RENAL TUBERCULOSIS: AN INCIDENTAL FINDING IN TYPE II DIABETES

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ABSTRACT

Background: Renal tuberculosis constitutes 14% to 41% of cases of extrapulmonary TB and with HIV pandemic; its prevalence is expected to increase. **Case report**: We report a case of a 51 year old diabetic/hypertensive female admitted via the A&E with complaints of vomiting and generalized malaise. She had hyponatraemia of 119mmol/l, creatinine 687µmol/L, and urea 28mmol/L. Her kidneys were normal sized and echogenic on ultrasound examination. She was treated for hyponatraemia with normal saline and discharged. She reported at the clinic 2 weeks later with complaints of fever, back pain and right sided neck swelling. Neck ultrasound revealed cystic mass in the lower part of the sternomastoid muscle, Xrays were normal, Urinary AFB done on four urine specimen collected over days was negative and Mantoux test was reactive. Spinal examination was unremarkable except for presence of tenderness at the T6 to T7 vertebrae. A diagnosis of extra pulmonary TB involving the cervical lymph nodes and spine was made and she was commenced on quadruple anti TB drugs which resulted in improvement of her renal function. **Conclusion:** The diagnosis of renal tuberculosis is usually difficult due to low yield of laboratory tests such as urine microscopy for AFB. High index of suspicion is often required in most patients.

Keywords: Renal Tuberculosis, Type II Diabetes, Gombe

INTRODUCTION

Tuberculosis is one of the oldest diseases known to man, affecting millions of people in all continents of the world. Although pulmonary involvement is the most commonly encountered disease, all other organs of the body can be affected by it. The World Health Organization (WHO) estimated that there were 10.4 million cases of tuberculosis worldwide in 2016 and about 90% of cases are in the developing countries of South East Asia and Africa.[1] In Sub-Saharan Africa tuberculosis is a leading cause of morbidity and mortality where up to a third of the populations are at risk of developing active tuberculosis.[1] Although pulmonary tuberculosis has traditionally been the major form of infection, extra pulmonary tuberculosis has assumed increased prevalence especially with the advent of the HIV epidemic.[2] Extrapulmonary tuberculosis involves invasion of tissues such as lymph nodes, gastrointestinal tract, genitourinary tract, nervous tissues as well as the skin.

Genitourinary tuberculosis is a common form of extra pulmonary tuberculosis acquired through haematogenous spread of the infectious particles from a primary site, usually the lungs.[3] Studies have found that genitourinary tuberculosis constituted 14 to 41% of all cases of extrapulmonary tuberculosis.[4-6] Although Sub Saharan Africa contributes 33% of the world TB burden, there is paucity of data on the prevalence of renal tuberculosis. Karsdtaedts et al[2] in South Africa, found that 46% of patients with extrapulmonary tuberculosis have identified risk factors for tuberculosis. And with the advent of HIV/AIDS

and the growing epidemic of diabetes mellitus, the prevalence of renal tuberculosis is set to increase in Africa. Type 2 diabetes is a known risk factor for both tuberculosis and chronic kidney disease. Due to the paucity of symptoms and diagnostic tools, it is difficult for diagnosis of renal tuberculosis to be made in our environment.[7] Thus many diabetic patients would be labeled to have diabetic nephropathy rather than alternative diagnoses such as renal tuberculosis.

CASE REPORT

A 51-year-old female nurse was admitted via the accident and emergency with complaints of generalized body weakness, vomiting and anorexia of 2 weeks duration. Her body weakness was gradual in onset and vomiting initially early morning, non-bloody, non-bilious. There was no abdominal pain or distension. She had no urinary symptoms. She had been diabetic and hypertensive for 7 and 10 years respectively and has been well controlled on glibenclamide and metformin for diabetes. She was found to have features of diabetic nephropathy during an out-patient clinic visit. About a month later she was seen at a private hospital where she was commenced on haemodialysis and had three sessions over one week. She had no history of alcohol or tobacco use.

On physical examination she was found to be pale, lethargic, with asterixis and no leg oedema. Her blood pressure was 100/60mmHg and heart sounds were normal. Laboratory test showed PCV 22%, creatinine 687µmol/L and urea was 28mmol/L, sodium 119mmol/L chloride 88mol/L, and bicarbonate 18mmol/L.

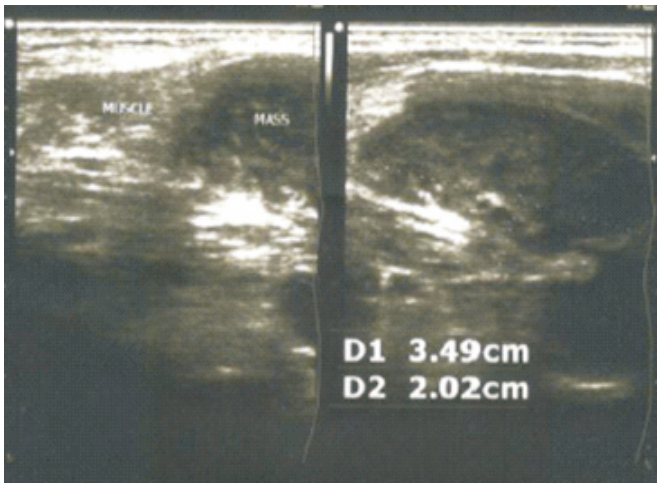

Figure 1: Neck ultrasound of neck showing cystic neck mass

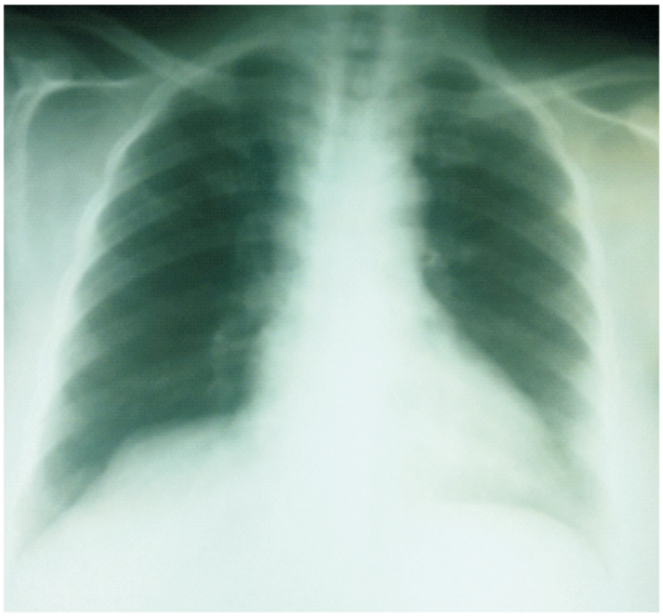

Figure 2: Showing chest Xray

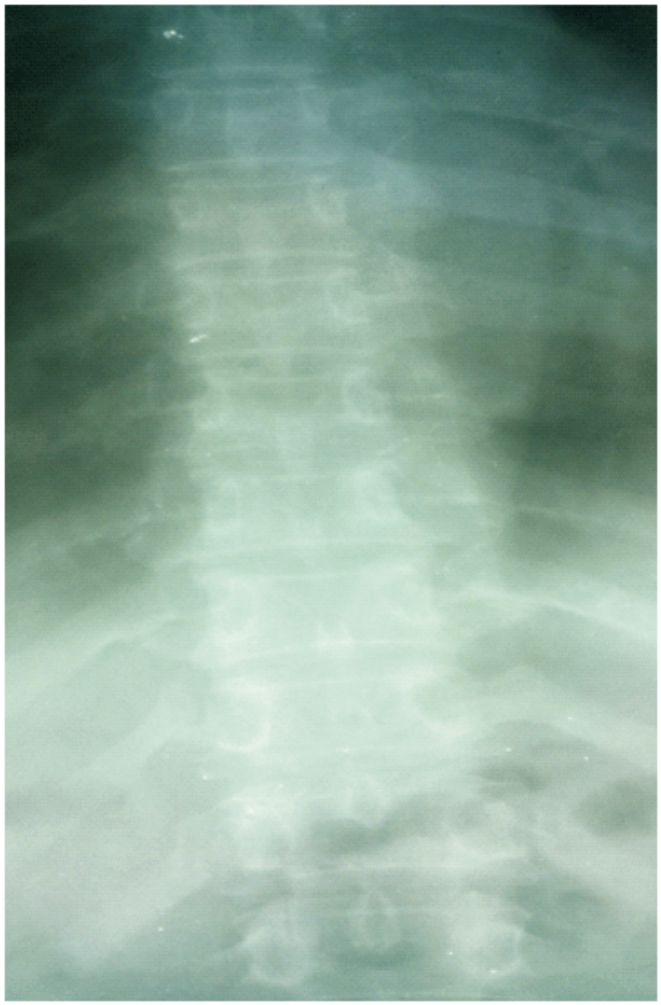

Figure 3: Showing the Xray of spine

Her renal ultrasound scan showed normal kidney sizes with increased echogenicity and no features of calcification or obstruction. She was non-reactive to HIV, Hbs Ag and HCV. Urinalysis showed proteinuria 3+, glucosuria 1+ and no haematuria. Her 24hour urinary sodium excretion was 145mmol/l and proteinuria 1.06g/day.

A diagnosis of hyponatraemia was made and she was treated with intravenous 0.9% saline augmented with table salt added to food. The sodium deficit was corrected over 4 days while on admission and she was discharged on the 5th day.

Two weeks after discharge she presented with complaints of right-sided neck swelling and dull aching back pain. There was no history of neck pain and no swallowing difficulty.
Examination revealed a cystic swelling measuring 2 x3 cm, fluctuant and non tender. Spinal examination was unremarkable except for presence of tenderness at the T6 to T7 vertebrae. Nervous system and chest examinations were unremarkable. Tuberculin skin test reaction was 24mm. Urinary AFB done on four urine specimen collected over days was negative. Facility for culture of mycobacteria is not available in our centre.

A diagnosis of extra pulmonary TB involving the cervical lymph nodes and spine was made. She commenced quadruple anti TB drugs.

There was resolution of the back pain and neck swelling after 3 months. Her renal function test at 7 months showed creatinine 210µmol/l, urea 12mmol/l and normal electrolytes.

Parameters	Month 0	Month 1	Month 2	Month 7
PCV (%)	22	27	28	35
Urea(µmol/L)	28	22	16	12
Creatinine (µmol/L)	628	433	255	210

DISCUSSION

Tuberculosis can involve only one organ such as the lungs, presenting as a localized disease or have a multi systemic involvement. Patient's background immunological competence has been found to be an important factor that determines whether the disease is limited to one organ or it spreads across various systems. Although pulmonary involvement is most common, extra pulmonary tuberculosis such as genitourinary tuberculosis is assuming greater importance especially in patients with compromised immune status.[8] Patients with genitourinary tuberculosis usually present with symptoms emanating from the site that is predominantly affected. Early granulomatous infection of the kidney may present as proteinuria, pyuria, and loss of kidney function. Isolated haematuria is another manifestation of renal tuberculosis. Lower urinary symptoms occur whenever the disease spreads to the ureter and bladder. A urinary symptom suggestive of urinary tract infection, accompanied by pyuria and haematuria with no bacterial growth, suggests urogenital tuberculosis. Advanced disease may cause obstructive uropathy, bladder defects and loss of kidney function.[9] Patients who have predominantly interstitial nephritis are likely to present with fluid and electrolyte imbalances. Our patient presented with generalized malaise and hyponatraemia suggesting that she had tuberculous tubulointerstitial nephritis (TIN). Glomerular diseases such as proliferative glomerulonephritis have been reported.[10] With the projected rise in number of persons affected by diabetes mellitus to more than 439 million by the year 2030, tuberculosis is set to raise along.[11] The natural history of tuberculosis in an immune competent individual comprises progression to a latent state of TB infection in which most bacteria are killed and the few that remain viable are in a latent state characterized by altered state of metabolism and persistence. To reach this state of balance, innate immunity and particularly mononuclear phagocytes in the alveolar spaces allow for the initial intracellular growth of the bacilli. As the mycobacterial burden increases, the adaptive immune response is deployed through

localized activation of T-helper (Th1) inflammatory response and recruitment of monocytes, lymphocytes, natural killer T cells and B lymphocytes for the formation of granulomas that kill most mycobactria and restrict the growth of those that remain viable.[12] In diabetic patients there is evidence of defective and delayed response to mycobaterium, thus patients with latent TB infection that develop diabetes have a tendency to develop active form of the disease.

This case brings to the fore the importance of strongly considering renal tuberculosis in diabetic

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