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General Medicine

UNMASKING RENAL AMYLOIDOSIS: NEPHROTIC PROTEINURIA IN THE WAKE OF PLEURAL TUBERCULOSIS

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ABSTRACT **Background:** Secondary (AA) amyloidosis is an infiltrative disease resulting from chronic inflammatory states such as infections, arthritis, or malignancies. Child presenting as Nephrotic proteinuria with histological evidence of De novo amyloidosis occurring is rare. **Case:** We report the case of a 15-year-old boy who developed pleural tuberculosis in 2018 completed 6 months of anti-TB therapy. In 2022, he presented with asymptomatic nephrotic range proteinuria and renal dysfunction. Kidney biopsy revealed features consistent with AA amyloidosis confirmed by histopathology and immunohistochemistry. **Conclusion:** This case highlights the rare occurrence of de novo AA amyloidosis following prior TB infection. Long-term follow-up with past infections is essential for early detection of such complications.

KEYWORDS : Amyloidosis, Secondary amyloidosis, Tuberculosis, Nephrotic range proteinuria,

INTRODUCTION

Nephrotic-range proteinuria in children is most commonly attributed to primary podocytopathies. In contrast, AA (secondary) amyloidosis is a systemic disease characterized by the deposition of serum amyloid A protein in various organs, including the kidneys. It typically occurs in the setting of ongoing chronic inflammation or infection, and its manifestation after apparent resolution of the underlying condition is uncommon. In India, AA amyloidosis secondary to infections such as tuberculosis remains a notable aetiology. We report a case of AA amyloidosis in a child with a remote history of pleural tuberculosis, presenting four years after completion of anti-tubercular therapy.

Case Presentation

A 15-year-old boy with no prior comorbidities presented in November 2018 with fever, dyspnoea, and right-sided pleuritic chest pain. Diagnostic pleurocentesis revealed an exudative pleural effusion with adenosine deaminase (ADA) >30 U/L. GeneXpert testing confirmed *Mycobacterium tuberculosis* (rifampicin-sensitive). He was initiated on a standard 6-month anti-tubercular therapy (ATT) regimen, with subsequent follow-up showing complete clinical and radiological resolution of tuberculosis. In April 2022, approximately four years post-treatment, he was found to have an asymptomatic elevation in serum creatinine (2.4 mg/dL) during routine evaluation. Urinalysis showed 3+ proteinuria, with a spot urine protein-to-creatinine ratio of 4.3, indicative of nephrotic-range proteinuria. A renal biopsy was performed to determine the underlying aetiology. Histopathological examination demonstrated mesangial and vascular deposition of pale eosinophilic, amorphous, waxy material. The deposits were negative on periodic acid–Schiff (PAS) and silver stains, but Congo red staining was positive, exhibiting characteristic apple-green birefringence under polarized light. Immunohistochemistry confirmed strong positivity for serum amyloid A, consistent with AA (secondary) amyloidosis. Immunofluorescence studies were negative for immune complex deposition. A whole-body PET-CT scan showed no evidence of active infection or inflammatory foci. The patient was managed conservatively with anti-proteinuric measures, including renin-angiotensin system blockade. Renal function normalized during follow-up, and he remains under regular monitoring.

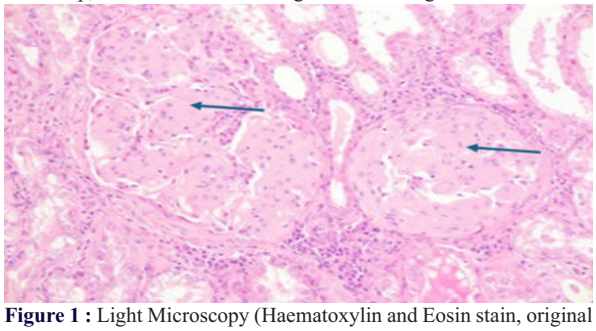

Figure 1 : Light Microscopy (Haematoxylin and Eosin stain, original magnification ×400)

This renal biopsy section shows glomeruli and adjacent blood vessels exhibiting deposition of pale eosinophilic, amorphous, acellular material within the mesangium and vessel walls (arrow). The material is weakly PAS positive and lacks silver stain affinity (not shown), suggesting amyloid deposition. There is also mild interstitial inflammation and tubular atrophy.

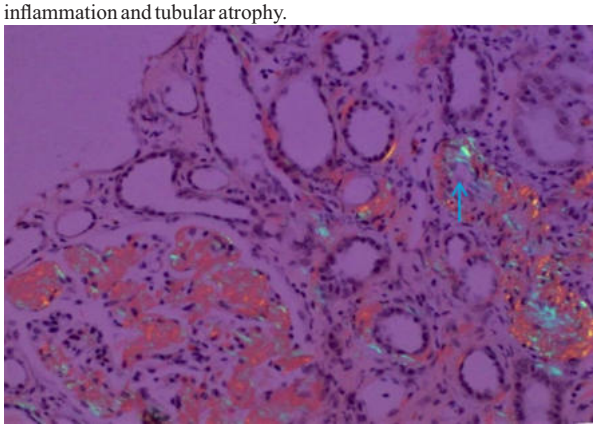

Figure 2: Congo Red Stain With Polarized Light Microscopy (original magnification ×400)

The same renal biopsy specimen demonstrates the classic **apple-green birefringence** of amyloid deposits in glomerular and vascular regions(arrow) under polarized light following Congo red staining. This confirms the presence of amyloid fibrils, consistent with a diagnosis of AA amyloidosis.

DISCUSSION

AA amyloidosis is a well-recognized complication of chronic inflammatory conditions and persistent infections, including tuberculosis (TB). Although more frequently reported during active disease, its occurrence after successful treatment is uncommon but documented. In a cohort of 43 adult TB patients, amyloidosis developed within 2 months to 7 years (mean, 2.25 years), despite adequate TB therapy in most cases (1). A Brazilian case involving a 46-year-old woman with concomitant renal granulomas and AA amyloid deposition during TB treatment further highlights the value of renal biopsy in detecting amyloid, even when Ziehl–Neelsen staining is negative (2).Childhood renal amyloidosis secondary to TB, as reported in two paediatric cases (a 10-year-old girl with abdominal TB and a 5-year-old boy with disseminated TB), both presented with nephrotic syndrome and responded to anti-TB therapy—with remission of proteinuria (3). These examples parallel our case: a 15-year-old treated for pleural TB, who developed nephrotic-range proteinuria four years later, and was found to have AA amyloidosis on biopsy.

Multiple studies identify TB as a leading cause of AA amyloidosis in

endemic regions. In South Africa, TB accounted for approximately 60% of AA amyloidosis cases-higher than earlier reports-highlighting its continued relevance in developing countries (4). In a cohort of 73 AA amyloidosis patients, TB-related amyloidosis was associated with more severe proteinuria, lower serum albumin, and poorer prognosis compared to other aetiologies (5).Interestingly, there are reports of late relapse or recurrence of AA amyloidosis many years after initial recovery. One patient with TB-associated AA amyloidosis remained proteinuria-free for 25 years but developed end-stage renal disease due to recurrent amyloid deposition (6). This underscores the insidious nature of ongoing amyloidogenesis, even in the absence of active infection, and the necessity for extended surveillance. Our patient exhibited normalization of renal function upon conservative management and renin–angiotensin system blockade, aligning with literature advocating for early diagnosis, anti-proteinuric therapy, and control of inflammatory stimuli. PET-CT confirmed absence of a reactivating foci.

CONCLUSION

AA amyloidosis should be considered in the differential diagnosis of patients presenting with unexplained proteinuria, particularly those with a history of chronic inflammatory conditions or infections such as tuberculosis. Early recognition is critical, as the disease may present insidiously, even years after apparent resolution of the underlying trigger. Definitive diagnosis requires kidney biopsy, helping to distinguish AA amyloidosis from other glomerular pathologies. Prompt diagnosis allows for timely initiation of anti-proteinuric therapy and close monitoring, which may help preserve renal function. Long-term follow-up is essential to assess for disease progression and identify any recurrence or reactivation of the underlying inflammatory condition.

Conflict of Interest Statement: None declared.

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