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CASE REPORT

Diagnostic Role of Tomography in Addison's Disease due to Adrenal Tuberculosis: A Case Report

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Abstract: ***Introduction*:** Adrenal tuberculosis remains the main cause of primary adrenal insufficiency (PAI) in tuberculosis (TB)-prevalent regions. This case report details the presentation of PAI due to adrenal TB, where the etiological diagnosis involves Abdominal Computed Tomography (CT).

***Case Report*:** A 37-year-old Peruvian woman with a history of TB contact displayed symptoms of adrenal insufficiency. PAI diagnosis was established, and CT imaging unveiled bilateral adrenal enlargement with calcifications. Treatment with prednisone and anti-TB therapy led to symptomatic improvement. Unfortunately, she succumbed to pneumonia after ten months of follow-up.

***Discussion*:** Adrenal TB must be considered in endemic regions and in the presence of a TB history. CT serves as a valuable diagnostic tool, particularly in settings with limited resources, revealing adrenal enlargement and calcifications.

***Conclusion*:** In patients with PAI, epidemiological history of TB, and when a rapid biopsy is not feasible, CT proves to be a valuable diagnostic method.

Keywords: Addison's disease, adrenal gland neoplasms, endocrine tuberculosis, diagnostic imaging, X-ray computed tomography, anti-TB therapy.

1. INTRODUCTION

Primary adrenal insufficiency (PAI), also known as Addison's disease, is an endocrine disorder characterized by an inadequate production of adrenal cortical hormones due to the impairment or direct destruction of the adrenal glands [1, 2]. This condition typically presents between the ages of 20 and 50, showing a higher prevalence in women [1, 3]. The prevalence of the disease ranges from 100 to 140 individuals per million people [4, 5], which has doubled since the last century [6]. The incidence of PAI is 4-6 cases per million adults per year in Western societies [2, 5].

Historically, tuberculosis (TB) was the primary cause of PAI; however, currently, the most prevalent causes are autoimmune adrenalitis in adults, constituting up to 90% of cases in Western countries, and congenital adrenal hyperplasia in children [1, 2]. Although TB continues to be the leading cause in developing countries and/or those with a high prevalence of this infection [1, 7], it typically does not clinically manifest until more than 90% of the adrenal glands have been destroyed [8, 9]. The primary site of TB infection is the lungs, although it can also affect other organs (Fig. **1**) [10]. TB can also involve endocrine glands, including the

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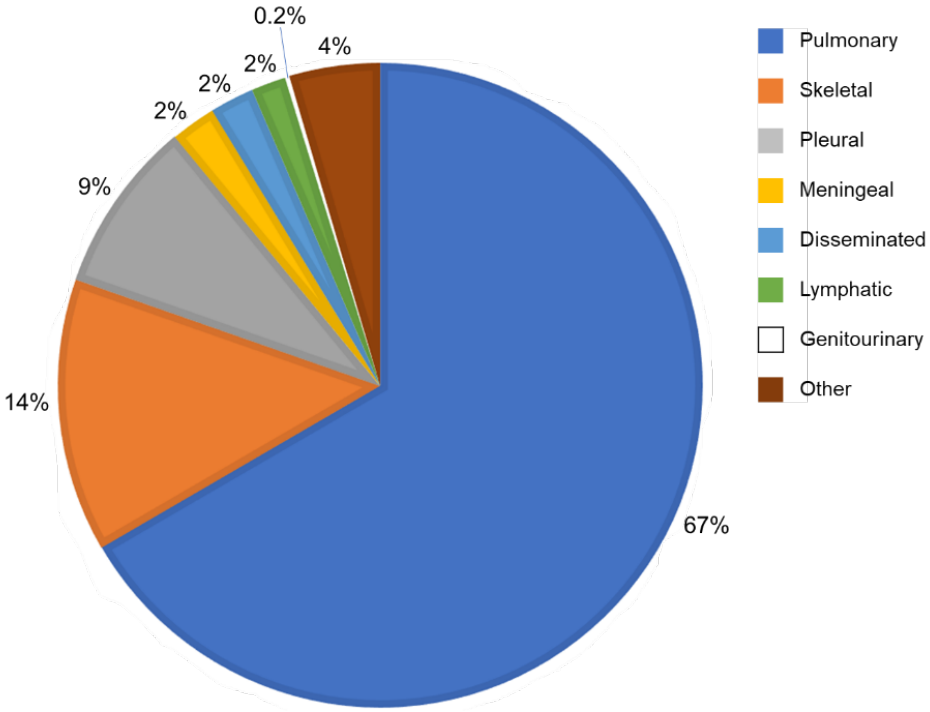

Fig. (1). Distribution of TB infection sites. *(A higher resolution / colour version of this figure is available in the electronic copy of the article).*

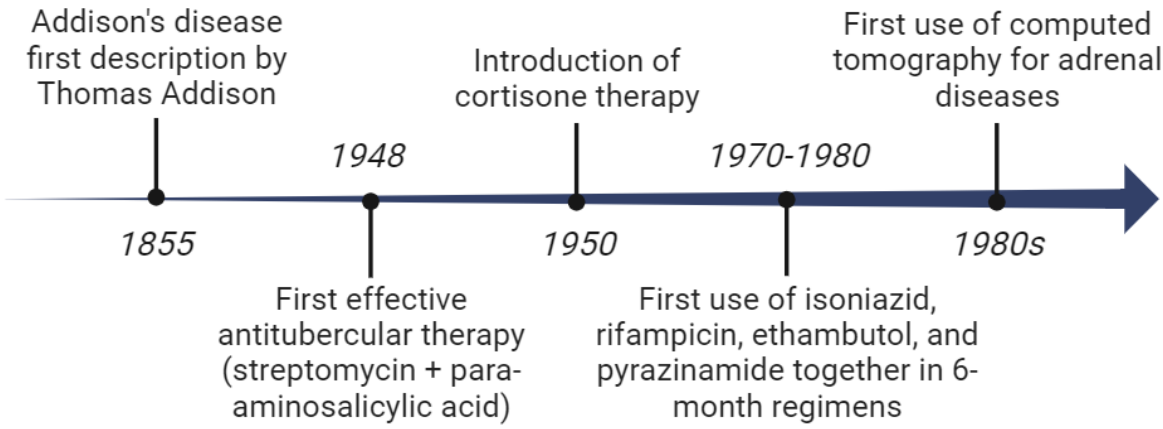

Fig. (2). Timeline of key historical milestones regarding PAI caused by adrenal TB. *(A higher resolution / colour version of this figure is available in the electronic copy of the article).*

hypothalamus, pituitary gland, thyroid, gonads, and pancreas, with the adrenal gland being the most commonly affected among these [11, 12]. Adrenal involvement is observed in between 0.3% and 5% of TB patients [13].

Findings on computed tomography (CT) can support the clinical-etiological diagnosis of PAI. In Mycobacterium tuberculosis infection, an increase in the size of the adrenal gland is evident, initially in one gland and then bilaterally, with the presence of microcalcifications and granulomatous infiltration. The etiological diagnosis of adrenal TB is confirmed with a biopsy of the gland [12]. Despite advancements in the diagnosis and treatment of PAI, challenges persist in achieving a timely diagnosis and preventing complications or death [3, 4, 6]. Fig. (**2**) provides a timeline illustrating key events related to PAI caused by adrenal TB [14-16].

We present the case of a female patient with PAI caused by adrenal TB. The diagnosis was established through epidemiological data and characteristic tomographic findings, underscoring the significance of this imaging modality as an alternative diagnostic method in hospitals where histopathological examination is not feasible.

2. CASE REPORT

A 37-year-old Peruvian woman with a prolonged epidemiological history of contact with TB patients during several

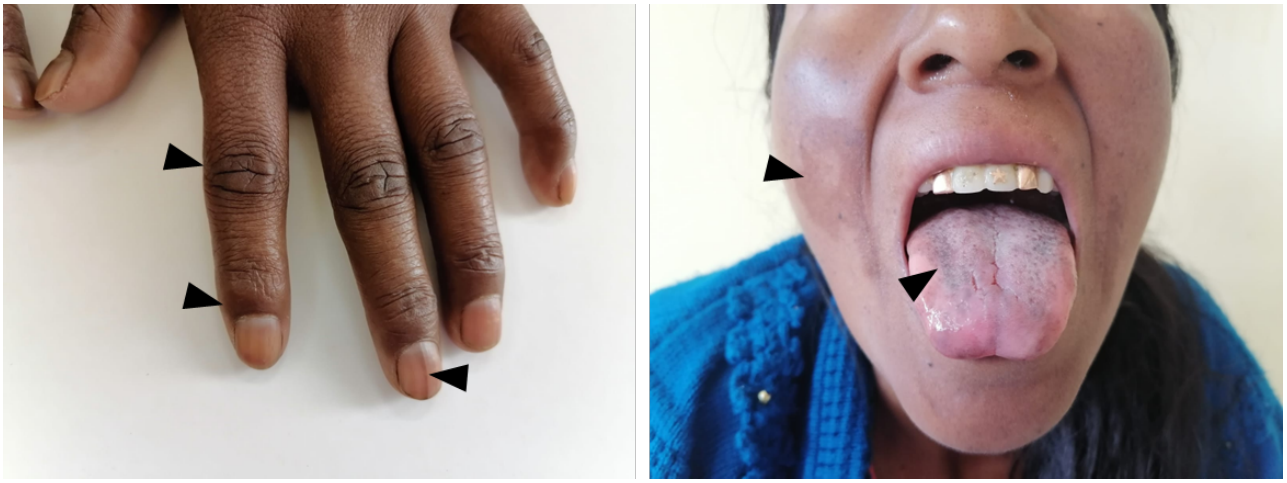

Fig. (3). Hyperpigmentation of skin and mucous membranes at the time of PAI diagnosis. Hyperpigmentation was observed on the back of the fingers, nail beds, face, and tongue (black arrowheads). *(A higher resolution / colour version of this figure is available in the electronic copy of the article).*

years of agricultural work was presented. She began experiencing symptoms four years ago, including abdominal pain, cold intolerance, excessive sleepiness, asthenia, fatigue, arthralgias, lower back pain, a weight loss of 18 kg, hyporexia, nausea, constipation, oligomenorrhea, orthostatic hypotension, dyspnea, and emotional lability, all of which worsened over time. Two years ago, skin and mucous membrane darkening, along with amenorrhea, were added to her symptoms. Seeking medical attention due to the intensification of symptoms, extreme weakness, and anorexia, she was found to be in a physically and nutritionally compromised state. On physical examination, her heart rate was 62 beats per minute, respiratory rate was 14 breaths per minute, temperature was 36.2°C, blood pressure was 80/50 mmHg, oxygen saturation was 98%, weight was 37 kg, and height was 1.50 m. Additionally, darkening of the skin on the face, trunk, hands, feet, nail beds, tongue, gums, and other mucous membranes was observed (Fig. **3**), along with the loss of pubic and axillary hair, diminished cardiac sounds and orthostatic changes. However, respiratory, abdominal, and neurological examinations were normal.

Among the conducted tests, the complete blood count, fasting blood glucose, lipid profile, bilirubin, sodium, and potassium were found to be within normal ranges. The chest X-ray (Fig. **4**) revealed no abnormal findings, and acid-fast bacilli were not detected in sputum and urine. Tests for syphilis, hepatitis B, and HIV yielded negative results.

Among the abnormal results, notable findings included: aspartate aminotransferase 56 U/L, alanine aminotransferase 42 U/L, total proteins 5.39 g/dl, albumins 3.09 g/dl, urea 46 mg/dl, creatinine 1.12 mg/dl, adrenocorticotropic hormone (ACTH) at 8 a.m. > 2000 pg/ml (reference range: 7.2 - 63.3), and cortisol at 8 a.m. 0.87 ug/dl (reference range: 6.2 - 19.4). Bradycardia was observed in the electrocardiogram.

Non-contrast-enhanced multislice spiral CT revealed diffuse bilateral enlargement of the adrenal glands, predominantly on the left side, with nodular, lobulated, irregular, and heterogeneous characteristics. The longitudinal and transverse diameters were 21 x 13 mm and 15 x 9 mm, respectively, in the left and right glands. Coarse calcifications were observed in both the central and peripheral areas. No alterations were noted in the adjacent fat planes, and there was no infiltration of neighboring organs (Fig. **5**).

Although consideration was given to performing an adrenal biopsy, it proved unfeasible due to the unavailability of specialists at our hospital and the patient's inability to travel to the referral center.

The patient was diagnosed with PAI and adrenal TB and initiated treatment with isoniazid, rifampicin, ethambutol, and pyrazinamide, along with prednisone at a daily dose of 5 mg, experiencing partial improvement in symptoms. However, she discontinued prednisone after 20 days due to the loss of her medications, resulting in symptom exacerbation. During this episode, her blood pressure dropped to 60/40 mm Hg, leading to hospitalization with the diagnosis of an adrenal crisis. She received a 100 mg bolus of intravenous hydrocortisone, followed by 50 mg of hydrocortisone every 6 hours, along with fluid therapy. The patient showed a rapid improvement in blood pressure and a gradual decrease in discomfort. She was discharged on the third day with reinforced education provided.

After two months, she entered the continuation phase of TB treatment with isoniazid and rifampicin, accompanied by 7.5 mg of prednisone daily. The prednisone dose was increased to 12.5 mg, achieving a significant clinical improvement without evidence of metabolic abnormalities. She completed the anti-TB treatment at 6 months, reaching a weight of 42 kg and experiencing a total improvement in symptoms, including a reduction in skin and mucous membrane hyperpigmentation.

The patient continued with hormonal replacement therapy and attended follow-up appointments at the Non-Communicable Diseases clinic every 3 months. During these check-ups, her blood pressure remained within normal parameters, with no evidence of metabolic abnormalities or recurrence of initial symptoms. Her weight reached 48 kg.

The patient moved to the highlands, where she passed away at the tenth month of follow-up due to pneumonia,

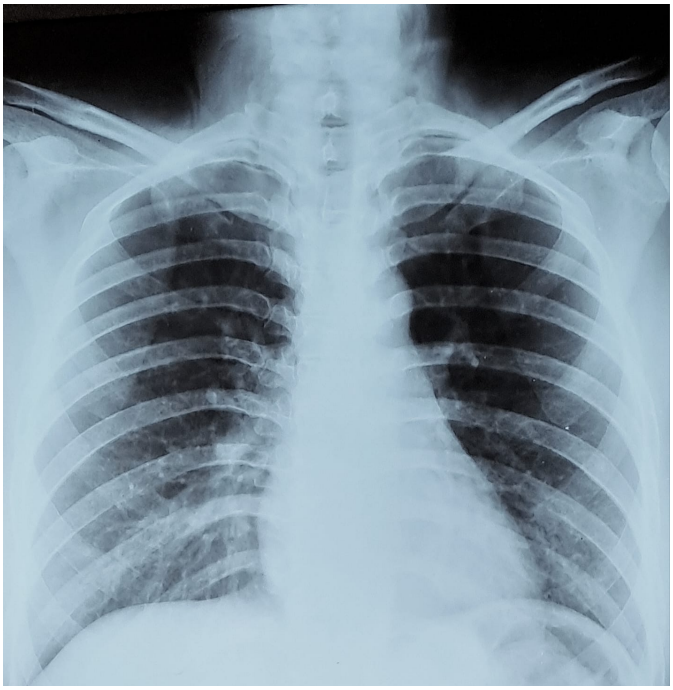

Fig. (4). Posteroanterior chest X-ray. No signs suggestive of pulmonary TB were evident. *(A higher resolution / colour version of this figure is available in the electronic copy of the article).*

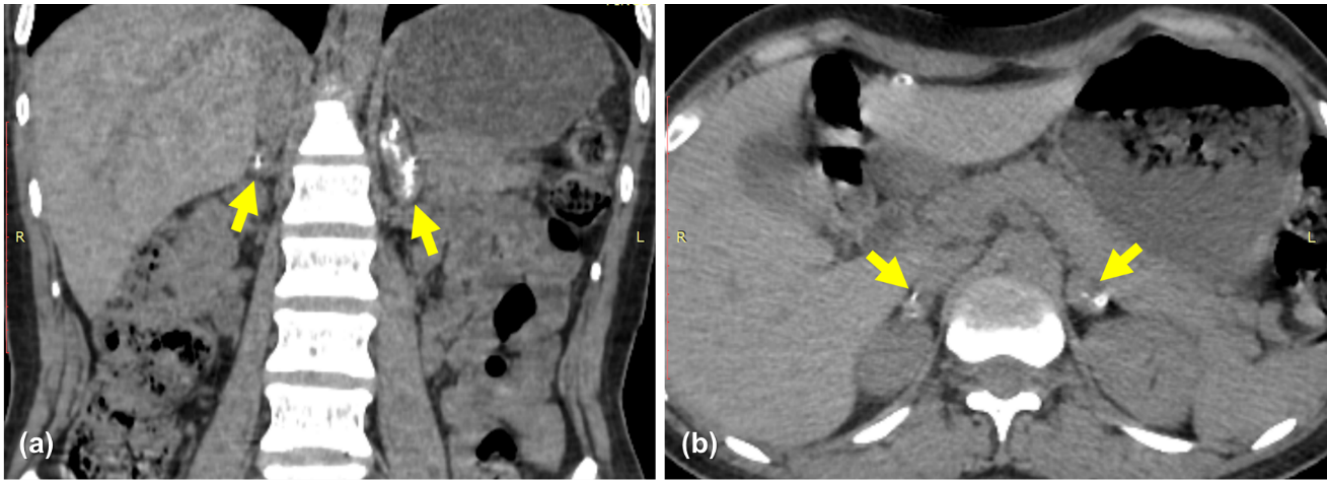

Fig. (5). Non-contrast-enhanced multislice spiral CT of the adrenal glands. Enlarged adrenal glands with calcifications were evident in the (**a**) axial and (**b**) coronal planes (yellow arrows). *(A higher resolution / colour version of this figure is available in the electronic copy of the article).*

aggravated by the difficulty in obtaining early medical attention due to the distance from her residence to a health center.

3. DISCUSSION

Once the diagnosis of PAI is confirmed, it is crucial to determine its etiology. It is recommended to measure autoantibodies against 21-hydroxylase, especially in regions with a high prevalence of autoimmune adrenalitis [2]. Additionally, a CT scan of the adrenal glands is recommended to rule out inflammatory, infiltrative, hemorrhagic, or metastatic processes. In areas with a high prevalence, an investigation for TB and HIV infection should also be conducted [2].

The possibility of adrenal TB should be particularly considered in patients residing in areas with a high prevalence of TB and those with a history of TB, either with active disease or a positive tuberculin test, who present the clinical manifestations described for PAI [7, 8]. Adrenal TB typically occurs through early hematogenous spread and is often associated with extra-adrenal infection in the majority of cases, with pulmonary and genitourinary TB being the main manifestations [8]. Tuberculous PAI clinically manifests late, occurring 10 to 15 years after the initial adrenal infection [8, 11]. Symptoms of adrenal TB include anorexia (75-100%), fever (72-94%), weakness (72-100%), fatigue (70-100%), hyperpigmentation (65-94%), gastrointestinal symptoms

(58-92%), arterial hypotension (50-90%), salt craving (5-16%), dizziness (4-12%), vitiligo (10-20%), and myalgias/arthralgias (5-10%). Rarely, patients may experience the acute onset of an adrenal crisis, which can be potentially life-threatening [8].

At the onset of adrenal TB, cortisol increases due to the chronic inflammatory state associated with the infection, leading to an upsurge in the production of inflammatory mediators and the release of ACTH. As a compensatory mechanism to maintain hormonal production, the size of the adrenal gland increases despite the direct destructive effect of the bacillus [9]. If the inflammatory process persists, it progresses to necrosis and destruction of adrenal cortical tissue, explaining the disease's presentation in advanced stages [9].

Non-invasive diagnostic tools, such as CT scans, Magnetic Resonance Imaging (MRI), and Positron Emission Tomography (PET) [8], support the clinical-etiological diagnosis of PAI [13].

In the early stages of the disease (within the first 2 years) and when active, over 80% of adrenal TB cases exhibit enlargement in both adrenal glands on CT scans, with no alteration in their contour [7, 17-19]. These glands show hypodense or isodense central areas indicative of caseous necrosis, with hypodense peripheral enhancement after contrast administration [8, 18]. Adrenal gland enlargement begins in one and extends to both [13]. CT sensitivity for adrenal TB is 91% with bilateral involvement and specificity of 99% with preserved contours [20]. In cases of long-term or inactive disease, adrenal tissue degenerates and is replaced by fibrous tissue, evidenced by atrophy, irregular margins, and diffuse calcification [7, 8, 17-19]. In this phase, peripheral enhancement in contrast-enhanced CT is unlikely due to reduced granuloma and caseous necrosis [12]. After completing anti-TB therapy, fibrotic, scarred, or calcified tissue appears as isodense or calcified lesions in the center of the glands [18], and in such cases, initiating anti-TB treatment is not recommended [21]. Adrenal primary tumor cases are usually unilateral and very rarely show calcifications or low attenuation center with peripheral rim enhancement. For adrenal tumors, the sensitivity of CT is 91%, and specificity is 94%, with unilateral involvement and mass-like appearance [20]. On the other hand, PAI derived from autoimmune adrenalitis manifests in CT as glandular atrophy without calcifications [22]. Considering the duration of the disease, adrenal atrophy would be expected in CT; however, in our patient, we observed bilateral adrenal enlargement in addition to calcifications. This suggests that the classical description of adrenal TB images on CT does not always align with patient findings, indicating potential discordance between the findings and the duration of the disease.

MRI can also provide useful clues for diagnosis, with findings similar to those described for CT, although calcifications are more evident in the latter [18]. Information available on PET imaging for patients with adrenal TB is limited [8].

The differential diagnosis of adrenal enlargement encompasses various conditions, such as TB, malignancy, hemorrhage, fungal infection, amyloidosis, sarcoidosis, adenoma, hemangioma, and hyperplasia [8, 11, 21]. Confirmation of the diagnosis of adrenal TB is achieved through histopathological studies [13], so tissue samples must be obtained for microbiological and pathological analysis, especially when adrenal involvement is the sole evidence of TB [7]. CT-guided needle aspiration is considered the optimal technique for obtaining adequate samples from the adrenal gland [7]. Polymerase chain reaction (PCR) and culture of these samples for mycobacteria may not consistently yield positive results, so a combination of histopathology, PCR, and culture may be necessary to confirm the diagnosis [8]. Due to a lack of availability in our setting, histopathological confirmation could not be performed.

The diagnosis of adrenal tuberculosis resulting in PAI can be established without resorting to adrenal biopsy in the presence of certain clinical and radiological findings. Particularly, bilateral adrenal enlargement on CT, along with evidence of active extra-adrenal TB, becomes relevant, especially in areas with a high TB burden [17]. The combination of PAI clinical features, origin from an endemic TB country, an epidemiological history of contact with TB patients, and radiological findings contributed to establishing a diagnosis of adrenal TB in our patient, leading to the administration of anti-TB therapy in addition to corticosteroid therapy.

The goals of treating PAI are to establish an optimal glucocorticoid dose, improve quality of life, and prevent complications. Hormone replacement aims to restore normal physiology, adjusting the dose based on the patient's symptoms [1]. Hydrocortisone is the glucocorticoid of choice. Alternatively, prednisolone or prednisone can be used, but the use of dexamethasone is not recommended [1, 5]. Monitoring should be conducted at least annually, assessing the clinical response (body weight, postural blood pressure, edema, energy levels, and obvious signs of over- or under-replacement) [2, 5].

Treatment guidelines for adrenal TB are similar to those for pulmonary TB [8, 23]. The adrenal cortex has considerable regenerative capacity [24], and anti-TB treatment improves adrenal enlargement but does not generally reverse PAI [7, 8]. Our patient received anti-tuberculosis treatment for 6 months with a good clinical outcome.

CONCLUSION

The possibility of adrenal tuberculosis as a cause of PAI should be considered in patients from endemic TB areas with a history of active or latent TB exhibiting the described clinical manifestations of PAI. CT imaging can serve as an alternative diagnostic method in settings where a rapid biopsy is not feasible. We suggest conducting further analytical observational studies to facilitate the implementation of CT as the primary diagnostic method for adrenal TB in cases of adrenal insufficiency where biopsy is not feasible.

LIMITATIONS

A chest CT could not be requested to assess pulmonary involvement. Additionally, the Tuberculin Skin Test with purified protein derivative, interferon-gamma detection, and the measurement of dehydroepiandrosterone, aldosterone, renin, thyroid hormones, and sex hormones could not be

performed. Oral hydrocortisone and fludrocortisone were not available.

AUTHORS' CONTRIBUTIONS

J.E.Q.A. contributed to the conceptualization, methodology, investigation, writing the original draft, resources, and project administration. M.C.D.V., S.E.Z.A., A.N.Z.A., and C.M.Q.G. participated in the investigation and writing of the original draft. E.R.G.O., J.R.R., L.A.C.U., S.P.S., L.A.R.H., and J.P.I. took part in the investigation, writing of the review, and editing. M.J.C.Z. contributed to the conceptualization, methodology, writing, reviewing, editing, and project administration.

LIST OF ABBREVIATIONS

CT = Computed Tomography

MRI = Magnetic Resonance Imaging

PAI = Primary Adrenal Insufficiency

TB = Tuberculosis

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the ethics committee, Comité Institucional de Ética en Investigación de la Facultad de Medicina de la Universidad Nacional de Trujillo, of the Universidad Nacional de Trujillo, Perú (approval Number: Of. N: 353-2023-UNT-FM-C.E).

HUMAN AND ANIMAL RIGHTS

This case report adheres to the ethical principles outlined in the Declaration of Helsinki.

CONSENT FOR PUBLICATION

Informed consent was obtained from the patient for publication of clinical details and images.

AVAILABILITY OF DATA AND MATERIALS

The data and supportive information are available within the article.

STANDARDS OF REPORTING

CARE guidelines were followed.

FUNDING

None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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Declared none.

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