

A Rare Case of Bilateral Adrenal Hemorrhage

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Abstract

Bilateral adrenal hemorrhage is a rare condition that may lead to acute adrenal crises, shock, and death, if untreated. It is associated with a 15% mortality rate and 55–60% mortality, if secondary to Waterhouse–Friderichsen syndrome.^[4] It can present with non-specific clinical and laboratory findings; hence, early recognition and treatment is mandatory. We report a 57-year-old female who presented with abdominal pain and vomiting found to have urosepsis causing bilateral adrenal hemorrhage.

Key words: Bilateral adrenal hemorrhage, Urosepsis, Acute Adrenal crisis

INTRODUCTION

Adrenal hemorrhage is a life-threatening condition that can present with non-specific symptoms such as abdominal pain, nausea, vomiting, and fatigue.^[1,2] Rarely, adrenal hemorrhage can be picked up as an incidental finding on imaging, without any symptoms. Examination findings are fever (most frequent), tachycardia, skin hyperpigmentation, and shock in severe bilateral adrenal hemorrhage.

Most cases are caused by acute, stressful illness (e.g., infection, acute coronary syndrome, heart failure, Waterhouse–Friderichsen syndrome - meningococcal septicemia, etc.). Other causes include blunt trauma and thromboembolic diseases such as antiphospholipid antibodies (APLA), anticoagulant use,^[3] thrombocytopenia, pregnancy complications,^[7] ACTH use, tuberculosis and rarely acute pancreatitis.^[6] A multicentric case–controlled study was done to study the major risk factors associated with bilateral adrenal hemorrhage. Thrombocytopenia, sepsis, and heparin use were identified as major risk factors.

CASE REPORT

A 57-year-old female, who is a known case of systemic hypertension on treatment, came with complaints of abdominal pain for 1 week, which was dull aching type of pain, non-localized. She also complaints of vomiting, non-bilious, non-projectile, and multiple episodes per day contains food particles for the past 1 week. Patient did not give any history of fever, burning micturition, or substance abuse. There was no history of any anticoagulant use, tuberculosis or trauma.

On examination, the patient was conscious, oriented to time, place, person, vitals stable, and afebrile. There was no skin hyperpigmentation or rashes present. Per abdomen, there was no organomegaly, epigastric tenderness was present. CNS examination revealed no focal neurological deficits.

All laboratory investigations were done which showed sodium -110, potassium - 4.2, chloride - 79, bicarbonate - 21. Serum osmolality-223, urine spot sodium-100, serum cortisol was 1.85. Echocardiogram done showed no regional wall motion abnormality with an ejection fraction of 64%. Ultrasound abdomen was done, which showed bilateral mild hydronephrosis, after which a CT whole abdomen was done which showed splenomegaly and bilateral adrenal haemorrhage (figure-1). CT thorax done showed minimal left pleural effusion. Urine culture showed Klebsiella. A diagnosis of bilateral adrenal hemorrhage due to urosepsis was made and the patient was managed

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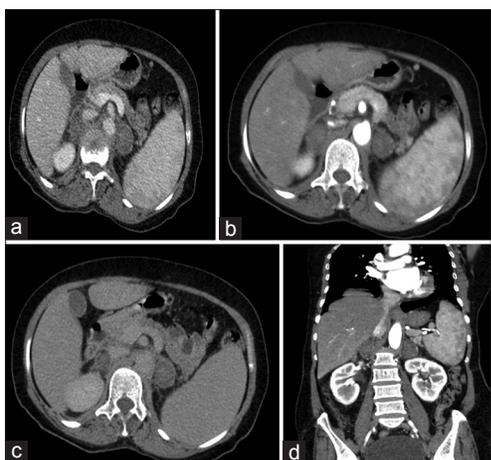


Figure 1: (a-d) CT abdomen showing bilateral adrenal haemorrhage

in ICU. APLA workup was done and was found to be negative. ANA/double-stranded DNA was negative. The patient was treated with injection meropenem 1 g IV BD, injection hydrocortisone, and other supportive measures in ICU. The patient improved symptomatically and was shifted back toward. The patient was discharged with T. Hisone 10 mg 1-0-1/2 and was advised to follow-up.

DISCUSSION

Bilateral adrenal hemorrhage can be caused by a number of conditions. In our patient, the underlying cause was urosepsis. The underlying mechanism in a non-traumatic adrenal hemorrhage is unclear. Adrenal gland has a poor venous drainage, but the arterial supply is rich. During stress, there is increased ACTH secretion which causes increases arterial blood flow, exceeding the venous drainage capacity causing adrenal hemorrhage. Adrenal vein thrombosis is another proposed mechanism, which occurs in association with primary APLA, sepsis, and heparin-induced thrombocytopenia. For the hormone deficiency to be clinically evident, 90% of the adrenal tissue must be destroyed. Hence, minor adrenal hemorrhage may go unnoticed.

CT abdomen is the investigation of choice in diagnosing adrenal hemorrhage. Hyponatremia, hyperkalemia, and hypoglycemia are presented in most cases; however, their absence does not exclude the diagnosis. High ACTH and low cortisol are diagnostic of primary adrenal insufficiency.

Acute adrenal insufficiency is a medical emergency. Supportive care is essential which includes fluid and electrolyte correction, monitors and stabilizes blood pressure and blood transfusion in case of severe hemorrhage.

If acute adrenal insufficiency is clinically suspected, hydrocortisone should be given without delay (100 mg bolus injection, followed by 200 mg per 24 h either as a continuous infusion or 50 mg every 6 h) along with intravenous fluid resuscitation, after withdrawing samples for cortisol assay, without waiting for the results. Treating the underlying cause is essential to the management of adrenal haemorrhage

CONCLUSION

Adrenal hemorrhage is a serious condition, which can present with non-specific signs and symptoms; hence, prompt diagnosis and treatment is required. It could be due to a number of causes. In our patient, the underlying etiology was urosepsis.

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