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A Rare Presentation of a Common Disease: A Case Report

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ABSTRACT

Addison's disease (AD) or primary adrenal insufficiency was first described by Thomas Addison in patients with adrenal tuberculosis. Over the past several decades, along with the introduction of anti tuberculous treatment, the incidence of both have declined. The most common symptoms are non specific, therefore the diagnosis is often delayed and patients may first present with a life threatening crisis. Here we report a case of Addison's disease which on further workup was found to be due to one of the most common infections in India-Tuberculosis.

Keywords: Addison's disease, Tuberculosis, hyperpigmentation

CASE REPORT

A 65 year old male patient who was a watchman by occupation was admitted with complaints of Fever on and off for past 1 week, Breathlessness on severe exertion for 2 weeks, generalized darkening of the skin with significant loss of weight and loss of appetite for the past 1 year. There was no history of any drug intake, comorbidities or surgeries in the past. On examination, the patient had diffuse hyperpigmentation involving both hands, foot including the palmar creases.

Vitals

PR-96/min. regular, normal. BP- 90/60 mmHgin right upper limb in sitting posture. Standing Bp - 70/40 mmHg in right upper limb, which showed that the patient had significant postural hypotension.

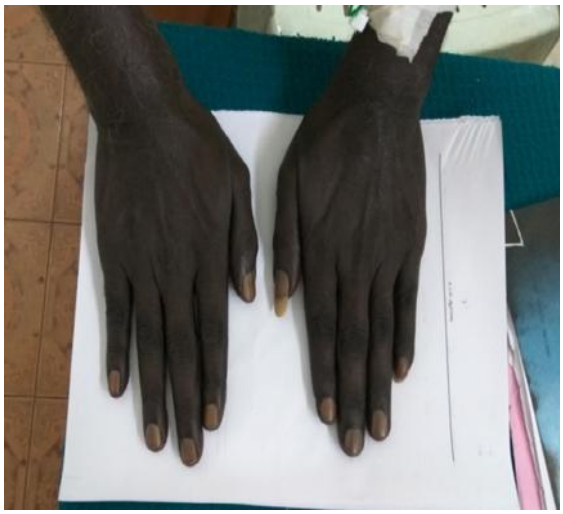
Cardiovascular system- first and second heart sounds heard, no murmurs/other events.

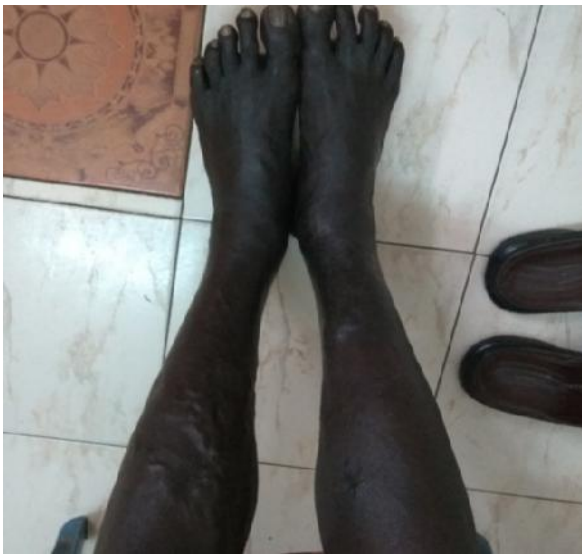
Respiratory system- normal bilateral vesicular breath sounds heard.

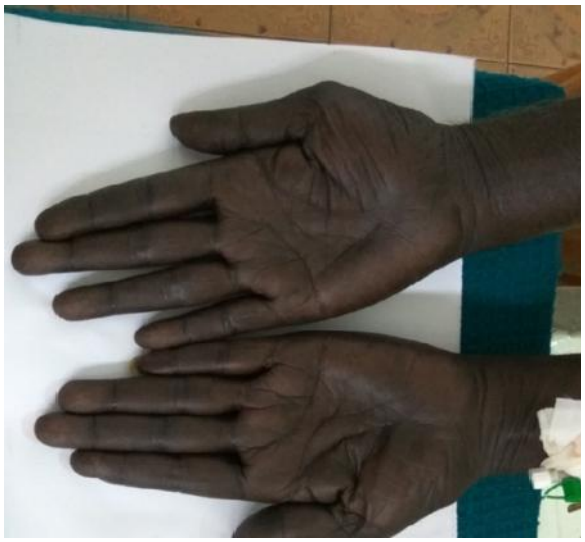
Abdomen- noorganomegaly, External genitalia was normal

Central nervous system- clinically normal

Fundus examination- both eyes- normal







INVESTIGATIONS

Complete hemogram- Tc-4800, HB-11.8mg/dl, platelet- 3,23,000/mm3- was normal.

ESR-65mm/at the end of 1st hour; RBS- 70mg/dl. UREA-50à40à32 mg/dl.

CREATININE-1.1à1.4à1.0 mg/dl, indicating prerenal failure which recovered.

NA- 117à142à135mEq/dl, K- 8.3à4.2à5.1mEq/dl- indicating hyponatremia and hyperkalemia.

LFT – Normal. URINE ROUTINE - NORMAL

ECG and CXR- normal., HIV 1 & II – non reactive.,

Now to find out the causes of diffuse hyperpigmentation, we proceeded with the following investigations

- PERIPHERAL SMEAR- normal (anemia of chronic diseases, hematological malignancies)
- PT/APTT/INR- NORMAL
- USG ABDOMEN- NORMAL — to r/o hemochromatosis
- SE.FERRITIN- 302.4 nanogm/dl (22-322)
- VIT B12–445 pgm/ml – normal (200-900) — to r/o megaloblastic anemia/
- FOLATE LEVEL- 30.9 nmol/L- normal (4.5-45.3) Folate deficiency
- Serology for HEPATITIS A & B- Negative
- OGD SCOPY & COLONOSCOPY- Normal-------to r/o Peutz Jegher syndrome
- USG NECK- NORMAL
- F.T3- 2.56 pgm/gl(2.3-4.2)
- F.T4- 1.06 ng/dl(0.89-1.76)
- TSH- 4.45 uIU/ml(0.5-6.0)--→ to r/o hyperthyroidism

In view of hyponatremia, hyperkalemia, postural hypotension and diffuse hyper pigmentation, we did

SE.ACTH- 927.0 pgm/ml (< 46) ---elevated

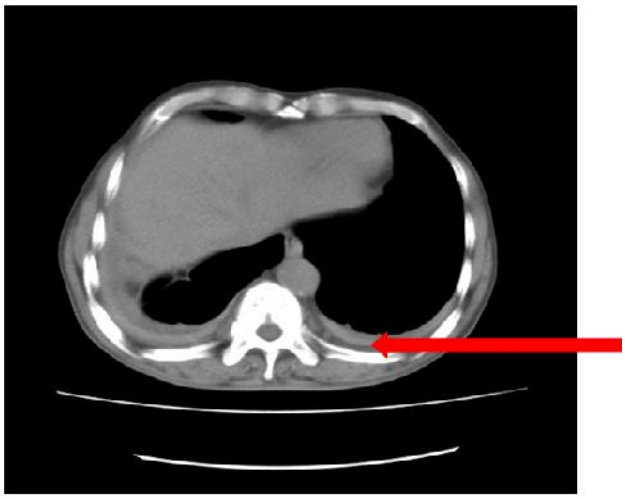
- SE. CORTISOL- 2.01 microgm/dl (4.3-22.4)---reduced
- SHORT CO-SYNTROPIN TEST WAS POSITIVE(a rise of cortisol levels >20microgm/dl after 30 min of injection of ACTH)
- SE.RENIN/ALDOSTERONE RATIO ELEVATED

Thus a primary adrenal insufficiency was confirmed. To find out the cause for it, we proceeded with the following

- ANA- 0.2 (<1- NEGATIVE)
- ADRENAL AUTO ANTIBODIES- NEGATIVE — to r/o autoimmune
- ANTI-THYROID PEROXIDASE ANTIBODY- NEGATIVE — Adrenalitis
- CT BRAIN- NORMAL STUDY
- Se. Uric acid- 6.0 mg/dl — to r/o any underlying
- Se. LDH- 18 IU/l — malignancy
- Se. calcium- 10.1 mg/dl

CT CHEST WITH CONTRAST

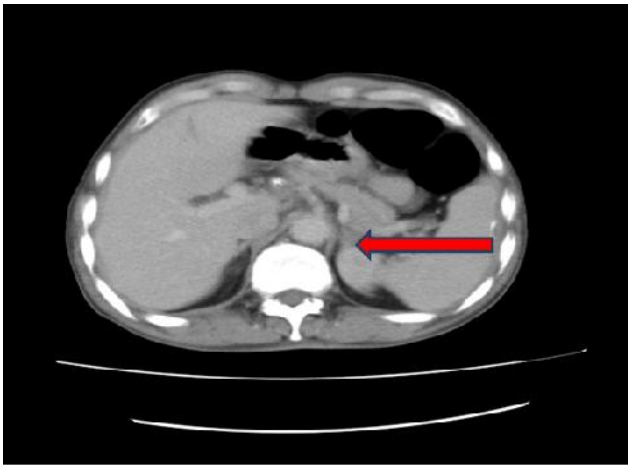
BILATERAL PLEURAL THICKENING WITH MINIMAL RIGHT PLEURAL EFFUSION



MANTOUX TEST- 30MM-STRONGLY POSITIVE

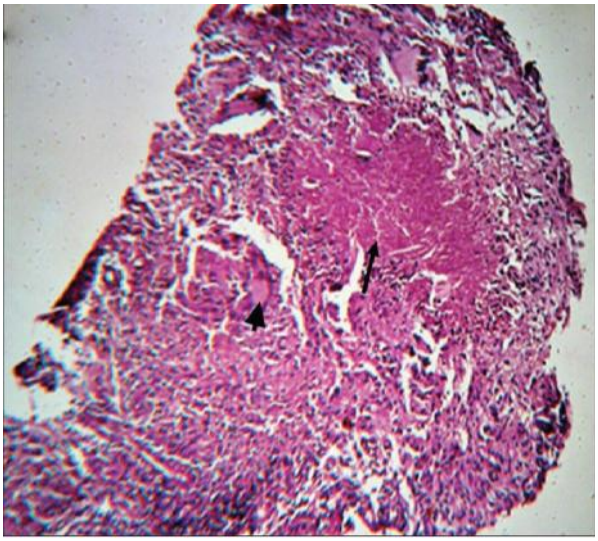
CECT ABDOMEN WITH CONTRAST & ADRENAL CUTS….

- There is hypertrophy with nodular thickening of (L) adrenal gland
- There is minimal nodular thickening with cystic changes noted in ® adrenal gland
- There are few enlarged paraaortic, retrocaval and calcified periportal lymph nodes noted
- Prominent extrarenal pelvis noted
- Imp- bilateral adrenal nodularity suggestive of Tuberculous etiology



CT GUIDED BIOPSY OF PLEURAL THICKENING WAS DONE

Multiple sections showed GRANULOMAS WITH CASEOUS NECROSIS & LANGERHAN'S CELLS., features suggestive of TUBERCULOSIS



THE FINAL DIAGNOSIS

ADRENAL TUBERCULOSIS DUE TO DISSEMINATED TUBERCULOSIS RESULTING IN PRIMARY ADRENAL INSUFFICIENCY-ADDISON'S DISEASE.

TREATMENT GIVEN

1. IV fluids to correct the prerenal failure
2. Inj. Hydrocortisone 100mg iv TDS followed by oral prednisolone
3. T. fludrocortisone 0.1 mg per day
4. CAT 1 ATT
5. Supportive measures

DISCUSSION

Adrenal sufficiency is a condition in which there is destruction of the adrenal cortex and subsequent reduction in the output of adrenal hormones i.e glucocorticoids - cortisol and mineral corticoids - aldosterone. There are two types of adrenal insufficiency Primary- adrenal cortex destruction due to autoimmune etiology; Secondary-adrenal destruction due to infection, infiltration, infection, malignancies etc

Today in developed countries, primary adrenal insufficiency is a relatively rare disease. Betterle et al, noted a high level of autoimmune form of the disease from European studies., which was most common and ranged from 44.5- 94% of all cases, compared with Addison's disease due to tuberculosis or other causes, which ranged from 0-33.3%.

The symptoms of primary adrenal insufficiency manifest when more than 90% of the adrenal glands have been destroyed. For this reason, Addison's disease due to TB manifest relatively late in life, predominantly in persons aged 40-60 years.

The major CT finding in adrenal TB are bilateral enlargement of the adrenal glands with calcification. When adrenal TB is active, enhanced CT shows enhancement of the peripheral rim of the gland showing a lower degree of attenuation. But, idiopathic Addison's disease is not characterized by either adrenal enlargement or calcification on CT. The enlarged adrenals gradually shrink because of calcification and fibrosis.

The best way to confirm the diagnosis is by a biopsy of the gland itself, which was deferred due to multiple reasons, especially when the size < 4 cm, the risk of internal organ damage and hemorrhage is high.

CONCLUSION

Adrenal TB is a rare endocrinal disorder, in the developed nations it usually related to autoimmune disorder but in the developing nations it is widely associated with tuberculosis. It is an important disease entity that must be identified early and treated promptly and aggressively.

Here is a case which presented with generalized hyperpigmentation and classical signs of mineral corticoid and glucocorticoid deficiency with no sex steroid deficiency. On further workup we found it to be primary adrenal insufficiency, and by ruling out all other possible causes in this age group, the etiology was narrowed down to tuberculosis. Correlating with the pleural thickening and positive biopsy from the pleural thickening, the diagnosis was narrowed down to Addison's disease due to disseminated TB.

This highlights the varied and rare presentation of adrenal tuberculosis which is not much reported in literature and the fact that tuberculosis still continues to be the most common treatable infection for Addison's disease.

REFERENCES

1. Betterle C, Dal Pra C, Mantero F, Zanchetta R (2002) Autoimmune Adrenal Insufficiency and Autoimmune Polyendocrine Syndromes: Autoantibodies, Autoantigens, and Their Applicability in Diagnosis and Disease Prediction. Endocr Rev 23: 327-364.

2. Nomura K, Demura H, Saruta T (1994) Addison's disease in Japan: characteristics and changes revealed in a nationwide survey. Intern Med 33: 602-606.

3. Falorni A, Laureti S, De Bellis A, Zanchetta R, Tiberti C, et al. (2004) Italian Addison Network Study: Update of Diagnostic Criteria for the Etiological Classification of Primary Adrenal Insufficiency. J Clin Endocrinol Metab 89: 1598-1604

4. Gordon HW, Robert GD (2008) Disorders of the adrenal cortex: Harrison's Principles of Internal Medicine (17ed) The McGraw-Hill companies New York.

5. Guo YK, Yang ZG, Li Y, Ma ES, Deng YP, et al. (2006) Addison's disease due to adrenal tuberculosis: Contrast-enhanced CT features and clinical duration correlation. Eur J Radiol 62: 126-131.

6. Sun ZH, Nomura K, Toraya S, Ujihara M, Horiba N, et al. (1992) Clinical significance of adrenal computed tomography in Addison's disease. Endocrinol Jpn 39: 563-569.

7. Hauser H, Battikha JG, Wettstein P (1981) Pathology of the adrenal glands. Common and uncommon findings in computed tomography. Eur J Radiol 1: 215-226.

8. Long Wang, Yanq J (2008)Tuberculous Addison's Disease Mimics Malignancy in FDG-PET Images. Inter Med 47: 1755-1756.

9. Jhala NC, Jhala D, Eloubeidi MA, Chhieng DC, Crowe DR, et al. (2004) Endoscopic ultrasound-guided fine-needle aspiration biopsy of the adrenal glands: analysis of 24 patients. Cancer 102: 308-314.

10. Mazzaglia PJ, Monchik JM (2009) Limited value of adrenal biopsy in the evaluation of adrenal neoplasm: a decade of experience. Arch Surg 144: 465-470.

11. Nigam R, Bhatia E, Miao D, Yu L, Brozzetti A, et al. (2003) Prevalence of adrenal antibodies in Addison's disease among north Indian Caucasians. Clin Endocrinol 59: 593-598

12. Higashi T, Fukuhara S (2009) Antibiotic prescriptions for upper respiratory tract infection in Japan. Intern Med 48: 1369-1375.

13. Ang D, Hsu AA, Tan BH (2006)Fluoroquinolones may delay the diagnosis of tuberculosis. Singapore Med J 47: 747-751.

14. Ginsburg AS, Hooper N, Parrish N, Dooley KE, Dorman SE, et al. (2003)Fluoroquinolone resistance in patients with newly diagnosed tuberculosis. Clin Infect Dis 37: 1448-1452.

15. Long R, Chong H, Hoeppner V, Shanmuganathan H, Kowalewska-Grochowska K, et al (2009) Empirical treatment of community-acquired pneumonia and the development of fluoroquinolone-resistant tuberculosis. Clin Infect Dis 48: 1354-1360.