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Primary Adrenal Insufficiency with Autoimmune Thyroditis.

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Abstract: *A 30year old female,known case of hypothyroidism since 4 months on Levothyroxine therapy,had came with circulatory collapse.She also had weight loss and hyperpigmentation of skin since one year.*
Initially patient was hemodynamically unstable, and also had reduced urine output, patient was started with fluid resuscitation and adequate inotropic support. Appropriate glucocorticoid and mineralocorticoid replacement was started with rapid clinical response.
The probable diagnosis was put as primary adrenal sufficiency with acute kidney injury.
Keyword: *Primary adrenal insufficiency, hyperpigmentation, hyperkalemia, APS-2, Autoimmune thyroiditis, Autoimmune adrenalitis.*

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I. Introduction

Addisons disease is a rare endocrine disease caused by inadequate secretion of renal cortical hormones –cortisol,aldosterone and androgens.

Cortisol,the main hormone affected in this disorder is important in body's ability to cope with stressful situations such as infection,hypotension, and surgical procedures.Also involved and with an overriding influence on the adrenals is hypothalamic-pituitary adrenal axis. Addisons disease is a term restricted to primary adrenocortical insufficiency. Primary adrenal insufficiency can be dreadful as cortisol secretion cannot be increased on demand at all.

The prevalence of adrenal insufficiency is 5 in 10,000 of general population.

Hypothalamic –pituitary origin of disease is most frequent with a prevalence of 3 in 10,000, whereas primary adrenal insufficiency has a prevalence of 2 in 10,000. Approximately one half of latter cases are acquired mostly caused by autoimmune destruction of adrenal glands, the other half are genetic.

Primary adrenal insufficiencymostly caused by autoimmune adrenalitis. Isolated autoimmune adrenalitis accounts for 30-40%whereas 60-70% develops adrenal insufficiency as a part of APS.

II. Case Report

A 30 yr old female G2P2L2 had sudden onset chest pain and palpitation since evening , decrease urinary output(since 2days) and also had complaints of weight loss of 4-5kg in last 4 months forwhich she was brought to the casualty. Patient also had complains of generalised body hyper-pigmentation which was accompanied with pigmentation in palmar-creases and oral mucosa. There was no history of breathlessness, giddiness, any episode of loss of consciousness. Patient also had no complains ofswelling over body, decreased appetite ,fever, nausea, vomiting, burning micturition.

Patient had a past history of hypothyroidism since 5 months on Levothyroxine therapy (Tab Thyrox 50ug OD) but she discontinued it since last 4days.There was no significant family history or drug history in the past.

In casualty, On examination patient was hemodynamically unstable, patient had tachycardia pulse rate was (110beats/min, regular, low volume) Blood pressure was 70 systolic taken on right arm supine position, her BMI was 17. She presented with Pallor+, but there was no lymphadenopathy and pedal oedema. The cardiovascular,respiratory,abdominal(no any mass palpated) and central nervous system were normal.Patient had an episode of hypoglycaemia in casualty(Blood sugar levels:56mg/dl).
The investigations done were as follows:

Parameters	26/5/18	27/05/18	29/05/18	30/05/18	1/06/18
HB	10.9	9.2			
TLC	23210	20840			
PLT	446000	423000			
UREA	82	42	40		
CREAT	2.2	1.9	0.8		

Sr Na	130	129	130	134	135
Sr K	6.6	6.2	5.8	5.2	3.8

SEROLOGY : NEGATIVE
FT3:4.65 , FT4: 0.86, TSH:>100
SERUM CORTISOL:5ug/dl(10-20ug/dl) collected in morning hours.
ANTI TPO antibodies 918.53(<=5.61)
ACTH levels:165.1 (7.2-63.3pg/ml
Serum aldosterone:2.24 ng/dl (supine:1.76-23.2; upright :2.52-39.2)
Chest XRAY (PA view) : No significant pathology.
CECT abdomen was suggestive of bilateral normal adrenal glands for age, right sided (13x2mm) and left sided (12x03mm)and there were no signs of calcification visualised.

III. Discussion

Primary adrenal insufficiency has an incidence of 0.8 per million and prevalence of 40-110 per million in USA and Europe.Often there is delay in diagnosis due to lack of suspicion on account of subtle nature of signs and symptoms in many patients.Usually presenting symptoms include weight loss(90%) , generalised weakness(20%), hyperpigmentation(91%) and hypotension(90%). Common laboratory imbalances includes hyponatremia(88%), hyperkalemia(64%) and anemia(40%).

IV. Secondary Adrenal Insufficency:

Hypothalamic-pituitary disease can leads to clinical infestations like visual impairment with bitemporal hemianopia caused by chiasmal compressions, and also this disease does not present with hyperpigmentation, hyperkalemia, hyponatremia.ACTH levels were also increased whereas in secondary adrenal insufficiency it is reducedl hence it is ruled out too.

APS-2 (Autoimmune Polyglandular Syndrome Type 2)

APS-2 is characterised by primary adrenal insufficiency associated with autoimmune thyroiditis,Diabetes mellitus(Type I), vitiligo, pernicious anemia and primary ovarian failure,however patient has not presented with the latter symptoms so APS-2 per se can be ruled out.

Triple A Syndrome/Congenital Adrenal Hyperplasia :

Triple A syndrome and congenital adrenal hyperplasia develops at childhood, but this patient is middle aged (30years).

Adrenal Infections:

Since Chest X-ray and CECT abdomen was normal tuberculosis is ruled out, furthermore serology(HIV,HBSAG,HCV) is nonreactive, infective pathology is by and large ruled out.

Adrenal Hemorrhage/ Lymphomas:

Since CECT suggests normal adrenal glands , hence these two are also not included as both have large adrenals on CT scan.

The patient presented with both acute and chronic symptoms consistent with primary adrenal insufficiency. Autoimmune adrenal insufficiency is generally an insidious process with first zona glomerulosa being affected,which may cause low aldosterone levels,followed later by zona fasciculate involvement with decrease in serum cortisol levels and increase in ACTH levels.

Since ANTI TPO antibodies were strongly positive which was suggestive of autoimmune thyroditis, hence autoimmune adrenalitiswas considered(Schmidt's syndrome)

Acute treatment included Intravenous Hydrocortisone (100mg)6hrly,with aggressive fluid resuscitation in the form ofnormal saline and inotropic support to treat hypotension,correction for Hyperkalemia was also initiated. Patient was started with tapered dose of Intravenous Hydrocortisone i.e.(50mg)6hrly once patient was hemodynamically stable and electrolytes were normal. Furthermore, she was subsequently shifted tooral Hydrocortisone. Mineralocorticoid treatment with Fludrocortisone(100ug)once daily was simultaneously started.

She was discharged home with sick day advise to double up her Hydrocortisone dose.She was also advised to keep IV Hydrocortisone with her in case of any emergency,and asked to follow up every monthly for monitoring of electrolytes, blood pressure and weight charting and thyroid profile every 3 monthly.

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