Case Report

Extrapulmonary Tuberculosis Complicated by Focal Segmental Glomerulosclerosis — A Rare Association

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Focal Segmental Glomerulosclerosis (FSGS) usually presents with reduced glomerular filtration rate, heavy proteinuria and has unfavourable prognosis. Numerous associations with FSGS are found. We encountered a case of FSGS associated with Tubercular Lymphadenopathy presenting with proteinuria, anasarca, nephropathy. 40-year-old female patient presented with Pyrexia of Unknown Origin (PUO) for 4 months and anasarca for 2 months, associated with generalised Lymphadenopathy. Routine evaluation showed Microcytic, Hypochromic anaemia, Raised ESR, Hypoalbuminemia, impaired Renal Function Test and Nephrotic Range Proteinuria. Malarial Parasite (MP), Microarray pooled DNA analyzer (MPDA) , Human Immunodeficiency Virus (HIV) 1&2, HBsAg, anti HCV Ab, Antinuclear Antibody (ANA), ANA profile was negative. Excision Biopsy from supraclavicular lymph node showed well-formed granuloma with wide areas of caseous necrosis ie, Granulomatous Lymphadenitis. Renal Biopsy showed Focal Global Glomerulosclerosis. Treatment included anti-Tuberculosis drugs which resulted in a partial improvement in renal function and proteinuria and regression of lymphadenopathy. Tuberculosis can cause renal disease in a number of ways. To the best of our knowledge, very few cases in which tuberculosis was associated with different forms of Glomerulonephritis have been reported in the literature, however, association of FSGS with Tuberculosis is extremely rare, making our case truly atypical. [*J Indian Med Assoc* 2021; **119(11):** 51-3]

Key words **: Extra Pulmonary Tuberculosis, Granulomatous Lymphadenitis, Nephrotic Syndrome, Focal Segmental Glomerulosclerosis.**

FSGS is responsible for 35% of cases of Nephrotic Syndrome in adults. Though viral infections [Parvovirus, HIV, Hepatitis B, Cytomegalovirus (CMV)] are commonly associated with FSGS, association of bacterial infection with FSGS is rare even after thorough literature search. We propose tuberculosis especially extrapulmonary tuberculosis may lead to FSGS, which we found in our patient.

Case Report

40 years not known diabetic/hypertensive female presented with low grade intermittent fever with evening rise of temperature & night sweats for last 4 months. There was no significant localising features. After 2 months of fever onset patient gradually developed generalised body swelling, associated with increased frothiness of urine. There was no hist. s/o congestive cardiac failure or cirrhosis of liver.

Discussion

Our patient has been suffering from pyrexia of unknown origin with anasarca. So, our differentials would be-

- Portal hypertension complicated by Spontaneous Bacterial Peritonitis
- Infective Endocarditis with Congetive Heart Failure
- Nephritic/ Nephrotic Syndrome

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Editor's Comment :

- When we encounter a case of FSGS, extensive search for the etiology is mandatory for initiating appropriate therapy.
- FSGS secondary to tuberculosis infection, is one of the rarest entities reported in available literature. These subsets of patients show improvement with anti-tubercular drug therapy without addition of immunosuppressive medications.

Examination :

- Moon facies
- Bipedal pitting pedal edema
- Raised Jugular Venous Pressure (8cm, prominent v wave)
- Significant Lymphadenopathy in left post triangle (2 in number, largest 3cm) & in right supraclavicular region & in central , anterior group of left axillary region & in right axillary region. They were discrete, firm, no fixity, no overlying skin changes, non tender.
- P/A-Spleen-palpable (3cm,firm,nontender), shifting dullness present
- Respiratory system- b/l pleural effusion
- Chorionic Villus Sampling (CVS) - no gallop, normally audible S1S2
- No abnormality in breast examination & in Fundoscopy.

Investigations :

- HB:10.7/ WBC:3600 (N 59L36M04E01B00)
- Erythrocyte Sedimentation Rate-107

- Ur-80
- cr-1.7 (e-GFR-37)
- LFT –WNL, ALB -2.2, Glb – 2.2
- Lipid Profile- Cholesterol-392 / TG-295 /HDL- 50
- Ft4/ TSH- 0.9/ 2.4
- Urine RE & ME- RBC-10-15 / granular cast/ protein 3+
- Urine c/s- neg.
- 24 hour urine protein -8.2g
- Integrated Counselling and Testing Centres / HBsAG /Anti Hepatitis C Virus – NR
- Sputum Acid-fast bacillus & Cartridge- Based Nucleic Acid Amplification Test - neg.
- USG W/A- Retroperitoneal Lymphadenopathy & splenomegaly, moderate ascites
- USG Doppler of spleno -portal axis: No evidence of Pulmonary Hypertension (PHT)
- Chest x Ray –b/l pleural effusion (Fig 1)

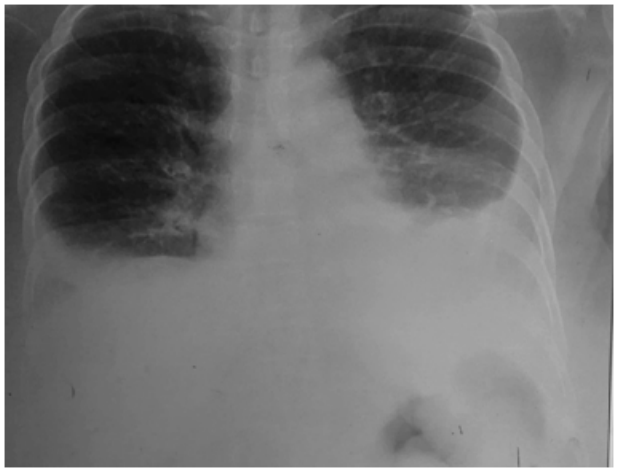


Fig 1

- 2D-ECHO-wnl
- Ascitic Fluid Study –High SAAG, cell count 90 (mainly mononuclear cells) CBNAAT- neg.
- Pleural Fluid Study –Transudative (light's criteria), ADA & CBNAAT- neg.

Differential Diagnosis :

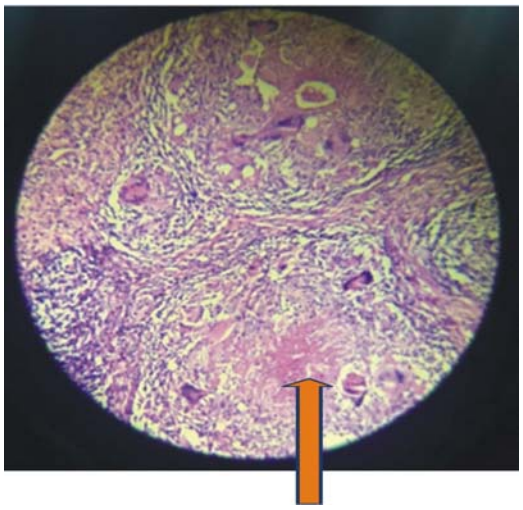
Pyrexia of unknown origin with Nephrotic Syndrome in a 40 years aged non diabetic, normotensive euthyroid patient.

Our Differentials would be —

- Connective tissue disorders- SLE, Sarcoidosis,
- Neoplastic disorder- Lymphoma
- Infective disorder- Tuberculosis
- Misc. Amyloidosis.

In Search of the Etiology, we performed —

- Excision Biopsy from supraclavicular lymph node (Fig 2)
- CBNAAT from lymph node tissue- positive
- Urine TB Polymerase Chain Reaction (PCR) - negative
- Autoimmune profile-



well formed granuloma with wide areas of caseous necrosis ie, Granulomatous lymphadenitis

Fig 2

ANA by Hep 2 Method & ANCA profile - neg
Low C3 (69.4) ,normal C4

- Renal biopsy-

Focal global glomerulosclerosis in 7 out of 15 glomeruli Immunofluorescence study was normal
Negative for congo red stain (Fig 3)

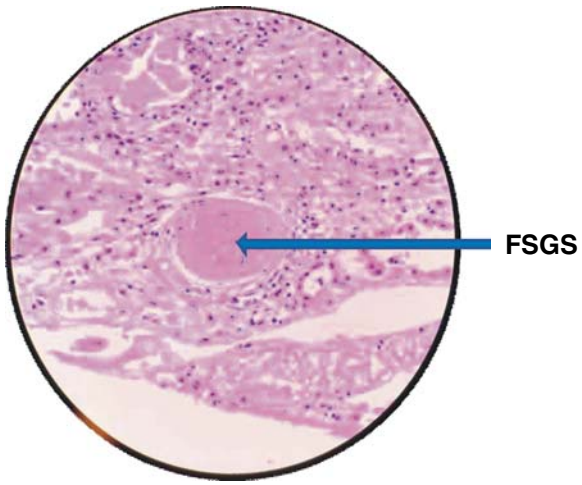


Fig 3

Provisional Diagnosis :

Tuberculous lymphadenopathy with FSGS

Treatment & Follow up:

- In view of tuberculous lymphadenopathy we started ATD
- After 6wks of therapy- the LNs regress.

Patient's clinical condition improved

24 hour urine proteinuria decreased from 8.2 gm to 1.35 gm

We continued therapy with ATD, ramipril, atorvastatin &this resulted in a partial improvement in renal function and proteinuria and regression of lymphadenopathy (Fig 4).

Repeat X-ray showed decreased pleural effussion

Discussion

- Extrapulmonary Tuberculosis may occur in 10-40% of cases having myriad of manifestations
- Tuberculosis can cause renal disease in a number of ways-
  - ♦ Genitourinary TB (direct invasion)
  - ♦ Chronic interstitial nephritis
  - ♦ Amyloidosis
- Renal involvement in Tuberculosis histologically shows epithelioid granuloma with or without caseation.
- The association of FSGS & Tuberculosis infection in the form of Extrapulmonary Tuberculosis is unique and has not been documented in the literature so far to date
- There is simultaneous existence of both FSGS and extrapulmonary tuberculosis in our patient without any preexisting conditions which might independently pre-dispose the patient to either of the two entities
- There occurs simultaneous improvement of both the entities solely with antitubercular treatment, without any immunosuppressive therapy.
- These indicates that the association may not be a mere finding but that they most probably are causally related.

Conclusion

- Extrapulmonary Tuberculosis is one of the aetiology of secondary FSGS

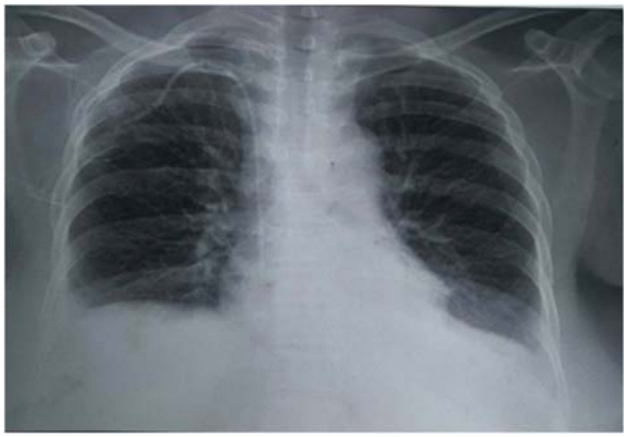


Fig 4

- Treatment with Immunosuppressive Therapy is not necessary in secondary FSGS
- Treating the cause is the mainstay of therapy, so a thorough search for the aetiology is must in all cases of FSGS

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