Case Report

CME: This article is offered for AMA PRA Category 1 Credit.™
See CME Quiz Questions on page 9.

Recognizing and Managing Adrenal Crisis in Urgent Care: A Case Report

Urgent Message: Adrenal crisis is uncommon in the urgent care setting, but it can be rapidly fatal if diagnosis is missed. Early administration of hydrocortisone and fluid resuscitation with correction of electrolyte abnormalities are essential to reduce mortality.

Clayton R. Halford, PA-C

Citation: Halford C. Recognizing and Managing Adrenal Crisis in Urgent Care: A Case Report. *J Urgent Care Med*. 2026;20(10):19-22

Keywords: adrenal crisis; urgent care; adrenal insufficiency; Addison disease; pediatric endocrine emergency; hyponatremia; hyperkalemia; hypoglycemia

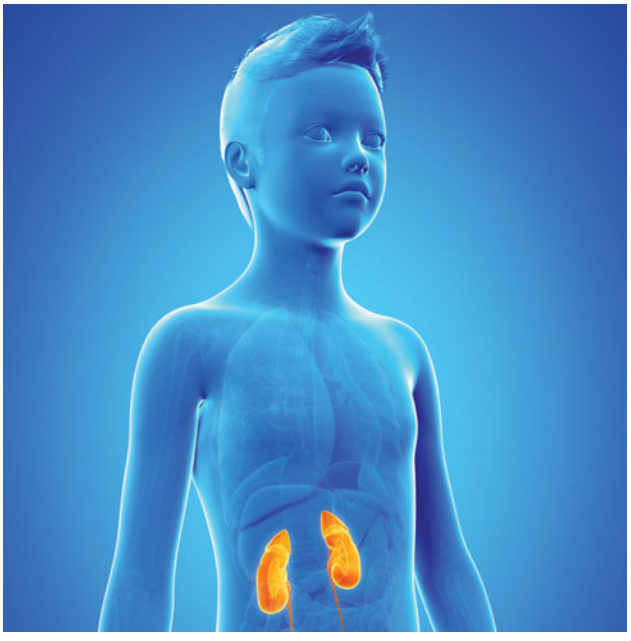
Abstract

Introduction: Adrenal (Addisonian) crisis is a rare but life-threatening endocrine emergency that can present to urgent care with nonspecific symptoms. Prompt recognition and early intervention with hydrocortisone, isotonic fluids, and correction of electrolyte abnormalities are critical to reducing morbidity and mortality.

Presentation: An 11-year-old previously healthy boy presented to urgent care with 24 hours of intractable vomiting, abdominal pain, fatigue, and dizziness, following 5 days of cold-like symptoms including cough, rhinorrhea, chills, and subjective fever. His parents had given him over-the-counter acetaminophen and ibuprofen with minimal relief.

Physical Examination: On examination, the patient appeared acutely ill and somnolent with intermittent lethargy. He was febrile, mildly tachycardic, and had dry mucous membranes. The abdomen was diffusely tender without peritoneal signs. Point-of-care glucose testing showed severe hypoglycemia. Intravenous access was established. Laboratory studies demonstrated hyponatremia, hyperkalemia, and evidence of dehydration. Because of the hyperkalemia, a 12-lead electrocardiogram was performed and demonstrated tall tented/peaked T waves most prominent in the precordial leads. Emergency medical services was activated for transfer to a higher level of care.

Diagnosis and Resolution: In the emergency department, the patient received stress dosing of hydrocortisone based on the suspected diagnosis of adrenal crisis.



Author Affiliation: Clayton Halford, PA-C, Intermountain Health Urgent Care/Instacare, Utah North Region; Department of Physician Assistant Medicine, Weber State University. The author has no relevant financial relationships with any ineligible companies.

He was also treated for hypoglycemia, electrolyte abnormalities, and dehydration. Further diagnostic evaluation confirmed an adrenal crisis precipitated by an influenza A infection. Treatment included oseltamivir, hydrocortisone, and fludrocortisone. Upon hospital discharge, he was prescribed daily glucocorticoid and mineralocorticoid therapy.

Conclusion: Urgent care clinicians should maintain a high index of suspicion for adrenal crisis in patients presenting with unexplained shock or with the characteristic triad of hyponatremia, hyperkalemia, and hypoglycemia. Prompt administration of corticosteroids, aggressive isotonic fluid resuscitation, and rapid transfer to a facility with intensive care capabilities are essential to reduce morbidity and mortality.

Introduction

Adrenal (Addisonian) crisis is a rare but life-threatening endocrine emergency that may present to urgent care or emergency settings with nonspecific symptoms, making early recognition particularly challenging.[1,2]

It results from an acute and severe deficiency of adrenal hormones—primarily cortisol and, in many cases, aldosterone—due to adrenal cortex failure.[3] Clinical manifestations often include profound fatigue, gastrointestinal distress, abdominal pain, altered mental status, and characteristic biochemical abnormalities such as hyponatremia, hyperkalemia, and hypoglycemia.[2,4]

Common precipitating factors include infection, surgery, trauma, abrupt withdrawal or reduction of chronic glucocorticoid therapy, and other acute physiological stressors.[2,5] Prompt recognition and immediate treatment with high-dose intravenous hydrocortisone and aggressive isotonic fluid resuscitation are critical to reducing morbidity and mortality.[2,3,5]

Case Presentation

A mother brought her 11-year-old, previously healthy boy to the urgent care for 24 hours of persistent, nonbloody, nonbilious vomiting. He reported associated diffuse abdominal pain, described as crampy and moderate in intensity, without radiation, and significant fatigue and dizziness, particularly when standing. Five days prior to presentation, he developed upper respiratory symptoms, including a nonproductive cough, rhinorrhea, chills, and subjective fever. His parents administered over-the-counter acetaminophen and ibuprofen with minimal improvement. He had not had any diarrhea, hematemesis, melena, or hematuria. He had no recent travel or sick contacts, and no history of similar episodes in the past. His past medical history and social history were unremarkable.

Physical Exam

On presentation to urgent care, the patient's vital signs were as follows: temperature of 37.8°C (100.0°F); heart rate of 122 beats per minute (BPM); blood pressure of 92/58 mmHg; respiratory rate of 22 breaths per minute; and oxygen saturation of 97% on room air.

- **General:** Ill and fatigued, lying quietly on the examination table with intermittent episodes of lethargy
- **Oral:** Dry oral mucous membranes
- **Lungs:** Shallow breathing, otherwise clear to auscultation bilaterally
- **Cardiovascular:** Tachycardic with regular rhythm
- **Abdominal:** Mild, diffuse tenderness without rebound, guarding, or peritoneal signs

Urgent Care Management

Based on the clinical presentation and vital signs, the triage nurse appropriately classified the case as urgent, prompting an immediate evaluation by a provider. An intravenous (IV) line was established, and point-of-care laboratory testing—including a complete blood count and a basic metabolic panel—was obtained **(Table 1).** A bedside fingerstick glucose measurement revealed hypoglycemia with a glucose level of 51 mg/dL. The patient appeared markedly somnolent, and 1 mg of intravenous glucagon was administered. A 500-mL bolus of normal saline was initiated. The hyperkalemia prompted a 12-lead electrocardiogram and cardiac monitoring, which revealed a narrow-complex tachycardia at 130 BPM and peaked T waves in leads V2–V4.

Differential Diagnoses and Medical Decision Making

A broad differential diagnosis included acute viral gastroenteritis, dehydration, sepsis, appendicitis, and adrenal insufficiency. Given the urgent care findings and clinical concern for potential adrenal crisis, emergency medical services was activated for immediate transfer to a hospital with pediatric intensive care (PICU) capabilities.

Emergency Department Course and Disposition

Upon arrival at the emergency department (ED), administration of intravenous hydrocortisone was provided based on the high suspicion for adrenal crisis. The patient was admitted to the PICU. Virology studies confirmed the presence of influenza A, the cause of his viral prodromal symptoms. This helped clarify the un-

Table 1. Laboratory Findings on Presentation

Test	Result	Normal Range (Age 11, Male)	Interpretation
Sodium (Na^+)	125 mmol/L	135–145 mmol/L	Low (Hyponatremia)
Potassium (K^+)	6.3 mmol/L	3.4–4.7 mmol/L	High (Hyperkalemia)
Bicarbonate (HCO_3^-)	15 mmol/L	22–28 mmol/L	Low (Metabolic acidosis likely)
BUN (Blood Urea Nitrogen)	28 mg/dL	5–18 mg/dL	High (Azotemia)
Creatinine	1.1 mg/dL	0.3–0.7 mg/dL	High for age (Possible renal impairment or dehydration)

derlying etiology of his adrenal crisis. An adrenocorticotropic hormone (ACTH) stimulation test was performed, which confirmed the diagnosis of primary adrenal insufficiency (Addison disease) with acute adrenal crisis. The patient responded appropriately to therapy and was discharged home 4 days later with maintenance hydrocortisone and fludrocortisone.

Epidemiology

Because adrenal crisis may present to urgent care with nonspecific symptoms, early recognition can be challenging.[6] For every 100 children with known adrenal insufficiency followed over 1 year, approximately 2.7–4.3 adrenal crisis events occur.[7]

Cortisol, Aldosterone, and Pathophysiology

Addisonian adrenal crisis results from an acute and severe deficiency of adrenal hormones, primarily cortisol and often aldosterone, due to failure of the adrenal cortex.[3] The adrenal glands, located atop each kidney, consist of the cortex and medulla. The cortex produces 3 major classes of steroid hormones: glucocorticoids (cortisol); mineralocorticoids (aldosterone); and adrenal androgens (dehydroepiandrosterone [DHEA]).[4]

Cortisol is essential for maintaining vascular tone, blood pressure, and glucose homeostasis. It supports the body's response to stress by increasing gluconeogenesis, mobilizing energy stores, and enhancing catecholamine-mediated vasoconstriction.[4] Aldosterone regulates sodium and potassium balance, thereby controlling extracellular fluid volume and blood pressure by promoting renal sodium retention and potassium excretion. DHEA is a steroid hormone that functions primarily as an inactive precursor that is converted into more potent sex steroids—testosterone and estradiol.[8]

In Addisonian crisis, the deficiency of cortisol and aldosterone leads to impaired stress response, hypotension, hypoglycemia, and electrolyte abnormalities, particularly hyponatremia and hyperkalemia.[4] These pathophysiologic disturbances correlate directly with this patient's presentation of hypoglycemia and electrolyte abnormalities, as well as his mild hypotension and fatigue.[4]

Crisis typically occurs during periods of physiological stress, such as acute illness, infection, trauma, or surgery, when the body's demand for cortisol increases but cannot be met due to adrenal failure. Without prompt treatment, this may progress to shock and multiorgan dysfunction.[8,9,10]

Pathophysiology of Adrenal Insufficiency

The hypothalamic-pituitary-adrenal (HPA) axis regulates cortisol production. The hypothalamus secretes corticotropin-releasing hormone (CRH), stimulating the pituitary gland to release ACTH, which in turn stimulates the adrenal cortex to produce cortisol. Disruption at any point—hypothalamus, pituitary, or adrenal gland—can lead to adrenal insufficiency.[3,11,12,13]

- Primary adrenal insufficiency (Addison disease) results from direct adrenal gland dysfunction, most commonly from autoimmune destruction, but may also result from infections such as tuberculosis. This leads to deficiencies in cortisol, aldosterone, and adrenal androgens.[3,11,12,13]
- Secondary adrenal insufficiency results from pituitary failure to produce ACTH, often due to pituitary tumors, surgery, radiation, hemorrhage, or infiltrative diseases. This leads to cortisol deficiency. Aldosterone production remains largely preserved because it is primarily regulated by the renin–angiotensin system.[3,11,12,13]
- Tertiary adrenal insufficiency (often glucocorticoid-induced) results from hypothalamic suppression of CRH, usually due to chronic exogenous glucocorticoid therapy or abrupt steroid withdrawal.[3,11,12,13]

In primary disease, loss of cortisol-mediated feedback results in elevated ACTH levels, whereas in secondary and tertiary disease, ACTH levels are low or inappropriately normal.[3,11,14,15,16] In this patient's scenario, diagnostic testing with an ACTH stimulation test was necessary to identify and confirm primary adrenal insufficiency leading to adrenal crisis.[13]

Clinical Presentation and Management

Clinical Presentation

The initial clinical presentation is often vague, with symptoms such as fatigue (67%), hyperpigmentation (50.4%), dehydration (33%), and hypotension (31%). Laboratory findings may include hyponatremia (55%), hyperkalemia (32.7%), and hypoglycemia (33.7%).[1,4]

In this patient, the symptoms of fever, hypotension, tachycardia, dehydration, and the characteristic electrolyte triad (hyponatremia + hyperkalemia + hypoglycemia) raised a strong suspicion for adrenal crisis secondary to previously unrecognized primary adrenal insufficiency (Addison disease). His history revealed a preceding 5-day viral illness, later confirmed to be influenza type A. This infection significantly increased his physiologic cortisol requirement, but due to underlying adrenal insufficiency, his adrenal glands were unable to mount an adequate stress response.

The Endocrine Society guideline and large pediatric cohort studies support that the above clinical and laboratory findings are typical at presentation and confirm that infections or acute illnesses are common precipitants of adrenal crisis in children with undiagnosed or untreated adrenal insufficiency.[2]

Management

Immediate administration of hydrocortisone should not be delayed in suspected adrenal crisis—even in outpatient or urgent care settings—as rapid treatment significantly reduces mortality. Guidelines emphasize initiation before laboratory confirmation because delays can lead to irreversible shock and death. Hydrocortisone (synthetic cortisol) is preferred over dexamethasone or other glucocorticoids because, at stress doses, it provides both glucocorticoid and mineralocorticoid activity, rapidly correcting hypotension, hyponatremia, and hyperkalemia. Its shorter half-life also allows easier titration as the patient stabilizes. If hydrocortisone is unavailable, dexamethasone may be used temporarily; however, its lack of mineralocorticoid activity necessitates concurrent mineralocorticoid replacement.[2,4,10]

Ethics Statement

The patient was unable to be contacted as contact information on record was no longer active. Therefore, patient demographics and nonessential details have been altered to protect anonymity.

Takeaway Points

- Adrenal (Addisonian) crisis is a rare but life-threatening endocrine emergency that may present with nonspecific symptoms. Prompt recognition and early intervention are critical to reducing mortality.
- Clinicians should consider adrenal crisis early in the differential diagnosis. Any ill patient with vomiting plus hypotension and/or altered mentation, along with the triad of hyponatremia, hyperkalemia, and hypoglycemia, warrants immediate empiric treatment for adrenal crisis.
- Increased physiologic stress—including acute illness, trauma, infection, or surgery—can unmask previously undiagnosed adrenal insufficiency. ■

Manuscript submitted November 13, 2025; accepted April 29, 2026.

References

1. Capalbo D, Moracas C, Cappa M, et al. Primary Adrenal Insufficiency in Childhood: Data From a Large Nationwide Cohort. *J Clin Endocrinol Metab*. 2021;106(3):762-773. doi:10.1210/clinem/dgaa881
2. Bornstein SR, Allolio B, Arlt W, et al. Diagnosis and Treatment of Primary Adrenal Insufficiency: An Endocrine Society Clinical Practice Guideline. *J Clin Endocrinol Metab*. 2016;101(2):364-389. doi:10.1210/jc.2015-1710
3. Vaidya A, Findling J, Bancos I. Adrenal Insufficiency in Adults: A Review. *JAMA*. 2025;334(8):714–725. doi:10.1001/jama.2025.5485
4. Rushworth RL, Torpy DJ, Falhammar H. Adrenal crisis. *N Engl J Med*. 2019;381(9):852-861. doi:10.1056/nejmra1807486
5. Martel-Duguech L, Poirier J, Bourdeau I, Lacroix A. Diagnosis and management of secondary adrenal crisis. *Rev Endocr Metab Disord*. 2024;25(3):619-637. doi:10.1007/s11154-024-09877-x
6. Dong J, Hahner S, Bancos I, Tomlinson JW. Clinical features, investigation, and management of Addison's disease. *Lancet Diabetes Endocrinol*. 2026;14(3):243-258. doi:10.1016/S2213-8587(25)00393-6
7. Hosokawa M, Ichihashi Y, Sato Y, et al. Incidence and Risk Factors for Adrenal Crisis in Pediatric-onset Adrenal Insufficiency: A Prospective Study. *J Clin Endocrinol Metab*. 2024;109(8):e1602-e1607. doi:10.1210/clinem/dgad753
8. Ganguly A. Primary aldosteronism. *N Engl J Med*. 1998;339(25):1828-1834. doi:10.1056/nejm199812173392507
9. Russell G, Lightman S. The human stress response. *Nat Rev Endocrinol*. 2019;15(9):525–534. doi:10.1038/s41574-019-0228-0
10. Beuschlein F, Else T, Bancos I, et al. European Society of Endocrinology and Endocrine Society Joint Clinical Guideline: Diagnosis and Therapy of Glucocorticoid-induced Adrenal Insufficiency. *J Clin Endocrinol Metab*. 2024;109(7):1657-1683. doi:10.1210/clinem/dgae250
11. Husebye ES, Pearce SH, Krone NP, Kämpe O. Adrenal insufficiency. *Lancet*. 2021;397(10274):613-629. doi:10.1016/S0140-6736(21)00136-7
12. Salvatori R. Adrenal Insufficiency. *JAMA*. 2005;294(19):2481–2488. doi:10.1001/jama.294.19.2481
13. Bancos I, Hahner S, Tomlinson J, Arlt W. Diagnosis and management of adrenal insufficiency. *Lancet Diabetes Endocrinol*. 2015;3(3):216-226. doi:10.1016/S2213-8587(14)70142-1
14. Hahner S, Ross RJ, Arlt W, et al. Adrenal insufficiency. *Nat Rev Dis Primers*. 2021;7(1):19. Published 2021 Mar 11. doi:10.1038/s41572-021-00252-7
15. Martin-Grace J, Dineen R, Sherlock M, Thompson CJ. Adrenal insufficiency: Physiology, clinical presentation and diagnostic challenges. *Clin Chim Acta*. 2020;505:78-91. doi:10.1016/j.cca.2020.01.029
16. Lewis A, Thant AA, Aslam A, Aung PPM, Azmi S. Diagnosis and management of adrenal insufficiency. *Clin Med (Lond)*. 2023;23(2):115-118. doi:10.7861/clinmed.2023-0067