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Renal Implications of Acute Endocarditis: Exploring the Dynamics and Management of Glomerulonephritis as a Secondary Complication

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Abstract

This article delves into the intricate landscape of glomerulonephritis secondary to acute endocarditis, a condition marked by the complex interplay of infectious, immunological, and renal factors. Addressing the evolving epidemiology and risk factors, it sheds light on the clinical manifestations that underscore the multifaceted nature of the disease. The diagnostic approach encompasses a comprehensive evaluation involving serological markers, blood cultures, and imaging studies, with renal biopsy crucial in assessing the severity of glomerular damage. The grim prognosis for renal function recovery necessitates nuanced therapeutic strategies, emphasizing the cautious use of immunosuppression and the importance of distinguishing this condition from other renal pathologies. The article highlights that a multidisciplinary approach involving cardiologists, infectious disease specialists, and nephrologists is paramount to developing tailored management plans. Exploring prevention and treatment strategies underscores the critical role of early initiation of pathogen-directed antibiotic therapy and vigilant monitoring for renal involvement. Ultimately, the article provides insights into the complexities of glomerulonephritis secondary to acute endocarditis, guiding early detection, prevention, and refined therapeutic interventions in this intricate intersection of infectious and renal pathology.

Keywords: Infective endocarditis; Glomerulonephritis; Renal complications

Abbreviations: IE: Infective Endocarditis; GN: Glomerulonephritis; RPGN: Rapidly Progressive Glomerulonephritis; ANCA: Anti-Neutrophil Cytoplasmic Antibodies; APSGN (Poststreptococcal Glomerulonephritis); MRSA (Methicillin-Resistant Staphylococcus aureus); ACE (Angiotensin-Converting Enzyme); ARB (Angiotensin Receptor Blocker); CIEDs (Cardiovascular Implanted Electronic Devices); RHD (Rheumatic Heart Disease)

Introduction

Acute Endocarditis is a severe and rapidly progressing inflammatory condition affecting the heart's inner lining, the endocardium. This disorder is characterized by the sudden onset of bacterial infection, leading to destructive lesions on the heart valves. It can include acute and subacute bacterial endocarditis

caused by viruses, fungi, and other microorganisms [1-5]. Due to the evolving nature of the infecting organisms over time, diagnosing the disease in its early stages can be challenging. It is frequently misdiagnosed or delayed until a severe infection has already occurred. Infective endocarditis by Staphylococcus aureus is gaining more relevance [2]. Given the damage to the valve caused by infection, there is a significant risk of embolization, stroke, and secondary complications. Glomerulonephritis, linked to infective endocarditis, is on the rise in the reported cases in the literature [2]. Glomerulonephritis is a category of renal disease marked by immune-mediated harm to the basement membrane, mesangium, or capillary endothelium, resulting in symptoms such as hematuria, proteinuria, and azotemia [4]. Acute Glomerulonephritis may arise from an inherent renal factor or a secondary condition that induces renal manifestations.

For example, acute post-streptococcal Glomerulonephritis is a typical illustration of acute Glomerulonephritis resulting from a streptococcal infection [3,4]. Likewise, Glomerulonephritis can also be triggered by a staphylococcus aureus infection. Though infective endocarditis-associated Glomerulonephritis is classified under immune complex-mediated Glomerulonephritis, confirming the presence of infected necessities culture verification, and pinpointing the infection focus depends on imaging techniques such as echocardiography [3]. Furthermore, some cases test positive for anti-neutrophil cytoplasmic antibodies [3,4]. Apart from these findings, IgG and C3 deposits on the basal membrane were identified [1]. This study aims to pinpoint the risk factors linked to Glomerulonephritis during the management of infective endocarditis and to examine the impact of GMN on patient survival and long-term kidney function. The information in this article may help diagnose, treat, and prevent infectious heart disease and better understand the disease.

Etiology and Pathogenesis

The renal implications of acute endocarditis involve a complex interplay of pathological mechanisms. While earlier theories emphasized embolism, abscess formation, and bacterial clump-related lesions, contemporary understanding suggests an immunological basis for glomerulonephritis development. Various histological types, including endocapillary proliferative glomerulonephritis, membranoproliferative glomerulonephritis, and crescentic glomerulonephritis, are identified, often accompanied by reduced complement levels and immune complexes. Pauci-immune staining patterns are observed in a substantial percentage of cases [6].

High-titer ANCA positivity is associated with more extensive glomerulonephritis. Immunofluorescence microscopy shows irregular, granular staining for IgG, IgM, and C3-positive cases, indicating immune complex formation, sometimes IgA, with dominance of IgM. Electron microscopy reveals subepithelial, subendothelial, and mesangial deposits, correlating with the extent of proliferation seen in light microscopy. The pathogenesis involves circulating immune complexes in infectious endocarditis, supported by immunofluorescence and electron microscopy findings. At the same time, ANCA may play a role in cases with little or no immune complex deposition [7].

Epidemiology and Risk Factors

The estimated incidence of Infective Endocarditis (IE) was 13.8 cases per 100,000 subjects per year, and IE accounted for 66,300 deaths worldwide, according to a study from 1990 to 2019 [8]. Bacterial, viral, and parasitic infections are the most important triggers for developing acute Glomerulonephritis (GN). Endocarditis-associated GN is classified as an infection-associated GN. A significant paradigm shift in the epidemiology and bacteriology of infection-associated glomerulonephritides has occurred over the past few decades; Streptococcus was the principal agent. However, the number of cases due to staphylococcus has increased. The two most common are Staphylococcus (53%) and Streptococcus (23%) [9,10]. High-risk patients include those with previous IE, patients with prosthetic valves, patients with congenital heart disease, and patients with ventricular assist devices. Patients at intermediate risk of IE include those who have rheumatic heart disease (RHD), non-rheumatic degenerative valve disease, congenital valve abnormalities such as bicuspid aortic valve disease, cardiovascular implanted electronic devices (CIEDs), and hypertrophic cardiomyopathy. The use of intravenous drugs, diabetes, HIV infection, malignancies, and other conditions that can suppress immunity are additional risk factors for acute IE [11].

Clinical Manifestations

Glomerulonephritis secondary to acute endocarditis presents a clinical tableau characterized by systemic and renal manifestations. Patients typically exhibit symptoms of infective endocarditis, such as fever, malaise, and cardiovascular symptoms indicative of valvular involvement [12]. Concurrently, renal involvement becomes apparent, often manifesting as hematuria, proteinuria, and varying degrees of renal impairment [12-16]. Hypertension may ensue due to volume overload and activation of the renin-angiotensin system. Edema can develop secondary to fluid retention. The underlying pathophysiology involves the formation of immune complexes, primarily composed of circulating antigens related to infective endocarditis, depositing within the glomeruli. These immune complexes incite an inflammatory response, leading to endothelial damage and subsequent glomerular injury [14-16]. The diagnostic approach includes clinical evaluation, serological markers, blood cultures, and imaging studies to confirm endocarditis. Renal biopsy may be employed in select cases to ascertain the severity of glomerular damage, with early recognition and management of infective endocarditis being paramount in mitigating renal complications [12-16].

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Diagnosis and Laboratory Evaluation

Laboratory evaluation of patients with glomerulonephritis associated with infective endocarditis should include renal function tests, urinalysis, and blood and urine cultures [17]. According to a 2015 report by Boils et al., the most common infectious agent found in blood cultures was S. aureus; the second was Streptococcus, and cultures were negative in 9% [18]. Trans-oesophageal echocardiograms and repeated blood cultures are recommended in patients with negative cultures [17]. Serologic findings in this study showed a decrease in complement activity in 56%, with reduced C3 only, reduced C4 only, or both in 37%, 3%, and 16%, respectively. Some patients also were reported with positive autoimmune serologies, mainly ANCA (28%) [18], and there is a report of positivity for PR3-ANCA and anti-GBM [19]. The primary pathology findings in endocarditis associated GN are focal crescents and necrotizing lesions. Immunofluorescence staining shows a variable degree of deposits comprising C3, with or without IgG. Patients with staphylococcal endocarditis might also have mild to moderate IgA deposits; however, a Pauci-immune pattern is also seen [17]. In the study mentioned above by Boils et al., electron microscopy shows mesangial (84%), subendothelial (45%), and subepithelial (35%) deposits, and rarely subepithelial humps [18].

Prevention and Treatment Strategies

Early initiation of pathogen-directed antibiotic therapy is essential in managing glomerulonephritis secondary to acute endocarditis, aiming to curtail bacteremia and the associated immune response contributing to nephritogenic formation [19]. The use of combination antibiotic regimens with bactericidal synergism is advocated to sterilize bloodstream infections rapidly. At the same time, definitive intervention, such as surgical debridement or device extraction, is crucial for addressing persisting infective foci, especially in infected cardiac valves. These measures aim to reduce circulating bacterial antigens, thus mitigating the perpetuation of immune complex generation [20]. It is imperative to concurrently avoid nephrotoxic co-exposures, such as moderating aminoglycoside courses, judicious use of IV contrast procedures, and limiting NSAID use. This approach preserves renal functional reserve in this vulnerable patient group [21]. Regular monitoring for signs of renal involvement, including urinalysis for hematuria and proteinuria, alongside serial assessment of serum creatinine for changes in filtration capacity, facilitates the early detection and treatment of glomerulonephritis before irreversible damage occurs [22]. Elevated serum levels of circulating immune complexes directly indicate active immune processes, highlighting the potential for renal deposition and injury if left untreated [23]. Some studies propose the initiation of prophylactic corticosteroid regimens at the diagnosis of endocarditis to mitigate downstream immune-mediated kidney injury potentially [24].

In cases where infective endocarditis patients exhibit signs of secondary glomerular disease, such as proteinuria or rising creatinine, first-line treatment involves corticosteroid agents like prednisolone at immunosuppressing doses (e.g., 1 mg/kg/day) to restrain inflammatory damage to the glomerular capillary units mediated by antibody binding, complement activation, and leukocyte influx [25]. Plasma exchange is a potent adjuvant therapy, directly eliminating circulating immune complexes and inflammatory mediators like cytokines, coagulation factors, and complement proteins [26]. Cytotoxic immunosuppressants like cyclophosphamide are recommended for aggressive or rapidly progressive glomerulonephritis resistant to the above interventions; this acts to arrest the proliferation and functional responses of leukocytes mediating glomerular inflammation while also impeding damaging proliferative changes in intrinsic glomerular epithelial and mesangial cells [27]. Simultaneous inhibition of the renin-angiotensin-aldosterone axis with ACE inhibitors or ARB medications is strongly advised, as this reduces proteinuria and intraglomerular pressure, thereby mitigating inflammatory glomerular injury [28]. In cases with substantial acute kidney injury and filtration compromise, temporary renal replacement therapy with hemodialysis may be required until the acute inflammation wanes and function stabilizes.

The prevention and treatment of glomerulonephritis as a secondary complication in the renal implications of acute endocarditis are critical considerations in contemporary medical practice. Early detection and the prompt initiation of appropriate antibiotic therapy are fundamental in preventing the progression of acute endocarditis to renal complications, such as post-streptococcal glomerulonephritis (APSGN) [21,31]. Ensuring the timely administration of antibiotics, guided by blood cultures and sensitivity testing, is imperative for eradicating the infectious agent and mitigating the risk of renal involvement. The infectious Methicillin-resistant strain of Staphylococcus aureus (MRSA) is often noted [21]. Rigorous monitoring of renal function through regular assessments of urine sediment, renal function tests, and imaging studies is essential for the early identification of glomerulonephritis in patients with acute endocarditis [30]. A contemporary multidisciplinary approach involving collaboration among cardiologists, infectious disease specialists, and nephrologists is crucial for developing a comprehensive management plan tailored to the individual patient's needs [27]. Treatment modalities may range from antibiotic therapy to oxygenation and dialysis, emphasizing ongoing surveillance for potential complications, including renal dysfunction, integrated into the long-term follow-up care of patients recovering from acute endocarditis; this underscores the importance of current research findings in shaping modern treatment strategies and the use of antibiotics [21].

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Prognosis

The prognosis for renal function recovery in infective endocarditis-associated glomerulonephritis (IEAGN) is generally grim, as evidenced by outcomes in 83 patients with Staphylococcus-related glomerulonephritis. Approximately half of these patients faced adverse outcomes, encompassing persistent nephropathy or kidney failure, remission, persistent renal dysfunction, progression to end-stage renal disease, death, and cases with no recorded outcome. Risk factors contributing to an unfavorable renal prognosis include older age, diabetes mellitus, pre-existing renal dysfunction, glomerulosclerosis, and interstitial fibrosis. Heart failure accompanying infective endocarditis can further prolong nephropathy, complicating the overall prognosis [32].

Distinguishing IEAGN from ANCA-associated glomerulonephritis is crucial for effective treatment planning. Clinical distinctions, as highlighted by Bonaci-Nikolic et al., such as age differences and specific clinical findings, guide this differentiation. While extensive immunosuppressive therapies are standard for ANCA-associated glomerulonephritis, they should be avoided in IEAGN. In cases of IEAGN with necrotizing lesions or crescent formation, immunosuppression does not improve renal prognosis and is associated with increased mortality. Steroids may be considered in cases of prolonged glomerulonephritis, but antibiotic therapy is crucial for eradicating the infection. Renal biopsy is a valuable tool for assessing the chronicity of glomerulonephritis in uncertain cases. This summary underscores the challenging prognosis in IEAGN and emphasizes the importance of tailored therapeutic approaches based on accurate differentiation from other renal conditions [33]. The prognosis for infective endocarditis-associated rapidly progressive glomerulonephritis (IEAGN-RPGN) remains uncertain, with treatment primarily focused on antibiotics due to the risk of exacerbating infection with immunosuppressive therapy. While antibiotics alone may lead to renal disease resolution, the generally poor prognosis prompts consideration of steroids, supported by some case reports indicating their efficacy. In a study involving 11 IEAGN-RPGN patients, a higher percentage of those receiving steroids achieved complete recovery compared to those with partial recovery, suggesting a potential benefit [33]. However, due to the limited sample size, the role of immunosuppressive therapy in improving the prognosis should be further investigated, carefully weighing risks against potential benefits. The overall prognosis is contingent on the timely identification and management of the underlying infection [34].

Conclusion

This comprehensive exploration of glomerulonephritis secondary to acute endocarditis illuminates the intricate interplay of infectious, immunological, and renal factors contributing to the disease's pathogenesis. The evolving epidemiology, risk factors, and clinical manifestations underscore the complexity of this condition, emphasizing the imperative for a multidisciplinary approach in its diagnosis and management. The grim prognosis for renal recovery necessitates nuanced treatment strategies, cautious use of immunosuppression, and a clear distinction from other renal conditions. The article provides insights into early detection, prevention, and tailored therapeutic interventions while underscoring the ongoing challenges and the imperative for continued research to refine our understanding and enhance treatment outcomes in this intricate intersection of infectious and renal pathology.

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