PULMONARY TUBERCULOSIS WITH NEPHROTIC SYNDROME

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PRESENTATION OF CASE

Forty-year-old male, coolie by occupation, residing at Chennai belonging to lower socioeconomic class presented to Department of Pulmonary medicine at Sree Balaji medical college and hospital, Chennai with complaints of swelling of both legs, abdominal distension, eye lid swelling, breathlessness, cough with expectoration and fever for 2 weeks. History of loss of appetite and significant loss of weight present. There was no history of chest pain, palpitations and syncopal attacks. Patient had past history of pulmonary tuberculosis five years back defaulted category 1 anti-tuberculosis therapy. Patient is a non- smoker, non-alcoholic, and not a tobacco chewer. No significant family history. No contact history of tuberculosis.

On general examination patient was conscious and oriented. Moderately built and poorly nourished with BMI of 17. Grade 2 pitting fast pedal oedema present. Grade 2 clubbing present. His vitals were pulse 90/min, regular, normal volume, condition of vessel wall normal and no radio-radial or radio-femoral delay, blood pressure of 90/70 mm/hg right upper arm sitting position, Respiratory rate was 24/min regular abdominal-thoracic type of respiration and temperature 100degree F. Head to foot examination not significant. On examination of respiratory system - upper respiratory tract was normal and lower respiratory tract on auscultation cavernous type of breath sounds was heard in right infraclavicular area with coarse crepitations. Examination of abdomen - Distended and flanks were full, umbilicus centrally placed and flushed to the surface, shifting dullness present and no palpable organo-megaly. Examination of cardiovascular system- first heart sound and second heart sound heard, no murmur heard. Examination of central nervous system no focal neurological deficit.

DIFFERENTIAL DIAGNOSES

Patient is belonging to lower socioeconomic status having past history of tuberculosis 5 years back now presenting with cough, fever and anasarca the possible differential diagnosis includes-

a) Recurrent pulmonary tuberculosis with hypo-proteinemia
b) Disseminated tuberculosis i.e. abdominal tuberculosis
c) Recurrent pulmonary tuberculosis with cor-pulmonale
d) Pulmonary tuberculosis with renal involvement

CLINICAL DIAGNOSIS

Recurrent pulmonary tuberculosis with anasarca under investigation

DISCUSSION OF MANAGEMENT

Patient was investigated with complete blood count analysis showing Hb-12gm. Liver function tests- normal. Renal function tests - creatinine 0.6mg/dl, urea-normal. Chest x-ray showed active infiltrates in right upper zone with cavity (figure 1). Sputum AFB - positive (+3). Sputum CBNAAT - positive but no rifampicin resistance. ultrasound abdomen showed ascites and mild echogenic kidneys. Serum protein - 3.6gm/dl. Serum albumin – 0.9gm/dl. Urine routine - albuminuria (+3). 24 hour urinary protein – 5 grams per day i.e. nephrotic range of proteinuria. Lipid profile - hyperlipidemia (300mg). ECG within normal limits. 2D ECHO – normal.

Based on above investigations patient was diagnosed as a case of sputum positive pulmonary tuberculosis with nephrotic syndrome. Patient was undergone a ultrasound guided renal biopsy and was sent for histopathological examination. HPE was suggestive of Congo red stain positive in glomeruli and arterioles and negative apple green birefringence (figure 2).

The final diagnosis was sputum positive pulmonary tuberculosis with secondary renal amyloidosis presenting as nephrotic syndrome.

Nephrologist opinion was taken and patient was put on salt restricted and high protein diet. Patient was treated conservatively with angiotensin converting enzyme inhibitors -Tablet perindropil 4mg once daily. Category 2 anti-tuberculous therapy was started with a modified renal regimen according to RNTCP. Patient was explained about poor prognosis of the disease.

Patient was followed up after 2 months, showed improvement in chest x-ray and respiratory condition. Albuminuria was reduced from 3+ to 2+. 24-hour urine protein improved from 5gm to 3.5gm. at the end of 8 months of completion of ATT sputum turned negative but albuminuria was persistent with no signs of improvement.

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Serum albumin	0.9gm/dl
Total protein	3.6gm/dl
Serum creatinine	0.6mg/dl
Urine routine micro	Albuminuria (+3)
24-hour urine protein	5grams per day (nephrotic range of proteinuria)
Ultrasound abdomen	Ascites with both kidneys appears echogenic
ECG	No abnormality
2D ECHO	No abnormality
Lipid profile	Hyperlipidaemia (300)
Chest x-ray (fig2.0)	Active infiltrates seen
Ultrasound guided renal biopsy (fig 3.0)	Congo red stain positive in glomeruli and arterioles and negative apple green birefringence

Table 1. Investigation Profile

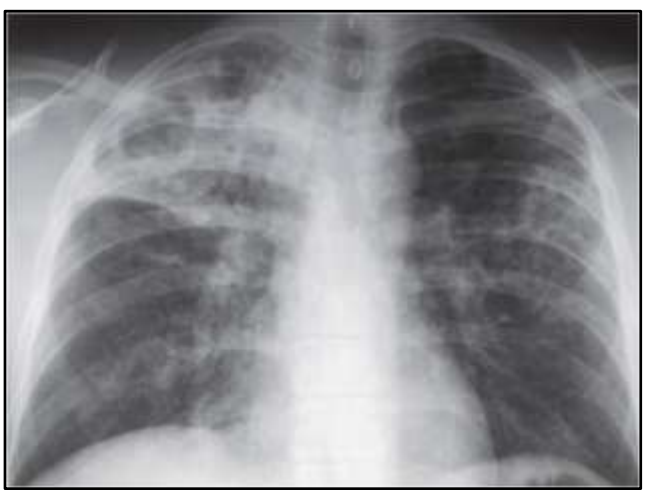

Figure 1. Chest x-ray PA View Showing Active Infiltrates with Cavity on Right Upper Zone

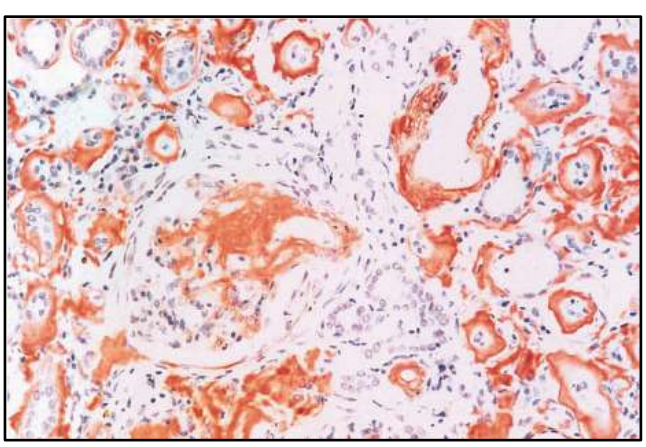

Figure 2. Congo Red Stain Positive in Glomeruli and Arterioles

Secondary Amyloidosis also known as reactive systemic amyloidosis. The amyloid deposits in this pattern are systemic in distribution and are composed of AA protein[1] Underlying conditions like tuberculosis, bronchiectasis and chronic osteomyelitis are cause of amyloidosis. Rheumatoid arthritis is one of the most common disease causing amyloidosis.[2] Heroin absuers and chronic skin infections associated with skin -propping of narcotics are responsible for amyloidosis.

In this context it is important to differentiate primary amyloidosis from secondary. This can be obtained by treating the deposit with potassium permanganate before congo red staining so that the apple green birefringence viewed in the polarized light is abolished in secondary when compared with primary amyloidosis.[3]

Amyloidosis in secondary to tuberculosis presents after years of exposure to the tubercle bacilli with long standing history of illness. Secondary renal amyloidosis is associated with a diverse range of disorders that usually includes chronic inflammatory disease or infectious diseases such as rheumatoid arthritis, osteomyelitis, Tuberculosis, chronic bronchiectasis, empyema, ulcerative colitis, carcinomas (most commonly renal cell carcinoma) and most recently in drug abusers, chronic skin infections, transmissible spongiform encephalopathies, Alzheimer's disease, and type-2 diabetes mellitus.[4,5] In these conditions, proinflammatory mediators/cytokines such as interleukin-1, tumor necrosis factor alpha, and interleukin-6, stimulate the synthesis of SAA in liver and other sites, that subsequently accumulates in renal tissue.[6] It is also to be noted that not all patients with chronic inflammatory disorders develop AA amyloidosis, and other factors such as genetic or environmental influences, specific properties of the precursor protein, macrophage activity, and the presence of 'amyloid enhancing factor' beside local tissue factors also play a role in amyloid fibril accumulation.[7]

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