



An important but easily overlooked medical complication of multiple trauma

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DECLARATIONS

Adrenal gland failure from blunt injury can be a serious complication of multiple trauma.

post event showed no calcification to suggest previous haemorrhage.

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Case reports

We present two cases of post-traumatic hypoadrenalism.

Case 1

A 53-year-old man was trapped under a hydraulic lift on the back of his lorry trailer. He was found to have spinal injuries with a comminuted unstable fracture of T6 and stable fracture of T12. He had further fractures of the left first rib, right second rib, and his sternum. He was admitted to the orthopaedic ward for conservative management with a spinal splint for five weeks. He remained stable until day 20 of his admission when he became hypotensive and tachycardic. Blood tests demonstrated hyponatraemia and hyperkalaemia. Despite fluid resuscitation and emergency treatment for hyperkalaemia he remained hypotensive and hyponatraemic.

An endocrine opinion was requested and he was found to have an abnormal short synacthen test (cortisol of 436 nmol/L falling to 398 nmol/L at 30 min). ACTH was high at 208 ng/L, and thyroid function tests were normal (TSH 2.02 mU/L, FT4 15.4 pmol/L) indicating an intact pituitary axis. His hypotension and electrolyte abnormality rapidly responded to glucocorticoid and mineralocorticoid treatment. A repeat short synacthen test has confirmed persistent hypoadrenalism (peak cortisol 175 nmol/L at 30 min). Although the adrenal glands lie retroperitoneally at the level of T12 which was fractured, a CT scan at the time of trauma showed no signs of adrenal gland damage. A repeat scan one year

Case 2

A 57-year-old man was injured after jumping out of his tilting lorry cab, which fell on top of him. He had fractures of T2 to T4 vertebrae, which were unstable and required urgent fixation. He also suffered a left rib fracture resulting in a pneumothorax. He had a long stay in ITU due to slow clinical progress, but even after discharge to the ward, had repeated admissions to HDU for recurrent episodes of hypotension and hyperkalaemia. Eventually 88 days after admission, an endocrine opinion was sought. A short synacthen test was performed which was grossly abnormal (basal cortisol of 7 nmol/L rising to 9 nmol/L at 30 min). Pituitary function testing showed an intact gonadotrophin and thyroid axis with normal prolactin (TSH 1.91 mU/L, FT4 18.3 pmol/L, FT3 5.5 pmol/L, LH 3.3 IU/L, FSH 5.6 IU/L, testosterone 11.6 nmol/L, prolactin 163 mIU/L).

After starting steroid replacement he improved significantly and was subsequently discharged home on steroid replacement. This diagnosis was confirmed as an outpatient three months later, with a persistent grossly subnormal repeat short synacthen test. He had two CT scans three years apart which showed no signs of damage to his adrenal glands.

Discussion

Primary adrenal insufficiency has a prevalence of 93–140 per 1 million people and an annual incidence of 4.7–6.2 per million people in Western populations.¹ Autoimmune adrenalitis is the cause in 80% of these cases.² Other causes of

hypoadrenalism are relatively rare and this may lead to delays in diagnosis, which can result in significant morbidity and occasional mortality.³

Our two patients had no symptoms or signs of adrenal gland failure prior to their injury. Their adrenal antibody tests were negative making autoimmune hypoadrenalism unlikely and the pituitary axes were intact, thereby excluding secondary hypoadrenalism. The location of their injuries was adjacent to the adrenal bed. Neither patient sustained a head injury. We suggest that the adrenal gland circulation was damaged by the trauma and led to adrenal gland dysfunction. In the second case there was a longer delay in diagnosis and by the time the endocrine team were contacted the patient had virtually no remaining adrenal reserve.

There have been few studies on the incidence of adrenal injury in patients with blunt trauma. CT abnormalities occasionally noted include indistinct enlarged adrenal glands, focal haematomas, and 'stranding' of peri-adrenal fat. Often co-existent thoracic, abdominal or spinal injuries are seen.^{4–6}

Patients with severe multiple injuries have many reasons for haemodynamic instability and delayed recovery. We believe that in patients with evidence of blunt trauma to the abdomen or spine, the possible development of post-traumatic hypoadrenalism should be considered and investigated.

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