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A Rare Case of Adrenal Tuberculosis Mimicking a Tumor: Diagnostic Pitfalls

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Abstract

Adrenal tuberculosis is a rare condition, accounting for 3% to 9% of adrenal insufficiency etiologies. It is responsible for a clinical table of acute or chronic adrenal insufficiency. We report a case of an adrenal mass with a misleading pseudotumoral radiological appearance in a patient initially addressed for preoperative evaluation of this "adrenal mass". The clinico-biological evaluation of this mass indicated a tubercular origin, thus avoiding an unnecessary surgery. This 43-year-old patient initially presented with non-specific abdominal pain, prompting an abdominal tomography in favor of a pseudotumoral right adrenal mass with an initial indication for adrenalectomy. Clinical evaluation revealed weight loss and diffuse melanoderma. The diagnosis of peripheral adrenal insufficiency was confirmed by a low cortisol level measured at 8 a.m. and a very high ACTH level. As part of the etiological investigation, the Quantiferon test proved strongly positive at 10 IU/ml. The diagnosis of adrenal tuberculosis was confirmed on the basis of these clinical, biological, and radiological findings. The patient was treated with hydrocortisone (30 mg/day) and antituberculosis therapy. The evolution is marked by clinical improvement—disappearance of asthenia, regression of melanoderma and normalization of blood pressure—and biological improvement after three months.

Keywords

Adrenal Mass, Pseudotumoral Aspect, Addison's Disease, Adrenal Tuberculosis

1. Introduction

Tuberculosis represents a major cause of primary adrenal insufficiency, accounting for 20% - 30% of cases, and remains a major public health concern in developing countries.

Addison's disease, or primary adrenal insufficiency, was first described in 1855 by Thomas Addison in patients with tuberculosis affecting the adrenal glands [1]. However, the incidence of adrenal tuberculosis has declined in recent decades primarily due to the introduction of anti-tuberculosis chemotherapy.

Despite these advancements, the diagnosis of adrenal tuberculosis is often delayed, as symptoms associated with adrenal insufficiency may not appear until several years after the hematogenous dissemination of tuberculosis [2].

Our case report presents an unusual manifestation of adrenal tuberculosis, with the ultimate objective of increasing awareness within the medical community about the various aspects of this prevalent condition in the Moroccan context.

2. Case Presentation

A 43-year-old male patient, with a history of chronic smoking and cured Hepatitis B (negative liver serology), initially presented with non-specific abdominal pain, prompting an abdominal tomodensitometry. The imaging favored a pseudo-tumoral right adrenal mass with an initial indication for adrenalectomy.

He was referred to us for preoperative evaluation of this "adrenal mass".

The interview revealed weight loss of 8 kg in two months, along with non-specific abdominal pain. Clinical examination showed hypotension at 90/70 mmHg, orthostatic hypotension, and diffuse melanoderma (**Figure 1**).

Figure 1. Melanoderma on the face, lips, and the inner surface of the cheeks.

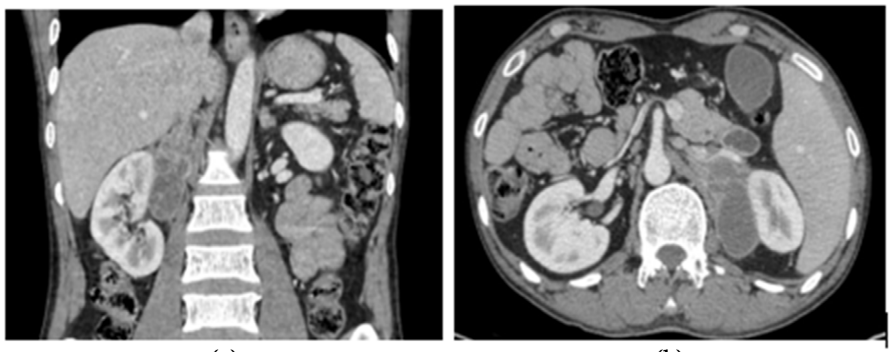
The diagnosis of peripheral adrenal insufficiency was based on a low 8-hour cortisol level of 52 µg/L (VN: 62–194) and a very high ACTH level. The ionogram indicated a tendency toward hyperkalemia and hyponatremia (natremia: 133 mmol/L, kalemia: 5.1 mmol/L) with a normal fasting blood glucose level of

4.93mmol/L (Table 1).

Table 1. Summary of our patient's biological parameters.

Parameters	Patient Values	Reference Range
blood ionogram		
-natremia	133 mmol/L	136 - 145
-kalemie	5.1 mmol/L	3.5 - 5
fasting blood glucose	4.93 mmol/L	3.89 - 5.83
8-hour cortisol	52 µg/L	62 - 194
Adreno Cortico Tropic Hormone (ACTH)	300 pg/mL	7.4 - 64.3

Adrenal computed tomography revealed a necrotic, multi-septate pseudo-adrenal mass measuring approximately 63 × 35 × 75 mm on the right, with micronodular atrophy on the left (Figure 2(a), Figure 2(b)).



(a) (b)

Figure 2. Adrenal tomography; coronal and axial sections showing lesion asymmetry: (a) On the right, a necrotic and multi-septate pseudo-adrenal mass measuring approximately 63 × 35 × 75 mm; (b) On the left, micronodular atrophy.

Etiological investigation, particularly the QuantiFERON test, was highly positive at 10 IU/mL (> 0.5 IU/mL). Thoracic tomography was normal, excluding concomitant pulmonary involvement.

The diagnosis of adrenal tuberculosis was made in consultation with the pneumo-phtisiologists, based on clinical signs of tuberculosis impregnation, radiological features highly evocative of a bacillary cause, and biological evidence—a very positive QuantiFERON level at 20 times the normal value.

The patient was treated with hydrocortisone-based hormone replacement therapy, at a dose of 30 mg/day, taking into account the interaction with the anti-tuberculosis treatment administered according to the 2RHZE/4RH protocol. An Addisonian card was issued to the patient, along with proper therapeutic education.

The evolution was marked by significant clinico-biological improvement, including the disappearance of asthenia, regression of melanoderma, normalization of blood pressure, and correction of hydro electrolytic disorders.

3. Discussion

Tuberculosis is an airborne infectious disease, primarily caused by the bacillus Mycobacterium tuberculosis. Although its usual site is the pulmonary parenchyma, this condition can also affect other organs [3].

The World Health Organization estimates that 3500 new cases of tuberculosis and 2900 deaths were recorded in Morocco in 2019. Tuberculous adrenal insufficiency is a rare disease, accounting for 10% of cases in Europe. It is caused by the destruction of both adrenal glands by caseous necrosis (90%). Adrenal tuberculosis is often bilateral in 70% of cases [4].

Addison's disease results from multiple causes, of which adrenal tuberculosis is a major etiology. This infection typically leads to bilateral enlargement of the glands (91% of cases according to Guo *et al.*), followed, in the chronic stage, by fibrosis and calcifications (incidence of 40% to 59%). Adrenal atrophy can occur after more than ten years of evolution [3] [5].

Although adrenal tuberculosis often occurs in the context of systemic dissemination, it is not uncommon for the adrenal glands to constitute the only infected organ. A large autopsy study also reported that the adrenal glands were the sole site of involvement in nearly 25% of cases [6].

The clinical presentation of adrenal tuberculosis is non-specific, with most affected patients being asymptomatic. Symptoms generally appear when there is significant destruction of both adrenal glands. Signs of adrenal insufficiency are often observed, accompanied by non-specific symptoms related to tuberculosis. Among the most frequent signs are fever, weakness, fatigue, nausea, vomiting, anorexia, weight loss, hypotension, and skin hyperpigmentation [3].

In some cases, adrenal tuberculosis is revealed during an episode of acute adrenal insufficiency or while investigating an adrenal mass.

Abdominal CT scan is the cornerstone of the radiological diagnosis of adrenal tuberculosis. It allows for the identification of typical features such as bilateral, pseudo-tumoral thickening of the glands, calcifications, and peripheral enhancement, often accompanied by a low-density center, which is a sign of caseous necrosis. This combination of signs is strongly suggestive of adrenal tuberculosis [3] [6].

Furthermore, the scanographic findings also make it possible to determine the evolutionary stage of the disease, and are characterized by [6]:

- Acute phase: Bilateral, homogeneous, asymmetric adrenal hypertrophy with cystic and typically calcific necrosis.
- Chronic phase: Bilateral atrophy.

In our observation, the asymmetrical radiological findings, combining a pseudo-tumoral mass on one side and atrophy on the other, suggest different evolutionary stages of adrenal tuberculosis. This unusual presentation constitutes a key element of the case, illustrating the coexistence of an active lesion and sequelar involvement.

Although Magnetic Resonance Imaging (MRI) shows similar signs, it allows for better visualization of granulomas and caseous necrosis, while CT scans remain more effective at detecting calcifications. However, MRI is often preferred, partic-

ularly in cases of allergy to iodinated contrast agents [3].

Echo- or scan-guided biopsy, not routinely performed and reserved for contentious cases, reveals an epithelioid giant cell granuloma with caseous necrosis [7].

Adrenal tuberculosis, a rare cause of adrenal insufficiency, is discovered later than the initial infection. Pathognomonic adrenal calcifications testify to the chronicity of the condition [3].

There are several conditions, such as adrenal autoimmunity, adrenal metastases, and non-Hodgkin's lymphoma, which must be distinguished from adrenal tuberculosis. Adrenal autoimmunity is the main cause of Addison's disease in developed countries. Laboratory tests, including the detection of adrenal antibodies, help to differentiate these conditions from adrenal tuberculosis.

Adrenal metastases are usually bilateral, but rarely cause Addison's disease. Bilateral pheochromocytoma must also be excluded, and specific features on MRI can distinguish it from adrenal tuberculosis [3].

The therapeutic management of adrenal tuberculosis is based on the following:

- Peripheral adrenal insufficiency: Therapy consists of hormone replacement therapy with hydrocortisone (25 - 30 mg/day) and fludrocortisone (50 - 100 µg/day) [8].
- The treatment for adrenal tuberculosis is similar to that of any other extrapulmonary tuberculous lesion. According to the WHO Tuberculosis Control Program, the standard therapeutic regimen combines four drugs during the intensive phase: isoniazid (5 mg/kg/day), rifampicin (10 mg/kg/day), pyrazinamide (30 mg/kg/day), and ethambutol (20 mg/kg/day). This treatment is generally followed by a consolidation phase with isoniazid and rifampicin, for a total treatment duration of 6 to 9 months [9].

Concomitant treatment of adrenal insufficiency and adrenal tuberculosis can be complex because rifampicin is a potent enzyme inducer of the cytochrome P450 system, which is involved in glucocorticoid metabolism, leading to decreased plasma levels [5].

In the absence of clear guidelines for dose adjustment, it is recommended to increase the hydrocortisone dose when used in combination with antituberculosis treatment. Adrenal function does not recover in most cases of tuberculous adrenal insufficiency; however, recovery is possible after appropriate and well-conducted antituberculosis treatment [10].

The evaluation of the response to treatment is based on clinical and radiological criteria. Clinically, therapeutic efficacy is demonstrated by the regression of initial symptoms and weight gain. Concurrently, CT scan follow-up reveals a regression of adrenal hypertrophy of the tumor-like syndrome. The clinical course of our patient appears very satisfactory, pending sufficient time for radiological monitoring [3].

4. Conclusions

Adrenal tuberculosis presents a real diagnostic challenge, as it can be confused with several other adrenal pathologies.

Our case report describes adrenal insufficiency occurring during the active, progressive phase of adrenal tuberculosis, in contrast to cases often reported in the chronic phase. It reveals an unusual pseudo-tumoral radiological presentation, which may mistakenly lead to surgical intervention. The case underscores the importance of multidisciplinary consultation for optimal patient management.

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Ethics Approval

Ethical approval was not required for this case report in accordance with institutional and national guidelines. Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Authors' Contributions

Authors 3 and 4 were involved in the clinical management of the patient. Authors 2 and 5 collected clinical, biological, and radiological data. Authors 1 and 5 conducted the literature review and drafted the manuscript. All authors critically reviewed and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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