela pericárdica, para aliviar o tamponamento ou obter tecido para diagnóstico.

Palavras-chave: Pericardite Urêmica. Hemodiálise. Doença Renal Crônica. Tamponamento Cardíaco. Toxinas Urêmicas. Inflamação Pericárdica.

RESUMEN

La pericarditis urémica es una condición inflamatoria que afecta al pericardio en pacientes con disfunción renal grave, particularmente en aquellos con enfermedad renal terminal. Los subproductos metabólicos tóxicos se acumulan en el torrente sanguíneo, provocando inflamación de este saco fibroelástico. Las manifestaciones clínicas comunes incluyen dolor torácico posicional, roce pericárdico y, en casos avanzados, signos de taponamiento cardíaco. En la actualidad, la pericarditis urémica es poco frecuente debido a las prácticas mejoradas de hemodiálisis, aunque su frecuencia decreciente puede contribuir a un diagnóstico tardío. La evaluación ecocardiográfica temprana es fundamental en los casos sospechosos para identificar derrame pericárdico o compromiso hemodinámico. Aunque la mayoría de los pacientes mejora con la intensificación de la diálisis, algunos requieren intervención quirúrgica, como la pericardiocentesis o la creación de una ventana pericárdica, para aliviar el taponamiento o para obtener tejido con fines diagnósticos.

Palabras clave: Pericarditis Urémica. Hemodiálisis. Enfermedad Renal Crónica. Taponamiento Cardíaco. Toxinas Urémicas. Inflamación Pericárdica.

1 INTRODUCTION

The pericardium consists of visceral and parietal layers separated by a potential space containing a small volume of lubricating fluid. (Rhabneh et al., 2025). Pericarditis has several etiologies, and can originate from a viral infection, the result of autoimmune processes, originating from tumor or metabolic processes, as in uremic pericarditis (Bhabneh et al., 2025).

Cardiovascular complications are the main cause of mortality in patients with chronic kidney disease (CKD). Among these complications, uremic pericarditis stands out as a relevant inflammatory manifestation, usually associated with advanced stages of CKD or the inadequacy of dialysis therapy.

Uremic pericarditis can occur before dialysis is started or even within the first eight weeks of establishment, especially in cases of subdialysis. Although its incidence has decreased with the improvement of dialysis techniques, the condition remains clinically relevant due to the risk of progression to cardiac tamponade and constrictive pericarditis (Peride et al., 2025; IMAZIO; ADLER, 2013).

Uremic pericarditis is a severe inflammatory manifestation that affects the pericardial sac due to severe renal dysfunction, and is frequently observed in patients with end-stage renal disease (ESRD) or severe azotemia who may have undergone inadequate dialysis therapy. As a consequence, the accumulation of uremic toxic metabolites that release pro-inflammatory markers and result in fibrous deposits in the pericardium and consequently uremic pericarditis may occur (Rhabneh et al., 2025; Peride et al., 2025).

Although its prevalence has declined significantly due to the modernization of renal replacement therapy techniques, the condition still has considerable incidence rates, ranging between 5% and 20% in dialysis populations (Badwan et al., 2023; Peride et al., 2025).

From a pathophysiological point of view, uremic pericarditis results from a systemic pro-inflammatory state triggered by uremic toxin retention, metabolic alterations, and fluid and electrolyte imbalances characteristic of advanced renal failure. Although the exact mechanisms have not yet been fully elucidated, evidence points to the activation of cytokine-mediated inflammatory pathways, such as interleukin-1, interleukin-6, and tumor necrosis factor-alpha, as well as oxidative stress and endothelial dysfunction, which promote progressive pericardial inflammation and favor the formation of pericardial

effusion, which is often voluminous and fibrinous (Rhabneh; Rout, 2025; Peride et al., 2025). Clinically, patients manifest positional chest pain, pericardial friction rub and, in critical situations, cardiac tamponade (Nabalawi, 2025). Proper management is essential, as late identification can lead to fatal complications or progression to constrictive pericarditis (Peride et al., 2025).

Recent studies suggest that uremic pericarditis may be related not only to the accumulation of nitrogenous metabolites, but also to the activation of inflammatore-mediated immune responses and interleukin-1 (IL-1)-dependent inflammatory cascades. This mechanism contributes to the fibrinous pericardial inflammation characteristic of the condition and provides pathophysiological basis for the use of IL-1-targeted therapies in refractory cases, such as anakinra (Nabalawi, 2025).

The diagnosis of uremic pericarditis is based on the correlation between the clinical presentation and laboratory and imaging findings in patients with uremia. The electrocardiogram may not show the classic alterations of acute pericarditis, such as ST-segment elevation or diffuse PR-segment depression, in addition to ST-segment depression and PR interval elevation in the AVR lead (ESC, 2015).

In this sense, the diagnosis is made when two of the four criteria are met: acute pleuritic chest pain that improves when sitting and leaning forward; pericardial friction; new generalized ST elevation or depression of the PR on the ECG; pericardial effusion (SILVA, B. M. et al. 2022).

Transthoracic echocardiography is the test of choice for diagnostic evaluation and follow-up, allowing the identification of the presence, volume, and hemodynamic impact of pericardial effusion. Laboratory tests often show elevation of urea and creatinine, as well as nonspecific inflammatory markers.

Despite the advancement of renal replacement therapies and the greater efficiency of dialysis techniques, uremic pericarditis remains clinically relevant due to the potential for progression to serious complications, such as cardiac tamponade and constrictive pericarditis, especially in contexts of late diagnosis or inadequate dialysis (Badwan et al., 2023; Peride et al., 2025). In addition, the clinical presentation may be atypical when compared to other etiologies of pericarditis, with a lower frequency of classic electrocardiographic alterations, which hinders early recognition of the condition. Thus, an in-depth understanding of its pathophysiological mechanisms, clinical manifestations, and therapeutic strategies becomes essential to optimize management and reduce

associated morbidity and mortality in patients with advanced renal disease. In view of this scenario, the present study aims to synthesize the current therapeutic approaches for uremic pericarditis, highlighting the fundamental role of dialysis and adjuvant pharmacological therapies.

2 METHODOLOGY

The present work is a bibliographic review of a narrative nature, elaborated with the aim of consolidating and examining contemporary scientific information about the "Therapeutic Approach to Uremic Pericarditis". For the theoretical foundation, a bibliographic survey was carried out in specialized databases, such as PubMed and StatPearls, using the search terms "Uremic Pericarditis" and "Therapy", structured according to the MeSH terminology and interconnected by Boolean operators. The selection criteria established included full-text studies, published in the last five years, written in English or Portuguese, that had direct relevance to the central theme. Duplicate publications, editorials without methodological rigor, and articles that did not specifically address the clinical management of the condition were discarded. The content analysis was segmented into two phases: the initial screening of abstracts and titles, followed by the thorough examination of the articles selected for the extraction of evidence. The data obtained were synthesized and organized in a descriptive manner to compose the body of the text.

3 RESULTS AND DISCUSSION

The primary and unquestionable therapeutic strategy for uremic pericarditis is the establishment or intensification of hemodialysis (Nabalawi, 2025; Rhabneh et al., 2025). The central goal of this intervention is the accelerated removal of toxic metabolites and the correction of fluid and electrolyte balance, which usually promotes symptom resolution in approximately 87% of cases after two weeks of intensive treatment (Badwan et al., 2023). In this context, the response to dialysis intensification may vary according to specific clinical characteristics. Badwan et al. (2023) describe the presence of systolic hypotension, leukocytosis, persistent fever, and voluminous pericardial effusions as predictors of treatment failure, which require close clinical surveillance and early consideration of invasive interventions. In addition, due to platelet dysfunction and the higher hemorrhagic risk in uremic patients, systemic anticoagulation should be avoided

whenever possible, especially in the presence of pericardial effusion. In cases refractory to conventional hemodialysis, the transition to peritoneal dialysis may represent a viable therapeutic alternative, and caution is also required in ultrafiltration in patients with large effusions, in order to avoid compromising cardiac filling. The use of advanced imaging methods can help in the evaluation of inflammatory activity and in the direction of treatment in selected situations. In patients with severe azotemia and with severe complications of uremia - symptomatic pericardial effusion, encephalopathy, severe refractory acidosis - dialysis should be started slowly in order to avoid the occurrence of the imbalance syndrome. In patients with larger pericardial effusions, dialysis should be performed carefully and with monitoring of hemodynamic status, to avoid inadequate cardiac filling (Badwan et al., 2023). A daily dialysis regimen for a period of 10 to 14 days is often recommended to ensure adequate clearance (Rhabneh et al., 2025; Peride et al., 2025). Evidence from recent clinical reports indicates that, when conducted appropriately and for a long time, intensification of hemodialysis for a period of up to two to three weeks may be sufficient to promote complete regression of pericardial effusion and resolution of symptoms, without the need for invasive intervention. The absence of clinical response after this interval should raise the suspicion of treatment failure and indicate early reassessment of management, including consideration of pericardiocentesis (Nabalawi, 2025). It is important to distinguish uremic pericarditis, which occurs before or up to eight weeks after the start of renal replacement therapy, from pericarditis associated with dialysis, which appears after this period and usually indicates insufficient dialysis or failure to adhere to treatment (Badwan et al., 2023; Peride et al., 2025). In this sense, the distinction between uremic pericarditis and dialysis-associated pericarditis has relevant clinical and therapeutic implications. While uremic pericarditis tends to occur before or within the first eight weeks after initiation of renal replacement therapy, the form associated with dialysis often reflects inadequacy of dialysis treatment, either due to insufficient clearance, adherence failures, or vascular access dysfunctions (Badwan et al., 2023; Peride et al., 2025). Recent studies reinforce that, in both scenarios, dialysis intensification is the initial therapeutic pillar, but the persistence of the inflammatory process after adequate optimization suggests a higher risk of complications, such as voluminous pericardial effusion and progression to constrictive pericarditis, requiring a more aggressive approach and rigorous monitoring (Rhabneh; Rout, 2025; Peride et al., 2025). In this context, pericarditis associated with dialysis is strongly related to the

inadequacy of dialysis treatment, either due to reduced clearance efficiency, shortening of sessions, or failures in adherence, resulting in persistent accumulation of uremic toxins. According to Peride et al. (2025), this scenario promotes a sustained inflammatory state, mediated by the release of pro-inflammatory cytokines, such as interleukin-1, interleukin-6, and tumor necrosis factor-alpha, in addition to oxidative stress and endothelial dysfunction, contributing to progressive pericardial inflammation, voluminous effusion, and increased risk of progression to cardiac tamponade or constrictive pericarditis.

It is from this perspective that the failure of the clinical response to dialysis intensification is associated with specific prognostic markers, which should alert to the need for early reassessment of the therapeutic strategy. Badwan et al. (2023) highlight that persistent hypotension, leukocytosis, high fever, and extensive pericardial effusions are related to a lower rate of clinical resolution with dialysis therapy alone. In addition, uremic patients have intrinsic platelet dysfunction and a higher risk of bleeding, which favors the occurrence of hemorrhagic effusions and limits the use of systemic anticoagulation, especially in the presence of significant pericardial effusion, reinforcing the need for serial clinical and echocardiographic surveillance in these cases.

In cases where symptoms persist despite optimal dialysis, pharmacological therapies become necessary. In severe uremic pericarditis with voluminous pericardial effusion, failure of clinical response after repeated intensive hemodialysis sessions for 7 to 14 days is associated with a higher risk of cardiac tamponade, and pericardiocentesis is indicated early in these cases. The presence of hemodynamic instability is an absolute indication for immediate drainage of the pericardial space (Nabalawi, 2025). The use of nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin and indomethacin, may aid in pain relief, although their efficacy in definitively eliminating uremic inflammation is limited and the risk of renal toxicity should be monitored in patients with residual function (Nabalawi, 2025; Rhabneh et al., 2025). Low-dose corticosteroids may be used in patients who cannot take nonsteroidal anti-inflammatory drugs (Rhabneh et al., 2025) (Lucas Seabra ADDED). Corticosteroids should be given in conjunction with colchicine when indicated. In addition, low-to-moderate dose regimens (prednisone 0.2-0.5 mg/kg/day) are more indicated than high-dose regimens (prednisone 1.0 mg/kg/day) (Peride et al., 2025). Colchicine is recommended in low, weight-adjusted doses to mitigate the risk of recurrence, but its use is restricted in cases of severe renal failure (Peride et al., 2025; Badwan et al., 2023). Emerging biologic therapies, specifically

interleukin-1 (IL-1) blockers, such as anakinra and rilonacept, have shown promising results in the management of refractory cases, significantly reducing pericardial inflammation and edema (Badwan et al., 2023; Peride et al., 2025), in addition to greater pain control, when associated with physiotherapy and low-dose opioids (Nabalawi et al,. 2025). Recent reports also highlight the role of anakinra not only in controlling refractory pericardial inflammation, but also in significantly improving pain in patients with associated metabolic diseases, when combined with non-pharmacological strategies, such as physical therapy, and the judicious use of low-dose opioids. This integrated approach may reduce the need for non-steroidal anti-inflammatory drugs, the use of which is particularly deleterious in patients with advanced chronic kidney disease (Nabalawi, 2025). The selection of drugs that will be used is a careful process, and should be done based on the patient's medical history in search of useful information for treatment, such as contraindications and comorbidities, risk of drug interaction, and previous treatments. (Peride et al., 2025). In this regard, for example, in patients who need antiplatelet therapy, the use of aspirin for the management of pericarditis may be more indicated than the use of other NSAIDs (Peride et al., 2025). It should also be emphasized that the use of pharmacological therapies in uremic pericarditis should be individualized considering the stage of renal disease and the patient's risk profile. Although non-steroidal anti-inflammatory drugs can be used for symptomatic pain control, their efficacy alone is limited and their use should be judicious due to the potential for residual renal toxicity (Badwan et al., 2023; Rhabneh; Rout, 2025). Colchicine, despite reducing recurrences in other forms of pericarditis, has important restrictions in patients with advanced renal failure, especially in patients with creatinine clearance less than 30mL/minute, and should be administered in reduced doses or avoided in selected cases (Peride et al., 2025). In refractory situations, biological therapies targeting interleukin-1, such as anakinra, emerge as promising alternatives, especially in persistent inflammatory conditions, although they still lack greater robustness of evidence in uremic populations (Badwan et al., 2023; Peride et al., 2025).

Management of acute complications requires more invasive interventions. Cardiac tamponade, which affects between 10% and 20% of dialysis patients with pericarditis, requires emergency pericardiocentesis (Peride et al., 2025). Pericardiocentesis is ideal in cases where pericardial effusion becomes symptomatic or when anti-inflammatory therapy is not effective. When related to drainage, it facilitates the approximation of the

pericardial layers, reducing the probability of recurrence (Peride et al., 2025). In situations of bulky and recurrent effusions, the creation of a pericardial window or, less frequently, pericardiectomy are viable surgical options to prevent chronic constriction (Badwan et al., 2023; Rhabneh et al., 2025). In addition, advanced imaging methods have taken on an increasing role in the evaluation of pericardial inflammatory activity and in the therapeutic direction in selected cases of uremic pericarditis. Although echocardiography remains the fundamental initial test for detecting pericardial effusion and signs of tamponade, cardiac magnetic resonance imaging and computed tomography allow for a more accurate characterization of pericardial thickening, edema, and active inflammation, aiding in prognostic stratification and therapeutic decision-making in refractory conditions (Badwan et al., 2023; Peride et al., 2025). This multimodal approach contributes to avoiding delays in the indication of invasive procedures and to personalizing clinical management.

Uremic pericarditis has relevant clinical and diagnostic particularities that help differentiate it from other etiologies of pericarditis. Unlike viral or idiopathic forms, classic electrocardiographic alterations, such as diffuse ST-segment elevation and PR-segment depression, are uncommon, since the inflammatory process does not directly involve the epicardium. Pericardial effusion associated with uremic pericarditis tends to be bulky, often rich in fibrin, and in some cases hemorrhagic, which explains the greater propensity to develop cardiac tamponade. Although pericardiocentesis is indicated as an emergency measure in the face of hemodynamic instability, this intervention does not constitute a definitive treatment, and the correction of the underlying cause through the adequacy of dialysis therapy is the main determinant of the resolution of the condition. The persistence of the inflammatory state can culminate in fibrosis and pericardial thickening, with progression to constrictive pericarditis, reinforcing the importance of early recognition of the condition and immediate optimization of dialysis (Rhabneh et al., 2025). Specific cases, such as tumor lysis syndrome presenting as uremic pericarditis, require vigorous hydration and strict uric acid control (Emidio et al., 2023). Additionally, the differential diagnosis is crucial, since viral etiologies, such as SARS-CoV-2 infection, can mimic uremic pericarditis in renal patients, requiring different treatment protocols (Silva et al., 2022). In addition, it is essential to emphasize the differential diagnosis of pericarditis in patients with advanced renal disease, since not all pericardial inflammatory conditions in this context are of uremic origin. Silva et al. (2022) reported a case of acute pericarditis associated with SARS-CoV-2 infection in a patient on peritoneal dialysis, highlighting that

the presence of typical electrocardiographic alterations, such as diffuse ST-segment elevation and PR-segment depression, associated with good dialysis adequacy (Kt/V > 1.7) and the absence of pericardial effusion, makes uremic etiology less likely. Unlike uremic pericarditis, in which electrocardiographic abnormalities are uncommon due to the absence of epicardial involvement, viral pericarditis often presents with such findings. These data reinforce the need for careful evaluation, since the therapeutic approach differs substantially between etiologies, avoiding both unnecessary intensification of dialysis and delay in appropriate anti-inflammatory treatment.

4 CONCLUSION

Uremic pericarditis is a relevant complication in patients with impaired renal function, and may, in less usual presentations, represent the initial manifestation of severe metabolic disorders, such as tumor lysis syndrome (TLS) (EMIDIO et al., 2023). This context reinforces the importance of systematic clinical evaluation and early recognition of the underlying etiology, since the diagnostic delay can significantly compromise the clinical evolution and prognosis of the patient.

The analysis of the literature shows that uremic pericarditis, although less frequent in the modern dialysis era, remains associated with high morbidity, especially when related to dialysis inadequacy or late recognition of the condition. Early intensification of renal replacement therapy is the main therapeutic measure and, in most cases, promotes satisfactory clinical resolution. However, the persistence of the inflammatory process, the presence of bulky pericardial effusions, and hemodynamic instability require additional interventions, including adjuvant pharmacological therapies and invasive procedures, highlighting the need for an individualized and multidisciplinary approach (Badwan et al., 2023; Rhabneh; Rout, 2025; Peride et al., 2025).

The therapeutic approach to uremic pericarditis should prioritize the treatment of the associated metabolic cause, with an emphasis on intensive intravenous hydration, correction of hydroelectrolytic disturbances, and strict control of hyperuricemia, fundamental measures for the recovery of renal function and for the resolution of the pericardial inflammatory process (EMIDIO et al., 2023). Although rasburicase is considered the drug of choice in the management of TLS, the use of allopurinol may represent a viable alternative in scenarios of unavailability, as long as it is associated with continuous clinical and laboratory monitoring.

The differential diagnosis between uremic pericarditis and pericarditis associated with dialysis should be made, excluding aortic and coronary syndromes and cardiac tamponade, in this way, dialysis treatment is intensified or started based on etiology, considering the use of anti-inflammatory drugs or corticosteroids based on the response to dialysis treatment, always observing a possible worsening of the patient's renal function due to the use of these drugs. (Badwan et al., 2023)

In addition, contemporary reports reinforce that uremic pericarditis remains a potentially fatal entity, even in the modern dialysis era, especially when associated with inadequate pain management and long-term use of nonsteroidal anti-inflammatory drugs. Early recognition of clinical signs, timely intensification of dialysis, and consideration of immunomodulatory therapies in selected cases are crucial for the prevention of severe complications and recurrence of the condition (Nabalawi, 2025).

Additionally, in patients with chronic kidney disease, the differential diagnosis of pericarditis should be carefully considered, since other etiologies, such as viral pericarditis associated with SARS-CoV-2 infection, may coexist or mimic the clinical picture (SILVA et al., 2022). Even so, the early identification of uremic pericarditis and the implementation of a therapeutic approach directed at the etiology remain decisive for the reduction of complications and the improvement of clinical outcomes.

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