CASE REPORT

Haematuria and loin pain, could this be tuberculosis?

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SUMMARY

We report a case of an 18-year-old Caucasian male presenting with haematuria and loin pain while working as a locksmith. He was systemically well with normal vital signs. Peripheral blood testing demonstrated renal failure, secondary to hydronephrosis, caused by haemorrhagic cystitis with no obvious cause for the obstruction. The patient was diagnosed with a urinary tract infection and treated with antibiotics. He responded well and his renal function improved. Four months later he re-presented with the above symptoms, weight loss and night sweats, bladder wall biopsy at this point confirmed tuberculosis.

BACKGROUND

Mycobacterium tuberculosis is one of the leading causes of morbidity and mortality due to infectious diseases worldwide. The latest WHO report states that there were 9.4 million new cases of and 1.7 million deaths caused by tuberculosis (TB) in 2009.[1] Miliary TB refers to clinical disease due to haematogenous spread of *M. tuberculosis*. Clinical manifestations can be non-specific and radiological findings may not be evident until late into the disease.[2] We report an interesting case that highlights the importance of the multitude of diverse TB presentations and considering TB as a differential diagnosis in atypical presentations. This case is of educational interest to professionals working within medical and surgical specialties. It raises awareness to the multitude of diverse TB presentations.

CASE PRESENTATION

An 18-year-old Caucasian male was referred to the urology clinic with a 2 week history of haematuria and left loin pain. There was no significant medical history. He lived with his family and worked as a locksmith for the previous 3 years. He was not taking any medication and had no family history. On examination he was systemically well with mild left loin tenderness.

INVESTIGATIONS

Peripheral blood testing revealed deranged renal function (urea 16.8 mmol/L, creatinine 567 µmol/L), normocytic anaemia, hypoalbuminemia, lymphocytosis and an erythrocyte sedimentary rate of 70. Subsequent renal ultrasound scan showed moderate left sided hydronephrosis with dilation of the upper ureter. An intravenous urogram performed showed a normal excretion of the right kidney but no evidence of excretion from the left after 2 h of contrast.

Cystoscopy revealed haemorrhagic cystitis, inflammatory changes with debris in the bladder, retrograde pyelogram showed dilation of the calyces but no obvious pelvic ureteric junction obstruction. A left ureteric stent was inserted to minimise the risk of obstruction. Treatment was initiated with antibiotics and the stent was later removed as his renal function improved. A follow-up scan demonstrated resolution of the hydronephrosis.

Four months later, he was acutely admitted with further haematuria, loin pain, night sweats and four stone weight loss. Repeat cystoscopy was performed with bladder wall biopsy. Results showed necrosis of the prostate with severe inflammatory reaction of the bladder, biopsies of which were smear positive for acid-fast bacilli (AFB), confirming a diagnosis of urogenital TB. Consequently further imaging was performed. A chest X-ray demonstrated changes compatible with miliary TB (figure 1).

Induced sputum samples returned positive for AFB. Urine culture and PCR failed to yield any organisms. A CT of the abdomen and pelvis identified evidence of bilateral renal TB while a CT of the head showed cerebral granulomas compatible with TB (figures 2 and 3).

Ultrasound of the patient's scrotum also showed evidence of TB. He was diagnosed with miliary TB with cerebral, respiratory and urogential tract involvement. It later transpired that this patient had no family history of TB, however he did come into contact with a patient with Isoniazid-resistant TB earlier in his life. He did not have the BCG vaccine. He had no risk factors for HIV and was found negative when tested.

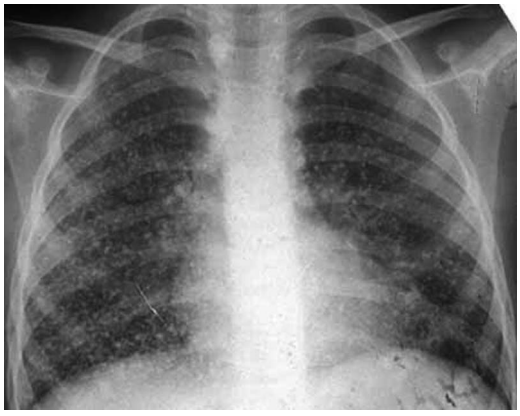

Figure 1 Chest X-ray demonstrating miliary TB.

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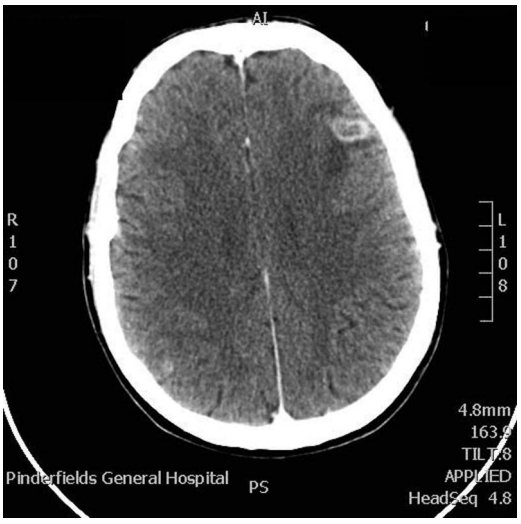

Figure 2 CT of cerebral granuloma, frontal lobe.

TREATMENT

Treatment was initiated with ethambutol 900 mg once a day, rifinah 300 two tablets once a day, pyrazinamide 2 g once a day, pyridoxine 10 mg once a day and prednisolone 40 mg once a day. Following that maintenance therapy with ethambutol 2400 mg, rifinah 300 three once a day plus an additional isoniazid 450 mg thrice weekly in view of him being exposed to isoniazid-resistant TB in the past.

OUTCOME AND FOLLOW-UP

Our patient was treated for 2 years in total. Clinical symptoms, serological tests and radiological imaging performed during the treatment period showed a good response to treatment and improved clinical outcome.

DISCUSSION

Miliary TB can occur due to a primary infection or secondary to reactivation of a latent focus. It is defined as being millet like seeding of TB bacilli in the lung (1–5 mm). It can occur in an individual organ (rare <5%) or a number of organs throughout the body (90%) including urogenital tract, brain, lung, liver and spleen.[3] It is an important disease to be aware of as it can mimic many other diseases. It can present in a diverse manner, commonly symptoms include fever, weight loss, cough, haemoptysis and night sweats but they are not necessarily specific. In some reports up to 50% of cases are not diagnosed until post-mortem.[4] The manifestations can be acute but are more likely to be subacute or chronic. In endemic areas miliary TB may be associated with reinfection.[3]

Development of miliary TB during primary infection can present with relatively acute onset and rapid clinical course. Acute disease may be fulminant, including multiorgan system failure, a syndrome of septic shock and acute respiratory distress syndrome. The subacute or chronic presentations of miliary TB are more common than acute disease.

The most common extrapulmonary sites of disease include the lymphatic system, bones and the liver. Twenty-five per cent of patients have meningeal involvement.[4] Diagnostic tests include peripheral blood testing (lymphocytosis, anaemia, hypoalbuminemia and a raised erythrocyte sediment rate). Cultures for mycobacteria include sputum, urine, blood and cerebrospinal fluid. Depending on organ involvement, appropriate radiological imaging and organ biopsy is undertaken if required. Hallmark histological appearances of miliary TB are necrotising granulomas and staining for AFB reveals rod-like structures in approximately 80% of specimens.[5] The disseminated nodules consist of central caseating necrosis and peripheral epithelioid and fibrous tissue. Radiologically, the nodules are not calcified.

A high index of clinical suspicion is important to obtain an early diagnosis and to ensure improved clinical outcomes. Early empirical treatment for possible but not yet definitive miliary TB increases the likelihood of survival and should not be withheld while investigation results are pending.[6]

Urogenital TB occurs in 30–40% of cases of extrapulmonary TB[5] and 2–20%[7] of those with pulmonary TB. It most frequently affects the kidneys. Renal infection is slowly progressive and highly destructive. Ureteral and bladder TB is secondary to descending infection through the urogenital tract. Ureteral stenosis is the main cause of delayed renal excretion. Symptoms arise when there is bladder impairment. The main symptoms include dysuria and haematuria in 37.9% and 35.6%, respectively. The diagnostic gold standard is urine culture, however PCR can be used in addition to cystoscopy and bladder biopsy.[7]

In general the same treatment regimens are used to treat pulmonary and extrapulmonary TB with varying course lengths and combinations of drugs. Pharmacological treatment includes rifampicin, isoniazid, ethambutol and pyrazinamide initially with the discontinuation of ethambutol and pyrazinamide for

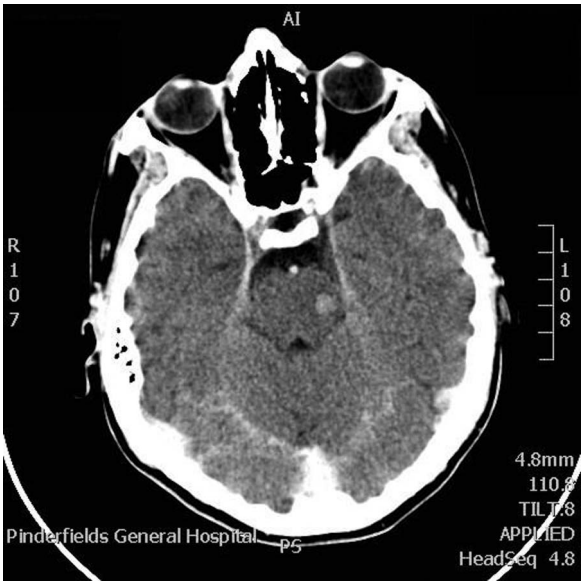

Figure 3 CT of cerebral granuloma.

Learning points

- Symptoms of haematuria, loin pain and acute renal failure raise differential diagnoses of urinary tract infection and renal calculi but have a high clinical index of clinical suspicion for tuberculosis (TB) and can also be a presenting feature of malignancy.
- Urogenital TB occurs in 2–20% of pulmonary TB cases.
- Miliary TB requires at least 12 months of treatment, longer if complicated as above.

the maintenance phase in some cases.[7] The WHO guidelines recommend a reduction in the treatment time to nine or six with four drugs. In complicated cases it can extend to 12–24 months.[8]

Competing interests None.

Patient consent Obtained.

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REFERENCES

1 [No authors listed]. WHO global tuberculosis control report 2010. Summary. *Cent Eur J Public Health* 2010;18:237.
2 Sharma K, Mohan A, Sharma A. Challenges in the diagnosis and management of military tuberculosis. *Indian J Med Res* 2012;135:703–30.
3 Maartens G, Willcox PA, Benatar SR. Miliary tuberculosis: rapid diagnosis, hematologic abnormalities, and outcome in 109 treated adults. *Am J Med* 1990;89:291.
4 Kim JH, Langston AA, Gallis HA. Miliary tuberculosis: epidemiology, clinical manifestations, diagnosis, and outcome. *Rev Infect Dis* 1990;12:583.
5 Sharma SK, Mohan A, Sharma A, *et al*. Miliary tuberculosis: new insights into an old disease. *Lancet Infect Dis* 2005;5:415–30.
6 Figueiredo AA, Lucon AM, Junior RF, *et al*. Epidemiology of urogenital tuberculosis worldwide. *Int J Urol* 2008;15:827–32.
7 Figueiredo AA, Lucon AM. Urogenital tuberculosis: update and review of 8961 cases from the world literature. *Rev Urol* 2008;10:207–17.
8 Kulchavenya E. Best Practice in the diagnosis and management of urogenital tuberculosis. *Ther Adv Urol* 2013;5:143–51.

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