Early presentation of renal amyloidosis in a patient with pulmonary tuberculosis

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Abstract

Amyloidosis is a potentially life-threatening disorder caused by deposition of insoluble fibrillar proteins in various tissues, which commonly results in organ dysfunction or failure. Amyloidosis, either primary or secondary, can be diagnosed by the presence of a beta-pleated sheet configuration on X-ray diffraction examination, a fine fibrillar nonbranching appearance on electron microscopy, and an apple-green birefringence when examined under polarized light after staining with Congo red. In developing countries, tuberculosis is still the commonest underlying cause for renal amyloidosis, with an incidence of 9%–11% among patients with tuberculosis. In contrast to the common belief, renal amyloidosis can be observed in patients with comparatively short-term tuberculosis and even after adequate treatment. Here, we report a case of renal amyloidosis in a patient with tuberculosis rousing clinical suspicion as the patient presented with a triad of pedal edema, proteinuria, and medical renal disease on ultrasound, which was confirmed later by biopsy as renal amyloidosis secondary to tuberculosis.

KEY WORDS: Tuberculosis, renal amyloidosis

Introduction

Amyloidosis is a potentially life-threatening disorder caused by deposition of insoluble fibrillar proteins in various tissues, which commonly results in organ dysfunction or failure. [1] Tuberculosis (TB) as a cause of secondary amyloidosis has been found in 50%–92.5% of cases. [2] Amyloidosis, either primary or secondary, is defined as a group of chronic infiltrative disorders that have in common a beta-pleated sheet configuration visualized on X-ray diffraction, a fine fibrillar nonbranching appearance on electron microscopy, and an apple-green birefringence when examined under polarized light after staining with Congo red. [3] The distinguishing of primary from secondary amyloidosis can be obtained by treating the deposit with potassium permanganate before

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Congo red staining so that the apple-green birefringence viewed in the polarized light is abolished in secondary when compared with primary amyloidosis. [1] Usually, amyloidosis secondary to TB presents after years of exposure to the tubercle bacilli with long-standing history of illness. Here is a case report of a patient showing renal amyloidosis secondary to pulmonary tuberculosis (PTB) within few months of the initiation of the treatment.

Case Report

A 32-year-old man presented with significant bilateral pedal edema gradually progressive in nature extending up to genital region, breathlessness that gets exacerbated on exertion, along with facial puffiness and abdominal distension for 1 month. As per records, he was a known case of PTB on Cat-I DOTS therapy since 5 months. The patient was vitally stable. Investigations revealed that CBC, LFT, serum creatinine, electrolytes, blood sugar, ECG, and 2D-ECHO were within normal limits. Serum HIV and other viral markers were negative. Chest X-ray film was suggestive of bilateral UZ-MZ patchy opacity with bilateral pleural effusion; ultrasonography revealed the presence of bilateral moderate pleural and peritoneal collection. Further investigations revealed significant

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proteinuria, hypoproteinemia, and hypoalbuminemia. FDP and D-Dimer were negative. Venous Doppler of both the lower limbs was suggestive of subcutaneous edema. Hence, deep vein thrombosis was ruled out. Diagnostic pleural and ascitic tapping was done, and reports were suggestive of transudative collection with TB-PCR and cytology being inconclusive. In view of the findings of hypoproteinemia, proteinuria, and refractory pedal edema, nephrologist consultation was taken, and as per his opinion, percutaneous renal biopsy was performed. Histopathological examination of biopsy slides showed characteristic features of renal amyloidosis. Suspecting renal amyloidosis secondary to TB, the slides were sent for examination under polarizing microscope, which showed the absence of apple-green birefringence in the slides pretreated with permanganate solution and, hence, confirmed the diagnosis of secondary renal amyloidosis. Patient was referred back to Nephrologist for further management where he was prescribed salt restriction and diuretics. Patient responded well to the treatment and was found to be symptomatically improving on follow-up.

Discussion

This report presents a case of renal amyloidosis with TB as a predisposing illness. Various studies have revealed that the development of renal amyloidosis in a patient with TB is independent of the duration of TB and irrespective of whether the patient is cured or not. The total time interval between diagnosis of TB (predisposing illness) and development of secondary amyloidosis may be a few months, and it may vary from 2 months to 7 years (mean, 27 months).^[4]

In this report, the patient presented with signs and symptoms, which are conclusive towards medical renal disease, i.e., pedal edema, proteinuria, and hypoalbuminemia; these findings have been found to be associated with renal amyloidosis.^[4,5] Hypercholesterolemia is also found to be a significant finding in patients of renal amyloidosis.^[3-5]

Radiological investigations also aid as supportive evidence towards the disease: chest X-ray suggestive of infiltrative TB lesions with pleural effusion. Similarly other radiological findings namely presence of paraspinal abscess, thoracic abscess, and destruction of ribs are also seen as characteristic features in patients with renal amyloidosis. [3]

Ultrasonography of abdomen suggested the presence of ascites and medical renal disease. [4,5] For the confirmatory diagnosis of renal amyloidosis and to differentiate whether it is primary or secondary renal amyloidosis, renal biopsy was the investigation of choice, which showed absence of apple-green birefringence in the slides, which were pretreated with permanganate solution, confirming it to be a case of secondary renal amyloidosis. [3-5]

For the stabilization and improvement in renal function, reduction in protein excretion and partial resolution of amyloid deposits (as assessed in some studies by scintigraphy with

radiolabeled serum amyloid P component [SAP])^[6] requires successful treatment of the underlying inflammatory process with surgical resection of the focus of infection or tumor.

Most patients progress to chronic renal failure, while histological regression and/or clinical remission has been observed in a few instances of secondary amyloidosis after adequate treatment of the predisposing disease. [7-9] Such remissions have occurred only in those patients in whom the blood urea and serum creatinine levels were at normal levels. [8]

The patient was continued on DOTs therapy for TB and referred to a nephrologist for further management where he was prescribed salt restriction and diuretics. The patient responded well to the treatment and was found to be symptomatically improving on follow-up.

Conclusion

Renal amyloidosis may occur in comparatively short-term tuberculosis and even after adequate treatment. Renal amyloidosis should be suspected clinically in patients presenting with a triad of pedal edema, proteinuria, and hypoproteinemia.

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