Clinical Critical Care

E-ISSN 2774-0048

VOLUME 33 NUMBER 1

JANUARY-DECEMBER 2025

Clinical Critical Care
eISSN 2774-0048

Clinical Critical Care
Volume 33, Article ID e250018, 5 pages
https://doi.org/10.54205/ccc.v33.275343

CASE REPORT

Overcoming combined shock: Hydrocortisone's role in adrenal insufficiency after unilateral adrenal gland injury - case report

Chompoonut Achavanuntakul, Karikarn Auksornchat

Department of Surgery, Thammasat University Hospital and Faculty of Medicine, Thammasat University, Pathum Thani, Thailand, 12120

OPEN ACCESS
Citation:
Achavanuntakul C, Auksornchat K. Overcoming combined shock: Hydrocortisone's role in adrenal insufficiency after unilateral adrenal gland injury - case report. Clin Crit Care 2025; 33: e250018.

Received: May 8, 2025
Revised: July 7, 2025
Accepted: July 22, 2025

Data Availability Statement:
All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author. (Chompoonut Achavanuntakul, email address: nut_jiew@hotmail.com)

Funding:
No funding

Competing interests:
No potential conflict of interest relevant to this article was reported.

Corresponding author:
Chompoonut Achavanuntakul
Department of Surgery, Thammasat University Hospital and Faculty of Medicine, Thammasat University, Pathum Thani, Thailand, 12120
Tel: (+66) 2-926-9523
Fax: (+66) 2-926-9530
E-mail: nut_jiew@hotmail.com

ABSTRACT:

Introduction: Adrenal gland injury is a rare traumatic condition. Adrenal insufficiency following adrenal gland injury is even more uncommon and often overlooked, particularly in the presence of hemorrhagic shock, which is the most frequent cause of shock in trauma patients. This misdiagnosis can lead to excessive fluid resuscitation and subsequent complications.

Case presentation: A 36-year-old male sustained blunt thoracoabdominal trauma following a motor vehicle accident. Abdominal computed tomography revealed a grade II pancreatic injury, a grade II splenic injury, a grade IV kidney injury, and a left adrenal gland hematoma. During admission, the patient presented with abdominal pain and generalized guarding. An exploratory laparotomy with splenic vein repair was performed. The patient developed hypotension, and fluid resuscitation was initiated along with the administration of noradrenaline. A cortisol level was measured at 7.86 µg/dL. Therefore, hydrocortisone was infused, resulting in a gradual reduction of vasopressor support and stabilization of the patient's hemodynamic status.

Conclusions: Severe trauma can result in combined shock. In cases of unilateral adrenal gland injury leading to low cortisol levels, prompt administration of hydrocortisone assists in improving the patient's condition, as demonstrated in this case.

Keywords: Adrenal gland; Combined shock; Hydrocortisone; Injury; Trauma

INTRODUCTION

Adrenal gland injury following trauma is rare, occurring in approximately 0.03% to 4.95% of trauma cases[1-5]. It is typically caused by blunt trauma resulting from high-impact forces such as motor vehicle collisions and pedestrian accidents[3,6,7]. While adrenal insufficiency can occasionally result from such injuries, it remains extremely rare and is generally life-threatening[5,8].

Most available data come from case reports. For instance, Naomi Szwarcbard et al.[9] reported three cases of bilateral adrenal gland hemorrhage following motor vehicle accidents, all of which resulted in fatal adrenal insufficiency. Other reports by N. Jimidar et al.[10] and Kahdi F. Udobi et al.[11] describe patients who developed bilateral adrenal hemorrhage after high-velocity motorcycle accidents, leading to adrenal insufficiency. All of these patients were successfully treated with cortisol replacement. Some case reports have described unilateral adrenal gland injury in patients without subsequent adrenal insufficiency.

Adrenal trauma is often accompanied by other organ injuries, including rib fractures, pneumothorax, kidney injury, splenic injury, and liver lacerations[1,4,12]. Some reports also describe concomitant head, chest, and pelvic injuries[6,13]. The mortality associated with adrenal gland injury remains unclear, with some studies indicating an increased rate[3,5,9], while others do not[2,12,13].

To date, there have been no reports of adrenal insufficiency following unilateral adrenal injury, which makes our patient's case particularly unique and of significant interest.

Patient consent was obtained, and the study was approved by the human research ethics committee of Thammasat University (medicine), project number MTU-EC-SU-0-269/67, prior to commencement.

KEY MESSAGES:

- In abdominal trauma, when hemorrhage alone does not fully explain the shock, consider the possibility of combined shock.
- This case underscores the importance of considering adrenal insufficiency in trauma patients, especially in the context of adrenal gland injury and shock.
- An administration of hydrocortisone led to hemodynamic stabilization and improved clinical outcomes in a patient with adrenal insufficiency.

CASE PRESENTATION

A 36-year-old male was brought to the emergency department following blunt thoracoabdominal injuries sustained in a motor vehicle collision with a truck approximately 70 minutes prior.

Upon initial assessment, the patient's vital signs were as follows: blood pressure 76/54 mmHg, heart rate 143 beats per minute, and respiratory rate 18 breaths per minute. The patient denied any significant medical history, current medications, or allergies.

Physical examination revealed blood and secretions in the patient's oral cavity. After suctioning the secretions, it became apparent that the airway was not patent. As a result, endotracheal intubation was performed. Examination of the trachea confirmed it was in a midline position; however, decreased breath sounds were noted bilaterally with subcutaneous emphysema on the right chest wall. Bilateral intercostal chest drains were inserted, and air was evacuated from both hemithoraces.

Massive transfusion protocol was activated, and the patient was provided with blood products, while investigations for the source of bleeding were initiated. No active external bleeding was seen. An extended focused assessment with sonography in trauma (eFAST) was done and revealed no evidence of intra-abdominal or intrapericardial hemorrhage; however, a loss of lung sliding sign was observed, suggestive of a bilateral pneumothorax.

After receiving 6 units of packed red blood cells and 4 units of fresh frozen plasma, the patient's vital signs improved, with blood pressure stabilizing at 111/68 mmHg and heart rate at 140 beats per minute. The patient was then sent for a comprehensive pan-computed tomography (CT) scan and was admitted to the intensive care unit (ICU).

CT imaging revealed no intracranial hemorrhage but showed multiple fractures, including skull and facial fractures, bilateral pulmonary contusions, pulmonary lacerations, bilateral pneumothorax, and fractures of the right 1st rib, right 7th rib, and left 6th-10th ribs. Additionally, the scan revealed a moderate amount of hemoperitoneum, retroperitoneal hemorrhage, mild diffuse jejunal bowel wall thickening, pancreatic injury (grade 2), splenic injury (grade 2), left kidney injury (grade 4), and diffuse enlargement and increased attenuation of the left adrenal gland, consistent with a left adrenal hematoma as shown in Fig 1.

The initial laboratory results demonstrated the following findings: hemoglobin (Hb) was 15.5 g/dL, hematocrit (Hct) 48.7%, and white blood cell (WBC) count 28,540/μL, with a differential showing 78% neutrophils, 17% lymphocytes, and 1% eosinophils. Renal function tests revealed a blood urea nitrogen (BUN) of 11 mg/dL and serum creatinine of 1.38 mg/dL. Electrolyte values were as follows: sodium 140 mmol/L, potassium 3.8 mmol/L, chloride 106 mmol/L, and bicarbonate 15 mmol/L. Calcium was measured at 8.9 mg/dL. Lactate levels were elevated at 12.7 mmol/L. The Dextrostix measured 32 mg% so a 50% glucose solution (50 mL) was administered as a bolus. Following the administration, the Dextrostix level was rechecked after 15 minutes, and it increased to 116 mg%.

During clinical observation, the patient developed abdominal pain and generalized guarding, so an exploratory laparotomy was performed. Approximately 300 mL of hemoperitoneum was noted, together with active oozing from the splenic vein and spleen parenchyma. No other active bleeding sources were identified. The splenic vein defect was repaired using a figure-of-eight suturing technique, and bleeding from the spleen was controlled with electrocautery and absorbable hemostatic agents. The estimated blood loss was 800 mL, and a total of 3,300 mL of isotonic fluids were administered intraoperatively. The patient developed hypotension, and fluid resuscitation was initiated. Noradrenaline was administered concurrently, with a maximum dose of 0.095 mcg/kg/min.

Due to concerns about adrenal insufficiency in the context of shock with adrenal gland injury, the patient's cortisol level was measured and found to be 7.86 μg/dL. A bolus of 100 mg hydrocortisone was administered intravenously, followed by a continuous infusion of 200 mg over the next 24 hours. This treatment led to a gradual reduction in the required noradrenaline dose, stabilization of the patient's hemodynamics, good urine output, and normalization of lactate levels. Hydrocortisone therapy was discontinued on day 6 of hospitalization. No further hemodynamic instability was noted.

On day 7, a 1 mcg ACTH stimulation test was performed, with cortisol levels measured before and at 20, 30, and 40 minutes after ACTH injection. The cortisol levels were 13.1, 13.8, 13.6, and 13.9 μg/dL, respectively. One

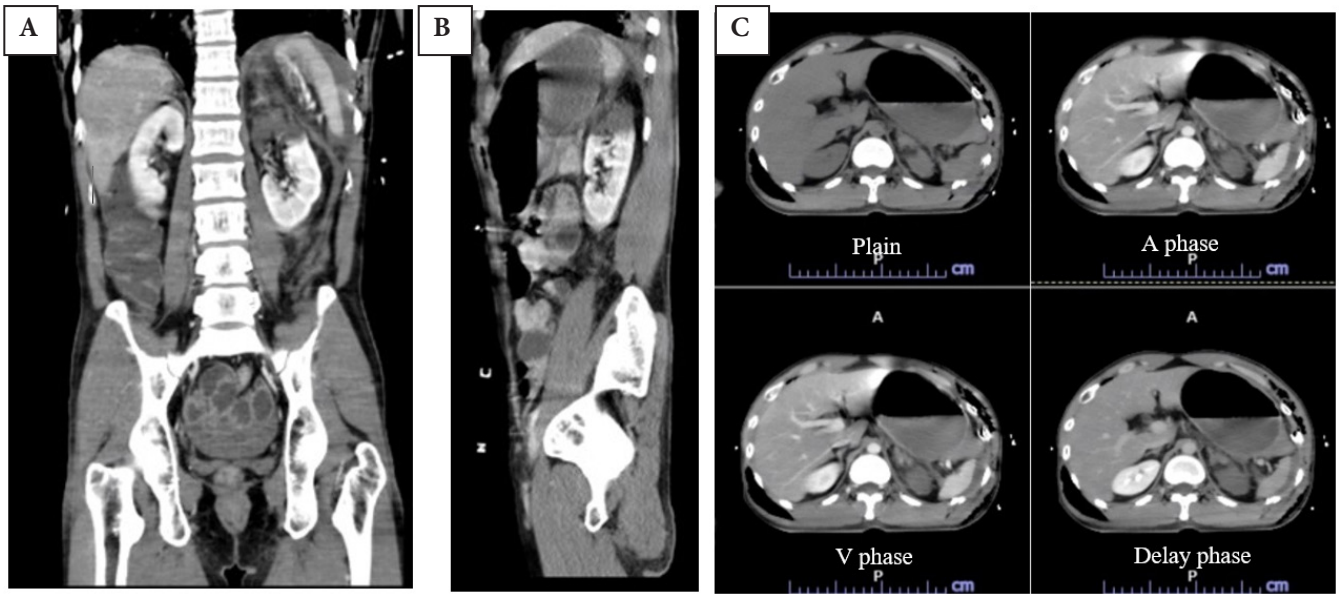

Figure 1. Initial abdominal computed tomography: diffuse enlarge and increased attenuation of left adrenal gland correlated with adrenal gland hematoma and left kidney segmental infarction. (A) Coronal view, V phase (B) Sagittal view, V phase (C) Axial view

day later, a 250 mcg ACTH stimulation test was performed, with cortisol levels measured before and at 30 and 60 minutes after ACTH injection. The results were 17.8, 23.2, and 21.8 μg/dL, respectively. These results demonstrated an appropriate increase in cortisol following ACTH stimulation, suggesting a relatively low cortisol level in acute stress but no evidence of long-term adrenal insufficiency.

During the administration, no hyponatremia, hypercalcemia, lymphocytosis, or eosinophilia was observed. In contrast, mild hyperkalemia was noted, with the maximum potassium level reaching 5.4 mmol/L on the first and second day of hospitalization. A single episode of hypoglycemia was observed, while subsequent glucose measurements ranged from 90 to 160 mg/dL.

The patient was discharged on day 9 of hospitalization without the need for permanent steroid supplementation.

DISCUSSION

In this case, the patient's unstable hemodynamics cannot be fully explained by the estimated blood loss, which is relatively low compared to the volume of fluids and blood components administered. Given this discrepancy, it is essential to consider other potential etiologies of shock beyond hypovolemic shock. Specifically, distributive shock resulting from adrenal insufficiency should be considered as a potential cause in case of adrenal injury. While differentiating among various causes of distributive shock, such as septic shock, neurogenic shock, or Critical Illness-Related Corticosteroid Insufficiency (CIRCI), is inherently challenging in acute clinical settings, several clinical and laboratory findings in this patient pointed toward adrenal insufficiency as the primary cause. The hemodynamic profile was notable for persistent hypotension that was unresponsive to fluids and vasopressors but improved rapidly following corticosteroid administration. Baseline cortisol levels were markedly low for the severity of the patient's condition, supporting a diagnosis of adrenal insufficiency rather than CIRCI. Infectious evaluation revealed no identifiable source of infection on physical examination or CT imaging, and the patient's clinical improvement occurred before infection source control, making septic shock less likely. Furthermore, there were no clinical or imaging findings suggestive of neurogenic shock, such as spinal cord injury or traumatic brain injury. Taken together, these findings strongly support adrenal insufficiency as the most plausible etiology of shock in this patient.

Previous reports have shown that adrenal insufficiency typically occurs in the setting of bilateral adrenal injury, with most unilateral cases managed conservatively without hormonal dysfunction. In contrast, our case highlights a rare instance of adrenal insufficiency following unilateral adrenal trauma, suggesting that impaired compensatory function of the contralateral gland, possibly due to stress-related hypoperfusion, hypovolemic shock, or subclinical dysfunction, may contribute to adrenal failure even in the absence of bilateral involvement. The comparison of the characteristics between this case and those reported in previous case reports is presented in Table 1.

Primary adrenal insufficiency is characterized by the adrenal glands' inability to produce steroid hormones[14]. Functional adrenal insufficiency refers to subnormal adrenal corticosteroid production during acute severe illness, typically transient in nature[14]. When the adrenal glands fail to mount an adequate stress response, this is referred to as relative adrenal insufficiency, which can be diagnosed by a random cortisol concentration of less than 25 μg/dL with a hemodynamic response to the prompt treatment. One potential etiology of adrenal insufficiency is traumatic hemorrhage. Secondary adrenal insufficiency is defined as inadequate adrenal gland function resulting from a lesion or dysfunction of the pituitary or hypothalamus.[14]

The high-dose short-term ACTH stimulation test is used to diagnose primary adrenal insufficiency[14]. This test involves administering 250 μg of ACTH via intrave-

Table 1. Case reports on adrenal gland injury.

Report (year)	Patient	Side of adrenal gland	Adrenal insufficiency	Management	Outcome
Kahdi F. Udobi et al. (2001)[11]	50-year-old female	Bilateral	Yes	Intravenous hydrocortisone	-
Y. Lin et al. (2013)[15]	32-year-old male	Left	No	Conservative	-
A. Lehrberg et al. (2017)[6]	53-year-old male	Right	No	Conservative	CT showed complete resolution of previous adrenal hemorrhage.
N. Jimidar et al. (2018)[10]	44-year-old male	Bilateral	Yes	Intravenous hydrocortisone	Persistent adrenal insufficiency.
I.M. Karwacka et al. (2018)[8]	73-year-old female	Right	No	Adrenalectomy	-
	65-year-old male	Left	No (hypercortisolemia, elevated normetanephrine)	Observation	CT showed decrease size of mass.
	45-year-old male	Right	No	Observation	-
	57-year-old female	Left	No	Observation	-
Y. Fadil (2021) [16]	47-year-old male	Right	-	Conservative	-
N. Szwarcbard et al. (2022)[9]	60-year-old male	Bilateral	Yes	Intravenous hydrocortisone	CT showed resolution of previous adrenal hemorrhages but a low-density mass, consistent with an adenoma. Normal adrenal biochemistry.
	88-year-old female	Bilateral	Yes	Intravenous hydrocortisone	CT showed complete resolution of previous adrenal hemorrhages. Persistent adrenal insufficiency.
	46-year-old male	Bilateral	Yes	Intravenous hydrocortisone	Persistent adrenal insufficiency.
Bulent Guvendi et al. (2023)[17]	42-year-old male	Bilateral	No	-	CT showed complete resolution of previous adrenal hemorrhages.
Our case	36-year-old male	Left	Yes	Conservative	-

nous or intramuscular injection, followed by measuring serum cortisol levels at baseline, 30 minutes, and 60 minutes after the injection. A serum cortisol level exceeding 18-20 μg/dL after ACTH administration generally rules out primary adrenal insufficiency[10,11].

Most clinical symptoms of adrenal insufficiency are nonspecific. Laboratory abnormalities such as hypoglycemia, hyponatremia, hyperkalemia, acidosis, lymphocytosis, and eosinophilia may be observed in chronic adrenal insufficiency[8,10,14].

Replacement therapy with glucocorticoids should be initiated promptly when adrenal insufficiency is suspected [14]. The recommended initial treatment is an intravenous bolus of 100 mg hydrocortisone, followed by a continuous infusion, with a total dose of 240-300 mg per day[5].

In this patient, serum cortisol during the shock period was measured at 7.86 μg/dL. Additionally, the 8:00 AM serum cortisol level was 17.8 μg/dL, which increased to 23.2 μg/dL following the ACTH stimulation test. The laboratory results also revealed an episode of hypoglycemia and hyperkalemia. After the proper treatment with hydrocortisone, the patient's hemodynamics improved, and recovery was achieved. All findings were consistent with relative adrenal insufficiency.

CONCLUSION

This case demonstrates the critical importance of recognizing adrenal insufficiency in trauma patients, particularly when adrenal gland injury occurs in the context of multi-organ trauma and shock. A temporary administration of hydrocortisone can significantly stabilize hemodynamics and reduce the need for vasopressor support, as demonstrated in this patient.

ACKNOWLEDGEMENT

We would like to express our sincere gratitude to our staff for their expert care and dedication in managing this complex case. We also would like to acknowledge the patient for their cooperation throughout the treatment process. Special thanks to Mrs. Sam Ormond for proofreading. This case would not have been possible without the collaborative effort of all involved.

AUTHORS' CONTRIBUTIONS

Conceptualization: CA, KA; Writing & editing: CA, KA.

REFERENCES

1. Falhammar H, Koskinen SK, Kistner A. Adrenal trauma experience at a major tertiary centre in Sweden: Clinical and radiological findings. Clin Endocrinol (Oxf). 2022;97:28-35.
2. Duncan AJ, Chahal IK, Sang HI, Ahmeti M. "Blunt adrenal injury does not affect outcomes in adult patients with isolated abdominal injury - A National Trauma Data Bank review(R)". Injury. 2024;55:111613.
3. Sengun B, Iscan Y, Doylu A, Sal O, Gok AFK, Sormaz IC, et al. Adrenal gland injury in trauma patients and its impact on clinical outcomes. Ulus Travma Acil Cerrahi Derg. 2024;30:546-33.
4. Raup VT, Eswara JR, Vetter JM, Brandes SB. Epidemiology of Traumatic Adrenal Injuries Requiring Surgery. Urology. 2016;94:227-31.
5. Truong H, Figler BD. Traumatic Adrenal Hemorrhage. A Clinical Guide to Urologic Emergencies. 2021. p 83-90.
6. Lehrberg A, Kharbutli B. Isolated unilateral adrenal gland hemorrhage following motor vehicle collision: a case report and review of the literature. J Med Case Rep. 2017;11:358.
7. Kunhivalappil FT, Hefny AF, Abu-Zidan FM. Management of blunt adrenal gland injury in a community-based hospital. Injury. 2019;50:1049-52.
8. Karwacka IM, Obolonczyk L, Sworczak K. Adrenal hemorrhage: A single center experience and literature review. Adv Clin Exp Med. 2018;27:681-7.
9. Szwarcbard N, Davis A, Bach LA, Hackman K. Adrenal gland haemorrhages following motor vehicle accident with resultant adrenal insufficiency. Endocrinol Diabetes Metab Case Rep. 2022;2022.
10. Jimidar N, Ysebaert D, Twickler M, Spinhoven M, Dams K, Jorens PG. Bilateral adrenal haemorrhage after a high energetic trauma: a case report and review of current literature. Acta Chir Belg. 2020;120:131-5.
11. Udobi KF, Childs EW. Adrenal crisis after Traumatic Bilateral Adrenal Hemorrhage. The Journal of TRAUMA Injury, Infection, and Critical Care. 2001;51:597-600.
12. DiGiacomo JC, Gerber N, Angus LDG, Munnangi S, Cardozo-Stolberg S. Blunt Adrenal Injury: Results of a State Trauma Registry Review. Am Surg. 2019;85:390-6.
13. Tanizaki S, Maeda S, Ishida H. Blunt adrenal gland injury: The impact of extra-abdominal injury. J Trauma Acute Care Surg. 2021;91:716-8.
14. Gerlach H. Adrenal insufficiency. In: Vincent J-L, Moore FA, Bellomo R, Marini JJ, editors. Textbook of Critical Care. 8 ed. 2024. p 1092-101.
15. Lin Y-H, Wu T. Isolated adrenal hemorrhage after blunt trauma: Case report and literature review. Urological Science. 2013;24:27-9.
16. Fadil Y, Bai W, Dakir M, Debbagh A, Aboutaieb R. Post-traumatic adrenal hematoma: A case report and revue of literature. Urol Case Rep. 2021;35:101534.
17. Guvendi B, Koksal A, Gozgec E, Ogul H, Kantarci M. Bilateral adrenal haemorrhage after blunt abdominal trauma. Br J Hosp Med (Lond). 2023;84:1.