EM CASE OF THE WEEK.

BROWARD HEALTH MEDICAL CENTER
DEPARTMENT OF EMERGENCY MEDICINE

BROWARD HEALTH
Care Warriors

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Adrenal Crisis

A 29 y/o female with PMH Addison's Disease and hypothyroidism presented to the ED with x 1 week of lethargy, decreased appetite, nausea and non-bloody emesis. She stated that she recently ran out of her medications, levothyroxine and hydrocortisone, and has not taken them in over 1 week. On exam patient appears to be lethargic in mild distress, tachycardic with cool extremities and dark, tan colored skin. Vital signs T: 98 HR: 77 RR: 22 BP: 61/30. Labs ACTH: 1307 Aldosterone: 1ng/dL Plasma Renin Activity: 11.97 lactic acid: 0.6 Na: 127 K: 4.5.

Which of the following is the most appropriate initial treatment for this patient's condition?

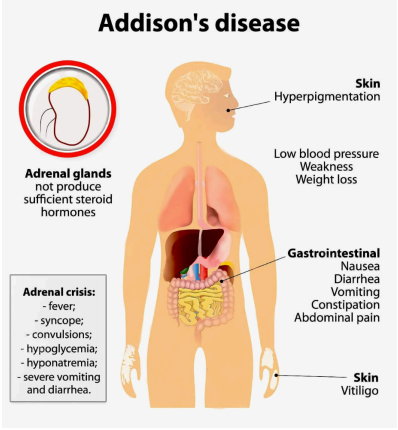
A. Hypotonic saline

B. Dexamethasone (4 mg IV bolus)

C. Hydrocortisone (100mg IV bolus)

D. Fludrocortisone

E. B and C



https://www.organsofthebody.com/adrenal-glands/addisons-diseases.php

EM Case of the Week is a weekly "pop quiz" for ED staff.

The goal is to educate all ED personnel by sharing common pearls and pitfalls involving the care of ED patients. We intend on providing better patient care through better education for our nurses and staff.

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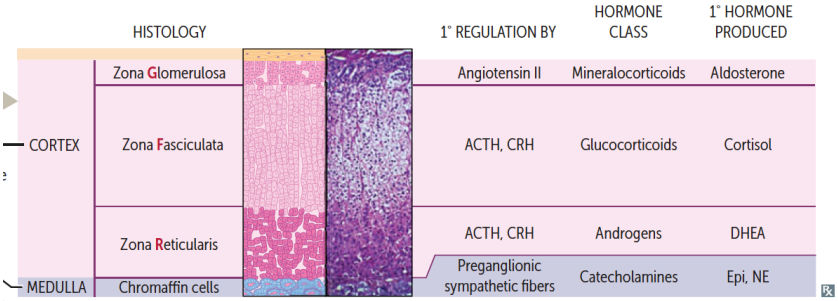
The correct answer is E. In a patient with a known history of adrenal insufficiency, hydrocortisone or dexamethasone IV bolus is appropriate. Hypotonic saline (A) would worsen the existing hyponatremia. Mineralocorticoid replacement (D) is unnecessary because IV fluids provide adequate sodium replacement in a timely manner.

Adrenal crisis, or acute adrenal insufficiency, predominantly presents as shock. Patients also may have nonspecific symptoms of nausea, vomiting, anorexia, fatigue, fever, confusion or coma [2]. It may occur in patients with chronic insufficiency during times of significant stress or infection, or in patients with secondary or tertiary deficiency receiving insufficient mineralocorticoid and glucocorticoid replacement.

Discussion

Common etiologies of primary adrenal crisis have transitioned from tuberculosis to autoimmune adrenalitis, infectious adrenalitis, hemorrhagic infarction, metastatic disease and drugs such as etomidate, ketoconazole and metyrapone.

Primary adrenal crisis typically presents with volume depletion and hypotension due to both mineralocorticoid and glucocorticoid insufficiency. Mineralocorticoids i.e. aldosterone promote sodium retention and vascular vasoconstriction. Glucocorticoid deficiency causes decreased vascular response to angiotensin II and norepinephrine. These patients may present with hyperpigmentation due to excess production of proopiomelanocortin (POMC) which is cleaved into ACTH and MSH. Treatment should not be delayed if symptoms are characteristic of a crisis. Appropriate labs include serum cortisol, ACTH, aldosterone, renin, and serum chemistry. Labs will be likely to show hyponatremia, hyperkalemia and occasionally hypoglycemia as well as elevated ACTH. Values should be used to confirm the diagnosis and/or differentials. After initial treatment, the underlying cause should be evaluated. Typically, a crisis is initiated by a viral or bacterial infection and should be treated accordingly.



First Aid USMLE Step 1 2017

Treatment

Adrenal crisis should be considered a life threatening emergency and treatment should not be delayed. It is more common in the setting of primary adrenal insufficiency but may also present in secondary or tertiary deficiency.

Initial management should include establishment of IV access with a large-gauge needle. Blood should be drawn for serum electrolytes, glucose and routine ACTH. Patient should begin infusion of 2-3L of isotonic saline or 5% Dextrose in isotonic saline. Patients require frequent hemodynamic monitoring and serum electrolytes to avoid fluid overload. A 4mg dexamethasone IV bolus should be given over 1-5 minutes, and then every 12 hours. Supportive measures should be continued as needed. Hydrocortisone should be used only if a patient has known history of adrenal insufficiency. Otherwise, hydrocortisone may interfere with plasma cortisol assays [1]

Once the patient is stabilized, continue IV saline at a slower rate over the next 24-48 hours. Possible infectious causes of the crisis should be investigated and treated accordingly. A short ACTH stimulation test should be conducted to confirm the diagnosis if history is unknown and the type of deficiency should be assessed. Parenteral glucocorticoids should be tapered over 1-3 days to oral glucocorticoid maintenance dose as tolerated. Mineralocorticoid replacement with fludrocortisone 0.1mg

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Primary Chronic Adrenal Insufficiency	Secondary or Tertiary Adrenal Insufficiency
Presenting sx are often non-specific including fatigue, weight loss, nausea, vomiting, abdominal pain, muscle and joint pain More specific presentations include hyperpigmentation due to increased POMC, postural hypotension due to mineralocorticoid deficiency, and salt craving. Reproductive changes exclusive to women include decreased axillary and pubic hair loss, loss of libido, and psychiatric symptoms including mania, depression, and psychosis [3] Common labs: hyponatremia, hypokalemia, occasionally anemia	No hyperpigmentation No hyperkalemia (aldosterone is present) Hypotension is less prominent Dehydration is absent but hyponatremia and volume expansion may be present GI symptoms are less prominent Possible manifestations of hypothalamic or pituitary tumor Lab abnormalities may be limited to hypoglycemia

Take Home Points

- Patients with adrenal crisis typically present with shock that is refractory to vasopressors and fluids.
- Common findings include nausea, vomiting, altered mental status, diarrhea, fever, fatigue, abdominal pain and most significantly hypotension.
- It is important to attain a thorough history to differentiate primary, secondary and history of long term steroid use to manage the patient properly.
- Classic lab findings include hyponatremia, hyperkalemia and/or hypoglycemia. Patients with secondary adrenal insufficiency may only be hypoglycemic due to impaired gluconeogenesis.

ABOUT THE AUTHOR

This month's case was written by Chelsea Kramish. Chelsea is a 4th year medical student from NSU-COM. She did her emergency medicine rotation at BHMC in July 2019. Chelsea plans on pursuing a career in Pathology after graduation.

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