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Infective Endocarditis - Associated Glomerulonephritis in an Adolescent with No Predisposing Cardiac Risk Factors: A Diagnostic Conundrum

[1] Syed Sanya Zuberia, [2] Joshika Madhav, [3] Anitha.P*, [4] Saji James, [5] Shruthi T.K, [6] Ajitha.P

[1, 2, 3, 4, 5, 6] Department of Pediatrics, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

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KEYWORDS

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ABSTRACT:

Infective endocarditis-associated glomerulonephritis (IEAGN) is significant due to its potential for rapid progression to acute kidney injury or chronic kidney disease if not promptly diagnosed and treated. Delayed intervention may result in irreversible renal damage and an increased risk of systemic embolic events, emphasizing the importance of early recognition and management. While the majority of cases occur in individuals with known predisposing conditions such as congenital heart disease, intravenous drug use, or the presence of indwelling medical devices, there are rare instances where IEAGN can develop in individuals with previously normal cardiac anatomy.

We report the case of an adolescent female who initially presented with clinical features suggestive of acute glomerulonephritis. A systematic evaluation subsequently revealed underlying infective endocarditis.

1. Introduction

Infective endocarditis-associated glomerulonephritis (IEAGN) is significant due to its potential for rapid progression to acute kidney injury or chronic kidney disease if not promptly diagnosed and treated. Delayed intervention may result in irreversible renal damage and an increased risk of systemic embolic events, emphasizing the importance of early recognition and management. While the majority of cases occur in individuals with known predisposing conditions such as congenital heart disease, intravenous drug use, or the presence of indwelling medical devices, there are rare instances where IEAGN can develop in individuals with previously normal cardiac anatomy.

We report the case of an adolescent female who initially presented with clinical features suggestive of acute glomerulonephritis. A systematic evaluation subsequently revealed underlying infective endocarditis.

2. Case Report

A 17-year-old developmentally normal female, with no known prior cardiac abnormalities, presented with a 14-day history of intermittent fever and myalgia. She was initially managed at an outpatient clinic with oral amoxicillin. Despite this, she continued to experience fever spikes and developed new-onset haematuria over three days. There were no associated complaints of oliguria, facial puffiness, or other urinary symptoms. On examination, her blood pressure was elevated at 140/90 mmHg, >99th percentile for age. There were no signs of meningeal irritation, and fundus examination was normal. No dependent edema or skin abnormalities were noted. Given the clinical presentation, acute glomerulonephritis (AGN) was suspected. Urine dipstick testing revealed 1+ proteinuria. Laboratory investigations showed an elevated blood urea nitrogen (20 mmol/L) and serum creatinine (1.2 mg/dL). A 24-hour urinary protein quantification revealed proteinuria of 1162.5 mg/day. Complement levels showed normal C3 (135.5 mg/dL) and a mildly elevated C4 (53 mg/dL). Anti-streptolysin O titers were within normal limits. The patient was started on oral nifedipine and intravenous benzathine penicillin. Despite symptomatic management, the patient continued to be febrile. On day five of admission, she developed pandigital clubbing and a grade three systolic murmur over the mitral area. Infective endocarditis was suspected, and transthoracic echocardiography confirmed vegetations on the anterior mitral leaflet with severe mitral regurgitation. Empirical intravenous antibiotic therapy with ceftriaxone, vancomycin, and gentamicin was initiated. Serial blood cultures were sterile. Subsequently, the patient developed lower back pain. MRI spine showed features

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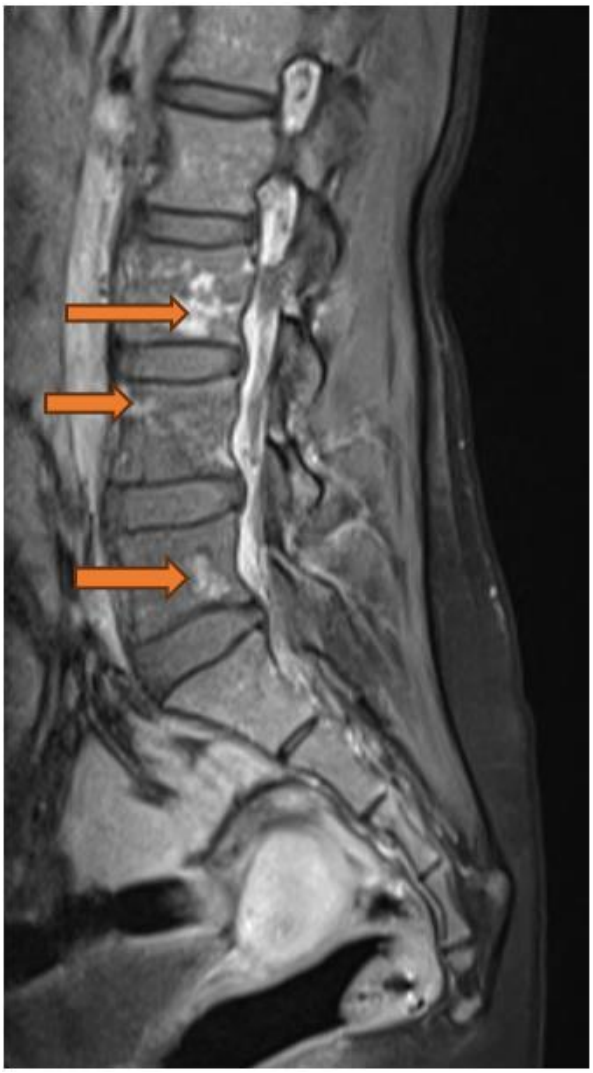
suggestive of infective spondylitis in lumbar region which was confirmed by a CT-guided vertebral biopsy. A multidisciplinary team comprising specialists in infectious diseases, cardiology, rheumatology, radiology, microbiology, nephrology, and orthopedics counselled the family regarding the nature and severity of the illness. She was prescribed a ten week course of oral clindamycin and rifampicin to treat both infective endocarditis and osteomyelitis. At follow-up, the patient demonstrated complete clinical recovery with resolution of symptoms.



MRI lumbosacral spine shows ill-defined areas of abnormal enhancement in L3, L4& L5

3. Discussion

The clinical spectrum of infective endocarditis-associated glomerulonephritis includes, Acute renal failure (79%), followed by acute nephritic syndrome (9%), rapidly progressive glomerulonephritis (6%), and nephrotic syndrome (6%).[1] Although IEAGN can occur at any age, it predominantly affects adults (30- 60 years of age),[2] particularly those with established risk factors. However, early presentation in young individuals without risk factors, such as in the current case, has seldom been reported.

The renal involvement in IE is not primarily determined by the size of the causative organism but rather by the deposition of bacterial antigens or immune complexes.[3] While embolic phenomena such as stroke, sudden-onset blindness, and pulmonary thromboembolism are well-recognized complications of infective endocarditis in paediatric populations, renal manifestations are less frequently reported. The pathognomonic features of IEAGN often overlap with the classical signs of infective endocarditis (IE). However, IEAGN tends to follow a more rapid and aggressive clinical course compared to the typically indolent progression of IE alone. In some cases, renal manifestations may serve as the initial indication of underlying infective endocarditis.[4]

Vertebral osteomyelitis in infective endocarditis typically affects the lower thoracic or lumbar spine. The condition often presents in adults with symptoms such as febrile lumbago or torticollis. The arterial route is considered the most plausible pathway for infection, as the segmental arteries supplying the vertebrae bifurcate to perfuse adjacent vertebral segments. To the best of our knowledge, there are currently no published case reports describing vertebral osteomyelitis secondary to infective endocarditis-associated glomerulonephritis (IEAGN).

Differentiating infective endocarditis-associated glomerulonephritis (IEAGN) from ANCA-associated glomerulonephritis is critical for determining the appropriate therapeutic approach. Studies have revealed that patients with ANCA-positive IRGN tended to be significantly younger and more frequently exhibited clinical features such as cardiac murmurs and splenomegaly. In contrast, hepatomegaly, pulmonary involvement, and peripheral neuropathy were significantly more prevalent in patients with ANCA-associated vasculitis.[5]

In IEAGN, *Staphylococcus* species are the most commonly implicated pathogens, accounting for approximately 53% of cases, followed by *Streptococcus*

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species in 23% of cases. Notably, up to 14% of cases may present with negative blood cultures, as observed in the present case due to prior antibiotic usage.[5] In addition to culture results, clinicians should assess the potential route of microbial invasion and investigate for any distal foci of infection. In cases where blood cultures are negative, a clinical decision must be made regarding whether to repeat the cultures or initiate empiric broad-spectrum antibiotic therapy, taking into consideration the patient's overall clinical condition. Long-term antibiotic therapy is generally required regardless of the specific pathogen identified, and in some instances, surgical intervention such as valve replacement may be necessary.[5]

The IEAGN requires a multidisciplinary approach that addresses both the underlying infection and the associated renal and cardiovascular complications. Treatment involves the administration of pathogen-directed intravenous antibiotics, based on culture sensitivity or the most likely causative organism. Renal complications are managed with supportive measures, including blood pressure control and the use of diuretics or angiotensin-converting enzyme (ACE) inhibitors, depending on renal function and volume status. In cases of heart failure, inotropic support may be required. The management of embolic complications—whether through anticoagulation or surgical intervention—depends on the location, severity, and nature of the embolism. Therapeutic strategies should be individualized based on the clinical severity of infective endocarditis and the degree of renal involvement. Ongoing monitoring with serial blood cultures and echocardiographic assessments is essential to evaluate treatment response and ensure adequate control of the infection.

The prognosis for renal recovery IEAGN is generally poor. In a cohort of 83 patients with glomerulonephritis secondary to *Staphylococcus* infection, approximately 50% exhibited persistent renal impairment or progressed to kidney failure, 36 patients achieved remission, 15 experienced ongoing renal dysfunction, 19 progressed to end-stage renal disease and 12 died.[5] Several risk factors have been associated with poor renal outcomes, including advanced age, pre-existing diabetes mellitus, baseline renal dysfunction, glomerulosclerosis, and interstitial fibrosis. Additionally, the presence of heart failure—commonly seen in infective endocarditis—may further exacerbate renal injury and contribute to a prolonged course of nephropathy.[5]

In summary, the prognosis of glomerulonephritis associated with infective endocarditis (IEAGN) is largely dependent on the prompt initiation of appropriate therapy, including targeted antibiotic treatment, timely surgical intervention when indicated, and comprehensive supportive renal care. Early recognition and management are critical for improving patient outcomes and minimizing the risk of progression to end-stage renal disease. To the best of our knowledge, there are no published case reports describing IEAGN as the initial clinical presentation in a paediatric patient with a structurally normal heart and without any known predisposing risk factors alongside vertebral osteomyelitis as a complication.

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