International Journal of Research in Medical Sciences
Kumar A et al. Int J Res Med Sci. 2026 Aug;14(8):3615-3620
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20262658

Case Report

Primary adrenal insufficiency secondary to ileocecal tuberculosis

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Received: 18 May 2026
Revised: 14 June 2026
Accepted: 17 July 2026

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ABSTRACT

Ileocecal tuberculosis, the most common form of gastrointestinal tuberculosis, is usually caused by Mycobacterium tuberculosis, which is also the leading infectious cause of adrenalitis. Adrenal involvement can result in primary adrenal insufficiency, presenting with weakness, fatigue, hypotension, electrolyte imbalance, and hyperpigmentation. We present the case of a 43-year-old male with recurrent vomiting, fatigue, orthostatic dizziness, and generalized hyperpigmentation. His blood pressure was 100/80 mmHg, and laboratory tests showed low serum cortisol, elevated ACTH, hyponatremia, and hyperkalemia—findings consistent with primary adrenal insufficiency. Imaging revealed bilateral adrenal enlargement, a thickened ileocecal junction, and enlarged mesenteric lymph nodes. Colonoscopy demonstrated a terminal ileal stricture with granulomatous inflammation, and adrenal biopsy showed caseous necrosis. GeneXpert confirmed rifampicin-sensitive M. tuberculosis, indicating disseminated tuberculosis originating from the ileocecal region. The patient was started on standard anti-tubercular therapy with corticosteroid replacement for Addison's disease. This case report highlights disseminated tuberculosis presenting as primary adrenal insufficiency secondary to ileocecal tuberculosis, an uncommon yet clinically significant association. Adrenal tuberculosis should be suspected with hyperpigmentation, electrolyte imbalance, and bilateral adrenal lesions; early diagnosis enables therapy, preventing adrenal crisis.

Keywords: Disseminated tuberculosis, Adrenal tuberculosis, Addison's disease, Ileocecal tuberculosis, Mycobacterium tuberculosis

INTRODUCTION

Tuberculosis, caused by Mycobacterium tuberculosis, is an aerobic, non-spore-forming, non-motile, acid-fast bacillus. India accounts for nearly one-eighth of the global tuberculosis burden, making it a major cause of morbidity and mortality.[1] The disease manifests variably depending on the site of infection. Ileocecal tuberculosis, the most common gastrointestinal form, constitutes 1–3% of all cases and may occur with or without pulmonary involvement. Infection usually follows ingestion of contaminated sputum or fluids, while Mycobacterium bovis may be transmitted through infected dairy products.[2] Patients may present with abdominal pain, anorexia, fever, altered bowel habits, or melena, along with weight loss, pallor, anemia, lymphadenopathy, and splenomegaly.[3] GeneXpert Mycobacterium tuberculosis / Rifampicin testing helps distinguish intestinal tuberculosis from Crohn's disease.[4]

Primary adrenal insufficiency (Addison's disease) is a rare endocrine disorder with an incidence of about four per million per year.[5] It results from destruction of the adrenal cortex, causing glucocorticoid and mineralocorticoid deficiency. Common causes include autoimmune disease, congenital adrenal hyperplasia, hemorrhage, and infections.[6] Though rare, M. tuberculosis remains the most frequent infectious cause.[7] We report a case of primary

adrenal insufficiency secondary to adrenal tuberculosis as part of disseminated infection from the ileocecal region.

CASE REPORT

A 43-year-old with no prior medical history presented with symptoms of recurrent vomiting, generalized fatigue, orthostatic dizziness, clinically significant weight loss (≥5% of baseline weight), and progressive hyperpigmentation involving the face, palmar creases, and oral mucosa. There was no history of diabetes mellitus, autoimmune disease, chronic steroid use, malignancy, or prior tuberculosis. The patient denied alcohol use or intravenous drug use. Nutritional assessment revealed mild undernutrition (BMI 18.6 kg/m²).

On examination, the patient was dehydrated, with diffuse hyperpigmentation. Blood pressure was 100/80 mmHg supine, with a postural drop to 86/60 mmHg on standing. Pulse rate was 96/min. There was no lymphadenopathy or organomegaly. Respiratory and cardiovascular examinations were unremarkable.

Laboratory investigations given in Table 1. The severely low cortisol with markedly elevated ACTH, along with hyponatremia and hyperkalemia, was diagnostic of primary adrenal insufficiency. ACTH stimulation testing was not pursued because it would not change immediate management and might postpone life-saving therapy.

Table 1: Laboratory investigation.

Parameters	Value	Reference range
Serum cortisol	29.4 nmol/l	140-190 nmol/l
Plasma acth	78 poml/l	7-11 pmol/l
Serum sodium	109 mmol/l	135-145 mmol/l
Serum potassium	6.5 mmol/l	3.5-5.1 mmol/l
Hiv-1 and 2 serology	Negative	-
Ana	Negative	-

An ultrasound of the abdomen revealed an enlarged right adrenal gland, suggesting a CECT for further evaluation. A CECT scan of the abdomen and pelvis was performed, revealing bulky bilateral adrenal glands (R>L), with few thin-walled hypodense lesions, the largest in the right measuring 2.8 x 2.7 cm and on the left measuring 2.2 x 1.4 cm (Figure 1). These lesions, on post contrast showed minimal peripheral enhancement, collapsed Ileocecal junction and distal ileal loop appears with wall thickening, with maximum thickness of 8.7 mm, for a length of ~ 4 cm. Multiple enlarged mesenteric lymph nodes seen in right iliac fossa, largest 13x10 mm along with multiple scattered mesenteric lymph nodes/nodules are seen an enlarged left external iliac lymph node measuring 12x10 mm was observed all these findings was suggestive of multi system involvement of tuberculosis (Figure 2). These findings suggested multisystem involvement, raising suspicion of disseminated tuberculosis.

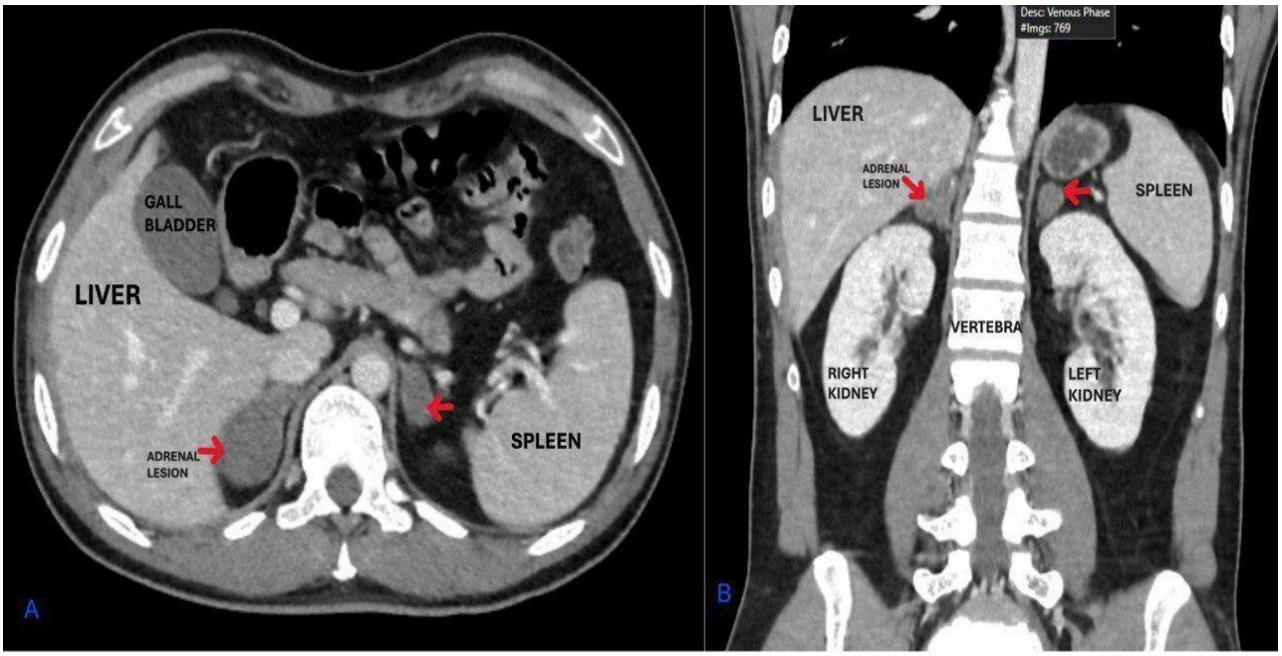

Figure 1: CT images highlighting adrenal gland enlargement. A) Axial CT shows bilateral adrenal glands appearing bulky (Right > Left). B) Coronal CT of the adrenal glands.

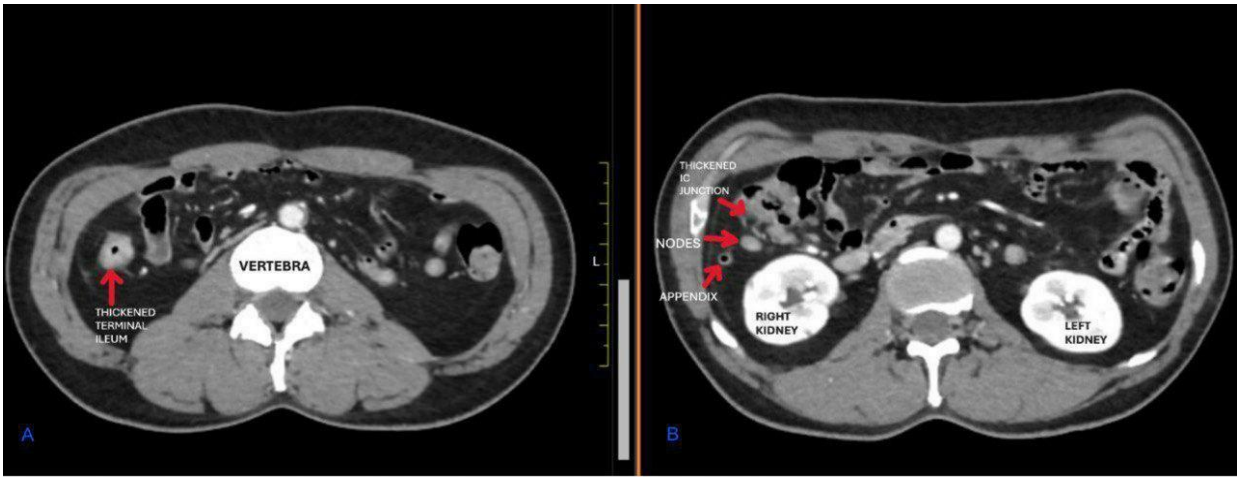

Figure 2: Axial CECT images. A) Axial CECT image showing thickened ileum thickness of 8.7 mm. B) Axial CECT image showing external iliac lymph node measuring 12x10 mm.

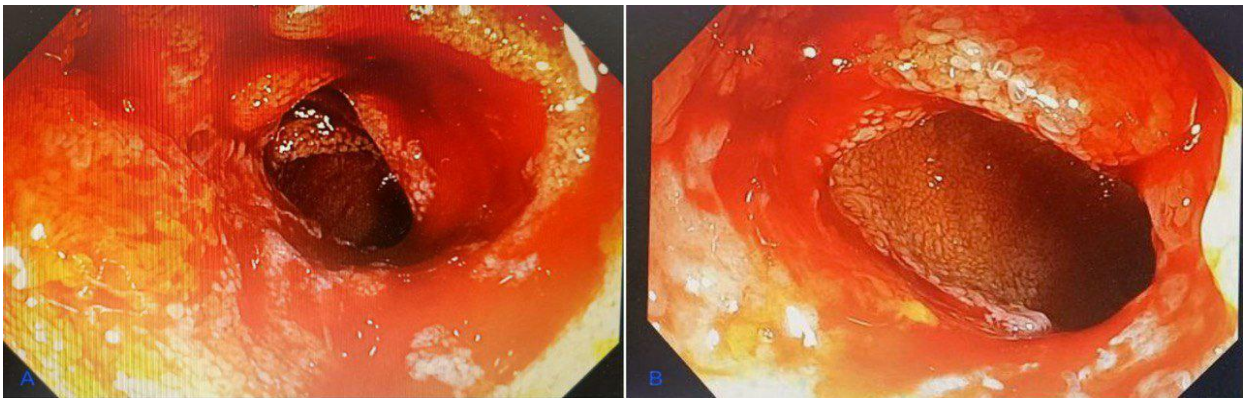

Figure 3: Colonoscopy findings. A) Colonoscopy image showing terminal ileum : 3 cm from the ileocaecal valve mucosa appears ulcerated with circumferential narrowing noted. B) Biopsy taken from the ulcer and around the stricture area.

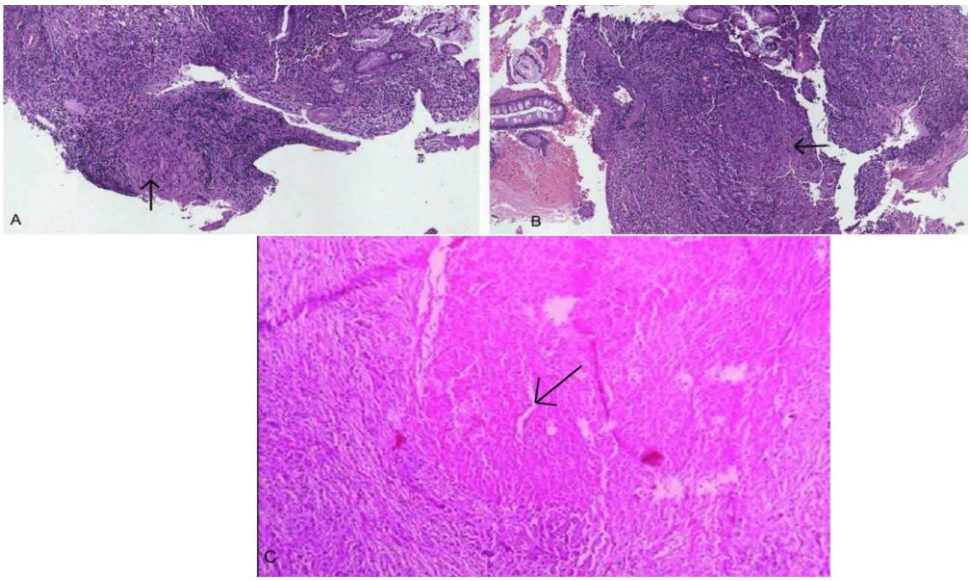

Figure 4 (A, B): Histopathology findings reveal an ileocaecal biopsy (H&E, X400) showing well-defined granuloma with giant cell in lamina propria. C) The right adrenal gland biopsy shows a granulomatous lesion with caseous necrosis.

Given these findings, a medical gastroenterology consultation was requested, and a colonoscopy and biopsy were conducted (Figure 3). The colonoscopy findings demonstrated a terminal ileal stricture, and subsequent biopsy revealed features of a granulomatous lesion in the ileum, favouring tuberculosis activity, with a score of D2C2A3 (Figure 4A and B). The right adrenal gland biopsy was performed and shows features of a granulomatous lesion with caseous necrosis, a feature characteristic of tuberculous adrenalitis (Figure 4C). Analysis with GeneXpert testing was positive for Mycobacterium tuberculosis, rifampicin-sensitive, indicating tuberculosis as an underlying etiology.

No pulmonary involvement was detected on chest radiography and sputum examination, consistent with extrapulmonary tuberculosis.

The patient was initiated on standard four-drug anti-tubercular therapy consisting of rifampicin (150 mg), isoniazid (75 mg), pyrazinamide (400 mg), and ethambutol (275 mg). Concomitantly, glucocorticoid and mineralocorticoid replacement therapy were started. The patient received intravenous hydrocortisone at a dose of 100 mg, followed by oral hydrocortisone at a maintenance dose of 20 mg per day in divided doses.

Due to persistent hyperkalemia and hypotension, fludrocortisone was added at a dose of 0.1 mg per day. Endocrinology consultation guided long-term steroid replacement therapy, including patient education regarding stress-dose adjustments.

At the three-month follow-up, the patient reported complete resolution of vomiting and dizziness. Serum electrolytes normalized, with sodium and potassium levels returning to the reference range. The patient gained 4 kg and reported a marked improvement in overall energy levels. Hydrocortisone was continued at the maintenance dose, and fludrocortisone was titrated based on blood pressure and electrolyte levels. Anti-tubercular treatment was continued according to national tuberculosis program guidelines.

DISCUSSION

Addison's disease, an adrenal insufficiency caused by inadequate steroid hormone production, may result from autoimmune, infectious, or infiltrative causes. The etiology varies geographically: autoimmune adrenalitis predominates in developed countries, whereas infectious causes—particularly tuberculosis—remain a significant cause in developing regions. Among these, the rare occurrence of Addison's disease secondary to ileocecal tuberculosis caused by Mycobacterium tuberculosis highlights important considerations in pathogenesis and management. This case underscores the rarity and clinical relevance of this association. Tuberculosis (TB) affects multiple organs, but extrapulmonary involvement of the adrenal glands is rarely reported. While adrenal insufficiency associated with active TB is well documented, the mechanism linking ileocecal tuberculosis to adrenal dysfunction remains poorly described. In this case, clinical findings demonstrated an association between ileocecal tuberculosis and adrenal insufficiency, as the patient presented with abdominal symptoms and also showed hormonal evidence of adrenal failure. Diagnosis typically relies on samples from secondary sites—such as sputum, urine, or intestinal biopsy—since isolating bacilli directly from the adrenal glands is often challenging.[8] Currently, the main diagnostic methods used are microscopy, Xpert Tuberculosis/RIF, and NAATs. Imaging CT scan is the mainstay for the diagnosis of gastrointestinal tuberculosis. Findings include asymmetric wall thickness of the terminal ileum, caecum. In chronic conditions, fibrosis and stenosis can be seen.[9] Colonoscopy can be done to obtain a biopsy, and it appears to have 80% diagnostic accuracy.[10] The D2C2A3 histological pattern reflects Degree of inflammation (D), Crypt architecture/Chronicity (C), and Disease Activity (A) on biopsy. While it indicates significant active disease, differentiation between intestinal tuberculosis and Crohn's disease requires additional evaluation of granuloma characteristics, caseation, and Acid-Fast Bacilli positivity, supporting the diagnosis of active intestinal tuberculosis when TB-specific features are present. In case of adrenal gland tuberculosis, CT scan imaging reveals bilateral mass-like enlargement, adrenal calcification, and peripheral rim enhancement with low attenuation in the centre part of the adrenal glands and confirmed by biopsy taken via CT-guided percutaneous fine needle aspiration.[11] As seen in this case, gastrointestinal symptoms preceded, deranged laboratory values, with hyperpigmentation of the skin indicative of adrenal insufficiency.

The pathogenesis of adrenal tuberculosis is believed to involve hematogenous and lymphatic dissemination. Mycobacterium tuberculosis may spread hematogenously from the gastrointestinal tract, leading to miliary seeding of the adrenal glands. Once within adrenal tissue, the bacilli can cause granulomatous inflammation and caseous necrosis, resulting in impaired cortisol production and, in severe cases, adrenal crisis.[12] Moreover, the hematogenous spread of tuberculous bacilli can occur years after the initial infection, complicating the diagnosis and treatment of secondary infections in the adrenal glands, enlarged mesenteric lymph nodes as seen in this case suggests that lymphatic dissemination plays a vital role, whereby Mycobacterium tuberculosis can migrate from intestinal lymph nodes to the adrenal glands through the thoracic duct.[13,14] This mechanism is supported by the interconnectedness of the lymphatic system, which facilitates the transfer of pathogens between regions. Another theory suggests that hematogenous spread may occur after initial lymphatic involvement, allowing the bacteria to breach vascular barriers and reach distant organs, including the adrenals.[15] Furthermore, the inflammatory responses triggered in the ileocecal region may enhance lymphatic drainage, potentially increasing the risk of dissemination. The intricate relationship

between tuberculosis and autoimmune diseases, particularly Addison's disease, has garnered significant theoretical attention. One compelling perspective highlights the concept of molecular mimicry, in which the immune response to Mycobacterium tuberculosis may inadvertently target self-antigens, leading to conditions such as Addison's disease. This autoimmune response is thought to occur when the immune system, once activated by TB infection, fails to differentiate between pathogenic and host antigens, resulting in an attack on the adrenal cortex.[16] Supporting this notion, research indicates a strong correlation between TB infections and the subsequent development of autoimmune disorders, suggesting a mechanism where chronic inflammation from TB exacerbates autoimmunity in genetically predisposed individuals.[17,18] Additionally, the role of cytokines in mediating autoimmune responses post-TB infection is noteworthy. Certain inflammatory cytokines released during TB infection may alter immune signalling pathways, thus fostering an environment conducive to autoimmunity.[19]

The differential diagnosis of bilateral adrenal enlargement with adrenal insufficiency includes autoimmune adrenalitis, the most common cause globally, which was excluded in this case due to the absence of autoimmune history and negative autoimmune markers. HIV-associated adrenal insufficiency was ruled out by negative serology, while fungal infections such as histoplasmosis or cryptococcosis were considered less likely given the absence of immunosuppression and the confirmation of tuberculosis. Metastatic malignancy and lymphoma were also unlikely, as imaging and histology favored an infectious etiology. Adrenal hemorrhage was excluded based on the chronicity of presentation and imaging findings.

The coexistence of intestinal tuberculosis, mesenteric lymphadenopathy, characteristic adrenal imaging, and GeneXpert-confirmed tuberculosis provides convincing evidence that tuberculosis was the primary cause of adrenal insufficiency in this patient.

Management of coexisting gastrointestinal and adrenal tuberculosis requires a combined approach by anti-tubercular therapy and steroids. Antitubercular therapy includes a standard four-drug regimen - isoniazid, rifampicin, pyrazinamide, and ethambutol. These four drugs are given thrice weekly for the initial 2 months, and additional four months, isoniazid, rifampicin are given. Response to ATT occurs by mucosal healing, but if any polyps and strictures are present, then other treatment modalities are required. For strictures, endoscopic balloon dilatation is used. Surgical therapy is needed only when there are complications.[20] To treat adrenal insufficiency, glucocorticoid analogues (prednisolone, hydrocortisone, dexamethasone given. Mineralocorticoid analogue (fludrocortisone), androgen analogue (dehydroepiandrosterone).[8]

CONCLUSION

From a public health perspective this case highlights the continued relevance of Tuberculosis as an endocrine cause of motility in endemic regions. Despite of small fraction of adrenal tuberculous cases, delayed diagnosis can result in severe complications. Early detection, expanding molecular diagnosis, improving nutritional support, and ensuring adherence to the National Tuberculosis Elimination Program (NTEP) are essential steps in reducing the disease burden. In Tuberculosis tuberculosis-endemic setting, integration of infectious disease and endocrine perspectives is crucial to facilitate timely recognition of symptoms and prevent life-threatening complications.

ACKNOWLEDGEMENTS

The authors sincerely acknowledge the support of Justice K. S. Hegde Charitable Hospital (KSHEMA), Mangalore.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Kumar A, Pal D, Ojha A. Primary adrenal insufficiency secondary to ileocecal tuberculosis. Int J Res Med Sci 2026;14:3615-20.