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Acute Adrenal Crisis: Importance of Perioperative Management in a Patient with Necrotizing Fascitis of Lower Limb.

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ABSTRACT: This is a case report of 65 year old male who presented with necrotising fasciitis of leg lower limb who underwent emergency fasciotomy and debridement and was discharge with uneventful postoperative period , only to return to the casualty in respiratory distress and in shock. With further evaluation in ICU, patient was found to be in acute adrenal crisis and on further detailed history it was found that patient was on steroids for chronic joint pain and he had stopped abruptly which had lead to the current scenario
Conclusion:Surgeons treating acute conditions should be aware of the HPA axis suppression and its appropriate therapy at emergency and further requirement of evaluation and referral for the best patient care
KEYWORDS: Necrotising Fascitis, Fasciotomy, Acute adrenal crisis, shock.

I. BACKGROUND

Adrenal crisis is one of the rare and lethal complications encountered after surgery. The incidence of adrenal crisis was 9.0 crises per 100 patient-years at risk.(1).The incidence of adrenal crisis appears to increase with age with one study reporting a rate of 24.3 admissions per million per year in patients aged 60–69 years(10). The hypothalamic–pituitary–adrenal axis has an important role in the body's ability to cope with stresses such as infections, hypotension, and surgery(2). Patients in adrenal crisis present with fever, tachycardia, and hypotension which might be similar to postoperative sepsis(1). Hence, high index of suspicion is required to diagnose the condition(1). We report here a patient with acute adrenal crisis, who presented with shock post discharge from the hospital following Debridement for necrotizing fasciitis.

II. CASE PRESENTATION

A 65 year old male patient presented with h/o swelling of left foot and leg since 3 days sudden onset gradually progressive associated with pain and bleb over the leg. Patient also gives h/o chronic joint pains for which he was on Tab. Methyl prednisolone 8mg for 1 year and had stopped the medication 3 days ago.

On admission vitals were stable, HbA1c was found to be 9%. Initial dose of 100mg Inj. Hydrocortisone was given and patient was taken up for emergency fasciotomy and debridement of left lower limb in view of compartment syndrome.
Postoperative period was uneventful and hence the patient was discharged on POD5
1 week later patient came back to casualty with h/o difficulty breathing and reduced responsiveness and on examination vital : tachycardia and hypotension suggestive of shock: local examination revealed wound was healthy with no signs of infection and probable cause for sepsis was ruled out.

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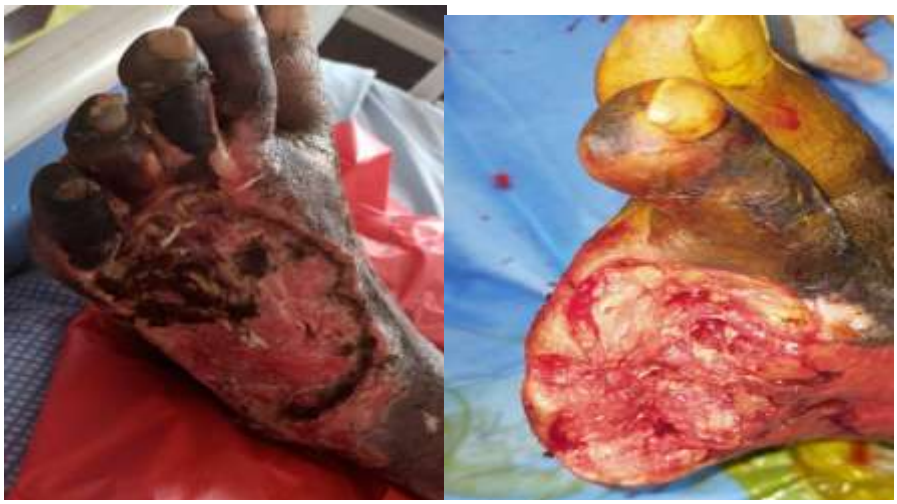
On further evaluation HbA1c was found to be 9% and Serum cortisol level was 4.4mcg/dl (Normal:5-23) and Patient was diagnosed to be in adrenal crisis and hence endocrinology opinion was taken and replacement dose of steroids were started and Insulin was given for control of blood sugars.

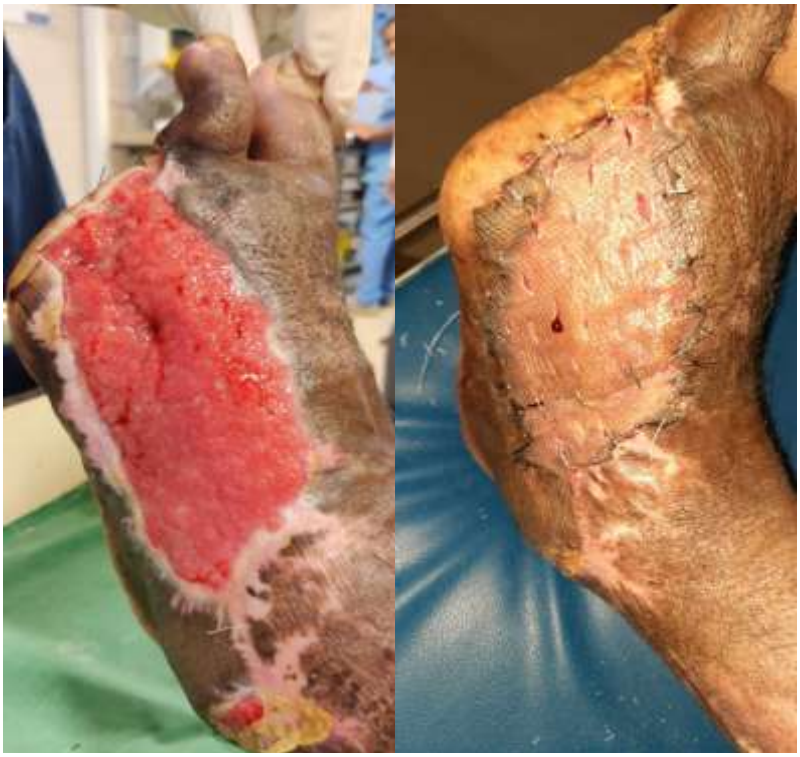
Patient improved symptomatically and was discharged later.

In view of dry gangrene of 3rd, 4th and 5th toe patient underwent disarticulation of the toes under SA under the cover of steroids .Postoperative period was uneventful.



Regular dressings were done and further after obtaining endocrinology fitness and under steroid cover patient underwent split skin grafting under spinal anaesthesia, which took up well with no further complications.

Patient was tapered off the steroids gradually and counselled regarding the harmful effects of abuse of steroids.



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III. DISCUSSION

Acute adrenal crisis may occur as a result of stress in patients not given supplemental glucocorticoids and known to have chronic adrenal insufficiency.(4)

However, the most severe cases are due to adrenal haemorrhage or infarction. Such patients are usually already seriously ill as a result of thromboembolic disease, coagulopathy (including antiphospholipid antibody syndrome), traumatic shock, severe burns, or sepsis(4).

The pathophysiology of adrenal damage is probably related to stress-induced increase in ACTH concentrations which increase adrenal blood flow to a degree that exceeds the capacity for venous drainage(5).Chronic exogenous glucocorticoid treatment can also impair pituitary regulation of cortisol production, often termed tertiary adrenal insufficiency(9).

Acute adrenal crisis in the post-operative period is an under diagnosed problem because of limited awareness and less exposure of the condition among surgeons . Meery H et al have documented transient cortisol deficiency in about 5% of the patient in the postoperative period. Patient with adrenal crisis present with fever, tachycardia and hypotension which might mimic other postoperative complication.

In our case since it was a case of necrotizing fasciitis it is even more confusing as the cause of this presentation is most commonly sepsis. The awareness about adrenal crisis is necessary and this is to be thought of when a patient who has undergone surgery presents with hypotension despite of fluid replacement.

Diagnosis of adrenal insufficiency is well established. The usual recommendation is to first measure early morning cortisol. If the concentrations are very low, the diagnosis is established. In the setting of primary and secondary adrenal insufficiency, random cortisol levels alone are usually not adequate in providing a diagnosis because of the wide fluctuations due to the pulsatile nature and diurnal variation of secretion(6) Cooper et al. stated that adrenal insufficiency appears to be likely when a random cortisol measurement is below 15 µg/dl during acute severe illness(8). If the concentrations are intermediate, an ACTH stimulation test should be performed. A normal test is based on intravenous injection of the entire vial (250 µg) with measurements of cortisol at 0, 30, and 60 min. A single dose of 0.25 mg of cosyntropin will stimulate the adrenal cortex maximally and to the same extent as 25 units of natural ACTH. The half-life of cortisol is in the range of 70 to 120 minutes. However, the half-life for cosyntropin is only 15 minutes. Administration is by intravenous or intramuscular injection and a rise in cortisol should generally be seen around 30 minutes after administration. Plasma cortisol levels usually peak about 45 to 60 minutes after injection and a normal response is an approximate doubling of the basal plasma cortisol value. A number of other agents may interfere with cosyntropin function and subsequent response. These include metyrapone, etomidate, ketoconazole, megesterol, and mitotate to name a few. In addition, agents such as rifampin and phenytoin may increase cortisol metabolism.

Once the diagnosis is made the treatment is simple by providing replacement steroids. Emergency treatment includes immediate administration of parenteral hydrocortisone via a bolus injection of intravenous (IV) hydrocortisone 100 mg, or intramuscular (IM) hydrocortisone, pending IV access. This bolus should be followed by 200 mg of hydrocortisone per 24 h, either via continuous IV infusion or alternatively in doses of 50 mg of hydrocortisone per IV/IM injection every 6 h(11,12). Prompt fluid management with isotonic fluids is important during the early management of adrenal crisis. The requirement of fluids may increase upto 4-5 litres during the first 24 hours. Patient should me monitored closely for the features of fluid over load especially in high risk individuals with renal failure, heart failure. If the patient is clinically stable, tapering of hydrocortisone to replacement doses can be initiated usually within 24–72 h. In patients with PAI, mineralocorticoid administration should be started as soon as total daily hydrocortisone dose is lower than 50 mg/24 h(9).

Patient education is the most important component in the prevention of adrenal crisis. After an acute presentation with an adrenal crisis, a regular review by an endocrinologist, in the months following diagnosis and then 6–12 months thereafter, is recommended(9).

Patient with cellulitis with compartment syndrome will require emergency surgery as a limb saving procedure. In patients with obvious focus of sepsis that is in case of cellulitis there are more chances that we miss out on adrenal crisis when it actually occurs because of the masking symptoms of sepsis that is hypotension and tachycardia. So high level of suspicion is necessary to identify the cause of shock in these patients and should be managed appropriately. A German study investigated the knowledge of adrenal crisis across both medical and surgical disciplines in a large university hospital, and reported that only 20 physicians (9.6%) correctly identified all situations

requiring hydrocortisone adjustment(13). In addition, internists, paediatricians and neurologists had more knowledge of AI treatment than anaesthesiologists or surgical doctors, who are potentially involved in the perioperative management of patients with AI(14).

IV. CONCLUSION:

We describe a case of adrenal crisis caused by the lack of adrenal reserve due to sudden withdrawal of Methyl prednisolone which had caused hypothalamopituitary adrenal axis suppression . Surgeons treating acute conditions should be aware of the HPA axis suppression and its appropriate therapy at emergency and further requirement of evaluation and referral for the best patient care

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