

# Adrenal gland haemorrhages following motor vehicle accident with resultant adrenal insufficiency

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## Summary

Adrenal gland haemorrhage is an uncommon, yet likely under-diagnosed complication of high-impact trauma, such as motor vehicle accidents (MVA). It usually occurs with multi-trauma and is associated with additional injuries to the ribs, liver, kidney, spleen and vertebrae. Trauma cases with resultant adrenal gland injury have higher mortality rates. Primary adrenal insufficiency as a result of bilateral adrenal haemorrhage is potentially fatal. We report three cases of life-threatening adrenal insufficiency following adrenal injuries sustained in MVA's. Case 1 was a 60-year-old-male who presented with acute haemodynamic instability on admission. Case 2 was an 88-year-old female on anticoagulation for atrial fibrillation, who developed haemodynamic instability 10 days into her admission. Case 3 was a 46-year-old male who developed hyponatraemia 2 weeks post-MVA. All were commenced on stress dose hydrocortisone replacement with improvement in clinical status. Only case 1 has had complete adrenal axis recovery, whereas the other patients remain on maintenance hydrocortisone replacement. Our cases demonstrate acute and subacute presentations of adrenal insufficiency following traumatic bilateral adrenal haemorrhages and highlight the importance of assessing adrenal morphology and function in any trauma patient with haemodynamic instability or hyponatraemia.

## Learning points:

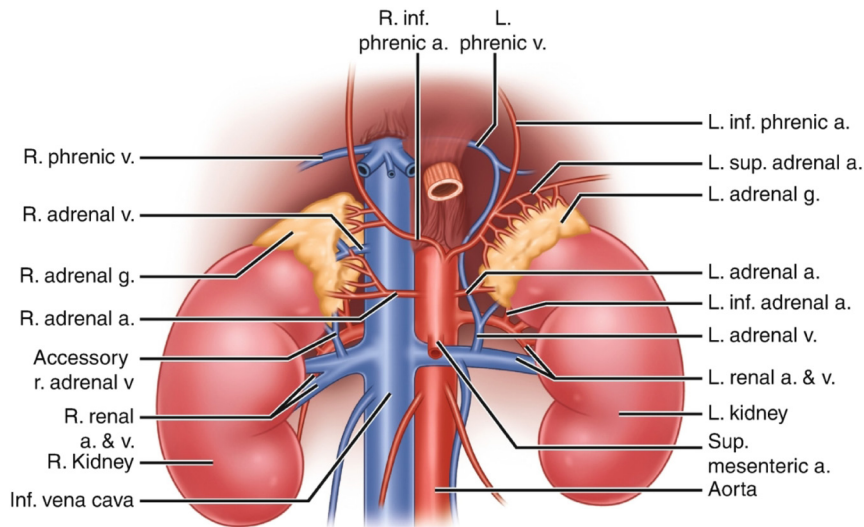
- Adrenal gland haemorrhage is an under-diagnosed consequence of high-impact trauma.
- Trauma patients with adrenal haemorrhage have a significantly increased mortality risk.
- Bilateral adrenal gland haemorrhage can result in life-threatening adrenal insufficiency requiring urgent glucocorticoid replacement.
- Biochemical assessment of the adrenocortical axis should be considered in all patients presenting with high-impact trauma following motor vehicle accidents.
- Given the potential for delayed presentation, any patients with new haemodynamic instability should have repeat biochemistry and/or imaging performed, even if initial adrenal imaging and investigations were normal.

## Background

The adrenal glands are small retroperitoneal organs located superiorly to the kidneys bilaterally, encased in fascia and surrounded by fat. They are supplied by a highly vascularised arterial plexus, formed by the inferior phrenic arteries, renal arteries and the aorta. Venous drainage is via

a single vein with the right adrenal vein draining directly into the inferior vena cava and the left draining via the renal vein (Fig. 1) (1, 2).

The adrenal cortex is responsible for steroid hormone production and is organised into three layers. The outer



**Figure 1**  
Adrenal gland arterial supply and venous drainage (2).

zona glomerulosa produces mineralocorticoids, the zona fasciculata produces glucocorticoids and the inner zona reticularis produces androgens (1). The adrenal medulla is the site of catecholamine production (1).

Although the adrenal cortex is vulnerable to traumatic damage, adrenal injuries may be overlooked in trauma patients. Trauma can result in impairment of one or more adrenal cortex hormones and life-threatening acute adrenal insufficiency may occur due to glucocorticoid deficiency (3, 4).

We present three cases of traumatic bilateral adrenal haemorrhage following motor vehicle accidents. The clinical presentations differed markedly between the cases, highlighting the need to remain vigilant for this rare consequence of high-impact trauma.

## Case presentation

### Case 1

A 60-year-old male, presented following a high-speed motor vehicle accident (MVA) with fractures of the C2 vertebra, mid-face (right Le Fort III and left Le Fort I), ribs, left acetabulum and bilateral talus bones. He was noted to have significant haemodynamic instability in the emergency department.

### Case 2

An 88-year-old female on dabigatran for atrial fibrillation presented following a high-speed MVA with T8, T9 and bilateral rib fractures, a left-sided subdural haematoma and a subcapsular splenic haematoma. She was haemodynamically stable throughout her week-long admission and was

discharged to rehabilitation. However, 3 days later she was found unresponsive with systolic blood pressure 65 mmHg and was readmitted for vasopressor support.

### Case 3

A 46-year-old male presented following a high-speed MVA with a diffuse axonal injury, intraventricular haemorrhage, frontozygomatic suture fracture, anterior longitudinal ligament tear with disc disruption to C4/5 and C5/6 and T6 vertebral body fracture with a T5-8 prevertebral haematoma. He was haemodynamically stable throughout his admission but developed hyponatraemia after two weeks.

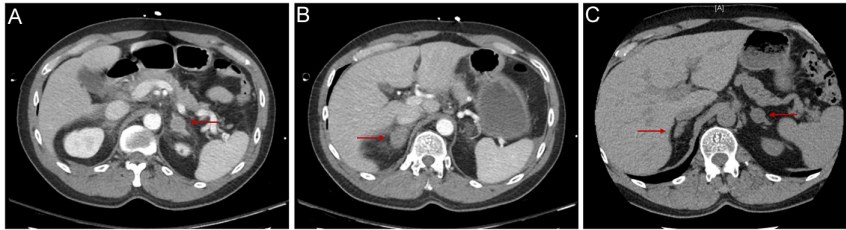
## Investigation

### Case 1

Initial trauma CT demonstrated hepatic and bilateral adrenal haemorrhages, measuring 20 × 22 mm on the left and 29 × 19 mm on the right (Fig. 2A and B). Given his haemodynamic instability, hydrocortisone was immediately commenced. To determine his adrenal status, the evening hydrocortisone was withheld on day five of admission. The following morning an 8:00 h cortisol was 57 nmol/L (100–540 nmol/L) with a concurrent increase in his noradrenaline requirements.

### Case 2

Initial trauma CT did not demonstrate any adrenal injury (Fig. 3A). Repeat abdominal CT performed on re-admission (10 days post-MVA) demonstrated new bilateral adrenal haemorrhages, measuring 19 × 25 mm on the left and



**Figure 2**

(A and B) Case 1. Initial CT abdomen demonstrating well defined nodular masses within both adrenal glands (50–80 HU) with retroperitoneal fat stranding. (A) Left side measuring 20 × 22 mm with an appearance consistent with bleeding into a pre-existing adrenal adenoma. (B) Right side measuring 29 × 19 mm. (C) Four-month follow-up CT demonstrating resolution of bilateral adrenal haemorrhages with a persisting left sided adrenal adenoma measuring 20 × 22 mm and normal right adrenal measuring 17 × 5 mm.

21 × 35 mm on the right (Fig. 3B). Serum cortisol was undetectable (<28 nmol/L) at 7:00 h the following morning and she was commenced on IV hydrocortisone 100 mg QID. Persisting primary adrenal insufficiency was confirmed 1 week later with an 8:00 h cortisol of 129 nmol/L and an ACTH level of 18 pmol/L (reference range 1.6–13.9 pmol/L).

### Case 3

Initial trauma CT did not demonstrate any adrenal injury (Fig. 4A). On day 15 of admission, he developed worsening hyponatraemia with a nadir serum sodium of 119 mmol/L and serum osmolality of 257 mOsm/kg. Concurrent urine sodium was 107 mmol/L and osmolality was 578 mOsm/kg. An afternoon short synacthen test demonstrated blunted adrenal response, with cortisol levels of 237, 272 and 305 nmol/L at 0, 30 and 60 min post-synacthen injection respectively. Repeat abdominal CT scan demonstrated new bilateral adrenal haemorrhages, measuring 20 × 20 mm on the right and 27 × 24 mm on the left (Fig. 4B). Two weeks later, aldosterone and renin levels were normal at 119.0 pmol/L (32.4–653.7) and 9.9 mIU/L (2.8–39.9), respectively.

## Treatment

### Case 1

In the emergency department, the patient was commenced on intravenous (IV) hydrocortisone replacement at stress

doses. Over the next 10 days, the hydrocortisone was weaned to an oral replacement dose of 20 mg mane and 10 mg at 14:00 h.

### Case 2

Following re-presentation to the hospital, the patient was commenced on IV hydrocortisone replacement at stress doses. Hydrocortisone was then weaned to an oral replacement dose of 20 mg mane and 10 mg at 14:00 h, and she was transferred back to rehabilitation.

### Case 3

In the setting of new hyponatraemia, the patient was treated with aliquots of hypertonic (3%) saline and fluid restriction. He was also commenced on IV hydrocortisone replacement at stress doses, which was weaned to an oral replacement dose of 20 mg mane and 10 mg at 14:00 h. The hyponatraemia began to improve the day after hydrocortisone was commenced and had returned to normal within a week.

## Outcome and follow-up

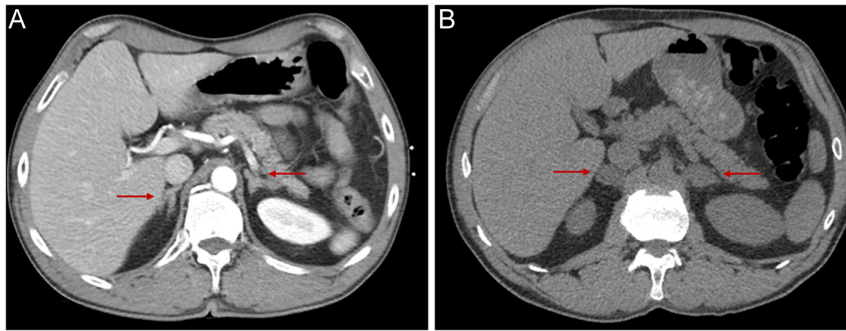
### Case 1

Three weeks following his MVA, after withholding the afternoon hydrocortisone, 8:00 h cortisol was robust



**Figure 3**

(A) Case 2. Initial CT abdomen demonstrating no adrenal lesion or haemorrhage. (B) Repeat CT scan following readmission demonstrating bilateral adrenal haemorrhages, measuring 19 × 25 mm on the left (54 HU) and 21 × 35 mm on the right (55 HU). (C) Four-month follow-up CT demonstrating resolution of bilateral adrenal haemorrhages with the left adrenal gland measuring 9 × 18 and 10 × 19 mm on the right.



**Figure 4**

(A) Case 3. Initial CT abdomen demonstrating no adrenal lesion or haemorrhage. (B) Repeat CT scan demonstrated new bilateral adrenal haemorrhages, measuring 20 × 20 mm on the right (18 HU) and 27 × 24 mm on the left (22 HU).

at 533 nmol/L with a normal ACTH of 7.48 pmol/L. Hydrocortisone therapy was ceased.

Four months later, repeat adrenal CT demonstrated resolution of the previous bilateral adrenal haemorrhages but a 20 × 22 mm low-density mass (5–13 Hounsfield units (HU)), consistent with an adenoma, remained within the left adrenal (Fig. 2C). Adrenal biochemistry performed at this time was normal: 8:00 h cortisol 333 nmol/L, ACTH 6.6 pmol/L, aldosterone 189 pmol/L (100–950 pmol/L), renin 12 mU/L (3.3–41 mU/L), aldosterone:renin ratio 16. Serum metanephrines and normetanephrines were also normal. The patient remains well and will have a repeat CT adrenal in 12 months to monitor the left adrenal adenoma.

### Case 2

Repeat abdominal CT scan 4 months later demonstrated complete resolution of the adrenal haemorrhages (Fig. 3C). However, the patient has persisting primary adrenal insufficiency, with an 8:00 h cortisol of 43 nmol/L and ACTH of 66.2 pmol/L and remains on hydrocortisone replacement.

### Case 3

Four months post-MVA, the patient had ongoing adrenal glucocorticoid insufficiency with a 9:00 h cortisol of 192 nmol/L and an ACTH of 33.4 pmol/L. Serum sodium was normal at 138 mmol/L. He remains on hydrocortisone at a reduced dose of 10mg mane and 10 mg at 14:00 h. He is yet to have repeat imaging performed.

## Discussion

We describe three cases of adrenal insufficiency following bilateral traumatic adrenal haemorrhages in the setting of high-speed motor vehicle accidents. Each case presented with a different clinical course: acute haemodynamic

instability, subacute haemodynamic instability and subacute hyponatraemia.

Adrenal gland haemorrhage is a rare but potentially fatal outcome of trauma (3). Such trauma is usually high-impact, such as an MVA or blunt-force trauma, and is most often accompanied by additional injuries to the ribs, liver, kidney, spleen and vertebrae (3). Indeed, isolated adrenal gland trauma is rare (3). A retrospective review of over 1.7 million trauma cases registered with the US National Trauma Data Bank from 2007 to 2011 found the incidence of adrenal gland trauma to be 0.44% (5). However, autopsy studies report adrenal injury in 7–26% of trauma patients, indicating that many cases go undiagnosed (3). The incidence of acute vs subacute haemorrhage has not been reported.

Trauma cases with associated adrenal gland injury have a mortality rate up to five times higher than those without adrenal gland injury, with the highest rates occurring in patients with bilateral adrenal involvement. Although the increased mortality is likely related to the overall severity of injuries, haemodynamic instability as a result of undiagnosed glucocorticoid deficiency may also contribute (4, 6). Adrenal haemorrhages are more commonly right sided, due both to potential compression between the liver and spine and transmission of increased venous pressure directly from the inferior vena cava (4). Non-traumatic causes of adrenal haemorrhage include sepsis (especially meningococcaemia), hypotension, antiphospholipid syndrome, disseminated intravascular coagulopathy, adrenal infection (e.g. tuberculosis and histoplasmosis) and post-instrumentation (7). These causes should be readily distinguishable from traumatic causes on history alone.

Adrenal haemorrhages can be easily identified radiologically. On CT scan, the most common imaging modality, they characteristically appear as a non-enhancing round or oval lesion with a density of 50–80 HU, often with surrounding stranding of the periadrenal fat (8, 9). The high initial attenuation then decreases over time. Adrenal size also returns to normal as the haemorrhage





resolves (8, 9). MRI is the most sensitive and specific imaging modality, however, it is less readily available and therefore less commonly used. Initially, lesions are T1 iso- or hyperintense and T2 hypointense and become T1 and T2 hyperintense sub-acutely. Chronic lesions have a haemosiderin rim which is T1 and T2 hypointense (10). Many haemorrhages resolve completely, but some may persist as a heterogeneous mass with a hypodense centre, and may develop calcifications after 12 months (9).

Primary adrenal insufficiency is the major consequence of bilateral adrenal haemorrhage. Patients are at highest risk if more than 90% of the gland is affected (3, 4, 7). Its incidence and time to clinical presentation have not previously been described, and as seen in our cases they can involve both acute and subacute presentations with either haemodynamic instability or hyponatraemia. Additional patient factors such as anticoagulation may also alter the clinical time course, as seen in case 2. Primary adrenal insufficiency can be diagnosed biochemically with a low morning cortisol and high morning adrenocorticotrophic hormone (ACTH) level and may be accompanied by hyponatraemia and hyperkalaemia (1).

Most cases of adrenal haemorrhage are managed conservatively, however transcatheter angiographic embolisation and surgical adrenalectomy are potential treatment options in the setting of severe or persistent haemorrhage (3, 5, 11). In the setting of bilateral adrenal haemorrhage, acute adrenal insufficiency will require stress doses of intravenous glucocorticoid replacement (1).

## Conclusion

These cases highlight the importance of recognising adrenal gland haemorrhage as a significant but likely underdiagnosed consequence of high-impact trauma. In cases where adrenal haemorrhage is noted on CT or there are clinical features of haemodynamic instability or persistent hyponatraemia, urgent assessment of the adrenocortical axis with rapid replacement of glucocorticoids is essential. Acute and subacute adrenal insufficiency is a serious complication of bilateral adrenal haemorrhage that must be considered in high-impact trauma patients, even if initial biochemical and radiological testing is normal.

### Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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### Patient consent

Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient/parent/guardian/relative of the patient.

### Author contribution statement

N S was involved in direct care of cases 1 and 3 and wrote the first draft of the manuscript. A D provided expert radiological advice. L B was involved in direct care of cases 1 and 2, provided expert input and reviewed the manuscript. K H was involved in direct care of cases 1 and 2, provided expert input and reviewed the manuscript.

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