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Case Study on the Impact of Prolonged Use of Steroid Cream on Serum Cortisol in a Young Man

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ABSTRACT

We present a case of tertiary adrenal insufficiency following a prolonged intermittent course of potent steroid cream for a difficult case of eczema. The patient presented to a teaching hospital with recurrent transient loss of consciousness, hypoglycaemia, and sepsis from pneumonia. Intravenous antibiotics and aggressive intravenous fluid were given, CT chest confirmed bronchopneumonia and tuberculosis (TB) was ruled out. Transient loss of consciousness and mild hypoglycaemia (3.2 mmol/L) was felt to be secondary to intravascular depletion from sepsis and the patient was discharged home after four days of intravenous treatment with a plan to continue the course of oral antibiotics to complete seven days of treatment in total. The patient had poor recovery at home with ongoing loss of appetite and energy, recurrent transient loss of consciousness mainly after meals, no seizure activity was noted, and rapid recovery within seconds was followed in each episode. Follow up appointment in clinic noted postural drop in blood pressure with normal renal function and electrolytes, 9 am cortisol was checked and with a very low 68 nmol/L result. This case highlights the importance of expecting true or relative cortisol deficiency in acutely unwell patients who present with syncope and hypoglycaemia in the absence of other common causes of transient loss of consciousness.

Keywords: Steroid; Serum Cortisol; Young Man

CASE PRESENTATION

A 17-year-old man with a past medical history of severe eczema presented to hospital with fever, productive cough, and shortness of breath. Chest X-ray and CT chest confirmed the presence of multiple areas of consolidation suggestive of bronchopneumonia, covid-19 PCR was negative, and TB was ruled out. Our patient was treated with intravenous ceftriaxone and azithromycin (hospital protocol for community acquired pneumonia) and aggressive intravenous fluid therapy. His blood results showed raised inflammatory markers, normal renal function, and HIV was also ruled out.

Symptoms of loss of appetite, lack of energy, recurrent transient loss of consciousness and low random glucose levels were felt to be part of the constitutional symptoms of sepsis, however, he was heavily investigated including Holter monitoring which showed two episodes of bradycardia 50-55 for few minutes without drop in

blood pressure nor symptoms. Transthoracic echocardiogram was essentially normal, as well as MRI brain and EEG.

The patient was discharged after four days on oral antibiotics, but symptoms of transient loss of consciousness were persistent interestingly during meals with prodromal symptoms, colour change, and rapid recovery.

Follow up review in the clinic noted postural hypotension (systolic blood pressure dropped by 17 mmHg only and diastolic blood pressure dropped by 6 mmHg only, however, patient was symptomatic). Hence 9 am cortisol was ordered which came back very low 68 nmol/L.

We started prednisolone 5 mg for convenience instead of hydrocortisone 10/5/5 mg as per patient preference and started fludrocortisone 100 mcg OD and treated this as potentially Addison disease. An MRI adrenal was performed which came back as normal and antibodies against 21-hydroxylase antibodies were negative.

Patient did feel much better on prednisolone with dramatic improvement in appetite and energy levels, no further postural drop in blood pressure, or transient loss of consciousness.

ACTH level came back a week later as low normal 11.74 pg/ml (reference 7.2 – 63 pg/ml), this result is not expected in primary hypoadrenalism and a secondary or tertiary hypoadrenalism felt to be more likely, pituitary gland was imaged and showed a probably incidental finding of 4 mm which did not show enhancement with contrast and rest of pituitary screen was normal including IGF-1, Prolactin, TSH, LH, FSH, testosterone, and T4.

A thorough history about the steroid cream was initiated. The patient gave a history of use of multiple steroid creams intermittently but over a period of six months on face, arms, and chest. The patient used beclomethasone (medium potency), mometasone (high potency), and clobetasol (super potent) alternating over the last six months.

ACTH stimulation test was arranged following an MDT discussion, results showed zero minutes cortisol 362 nmol/L, 30 minutes cortisol (after ACTH 250 mcg IM injection) 463 nmol/L, and one hour cortisol level of 558 nmol/L. This was felt to be a positive stress test and prednisolone was stopped.

It is important to say that the ACTH stimulation test was arranged after 3 days from last steroid cream use and 36 hours of last oral prednisolone tablet use.

Follow up in clinic showed no symptoms or signs of hypoadrenalism, and no postural drop in blood pressure.

DISCUSSION

Normal cortisol level at 9 am is usually between 275 to 555 nmol/L, level below 80 nmol/L is strongly suggestive of adrenal insufficiency.[1]

While 9 am cortisol level of more than 415 nmol/L excludes adrenal insufficiency.[2]

Primary adrenal insufficiency presents with low 9 am cortisol and simultaneous rise in ACTH, plasma aldosterone and renin levels should also be checked (were normal in our case). If ACTH level is low, this points towards secondary pituitary disease or tertiary hypothalamic disease as the cause of adrenal insufficiency.

Short ACTH stimulation test is performed by injecting 250µg cosyntropin IM or IV after withdrawing a sample for basal cortisol and ACTH. Further samples of cortisol are taken at 30 and 60 minutes. Adrenal insufficiency is excluded by an incremental rise in cortisol of >200 nmol/L and a 30 min value >550 nmol/L.

In acute adrenal crisis the priority is to treat with IV steroids and normal saline, short ACTH stimulation test can be performed within few days after which the hypothalamic-pituitary-adrenal axis can be compromised.[3]

Primary adrenal insufficiency will show raised ACTH and Renin levels, with low aldosterone, also, a raised potassium with a low sodium level.

Secondary or tertiary adrenal insufficiency will show a low or low normal ATCH level, no change in renin and aldosterone levels, sodium level can be reduced due to increased vasopressin level, but potassium level should be normal.[4]

Imaging the adrenal glands is indicated to rule out tumours, calcifications, or haemorrhage. Ruling out TB and HIV is important and checking for adrenaline antibodies (antibodies against 21-hydroxylase) is indicated in patients presenting with primary adrenal insufficiency. Pituitary imaging is important in patients with secondary adrenal insufficiency.[5,6]

Secondary adrenal insufficiency is due to low ACTH, this can occur in isolation or associated with panhypopituitarism.[7]

Isolated ACTH deficiency is a rare disorder.[8] In rare cases autoimmune lymphocytic hypophysitis with selective corticotrophin absence can occur.[9]

Traumatic brain injury more commonly causes growth hormone or gonadotropin deficiency, but ACTH deficiency leading to secondary adrenal insufficiency occurred in 8 percent of 611 patients in one meta-analysis.[10]

Another common cause of secondary adrenal insufficiency is chronic administration of opiates.[11]
Tertiary adrenal insufficiency can commonly occur due to suppression of CRH by suppressing the hypothalamic-pituitary-adrenal function by using high doses of glucocorticoids for a duration of 3 weeks or more.

High doses of glucocorticoids decrease hypothalamic CRH synthesis and secretion leading to pituitary corticotrophin reduction in size.[12]

Glucocorticoids may be taken via the oral, ocular, inhaled, transdermal, rectal, or parenteral routes and a thorough history regarding medications, creams, and supplements should be obtained.[13]

Learning points

-Acute physicians should be aware of the possibility of absolute or relative cortisol deficiency in patients presenting with syncope in a context of infection if other causes like cardiac, renal failure, or antihypertensives are ruled out.

-Thorough medication history taking if steroid deficiency is expected including other routes like topical potent steroids.

-Importance of ACTH stimulation test in assessing the integrity of adrenal glands and how to interpret the results.

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