ORIGINAL RESEARCH PAPER

General Medicine

KEY WORDS: Unusual presentation, Gastro enteritis, Addisonian crisis.

A DEVIOUS DISEASE DECEPTIVELY PRESENTING AS A STRAIGHT FORWARD GASTRO ENTERITIS.

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ABSTRACT

Internal Medicine is an amazing faculty in which frequently things are just NOT what (and how) they appear. It'll not be an exaggeration to say that it's a game of mental chess that we physicians play while dealing with cases. Here we describe a case who, prima facie, seemed to be a straightforward gastro enteritis, but turned out to be something totally different.

CASE REPORT:

A 32 Years old Male IT Professional presented with features of acute gastro enteritis. He had 6-8 episodes of vomiting with blood in vomitus since 1 day, 16-17 episode of watery loose stools, Fever with shivering and Cramps and pain in abdomen in epigastric and periumbilical region.

He was on no regular Medications. Besides Past history of Dengue associated SIADH in November 2019, there had been no other significant past illnesses/allergies/family history. He denied any vices.

ON EXAMINATION:

- Found to have severe dehydration with a dry tongue and poorly reverting skin folds pinches.
- Low BP-100/60mmHg initially and dropped further later on despite good oral intake.
- RBS-63mg/dl which was ascribed by him and his wife to a pathetically low oral intake.
- Fever- Oral temp 101F.

Despite sufficient IV fluid replacement (2-3 Liters of RL/DNS), BP did not rise as expected. Though he was conscious and oriented, He preferred to communicate minimally, seemed morose and preferred to lie undisturbed in bed. His lab reports were alarming too. Arterial blood gas analysis was suggestive of severe metabolic acidosis.

At this point of time, his original working diagnosis of "simple Gastro enteritis" was reviewed. Endotoxin induced gram negative septicemia was considered. Noradrenaline was started, IV fluids escalated and patient was shifted to ICCU for further management. Treatment was started with IV antibiotics, IV Dextrose, IV antacid, IV antiemetic and IV paracetamol. His BP was maintained at a MAP (mean arterial pressure) of 70-75.

On day 2 of ICCU stay, he had 2-3 episodes of hematemesis with around 50 ml of frank blood. OGD-scopy was done which was suggestive of retch induced gastropathy with antral and duodenal tears. PPI infusion and Vitamin K injections were given. By the end of day 2 hypotension, hyponatremia, relative bradycardia were persistent but not really too alarming.

At this point of time, Cortisol deficiency was thought of. Sample was sent to measure serum cortisol and thyroid hormone levels. Serum cortisol was found to be less than 1.

So, his diagnosis finally emerged as "acute Addisonian crisis"

Table of Investigations:

CBC	HB=15,PCV=46,WBC=6700,PLT=1,90,000 ,N=67%
CREATININE	0.9
ELECTROLYTES	136/4/103
PT/INR	16.2/1.21
T.BILI	1.2 D=0.16
SGOT/SGPT	26/25
ALK.PHOS	52
LIPASE	182
SR.CORTISOL	< 1 mg/dl
TRIPLE-H	Negative
CRP	106
CALCIUM	7.1
PHOSPHATE	2.4
PROLACTIN	7.71
LH	2.2
SERUM TESTOSTERONE	18.88
T3/T4/TSH	17/1.9/6.75
BLOOD C/S	No growth
STOOL:R/M	Normal
CB-NAAT COVID -19	Negative
PS-MP	negative
BUN	16

USG Abdomen and Pelvis:

- Mild hepatomegaly
- Few reactive lymph nodes in the right iliac fossa.

MRI Brain:

1. Pituitary measured 4.1 x 5.7 x 9.3 mm in size with a small appearing anterior gland and normal appearing posterior gland.
2. No other significant intracranial abnormality was detected.

CT Abdomen and Pelvis:

CT scan of the abdomen and pelvis revealed a subtle hypo density along the main portal vein and in its intra hepatic branches which raised possibility of peri-portal edema. There was a thin sliver of fluid seen surrounding the GB (? liver parenchymal disease). Adrenals were unremarkable.

Treatment Plan:
INJ Hydrocortisone 100 mcg was given empirically once sample was sent for serum cortisol measurement, even without waiting for the report. After serum cortisol was found to be low, IV hydrocortisone was continued. Thyroxin was given to correct hypothyroidism. He responded well to Hydrocortisone therapy and his vital parameters improved. Ionotropic support, IV dextrose and antibiotics all were stopped in the ensuing 1-2 days. His BP improved to 110/70 mmHg without ionotropic support and patient was shifted out of ICU to ward on day 4. IV steroids were tapered off and oral hydrocortisone 10 mg TDS was started and patient was discharged on seventh day of admission.

FOLLOW UP:
On reassessment in OPD, patient reported excellent recovery and resolution of all symptoms like vomiting, loose motion, low sugar and fever. Oral Hydrocortisone was continued at 5 mgs in morning and 2.5 mgs in evening. Thyroxine has been continued at 100 mcg. Currently patient is perfectly fine without recurrence of any symptoms.

Discussion:
The incidence of adrenal crisis is estimated to be 8 per 100 patient years in known patients with adrenal insufficiency. Prospective data indicate 0.5 adrenal crisis related deaths per 100 patient years. [1,6].

Adrenal crisis or acute adrenal insufficiency (also known as Addisonian crisis) is an endocrinologic emergency causing high mortality due to physiologic derangements resulting from an acute deficiency of cortisol. Early identification and management are necessary to avoid death. [2,3]

Acute adrenal insufficiency is seen more commonly in patient with primary adrenal insufficiency due to loss of both glucocorticoid and mineralocorticoid secretion.

It can present with nonspecific signs and symptoms often leading to misdiagnosis and delayed management. Infections are the major precipitating factor, but other causes like physical stress (such as surgery, trauma, adrenal hemorrhage), discontinuation of glucocorticoid therapy or even psychological stress can result in adrenal crisis. Emergency treatment involves prompt recognition and administration of parenteral hydrocortisone (100 mg immediately and every 6 hours), rehydration and management of electrolyte abnormalities. [1,5]

An adrenal crisis is the first presentation in most cases of hitherto undiagnosed Adrenal Insufficiency. A cross-sectional study of both primary adrenal insufficiency and secondary adrenal insufficiency showed that only 50% of the patient were diagnosed within first 6 months from the onset of symptoms with as many as 20% patients reporting symptom duration of more than 5 years before diagnosis. [4]

In many cases the disease has an insidious onset and diagnosis is made only when patient presents with acute adrenal crisis during an inter-current illness. It is a medical emergency manifesting as hypotension and acute circulatory failure. [5] And can mimic signs and symptoms of acute abdomen with abdominal tenderness, nausea, vomiting and fever. [6]

Secondary adrenal insufficiency is twice as common as primary adrenal insufficiency but rates of adrenal crisis are higher in in primary adrenal insufficiency than in secondary adrenal insufficiency. [7] A recent study reported that 65% of primary adrenal insufficiency patients had experienced an adrenal crisis as compared to 47% of secondary adrenal insufficiency patients. [8]

DIAGNOSIS:
ACUTE ADRENAL CRISIS WITH HYPOTHYROIDISM.

Conclusion:
This is a case of acute adrenal crisis with concomitant hypothyroidism. It presented deviously as a simple and straight forward gastro enteritis. Severe hypotension requiring ionotropic support, metabolic acidosis and low sugars aided in making a prompt diagnosis. Serum cortisol levels confirmed the suspicion. Once Cortisol was found to be low, other hormones too were assayed, which showed hypothyroidism as well.

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