Case Report

DOI: 10.7860/JCDR/2026/88524.23841

Internal Medicine Section

Silicotuberculosis with Suspected Distal Renal Tubular Dysfunction and Nephrogenic Diabetes Insipidus: A Case with an Unusual Association

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ABSTRACT

Silicosis is a chronic occupational lung disease associated with an increased susceptibility to tuberculosis. However, renal manifestations in the context of silica exposure are uncommon and remain incompletely understood. The present case is of a 52-year-old underground hard rock miner with a history of prolonged silica exposure who presented with fever, cough, progressive breathlessness, polyuria, and lower limb weakness. Radiological evaluation demonstrated bilateral reticulonodular opacities with calcified mediastinal lymph nodes. Tissue GeneXpert testing confirmed *Mycobacterium tuberculosis*, establishing the diagnosis of silicotuberculosis. During hospitalisation, the patient developed severe hypokalaemia with normal anion gap metabolic acidosis, raising suspicion of distal renal tubular dysfunction; however, definitive diagnostic parameters for distal renal tubular acidosis were incomplete. Persistent polyuria with low urine osmolality suggested possible nephrogenic diabetes insipidus, although confirmatory testing could not be performed due to clinical instability. Despite initiation of antitubercular therapy and intensive supportive care, the patient's condition deteriorated with progressive respiratory failure and refractory shock, ultimately resulting in death. This case underscores the diagnostic complexity of silicotuberculosis with concurrent electrolyte and renal abnormalities, while highlighting that the relationship between silica exposure, tuberculosis, and renal tubular dysfunction remains uncertain and likely multifactorial, warranting cautious interpretation.

Keywords: Acidosis, Hypokalaemia, Kidney tubular diseases, Occupational exposure, Tuberculosis

CASE REPORT

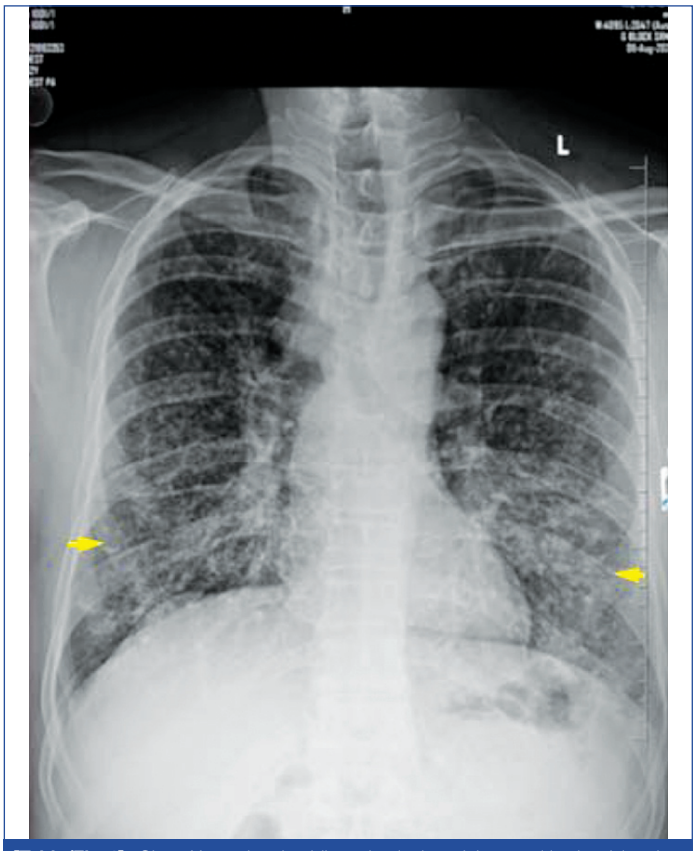
A 52-year-old male presented with a one-month history of productive cough, progressive breathlessness, and low-grade fever. He also complained of excessive thirst, increased urine output, and generalised weakness, particularly involving the lower limbs. There was significant weight loss and anorexia. He had worked as an underground hard rock miner for approximately 30 years with prolonged exposure to silica dust. There was no prior history of diabetes, hypertension, chronic kidney disease, or any significant past medical illness.

On examination, he was tachypnoeic with bilateral diffuse wheeze and coarse crepitations over both lung fields. Oxygen saturation on room air was reduced. Neurological examination revealed muscle power of 4/5 in all limbs with preserved tone and reflexes. There were no focal neurological deficits.

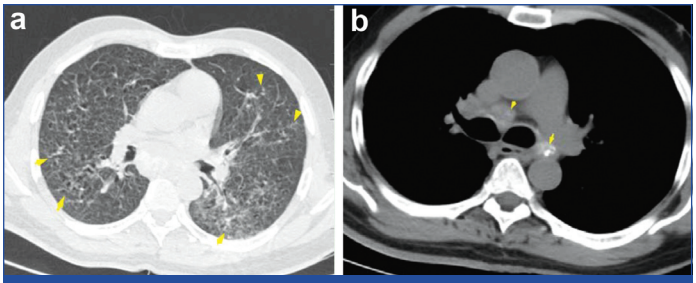
Laboratory investigations showed haemoglobin of 12.2 g/dL, total leucocyte count of 3650/mm^3, and platelet count of 371,000/mm^3. Serum creatinine was 1.8 mg/dL and blood urea nitrogen was 30 mg/dL. Serum sodium was 130 mmol/L, potassium was markedly reduced at 1.2 mmol/L, and bicarbonate was 17 mmol/L, chloride 103 with anion gap of 10 mmol/l. Liver enzymes were elevated (AST and ALT 144 IU/L each) with normal serum albumin. Chest radiograph demonstrated bilateral reticulonodular opacities involving the middle and lower zones [Table/Fig-1].

High-Resolution Computed Tomography (HRCT) of the thorax revealed diffuse centrilobular nodules with a tree-in-bud pattern, septal thickening, and calcified mediastinal lymphadenopathy [Table/Fig-2]. Angiotensin Converting Enzyme (ACE) and calcium levels were within normal limits.

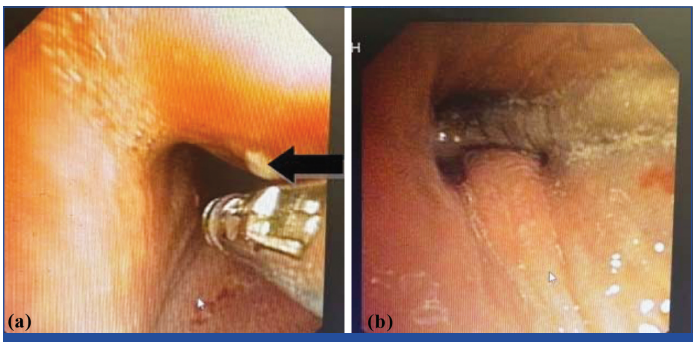
Initial sputum smear for acid-fast bacilli and GeneXpert were negative. Given persistent clinical suspicion, bronchoscopy with endobronchial ultrasound was performed. Anthracotic nodules were visualised [Table/Fig-3]. Tissue samples obtained during the procedure tested positive for *Mycobacterium tuberculosis* (low bacillary load) on GeneXpert assay, confirming silicotuberculosis. Standard antitubercular therapy was initiated with dose adjustment for renal function.



[Table/Fig-1]: Chest X-ray showing bilateral reticulonodular opacities involving the middle and lower zones.



[Table/Fig-2]: HRCT thorax a) Showing diffuse centrilobular nodules with tree-in-bud appearance; b) Calcified mediastinal lymphadenopathy.



[Table/Fig-3]: Bronchoscopic image showing anthracotic nodules within the bronchial tree a) Left main bronchus- necrotic nodule; b) Right upper lobe, anthracotic mucosa.

The patient's hospital course was complicated by profound hypokalaemia requiring aggressive intravenous potassium replacement. A spot urinary potassium level of 10.2 mmol/L was noted; however, this value alone was insufficient to definitively establish renal potassium loss in the absence of supportive indices such as urine potassium-to-creatinine ratio or Transtubular Potassium Gradient (TTKG). Therefore, the exact mechanism of potassium loss remains uncertain.

Arterial blood gas analysis confirmed metabolic acidosis. A urinary bicarbonate level <2 mmol/L and calcium/creatinine ratio of 0.2 suggested possible distal tubular dysfunction. However, key diagnostic parameters required to definitively establish distal renal tubular acidosis- including urine pH (expected >5.5 in systemic acidosis), urine anion gap, fractional excretion of potassium, and imaging evidence of nephrocalcinosis- were not available. Hence, the diagnosis of distal renal tubular acidosis remains probable but not definitive.

Extensive evaluation for secondary causes was undertaken. There was no history of offending drugs known to cause tubular dysfunction. Autoimmune work-up, including a comprehensive panel, was negative. However, it remains unclear whether the renal dysfunction was directly attributable to silica exposure, secondary to tuberculosis-related tubulointerstitial involvement, or a consequence of severe hypokalaemia itself. Thus, the causal relationship remains speculative and should be interpreted with caution.

Despite correction attempts, the patient continued to have persistent polyuria exceeding three litres per day with low urine osmolality and relatively elevated serum osmolality, raising suspicion of nephrogenic diabetes insipidus. However, confirmatory testing-including desmopressin response test, urine specific gravity measurement, serum sodium trend analysis, and Antidiuretic Hormone (ADH) levels- was not performed. Although a water deprivation test could not be completed due to clinical instability, this limitation has been appropriately acknowledged. Therefore, the diagnosis of nephrogenic diabetes insipidus remains suggestive but not definitive.

Respiratory status progressively worsened, necessitating non invasive ventilation and later intensive care admission. The patient developed hypotension requiring multiple inotropes and subsequently suffered cardiac arrest. Resuscitative efforts were unsuccessful.

DISCUSSION

Silicosis remains a significant occupational health concern, particularly among workers in mining and quarrying industries. Chronic inhalation of crystalline silica leads to persistent pulmonary inflammation, fibrosis, and impaired macrophage-mediated immunity, predisposing affected individuals to tuberculosis [1-3]. Silica particles induce macrophage apoptosis and impair cell-mediated immune responses, thereby facilitating reactivation or acquisition of *Mycobacterium tuberculosis* infection [1,3]. Patients with silicosis have been reported to have a two- to threefold higher risk of developing tuberculosis compared with the general population, as demonstrated in epidemiological studies by Ehrlich R et al., and Cowie RL [1,2]. Consequently, silicotuberculosis continues to represent an important and often underrecognised clinical entity, especially in developing countries [1,4].

Radiological differentiation between silicosis and pulmonary tuberculosis can be challenging because both conditions may present with diffuse nodular opacities and chronic respiratory symptoms [1,5]. The presence of calcified mediastinal lymph nodes, diffuse centrilobular nodules, and tree-in-bud appearance in a patient with prolonged occupational silica exposure should raise strong suspicion for silicotuberculosis [1,5]. In such cases, microbiological confirmation may be difficult, and tissue diagnosis is often required [4].

Renal involvement in silica exposure is increasingly recognised but remains underreported. Several studies have demonstrated an association between silica exposure and renal disease, including both glomerular and tubular pathology [3,6]. Proposed mechanisms include silica-induced autoimmune activation, chronic inflammatory injury, and direct tubular toxicity [3,6]. Experimental and clinical studies by Pollard KM have demonstrated that silica particles may act as immunological adjuvants, promoting autoantibody formation and immune-mediated renal injury [3]. In addition, chronic tubulointerstitial nephritis may develop secondary to persistent inflammatory activation [6]. Calvert GM et al., also reported increased mortality from renal diseases among silica-exposed workers, further supporting a possible association between silica exposure and renal dysfunction [7].

Tuberculosis itself can contribute to renal dysfunction through granulomatous interstitial nephritis, tubulointerstitial inflammation, and structural renal damage [8]. In rare instances, renal tuberculosis may manifest with distal renal tubular acidosis, characterised by impaired distal urinary acidification leading to metabolic acidosis and severe hypokalaemia [6,8]. Tubulointerstitial damage from chronic infection or granulomatous inflammation may additionally impair urinary concentrating ability and contribute to nephrogenic diabetes insipidus [8]. In the present case, the exact mechanism of renal tubular dysfunction remains uncertain and was likely multifactorial. The abnormalities may have resulted from silica-related tubular injury, tuberculosis-associated interstitial nephritis, severe hypokalaemia itself, or a combination of these factors [3,6,8].

The diagnosis of distal renal tubular acidosis and nephrogenic diabetes insipidus in this patient remained suggestive rather than definitive because complete confirmatory investigations could not be performed due to clinical instability. Nevertheless, persistent polyuria with low urine osmolality and severe refractory hypokalaemia strongly suggested underlying tubular dysfunction. Severe hypokalaemia is itself a recognised cause of reversible nephrogenic diabetes insipidus through downregulation of aquaporin-2 channels in the collecting ducts, thereby impairing water reabsorption [7]. Such electrolyte abnormalities may precipitate life-threatening complications including cardiac arrhythmias, respiratory muscle weakness, and haemodynamic instability [6,8].

To the best of current knowledge and available indexed literature, no well-documented cases of silicotuberculosis presenting simultaneously with suspected distal renal tubular acidosis and

nephrogenic diabetes insipidus have been reported [1,6]. This case therefore contributes to the limited literature describing multisystem manifestations of silica exposure and tuberculosis. It also underscores the importance of maintaining a high index of suspicion for renal involvement in patients with silicotuberculosis who develop unexplained metabolic acidosis, hypokalaemia, or persistent polyuria [6,8]. Early recognition and comprehensive metabolic evaluation may help prevent severe complications and improve clinical outcomes. However, given the incomplete diagnostic workup and overlapping pathophysiological mechanisms, the causal relationships should be interpreted cautiously.

CONCLUSION(S)

Silicotuberculosis presents substantial diagnostic challenges because of overlapping clinical and radiological manifestations. This case highlights a possible association between silica exposure, tuberculosis, and renal tubular dysfunction, although causality could not be definitively established due to incomplete diagnostic confirmation. In patients with prolonged silica exposure who develop unexplained metabolic acidosis, hypokalaemia, or polyuria, renal tubular abnormalities should be considered. Further studies are required to better elucidate the relationship between silica exposure and renal injury.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Mar 22, 2026
- Manual Googling: May 19, 2026
- iThenticate Software: May 22, 2026 (6%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Feb 25, 2026**
Date of Peer Review: **Apr 15, 2026**
Date of Acceptance: **May 25, 2026**
Date of Publishing: **Jul 01, 2026**