

RENAL COMPLICATIONS OF GOUT: A SYNTHESIS OF CURRENT EVIDENCE

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Background. Gout, a chronic inflammatory disease caused by the deposition of urate, involves not only the joints but also the renal structures, generating complications with severe evolution. Renal damage associated with gout is an underdiagnosed clinical entity, but it has an essential role in the progression of CKD.

Objective(s). This synthesis aims to perform a critical, systematic, rigorous and comprehensive review of the specialized literature in PubMed, Scopus regarding renal complications of gout.

Materials and methods. We conducted a narrative review of the literature published to date, using databases such as PubMed and Scopus. The inclusion criteria were: observational, clinical, and experimental studies addressing the pathophysiological mechanisms, clinical manifestations, diagnostic methods, and therapeutic options for renal involvement in gout.

Results. Pathophysiological mechanisms: deposition of urate crystals triggers interstitial inflammation with IL-1 β , TNF- α and macrophage recruitment, leading to tubular fibrosis. Decreased uric acid excretion favors recurrent crystalluria. Manifestations: nephrolithiasis (25–40%), acute papillary necrosis with renal failure and crystals in the sediment, chronic interstitial nephropathy with decreased eGFR. Diagnosis: ultrasound, non-contrast CT, eGFR CKD-EPI, proteinuria, microalbuminuria, hematuria, 24 h urinary pH, biopsy, but rarely used. Management: hypouricemic agents (allopurinol, febuxostat US <6 mg/dl), alkalinization, hydration and BP control.

Conclusion(s). Renal damage in gout is multifactorial, polyetiological and often underdiagnosed. Periodic screening of renal function, urine sediment examination and 24 h pH help in detection. Hypouricemic therapy, adjusted to eGFR, plus alkalinization and regular hydration, slow down the progression of lesions.

Keywords: Gout, chronic kidney disease, crystalluria, urinary Ph

CARDIOTOXICITY INDUCED BY CONTEMPORARY ONCOLOGIC THERAPIES: EARLY BIOMARKERS AND PRACTICAL CLINICAL ALGORITHMS.

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Background. Anticancer therapies (anthracyclines, anti-HER2 agents, PD-1/PD-L1 inhibitors) reduce cancer-related mortality; however, they are associated with cardiotoxicity. According to the ESC guidelines, hs-cTn and NT-proBNP are considered reliable biomarkers for the early detection of subclinical myocardial injury.

Objective(s). To evaluate cardiac biomarkers for the early identification and monitoring of cardiotoxicity caused by contemporary oncologic treatments, employing current clinical algorithms.