Case Report

Miliary Tuberculosis with Addisons Disease - in old case of pulmonary tuberculosis, and tuberculosis of seminal vesicles - A rare case report

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Abstract

Addison's disease is a rare endocrinal disorder that was first described by Thomas Addison in 1855. Addison's disease occurs as a result of a lack of production of adrenocortical hormones, which is a rare but fatal disease if left untreated. The two most common causes of Addison's disease are autoimmune adrenalitis and tuberculosis which refer to hypoadrenalism caused by total or near total destruction or dysfunction of both adrenal cortices. Usual manifestations involve chronic fatigue, muscle weakness, loss of appetite, nausea, vomiting, diarrhoea, hypotension, and hyper pigmentation of the skin. A substantial proportion of patients presenting with extra-pulmonary tuberculosis (TB) have urogenital TB (UG-TB), which is easily under diagnosed because of non-specific symptoms, which are chronic and have cryptic protean clinical manifestations. Most of the clinician are not aware of the possibility of UG – TB. Calcification of seminal vesicle found in this case is a rare condition, which is commonly associated with diabetes, hyperparathyroidism, and genitourinary tuberculosis. We here in report a rare case of adrenal insufficiency due to miliary tuberculosis involving adrenal gland, old pulmonary tuberculosis and genitourinary tuberculosis (seminal vesicles calcification) in a 31 year old male person. He presented with multiple episodes of vomiting, and giddiness which was

also accompanied with atypical hyperpigmentation. His symptoms resolved after starting anti tuberculous therapy.

Key words

Miliary Tuberculosis, Addisons Disease, Adrenal Insufficiency, Pulmonary Tuberculosis, Genitourinary Tuberculosis, Seminal Vesicles Calcification.

Introduction

Addison's disease is a rare endocrinal disorder that was first described by Thomas Addison in 1855 [1]. Addison's disease occurs as a result of a lack of production of adrenocortical hormones, which is a rare but fatal disease if left untreated [2]. The two most common causes of Addison's disease are autoimmune adrenalitis and tuberculosis which refer to hypoadrenalism caused by total or near total destruction or dysfunction of both adrenal cortices [3]. Usual manifestations involve chronic fatigue, muscle weakness, loss of appetite, nausea, vomiting, diarrhoea, hypotension, and hyper pigmentation of the skin [4]. A substantial proportion of patients presenting with extra-pulmonary tuberculosis (TB) have urogenital TB (UG-TB), which is easily under diagnosed because of non-specific symptoms, which are chronic and have cryptic protean clinical manifestations. Most of the clinician are not aware of the possibility of UG – TB [5, 6, 7, 8]. Calcification of seminal vesicle found in this case is a rare condition, which is commonly associated with diabetes, hyperparathyroidism, and genitourinary tuberculosis [9].

Tuberculosis is a major global health problem and may appear as a multisystem disease [10]. Pulmonary TB and extra-pulmonary tuberculosis are the two types of clinical manifestation of tuberculosis. The extra-pulmonary tuberculosis involves organs other than the lungs (e.g. lymph nodes, pleura, abdomen, genitourinary tract, joints, skin and bones, or meninges). Extra pulmonary sites account for 10 percent of tuberculosis cases [11]. Tuberculosis (TB) infects 316 Indians per 100,000 - way above the 193 predicted by World Health Organization (WHO) in 2019 [12]. Thomas Addison initially diagnosed Addison's disease, a rare endocrine ailment, in 1855 [1]. This condition is characterized by a deficit in the production of glucocorticoids and mineralocorticoids for a variety of reasons [1, 2]. It affects both men and women equally across all age groups [13]. Atopic autoimmune adrenalitis and Tuberculosis are the two most typical causes of Addison's disease [3].

Adrenal insufficiency can be of two types, (1) primary and (2) secondary [10]. In developed countries, the autoimmune type of adrenal insufficiency is the most common cause while in developing countries infectious agent like tuberculosis is the most common cause [3]. The patient usually complains of gastrointestinal upset with anorexia, nausea, vomiting, diarrhea, and hyper pigmentation. The characteristic physical symptom of Addison's disease is generalized cutaneous hyper pigmentation [14]. Hyper pigmentation is usually seen over pressure points like elbows and knees and on skin exposed to the sun [15]. Adrenocorticotropic hormone and beta-lipotropin, both of which can stimulate the formation of melanocytes, are the two hormones that produce hyper pigmentation [13]. Adrenal calcification and enlargement are commonly seen in Addison's disease associated with tuberculosis [16, 17, 18]. Thirty to forty percent of all cases of extra pulmonary tuberculosis are caused by genitourinary tuberculosis [19]. The latent period between pulmonary infection and clinical genitourinary tuberculosis is usually 22 years [20]. A substantial proportion of patients presenting with extra-pulmonary tuberculosis (TB) have urogenital TB (UG-TB), which is easily under diagnosed because of non-specific symptoms, which are chronic and have cryptic

protean clinical manifestations. Most of the clinician are not aware of the possibility of UG – TB [5, 6, 7, 8]. Many patients can be asymptomatic during the early stages of the disease. GUTB can refer to TB affecting the urethra, bladder, ureters, or kidneys in males and females. The scrotum, penis, testes, epididymis, or vas deferens in males, and vulva, vagina, cervix, uterus, ovaries, or fallopian tubes, in female are affected. The urinary tract TB occurs more often when compared to genital TB [21]. GUTB is more important as it is diagnosed late in the disease course, which may lead to complications such as urethral or ureteric strictures, renal failure, infertility. Male genital TB affects the seminal vesicles through canalicular spread. Seminal vesicle calcification is usually occurs in hyperparathyroidism, Tuberculosis, and diabetes mellitus [9].

Due to lack of awareness of physician and a lack of resources in patients for specialized investigations, endocrine disorders especially Addisons is frequently goes undiagnosed in the early stages of the disease, in developing countries like India [22]. One must become familiar with the varied presentations of these illnesses due to their low prevalence and atypical presentations [23-32].

Case report

A 31 year old male, lorry driver by occupation, presented with chief complaints of multiple episodes of vomiting, 10-15 episodes/day, green in color, of one day duration, complained of giddiness the next day, then developed, multiple episodes of non projectile vomiting.

History of present illness: Patient was apparently asymptomatic till a day earlier, then developed multiple episodes of vomiting 10-15 episodes, green color, history of outside food consumption was present. No history of loose stools, fever, pain abdomen. Complaints of giddiness the next morning, no history of headache, blurred vision.

Past history: No history of similar complaints in the past. No history of chronic cough, Not a known case of hypertension, diabtetes mellitus, thyroid, seizures, asthma, tubereculosis, epilepsy, CAD, COPD, history of sustained head injury 5 years ago, history of blood transfusion 3 years ago for weakness, no history of surgeries.

Personal history: Appetite – decreased, mixed diet, bowels and bladder regular, alocoholic +, smoker +, stopped 2 years ago, sleep - sound.
Family history: No history of similar complaints in the family.

General Examination
Patient is conscious, coherent, cooperative, answering to questions well. Patient is well built, well nourished, BMI – 16.9, No Pallor, No icterus, No cyanosis, no clubbing, No lymphadenopathy, No pedal edema. Vital data: Pulse-65/ mt, regular, normal volume, No radio radial delay, no radio femoral delay, all peripheral pulses felt. BP - 80/50 mm Hg, Rt arm supine position. RR- 16/min. Temp - 98.4^0 F, JVP - not raised. Tongue: dry, skin-dry, eyes-sunken; Face, Knuckles and Palms- dark hyper pigmentation ++.

Systemic examination
Respiratory system – lungs – clinically clear
Cardio vascular system: Heart sounds - S1, S2 heard, no murmurs heard.
P/A: Diffuse tenderness present, no organomegaly
CNS: No focal neurological deficit found
Tongue – dry, Skin – dry, Eyes – sunken, Face, Knukles And Palms – hyper pigmented.

Investigations: Hb-13.8 gm/dl, RBC count-5.3 millions/cumm, WBC count 15200 cells/cumm, Platelet count – 4.2 lakhs/cu mm., CUE: pale yellow, clear.

Date	Blood Urea (Mg/Dl)	S. Creatinine (Mg/Dl)
25/5/22	50	2.9
26/5/22	40	1.8

Random Blood Sugar: 102 mg/dl; Total T3 - 1.11ng/dl, total T4 - 11.7ng/dl, TSH - 0.69u IU/ml, ESR – 25 mm/hr, Serum Cortisol: 0.63 ugm/dl (Ref N - 4.30-22.4 ugm/dl), 24hr urine protein: 120 mg/24hr, 24hr urine volume: 3000 ml/24 hrs, Sputum AFB - Negative, Sputum C/S –Negative, CBNAAT – Negative, PTH - 63.8 pg/ml (Ref N- 14 to 65 pg/ml), Serum Electrolytes: Na+: 131 mmol/L, K+:7.0mmol/L, Cl- : 102 mmole/L, HIV: Negative, HbsAg: negative, HCV: negative

USG Abdomen: liver shows normal echo texture, span measuring 16 cm, gall bladder normal, spleen normal in size, Right Kidney 9x4.5cm, Left Kidney 9.2x4.3cm, Adrenal Gland: Right Side 4.0 x1.5cms, Left Side 3.0 x1.5cms, no other masses were seen.

CT Abdomen Adrenals: Well defined oval shaped hypodense lesion with foci of calcification noted in both limbs of adrenal glands on both sides measuring 4.0 x1.4 cm on right side; 2.7x1.3 cm on left side. Bilateral seminal vesicle calcifications noted.

Spine: Spondylolisthesis, L5 vertebrae over S1, with spondylolysis. Hypodense lesion with sclerotic margin noted in right sacral arch. Facet joint arthroplasty at L5-S1X Ray Chest PA View: Fibrocalcific lesion at lateral segment of right middle lobe.

Diagnosis

Miliary tuberculosis with Addisons disease - in old case of Pulmonary Tuberculosis, and tuberculosis of Seminal Vesicles.

Treatment given:

1. IVFluids: NS AND DNS
2. Injection METROGYL 500mg/IV/TID
3.Injection CIFRAN 200mg/IV/BD
4.Injection ZOFER 4mg/IV/TID
5.Injection OPTINEURON 1ampoule/IV/OD
6.Syrup SUCRALFATE 4tsp/TID
7. Injection HYDROCORT 100mg/IV/TID

Treatment suggested on follow-up

1. T.AKT4- 1 strip daily for 2 months followed by AKT 3 for 4 months
2. Tablet OMNACORTIL 20mg-OD
3. Syrup SUCRALFATE 4tsp/TID

Discussion

Addisons disease is a rare disease that affects 1 in 100,000 people [13]. Although tuberculosis only accounts for 7–20% of instances of Addisons disease, autoimmune illness accounts for 70–90% of the underlying conditions [32]. Fungal infection, haemochromatosis, metastatic neoplasm, and X-linked adrenoleukodystrophy are some of the additional causes of Addisons disease [1, 33]. In developing countries like India tuberculosis is the most common cause of Addisons disease [34]. Because Addisons disease is usually not diagnosed in its early stages, it can present a life-threatening crisis [13]. Addisons disease typically develops 32+/- 15 years after initial tuberculosis. Addisons disease is usually becomes evident slowly by tuberculosis [35].

The symptoms of Addisons disease begins gradually with chronic worsening of fatigue, loss of appetite, generalized weakness, hypotension and weight loss. Another characteristic physical finding is hyper pigmentation. The generalized, homogenous, brown hyper pigmentation that is seen in areas exposed to the sun, such as the face, neck, and back of hands, or areas exposed to repetitive pressure or friction, such as the elbows and knees, is associated to ACTH melanogenesis [36]. The patient had hyper pigmentation of palms, knuckles, and face. The clinical features of hypoadrenocorticism actually begin to appear after the destruction of at least 90% of the glandular tissue [13]. The initial symptoms for suspecting the diagnosis of Addisons disease were the presence of hyper pigmentation and hypotension. Investigations such as electrolyte disturbances were further pointers to diagnosis [4]. While hypoglycemia is uncommon, hyponatremia and hyperkalemia are frequently linked to Addisons disease. Patients typically complaints of experiencing gastrointestinal disturbances like anorexia, nausea, vomiting, and

diarrhoea. In Addisons disease associated with tuberculosis, the adrenal glands frequently calcify and enlarge [16, 17, 18].

Addisons disease is also associated with psychiatric symptoms such behavioral abnormalities, decreased motivation, and anxiety disorders. According to Anglin, et al., abnormalities in the electrophysiological, electrolyte, and metabolic activity may be related to the neuropsychiatric symptoms of Addisons disease, though the exact cause is unknown [37].

There may not be distinct symptoms for Addisons, and the symptoms can overlap with those of other endocrine system disorders [2]. Consequently, diagnosis is challenging [2].

A sickness or accident can exacerbate the condition and cause an Addisonian crisis. The symptoms of Addisons disease progress slowly and are usually ignored. Addisonian crisis symptoms include sudden penetrating pain in the lower back, abdomen, or legs, along with severe vomiting and diarrhoea, these symptoms are followed by dehydration, low blood pressure, and loss of consciousness [18, 38]. Extra-pulmonary TB commonly affects the adrenal glands. The loss of the adrenal glands by caseous necrosis results in tuberculous addisons disease [39]. Due to hematogenous or lymphogenous dissemination from the primary site of infection, the adrenal glands on both sides are frequently (70%) affected [40]. Occasionally the adrenal glands may be the only diseased organ. In a autopsy study, 25% of people with adrenal tuberculosis, the history, imaging modalities, determined the growth of adrenal gland with or without calcification [33]. For the diagnosis of adrenal insufficiency, the blood cortisol, serum ACTH, and fasting ACTH stimulation tests are important. The present patient met the diagnostic criteria for Addisons disease due to decrease in cortisol levels and a rise in ACTH levels [41]. Due to possible adrenal crisis attacks and the underlying conditions, life expectancy is decreased in Addisons disease. Patients with primary or secondary adrenal insufficiency have lower quality of life even though they get enough glucocorticoids and mineralocorticoids [42]. Physicians should be aware that Addisons disease may manifest during the follow-up of tuberculosis. Adrenal insufficiency due to tuberculosis in developing countries is an important cause .In this approach, a diagnosis can be made before an addisonian crisis starts, preventing sudden death [2].

GUTB is usually caused by the hematogenous spread of the mycobacteria during the early infection. These bacilli remain dormant in the urogenital tract and become activated in periods of immunosuppression [43]. Other routes of infection include lymphatic spread and sexual transmission [44]. Diabetes, advancing age, low BMI, concurrent cancers, immunosuppression, and kidney failure can increase the risk of reactivation of TB [45].

We herewith present a rare case of seminal vesicle tuberculosis that did not affect the kidneys, ureters, or bladder [46]. Although the exact incidence of seminal vesicle calcification is unknown, it is frequently linked to hyperparathyroidism, diabetes mellitus, and TB [9]. Our patient is non diabetic and his parathyroid hormone levels were normal. Hence, it is inferred that the cause of calcification of seminal vesicles is Tuberculosis. During the course of diagnosing the patient's addisons disease, seminal vesicle clacification is also became evident. Lab investigations in this case presented, hyponatremia and hyperkalemia.

Conclusion

Addison's disease is a rare endocrinal disorder, in developed nations as it is usually related to auto-immune disorder but in the developing nations it is commonly associated with tuberculosis [13]. Addison's disease caused by tuberculous adrenalitis presents with non-specific symptoms and onset is often insidious. Diagnosis is therefore frequently missed, and the chance of an adrenal crisis is increased. In the majority of tuberculosis-related Addison disease cases, the

adrenal function does not recover [39]. The significance of a systematic search to identify initial cases of genitourinary tuberculosis independent of symptoms, must be stressed in these cases because this patient also has seminal vesicle calcification (genitourinary tuberculosis) [11]. In conclusion, we emphasise the significance of a thorough endocrine investigation. Neonatal hypothyroidism, adrenal insufficiency following TB, and pituitary damage following episodes of postpartum haemorrhage are a few examples of undiagnosed endocrine problems. Awareness will aid in early detection, which can stop many fatalities. We are reporting a rare case of Addisons disease caused by tuberculosis, which was also accompanied with atypical hyper pigmentation, pulmonary tuberculosis, and genitourinary tuberculosis (seminal vesicles calcification).

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