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Massive cerebral tuberculomas, Pott's disease and hypercalcaemia secondary to *Mycobacterium bovis* in a patient with chronic kidney disease on peritoneal dialysis

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SUMMARY

Tuberculosis (TB) is still a health problem in developing countries. Pulmonary involvement remains the most common clinical presentation. However, multiorgan involvement can be life-threatening. We present the case of a young woman on peritoneal dialysis who was admitted to hospitalisation for hypercalcaemia and low back pain. In his biochemical evaluation, suppressed intact parthyroid hormone (iPTH) and elevated 1,25-hydroxyvitamin D were detected. On a lumbar CT scan, a hypodense lesion in vertebral bodies compatible with Pott's disease was found. Positive cultures for *Mycobacterium bovis* were obtained in bronchoalveolar lavage and peritoneal fluid, for which specific treatment was initiated. Due to neurological deterioration, a CT scan was performed showing the presence of multiple tuberculomas. Retrospectively, the lack of an etiological diagnosis of chronic kidney disease, the initiation of dialysis 8 months before and the clear evidence of long-standing TB strongly suggest mycobacterium infection as the cause or trigger for the rapid decline in kidney function.

BACKGROUND

According to the Global Tuberculosis Report 2022, an estimated 10.6 million people worldwide were diagnosed with tuberculosis (TB) in 2021 and 1.4 million people died from the disease. In 2020 and 2021, one-quarter of the world's population is estimated to be infected with *Mycobacterium tuberculosis (M. tuberculosis)*.[1] However, while *M. tuberculosis* is the main infectious agent causing TB, cattle are the primary host of *Mycobacterium bovis (M. bovis)*, so they have a zoonotic risk. Most research has been conducted in developing nations wherein human interaction with animal species lacking a proper bovine TB registration system significantly elevates the contagion risk.[2] In some developed countries, such as the UK, there has been a substantial reduction in the incidence of *M. bovis* since the second half of the 20th century. The first intervention has been related to the pasteurisation of milk since 1930[3 4] and the second is the screening of cattle herds with the tuberculin test.[5] Compared with *M. tuberculosis*, *M. bovis* has a wider spectrum of hosts. Infections have been documented in most domestic animals and many wild mammals.[6 7] It is widely accepted that all terrestrial mammals are vulnerable to infection.

Nonetheless, the severity of the illness will be contingent on innate immunity, immunological memory and pathogenic burden.[6] From 2009 to 2019, a global prevalence rate of *M. bovis* infection in humans of 12% has been estimated.[8] The main transmission routes of *M. bovis* include uncooked meat, unpasteurised milk and contact with livestock waste.[9]

When the body is infected with TB bacteria, the immune system responds by creating an inflammatory process. This process results in the formation of a granuloma, a mass of cells that encloses the infected cells and slows down the replication of the TB bacteria, thereby leading to latent infection. However, in individuals with weakened immune systems, the immune response can continue and progress to active primary TB disease. This can cause tissue destruction in the lungs and spread TB bacteria to other body parts.[10]

TB caused by *M. bovis* is clinically indistinguishable from TB caused by *M. tuberculosis*. Although in countries where bovine TB is uncontrolled, extrapulmonary forms are more frequent.[11] Classical manifestations include pulmonary disease which can be suspected in patients with productive cough, haemoptysis, fever and weight loss, who have a history of infection or contact with patients with TB. In areas where TB is common, Ziehl-Neelsen stain can be used for microbiological diagnosis. The staining of sputum, lavage or bronchioalveolar aspirate specimens has been reported to have a sensitivity of 70%. Although culture is still considered the most reliable method in these countries, it has the disadvantage of taking 2–6 weeks to yield results which delays prompt treatment.[12] Fortunately, molecular tests such as mycobacterial nucleic acid amplification tests are available to diagnose active TB. In particular, the mycobacterial direct amplification and molecular mycobacterium/rifampicin resistance (MTB/RIF) tests diagnose TB and provide information on drug sensitivity including rifampicin.[1]

The purpose of this document is to describe a case of disseminated TB in a patient with chronic kidney disease (CKD) who initially presented with asymptomatic hypercalcaemia.

CASE PRESENTATION

A woman in her 30s on peritoneal dialysis (PD) was diagnosed with CKD 8 months before her first hospital admission without a specific examination to determine aetiology. She was then referred to our medical centre to begin a renal transplant protocol. During the biochemical examination at the nephrology outpatient clinic, hypercalcaemia(total calcium [tCa]: 11.9 mg/dL, ionised calcium [iCa] 1.68 mmol/L), mild lumbar ache and signs of malnutrition were found leading to her hospitalisation. On admission, the patient's vital signs were within normal range with a weight of 48 kg and a height of 1.70 m. An abdominal examination revealed the presence of a technically well-placed catheter without infection or dysfunction during PD exchanges. Laboratory tests on admission were unremarkable except for hypercalcaemia, hypoalbuminaemia (2.7 g/dL), anaemia (haemoglobin 8.9 g/dL) and mild lymphopenia ($0.69\times10^3/mm^3$), complement C3/C4 were normal, HIV serology test and all tested autoantibodies (ANAs, dsDNA, SSA, SSB, PR3, MPO) were negative.

INVESTIGATION

The parathyroid hormone (PTH) levels were properly suppressed (4.77 pg/mL) ruling out primary hyperparathyroidism. Levels of parathyroid hormone-related peptide (PTHrp) were absent, but levels of 1,25-hydroxy vitamin D were found to be abnormally high (105 pg/mL, normal range: 18–65 pg/mL). Management was initiated without hyperhydration due to anuria and was maintained with low osmolarity dextrose PD exchanges with an improvement of hypercalcaemia but without achieving normal levels.

Due to persistent lumbar pain, a lumbar MRI revealed hypointense lesions in L2-L3 vertebral bodies (compatible with Pott's disease) extending to the surrounding soft tissues (see figure 1), calcified thoracic lymphatic nodules and hepatomegaly.

A few days after admission, the patient began with a non-productive cough and slight oxygen desaturation (SO2 90%). Physical examination of the chest revealed bilateral crepitating pulmonary rales. The chest CT scan revealed bilateral diffuse micronodular lesions. Subsequently, a bronchoscopy with bronchial lavage was performed and polymerase chain reaction (PCR) testing along with a positive culture for *M. bovis* confirmed TB. Treatment was initiated with a combination of isoniazid, rifampin, pyrazinamide and ethambutol as a combined pill (the only anti-tuberculous drug available in the hospital during the patient's illness). After a thorough evaluation, the medical team concluded that the patient was in a stable condition and, therefore, be discharged from the hospital.

Unfortunately, 1 month later, the patient presented at the emergency department due to abdominal pain and catheter dysfunction. The peritoneal effluent was cloudy with 380 cells/mm^3, lymphocyte predominance (70%), normal proteins (2.5 g/dL) and low glucose (58 mg/dL). PD fluid cultures were obtained and PD exchanges were temporarily halted. The patient experienced acute confusional syndrome and episodes of psychotic breaks hours after hospitalisation. These episodes included aggression and self-harm alternating with periods of indifference to the environment. A lumbar puncture was performed and the central nervous system (CNS) fluid showed 154 cells/mm^3, lymphocyte predominance (60%), hyperproteinorrachia (360 mg/dL) and a cerebrospinal fluid (CSF)/blood glucose ratio of 0.28. CT scans of the brain detected multiple lesions in the cerebral cortex compatible with disseminated tuberculomas (see figure 2). *M. bovis* was also found in the PCR analysis of cerebrospinal and peritoneal fluid cultures.

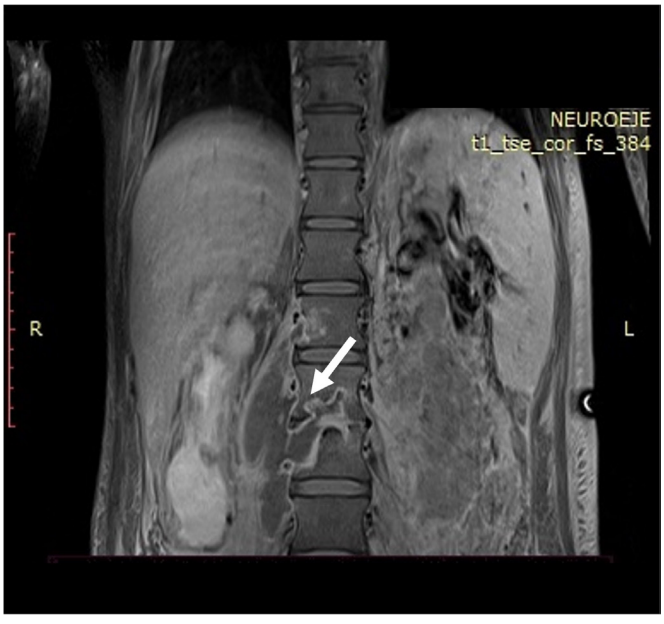

Figure 1 MRI of the lumbar spine showed extensive lesions in vertebrae L2-L3 that invade adjacent soft tissues (arrow) compatible with Pott's disease due to *Mycobacterium bovis*.

DIFFERENTIAL DIAGNOSIS

The present case shows several clinical problems to be addressed. In the first place, hypercalcaemia was the main reason for the first admission to the hospital. The initial step will always be to verify that iCa levels are elevated, then to quantify iPTH because primary hyperparathyroidism must be ruled out. In the present case, iPTH levels were adequately suppressed, so the analysis of PTHrp is usually the next step to consider. Due to the absence of PTHrp to explain hypercalcaemia, the differential diagnosis undoubtedly lies in pathologies with overproduction of 1,25-hydroxy vitamin D such as granulomatous diseases (eg, sarcoidosis, lymphoma, TB).[13] Hypercalcaemia in TB is caused by macrophages' extrarenal metabolism of the nutritional 25-hydroxy vitamin D through the alpha-1-hydroxylase pathway. This leads to increased absorption of calcium and phosphate in the enterocyte and increased calcium reabsorption in the renal tubules, ultimately resulting in hypercalcaemia.[14]

Within the differential diagnoses considered during the approach to this patient, focusing on neurological manifestations, given the characteristics of our population the possibility of TB in the CNS should be considered. Clinical manifestations may begin with a prodrome of up to 4 weeks with fatigue and general malaise. Alterations in CSF circulation are common, resulting in the presence of hydrocephalus, lethargy, loss of consciousness and seizures. The results of CSF studies often show decreased glucose levels and increased levels of proteins, lactate and lymphocytes. It is worth noting that mycobacterial cultures are essential for diagnostic confirmation. Contrast-enhanced MRI is considered the modality of choice for detecting CNS TB due to its superior sensitivity and specificity compared with CT.[15]

TREATMENT

During her first hospitalisation, the treatment of hypercalcaemia represented a challenge due to the relative contraindication for

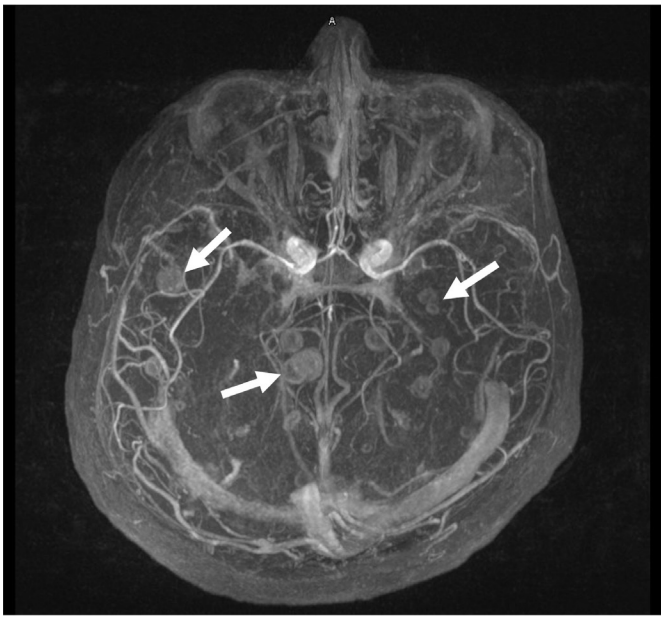

Figure 2 Brain CT scan showing multiple rounded masses distributed throughout the cerebral cortex (arrows) compatible with multiple tuberculomas.

the use of high doses of fluids due to pre-existing nephropathy. It was decided to adjust the PD scheme to three daily exchanges with 1.5% dextrose solution obtaining a gradual improvement in iCa in 5 days (from 1.68 mmol/L to 1.20 mmol/L). Once *M. bovis* infection was diagnosed, treatment was started with a combined pill of rifampicin 150 mg, pyrazinamide 400 mg, ethambutol 300 mg and isoniazid 75 mg 4 tablets daily for 6 days a week during the induction phase. Once tuberculomas were detected, and due to the aggressive clinical presentation, the evidence of potential efficacy and synergy in cases of *M. bovis* infection and the absence of susceptibility report at that moment, intravenous levofloxacin (750 mg once a day) and linezolid (600 mg two times a day) were prescribed until the susceptibility analysis was available plus intravenous dexamethasone 20 mg daily for 7 days. Due to the isolation of mycobacteria in the peritoneal effluent, it was decided to suspend PD and place a catheter for haemodialysis; however, given the nutritional status and the low biochemical requirement, it was maintained with one conventional haemodialysis session per week.

OUTCOME AND FOLLOW-UP

Unfortunately, the patient's neurological condition worsened and required mechanical ventilation. Despite efforts to improve his haemodynamic status, the patient died a few hours later.

DISCUSSION

M. bovis is the primary causative agent of TB in cattle and several other domestic and wild mammalian species characterised by a chronic and progressive respiratory disease.[16 17] Although *M. tuberculosis* primarily affects humans, *M. bovis* has a broader host range that includes humans. In developed countries, *M. bovis* infections account for a relatively small proportion (0.5–7.2%) of patients with confirmed TB diagnoses.[18] By contrast, in developing countries, *M. bovis* infections are likely a significant public health concern;[11 19] unfortunately, due to the shortage of laboratories capable of isolating and distinguishing this organism from other *M. tuberculosis* complex bacteria, it is difficult to ascertain the proportion of human TB cases attributable to *M. bovis* in most developing countries. However, it is believed to be higher in developing countries than in industrialised ones.[20] Some experts have estimated that *M. bovis* could account for 10–15% of the new cases of human TB that occurred in developing countries in the 2000s. Although historical evidence suggests that *M. bovis* does not establish itself in humans as readily as *M. tuberculosis,*[21] it is still infectious for humans. It poses a significant zoonotic risk.[3] Although the patient lived in an urban environment, she had relatives from rural areas who raised farm animals and livestock where she could have acquired the infection.

In a recent meta-analysis involving 1.5 million subjects, it was determined that the incidence of TB in patients with CKD varies widely, ranging from 60 cases per 100 000 in the UK to 19 720 cases per 100 000 in China. The pooled incidence was estimated at 3718 cases per 100 000. Patients undergoing renal replacement therapy, such as haemodialysis or PD, had a higher incidence of TB than predialysis patients (5611/100 000 and 2700/100 000 vs 913/100 000, respectively). It is important to note that individuals with CKD are more likely to have extrapulmonary disease.[22] The pooled prevalence of latent TB in patients with CKD, obtained from 53 studies and 12 772 subjects, was found to be 30.3% (95% CI 25.5% to 34.8%). The prevalence in predialysis patients was 17%, in haemodialysis patients 34.8% and in PD patients 25%.[23]

It is known that patients with TB can experience hypercalcaemia usually caused by an increase in the expression of extrarenal alpha-1-hydroxylase from macrophages present in granulomas.[14] This enzyme promotes the formation of 1,25-dihydroxy vitamin D and calcium reabsorption from the intestinal mucosa. Hypercalcaemia can cause haemodynamic acute renal injury by inducing vasoconstriction of the afferent arteriole. Additionally, it can induce polyuria by reducing the osmolarity of the renal medulla which impedes the normal mechanism of urinary concentration.[24]

Among the extrapulmonary manifestations in PD patients, peritonitis undoubtedly stands out as a complication with prognostic implications. In an analysis of Thomson *et al*, which reviewed 216 cases diagnosed with *M. tuberculosis* peritonitis, a significant delay in diagnosis of up to 6 weeks was reported. Risk factors for mortality included age (> 50 years), male gender and the need for PD catheter removal.[25]

While TB is a rare cause of infectious peritonitis (< 3%), descriptions of peritonitis caused by *M. bovis* are not extensive with clinical cases reported in patients with cirrhosis or mimicking abdominal cancer but without any report in the literature to our knowledge of peritonitis by *M. bovis* in a patient undergoing PD.[26–29]

Rohit and Abraham analysed 92 peritoneal effluents from patients with continuous ambulatory peritoneal dialysis (CAPD) or automated peritoneal dialysis (APD) showing a 6.52% rate of tuberculous peritonitis.[29] Abraham *et al* found that molecular diagnostic techniques produced 82% of positive cases while only 20% were positive through peritoneal fluid cultures and smears.[30] In another analysis of 50 cases of tuberculous peritonitis in patients with CAPD, a 4-week treatment delay was a determining factor for patient clinical outcomes including mortality.[31]

Causes predisposing patients with PD to tuberculous peritonitis include cellular immunity, high glucose concentration in dialysis fluid and alterations in peritoneal pH that

can induce phagocytosis deficit.[32] The dissemination route is usually haematogenous with the reactivation of latent tuberculous foci in the peritoneum being common and rarely translocation from the infected intestine or salpingitis.[33] Regarding treatment, it has been reported that treatment of tuberculous peritonitis lasting less than 12 months can increase the risk of recurrence.[29 32]

Among patients with TB, less than 2% show CNS involvement. Tuberculomas are an infrequent manifestation in developed countries and somewhat more common in developing countries. Tuberculomas are slow-growing lesions with varied clinical manifestations, typically involving altered mental status and seizures.[34] In the present clinical case, the massive and unreported presence, in the literature to our knowledge, of disseminated cerebral tuberculomas stands out explaining the patient's episodes of psychosis and seizures during the latter period of her hospitalisation. The CT scan showed lesions along the entire cerebral cortex.

In an interesting analysis, González-Duarte *et al* evaluated 64 patients without CKD diagnosed with CNS TB from 1999 to 2009. 44% of patients had neurological symptoms as the only manifestation of TB, 38% had neurological and systemic symptoms and 18% had neurological symptoms within 1 month of diagnosis of systemic TB. Only 32% of the patients had focal neurological symptoms, such as stroke, due to tuberculomas. 84% of the cases had a positive culture. 46 were caused by *M. tuberculosis* and only 8 cases were positive for *M. bovis*. Interestingly, isolation of *M. bovis* was significantly associated with brain lesions (p=0.03) and neurological sequelae (p=0.02) in the multivariate analysis.[35] Although the outcomes of *M. bovis* tuberculomas in dialysis patients have not been published, results like those reported by González-Duarte *et al* are expected, especially due to the high comorbidity accompanying patients on kidney replacement therapy.

Another interesting aspect of the present case is the 'undetermined' aetiology of CKD. The patient's residence in an endemic area for TB and the short course of chronic nephropathy with a fatal outcome suggests a strong interrelation between the two pathologies. The variety of renal conditions related to TB has been widely described, including immunoglobulin A nephropathy,[36 37] pauciimmune antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis,[38 39] ANCA-negative crescentic glomerulonephritis,[40 41] collapsing glomerulopathy,[42 43] membranous nephropathy,[44] double-positive Goodpasture syndrome[45] and membranoproliferative glomerulonephritis with dominant C3 deposits.[46 47] In fact, in patients with TB, progressive deterioration of renal function, proteinuria associated with low serum levels of C3 or isolated glomerular C3 deposits and absence of immunoglobulins or immune complexes by immunofluorescence in the context of C3 glomerulopathy should establish active mycobacterium infection as the primary causal option.

Undoubtedly, the multisystemic and massive expression of *M. bovis* infection reported in this document highlights the importance of addressing each clinical and biochemical condition to integrate differential diagnoses that, if diagnosed promptly, can improve the prognosis of patients in case of an appropriate therapeutic response.

Learning points

- Identifying *Mycobacterium bovis* in developing countries can be a difficult task, and it still poses a zoonotic risk.
- Patients with chronic kidney disease are more likely to have extrapulmonary tuberculosis (TB).
- Increased alpha-1-hydroxylase and 1,25-dihydroxy vitamin D expression in granuloma macrophages may cause hypercalcaemia in TB.
- Cerebral tuberculomas are a rare manifestation of TB, but their clinical impact can be aggressive and potentially lethal.
- TB can cause various histological patterns of glomerulonephritis, potentially leading to loss of renal function if not promptly treated.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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