

Case Report

Perioperative adrenal crisis unmasking previously undiagnosed secondary adrenal insufficiency in a young woman with acute appendicitis: a case report

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ABSTRACT

Secondary adrenal insufficiency often presents with vague, nonspecific symptoms and may remain undiagnosed until physiological stress precipitates adrenal crisis. A 19-year-old female presented with right lower quadrant abdominal pain, nausea, and light-headedness. Imaging findings were suspicious for acute appendicitis, and laparoscopic appendectomy was performed. Further history revealed a three-year history of intermittent dizziness, headaches, poor appetite, and generalized weakness that had never been evaluated. On admission, she had marked bilateral lower limb weakness despite unremarkable magnetic resonance imaging of the brain and spine. Laboratory evaluation demonstrated a morning cortisol level of 1.12 µg/dl, and subsequent testing revealed suppressed adrenocorticotrophic hormone levels, consistent with secondary adrenal insufficiency. Following surgery, the patient developed hypotension, tachycardia, and tachypnoea despite initial management, raising concern for perioperative adrenal crisis. She responded to intravenous fluids and stress-dose hydrocortisone with rapid haemodynamic stabilization and gradual improvement in symptoms and mobility. This case highlights the diagnostic challenge of secondary adrenal insufficiency in young patients with longstanding nonspecific symptoms, particularly in the absence of characteristic electrolyte abnormalities. It also emphasizes that acute illness and surgical stress may unmask previously unrecognized adrenal insufficiency and underscores the importance of prompt recognition and glucocorticoid therapy to prevent potentially life-threatening complications.

Keywords: Secondary adrenal insufficiency, Adrenal crisis, Acute appendicitis, Perioperative complications, Glucocorticoid therapy

INTRODUCTION

Secondary adrenal insufficiency results from inadequate adrenocorticotrophic hormone (ACTH) secretion, leading to deficient cortisol production. Unlike primary adrenal insufficiency, mineralocorticoid secretion is generally preserved because aldosterone production is primarily regulated by the renin-angiotensin-aldosterone system. Consequently, patients may have normal serum sodium and potassium levels and lack the classical biochemical

abnormalities associated with primary adrenal insufficiency, making diagnosis challenging.^{1,2}

Clinical manifestations are often nonspecific and include fatigue, dizziness, anorexia, nausea, weakness, weight loss, and abdominal symptoms. These features may be attributed to more common conditions, resulting in delayed diagnosis.^{1,2} In patients suspected of having central adrenal insufficiency, an 8-9 am serum cortisol concentration is an important initial investigation. A

cortisol concentration below 3 µg/dl is considered indicative of adrenal insufficiency, whereas intermediate values generally warrant dynamic testing.³

Adrenal crisis is a life-threatening emergency that may occur in patients with established adrenal insufficiency or may be the initial manifestation of previously undiagnosed disease. Physiological stress, including infection, trauma, and surgery, can precipitate crisis when an adequate cortisol response cannot be mounted.^{4,5} Prompt administration of parenteral glucocorticoids and intravenous fluids is essential, and treatment should not be delayed while awaiting diagnostic confirmation.^{4,6}

We report a young woman with chronic unexplained dizziness and nonspecific systemic symptoms who presented with suspected acute appendicitis. Her postoperative deterioration following laparoscopic appendectomy led to the diagnosis of previously undiagnosed secondary adrenal insufficiency and suspected perioperative adrenal crisis.

CASE REPORT

A 19-year-old woman with no significant past medical history presented to the emergency department with acute right-sided abdominal pain associated with nausea and light-headedness. The symptoms began while she was lying in bed. Further history revealed a 3-year history of intermittent dizziness and headaches, particularly during menstruation, which had never previously been evaluated. During the preceding 2 weeks, she had developed persistent dizziness, nausea, anorexia, generalized weakness, intermittent blurred vision, and constipation. She denied a positional component to the dizziness, syncope, or loss of consciousness. She denied tobacco, alcohol, or illicit drug use. Her last menstrual period was 20 days before presentation.

On examination, she was hemodynamically stable. Abdominal examination revealed right lower quadrant tenderness with a positive Rovsing sign but no guarding or rigidity. Murphy sign was negative. Neurological examination demonstrated marked bilateral lower-extremity weakness, with motor strength of 1/5 bilaterally. Sensation and reflexes were intact, rectal tone was preserved, and there was no perineal numbness.

Laboratory investigations showed a normal complete blood count, including a white blood cell count of 7000/mcl, hemoglobin of 12.9 g/dl, and platelet count of 299,000/mcl. Serum sodium, potassium, calcium, magnesium, and phosphate were normal. Thyroid function tests, HbA1c, vitamin B12, folate, C-reactive protein, lactate dehydrogenase, lactate, and troponin were also normal. Creatine kinase was 54 U/l, while aspartate

aminotransferase was mildly elevated at 37 U/l. Urinalysis showed trace ketones, and a urine pregnancy test was negative. The modified Alvarado score was 4.

Computed tomography of the abdomen and pelvis demonstrated a non-visualized appendix with mild periceal inflammatory changes extending toward the right adnexa, suggestive of acute appendicitis versus a ruptured ovarian cyst. Pelvic ultrasonography demonstrated a normal uterus and left ovary, a complex right ovarian corpus luteum cyst, bilateral adnexal free fluid, and right lower quadrant inflammatory stranding with a lobulated hypoechoic structure adjacent to the right ovary. Appendicitis could not be excluded.

She underwent laparoscopic appendectomy for suspected acute appendicitis. Postoperatively, she developed hypotension, tachycardia, and tachypnea, raising concern for perioperative adrenal crisis. She received a 1 litre normal saline bolus followed by maintenance intravenous fluids, hydrocortisone 50 mg every 6 hours, and oxygen at 2 l/min via nasal cannula. Endocrinology was consulted, and she was subsequently transitioned to oral hydrocortisone 30 mg in the morning and 15 mg in the afternoon. Persistent instability occurred on postoperative day 1, prompting further endocrine evaluation.

A morning serum cortisol concentration was markedly low at 1.12 µg/dl. ACTH concentrations on two consecutive days were 4 pg/ml and <1.5 pg/ml, respectively. The combination of profound cortisol deficiency and inappropriately low ACTH supported a diagnosis of central adrenal insufficiency.³ Her normal serum potassium and absence of significant electrolyte abnormalities were also compatible with secondary rather than primary adrenal insufficiency.

Magnetic resonance imaging of the brain and whole spine showed no abnormal enhancement or significant structural abnormality. No clear underlying cause of the secondary adrenal insufficiency was identified during the admission. There was no reported history of chronic glucocorticoid therapy or other significant medical illness.

Following continued glucocorticoid replacement, her vital signs stabilized, tachycardia and tachypnea resolved, and supplemental oxygen was discontinued. Hydrocortisone was tapered to an oral maintenance regimen of 20 mg in the morning and 10 mg in the afternoon. Her lower-extremity strength and functional status improved with physiotherapy, and she regained the ability to ambulate. She was discharged on maintenance hydrocortisone with outpatient endocrinology follow-up.

The timeline of the patient's clinical course is shown in Figure 1.

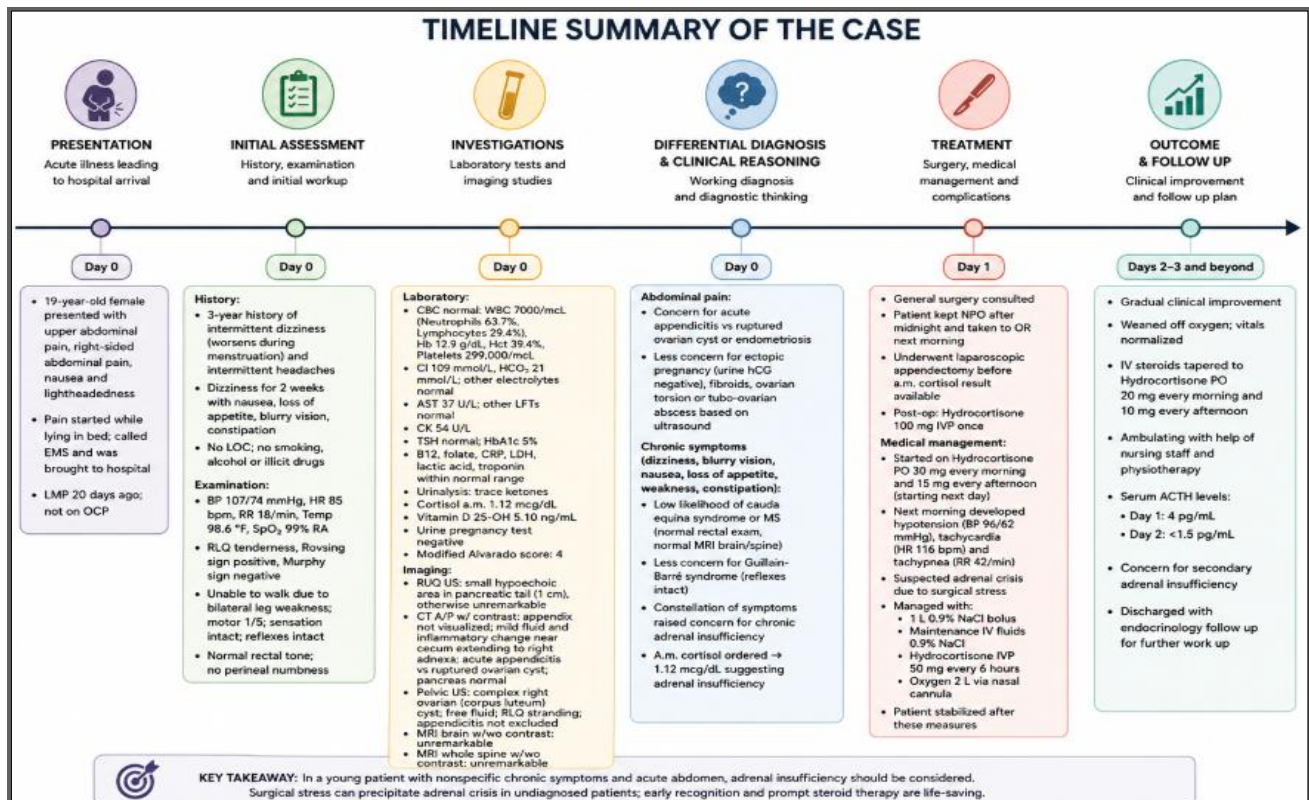


Figure 1: Clinical timeline of presentation, investigation, management, and outcome in a case of perioperative adrenal crisis unmasking secondary adrenal insufficiency.

DISCUSSION

Secondary adrenal insufficiency results from impaired ACTH secretion and subsequent cortisol deficiency. Because aldosterone secretion is generally maintained, patients with central adrenal insufficiency may not develop hyperkalemia or other classical biochemical features of primary adrenal insufficiency.^{1,2} This distinction is clinically important because normal electrolytes may not exclude clinically significant cortisol deficiency.

Our patient had several years of intermittent dizziness and headaches followed by 2 weeks of persistent dizziness, nausea, anorexia, weakness, and constipation. These symptoms are nonspecific and may overlap with gastrointestinal, neurological, gynecological, and functional disorders. A high index of suspicion is therefore required when symptoms are persistent and unexplained.^{1,2,10}

The biochemical findings strongly supported central adrenal insufficiency. The morning cortisol concentration of 1.12 µg/dl was substantially below the threshold considered indicative of adrenal insufficiency in the Endocrine Society guideline for central adrenal insufficiency.³ The ACTH concentrations of 4 pg/ml and <1.5 pg/ml were inappropriately low in the setting of profound cortisol deficiency, supporting impaired

hypothalamic-pituitary stimulation rather than primary adrenal gland failure.³

A corticotropin stimulation test is generally recommended when morning cortisol concentrations are in an intermediate range.³ In this patient, the cortisol concentration was markedly low and she developed acute postoperative instability requiring urgent glucocorticoid therapy. In suspected adrenal crisis, treatment should not be delayed for confirmatory testing.^{4,6} The normal electrolyte profile is an important feature of this case. In primary adrenal insufficiency, aldosterone deficiency may result in hyponatremia and hyperkalemia. In secondary adrenal insufficiency, mineralocorticoid secretion is generally preserved, allowing patients to present with relatively normal electrolyte concentrations.^{1,2}

The patient's postoperative deterioration is particularly relevant. Surgical stress normally activates the hypothalamic-pituitary-adrenal axis and increases cortisol production. Patients with adrenal insufficiency cannot mount this response adequately, making surgery a potential precipitant of adrenal crisis.^{4,5,9} Perioperative guidelines emphasize the importance of recognizing adrenal insufficiency and providing appropriate glucocorticoid supplementation during surgical stress.⁵

The development of hypotension, tachycardia, and tachypnea after surgery, together with markedly low cortisol and inappropriately low ACTH, was considered

consistent with perioperative adrenal crisis. Although postoperative instability has several possible causes, including hypovolemia, bleeding, infection, anesthetic effects, and cardiopulmonary complications, the biochemical findings and subsequent improvement with glucocorticoid replacement supported adrenal insufficiency as a major contributor.

Emergency treatment of adrenal crisis consists of parenteral hydrocortisone and intravenous fluid replacement.^{4,6} Recommended adult treatment includes an initial parenteral hydrocortisone dose followed by approximately 200 mg over 24 hours, administered by continuous infusion or divided doses.⁵ In this case, hydrocortisone 50 mg every 6 hours was administered together with intravenous saline, followed by transition to oral replacement after clinical stabilization.

The cause of the patient's secondary adrenal insufficiency remained uncertain. Potential causes of central adrenal insufficiency include pituitary or hypothalamic disease, traumatic brain injury, infiltrative disorders, pituitary surgery or irradiation, and suppression of the hypothalamic-pituitary-adrenal axis by exogenous glucocorticoids.^{2,3,7} The patient had no reported history of chronic glucocorticoid use, and brain and spine imaging did not reveal a structural abnormality. Further outpatient endocrine assessment, including evaluation of other pituitary hormone axes when clinically indicated, would therefore be appropriate.

The marked bilateral lower-extremity weakness was an unusual feature. Sensation and reflexes were preserved, creatine kinase was normal, and neuroimaging did not identify a structural cause. Her functional status improved following glucocorticoid replacement and physiotherapy. Although a direct causal relationship between cortisol deficiency and the neurological findings cannot be established, severe glucocorticoid deficiency can contribute to generalized weakness and impaired physical function.^{2,10}

This case highlights the importance of considering adrenal insufficiency in patients with chronic unexplained symptoms, particularly before or during major physiological stress. Early recognition is important because adrenal crisis can be fatal if treatment is delayed.^{4,9} Patient education after diagnosis is also essential, including instructions regarding stress-dose glucocorticoids and emergency treatment during significant illness or physiological stress.^{4,8}

CONCLUSION

Secondary adrenal insufficiency should be considered in young patients with chronic unexplained dizziness, anorexia, nausea, fatigue, and generalized weakness, even

when routine laboratory investigations and serum electrolytes are normal. Profoundly low morning cortisol with inappropriately low ACTH supports central adrenal insufficiency. Surgical stress may unmask previously compensated disease and precipitate adrenal crisis. Prompt glucocorticoid replacement and fluid resuscitation are essential when adrenal crisis is suspected, and treatment should not be delayed while awaiting diagnostic confirmation.

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