ORIGINAL RESEARCH PAPER

Volume-7 | Issue-6 | June-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE OF HIV ASSOCIATED NEPHROPATHY

General Medicine

Dr Aron V Thomas Post Graduate In General Medicines At SNMC and HSK HOSPITAL BAGALKOT . Karnataka

Dr Naveen Kulkarni* Associate Professor In General Medicines At SDM Medical College Dharwad. Karnataka *Correspondence author

ABSTRACT

Infection with the HIV may lead to a wide spectrum of anatomic and functional derangements in multiple target organs, such as the kidney. HIV associated nephropathy(HIVAN) is the most important cause of chronic renal failure and is seen in those with low CD4 counts. Progression to end stage renal disease is more rapid with most other causes of CRF. HIVAN can have normal kidney sizes or increased kidney size, usually they present with nephrotic syndrome[1].

KEYWORDS

Nephrotic, Albuminuria, Immunodeficiency

INTRODUCTION

In HIV infection kidney diseases may be direct consequences of HIV infections, opportunistic infections, neoplasms or drug related. Overall microalbuminuria is seen in 20% of untreated HIV infected patients, significant proteinuria is seen in closer to 2% HIV associated nephropathy was first described in IDUs and was initially thought to be IDU nephropathy in patients with HIV infection, it is now recognised as a true direct complications of HIV. Over 90% of reported cases have been in African-American or Hispanic individuals[2]. HIVAN should be considered in Caucasians with HIV complicated by nephrotic syndrome and renal failure[3]

CASE REPORT

A 35 year female admitted with history of fever with chills for the past 1 week, intermittent type associated with chills. History of breathlessness on exertion for the past 1 week. She reported weight loss with decreased appetite . She was a known case of HIV infection on treatment for the past two and half years (Tenofovir, Lamivudine and Efavirenz) her CD4 count in 2016 October was 396. She complained of pain abdomen over the right side lower abdomen for the past 1 week. No history of urinary disturbances, bowel disturbances, cough, headache, vomiting, chest pain, pedal oedema.

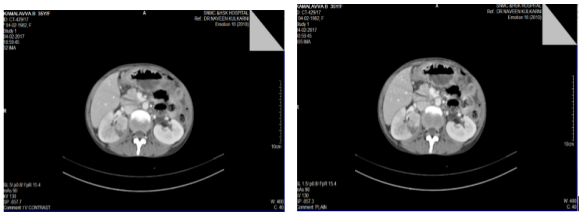
On examination, she had pallor, platynychia, vitals were normal. On systemic examination, there were crepitations in the right infra axillary and inter scapular area, breath sounds were diminished in these areas as compared to left side. In left second and third intercostal space systolic murmur was heard. There was no hepatosplenomegaly. Nervous system examinations was essentially within normal limits.

Investigations revealed hemoglobin – 4gram% , WBC count - 31,000 cells/cubicmm, DLC polymorphs-86%, Lymphocytes-10%, Eosinophils- 4%. Platelet count was 2.35 Lakhs. ESR -80 mm at the end of first hour. Peripheral smear showed Microcytic hypochromic anemia with neutrophilia. Urine examination- 10-12 pus cells /HPF, albumin 2+. Reticulocyte count 0.3% Serum ferritin 45.88 ng/ml. RBS, serum creatinine, electrolytes, LFT were within normal limits. TSH was 10.86 µIU/ml . Malaria antigen test was negative.

Chest X-ray revealed no significant abnormalities, ECG was essentially within normal limits. USG abdomen and pelvis - no sonological abnormalities.

DISCUSSION

A diagnosis of anaemia with urinary tract infection was made. She was treated with antibiotics and blood transfusions. She continued to have fever intermittently and pain abdomen on second day and third day. Due to persistent pain abdomen she was advised to undergo further evaluation with CT- abdomen and pelvis. Meanwhile urine and blood culture revealed no growth. She underwent CT abdomen and pelvis. CT scan showed bilateral kidneys enlarged with renal infarcts in right kidney.



HIV associated nephropathy can be an early manifestation of HIV infection and can also be seen in children. Proteinuria is the hallmark of this disorder. Edema and hypertension are rare. Ultrasound examinations revels enlarged hyperechogenic kidney. Definitive diagnosis is obtained through renal biopsy. The incidence of this disease in patients receiving ART has not been well defined. This is the leading cause of end stage renal disease in patients with HIV infection[2].

HIVAN can occur at any stage of HIV infection: although initial reports described the renal disease in association with clinical AIDS, this form of nephropathy has been often observed in otherwise asymptomatic HIV- infected individuals; at the time of its clinical presentation, a number of patients do not have any other manifestation of HIV disease. Clinically, nephrotic syndrome with heavy proteinuria is a cardinal manifestation of HIVAN, and nephrotic range proteinuria is observed in 90% of cases, but not all patients demonstrate all the classic criteria of nephrotic syndrome (as in the reported case); peripheral edema and hypercholesterolemia may be absent. At the time of first presentation, renal insufficiency with increased serum urea and creatinine levels is reported in only 10% to 20% of cases, such as hematuria and arterial hypertension[4]. HIVAN is seen in AIDS with nephrotic range proteinuria and large kidneys on USG and low Cd4[5]

Anemia is the most common hematologic abnormality in HIV infected patients. While generally mild, anemia can be quite severe and require chronic blood transfusions. Among the specific reversible causes of anemia in the setting of HIV infection are drug toxicity fungal and mycobacterial infections, nutritional deficiencies and parvovirus B19 infections[4].

Thyroid function may be altered in 10- 15% of patients with HIV infection. Both hypo and hyper thyroidism may be seen. The predominant abnormality is sub clinical hypothyroidism[6].

Renal disease may be the result of direct infection of the kidney and lower urinary tract or may present as secondary amyloidosis. Patients present with dysuria, hematuria, or flank pain. More

than 90 percent of asymptomatic patients have sterile pyuria with or without microscopic hematuria. Abdominal CT scan may reveal renal calcifications, calculi, scarring, hydronephrosis, or evidence of extrarenal disease (e.g., ureteral strictures; contracted bladder; calcifications in the vas deferens, seminal vesicles, or prostate). Renal function usually is preserved, except in the setting of tuberculous interstitial nephritis[7].

Based on her symptoms, other blood investigations and CT findings a diagnosis of HIV with severe anaemia with sub clinical hypothyroidism with HIV associated nephropathy with probability of tuberculosis was made. Diagnosis of renal tuberculosis is based on pyuria in absence of infection as judged by culture and sensitivity on routine media and lower UTI not responding to conventional antibiotics[8]. She was given blood transfusions. She was initiated with anti- tubercular drugs along with levothyroxine and continuation of ART. In view of active infection and ATT initiation patient was not started with prednisolone and ACE inhibitors which are considered to be treatment options for HIV associated nephropathy. Patient insisted for discharge in view of personal reasons. Thus she was advised for regular follow up with urine examination and blood examinations.

HIV associated nephropathy requires renal biopsy for confirmation. In settings where renal biopsy is not possible, due to various reasons, HIVAN can be considered in patients who present with abdomen and urinary symptoms, urine analysis showing proteinuria and with imaging suggestive of increase in kidney sizes. Renal biopsy remains gold standard for diagnosing HIVAN. Suspicion remains if constant proteinuria and decrease in creat clearance level[9]

REFERENCE

1. G.Maartens; Davidson's principles & practice of medicine,22nd edition. Part 2 Practice of medicine: chapter 14 HIV infection and AIDS, Presenting problems in HIV infection, Renal disease 405.
2. Longo DL, Fauci SA. In: Kasper DL, Fauci AS, Hauser SL,Longo DL, Jameson JL, Loscalzo J, editors. Harrison's principals of Internal Medicine. 19th ed. Vol 2. New york: McGraw Hill; 2015. P.1258-9
3. Yalavarthy R et al. Int J STD AIDS. 2008 Nov; 19(11) 789-90
4. Longo DL, Fauci SA. In: Kasper DL, Fauci AS, Hauser SL,Longo DL, Jameson JL, Loscalzo J, editors. Harrison's principals of Internal Medicine. 19th ed. Vol 2. New york: McGraw Hill; 2015. P.1262
5. Craig GA et al. The acute, the chronic and the news of HIV related renal disease in Africa. Kidney international Vol 78. Issue 3, 1 august2010,239-245
6. Longo DL, Fauci SA. In: Kasper DL, Fauci AS, Hauser SL,Longo DL, Jameson JL, Loscalzo J, editors. Harrison's principals of Internal Medicine. 19th ed. Vol 2. New york: McGraw Hill; 2015. P.1259
7. Calza, Leonardo; Manfredi, Roberto; Chiodo, Francesco. HIV-Associated Nephropathy With Peripheral Edema, Arterial Hypertension, and Hyperlipidemia: Case Report and Literature Review. Infectious Diseases in Clinical Practice:November 2002 - Volume 11 - Issue 8 - pp 459-464
8. John BE et al. Tuberculosis and Kidney. J Am Soc Nephrol 12.1307-1314;2001
9. Gafencu M et al. Late onset of HIVAN in ayoung female. Rom J Morphol Embryol 2014;55(4):1463-7