Bilateral Adrenal Hemorrhage after Blunt Abdominal Trauma

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INTRODUCTION

Adrenal insufficiency associated with bilateral adrenal hemorrhage after blunt torso trauma is uncommon and may not be suspected as a cause of hemodynamic instability in a patient with multi-system trauma. Without appropriate treatment, acute adrenal failure is associated with major complications and death.

Only a few cases of bilateral adrenal hemorrhage after blunt trauma have been reported.1-4 We present a patient who suffered multiple injuries in a motor vehicle collision, including bilateral adrenal hemorrhage. The patient’s injuries resulted in adrenal insufficiency, which required steroid supplementation.

CASE REPORT

A 52-year-old male was evaluated at an American College of Surgeons-verified level I trauma center after suffering injuries in a motor vehicle collision. On presentation, the patient had a Glasgow Coma Scale Score of 3T (i.e., no eye opening, no verbal response, no motor response, and intubated), a blood pressure of 126/114 mmHg, and a heart rate of 111 bpm. The patient underwent computed tomography (CT) of the head, cervical spine, chest, abdomen, and pelvis. These radiographic studies revealed bilateral adrenal injury (Figure 1), a splenic laceration (Figure 2), left renal laceration, a lung contusion, rib fractures, cerebral intraparenchymal and subarachnoid hemorrhages, a condyle fracture of the first cervical vertebrae, and pubic rami fractures.

The patient underwent splenic embolization, which was unsuccessful. The patient subsequently underwent emergent exploratory laparotomy and splenectomy. Despite surgical control of intraabdominal bleeding and aggressive resuscitation with crystalloids and blood transfusions, the patient remained hemodynamically unstable. An adrenocorticotropic hormone (ACTH; cosyntropin) stimulation test was performed due to suspected adrenal insufficiency and the results were consistent with that diagnosis. The patient was treated with hydrocortisone and fludrocortisone. The patient’s blood pressure stabilized after the first dose of intravenous steroids.

The patient subsequently underwent tracheostomy and gastrostomy tube placement. Ten days after his initial presentation, a repeat cosyntropin test and a 24-hour urine cortisol level were obtained. The patient’s cortisol level was 1 mcg/dL prior to the administration of cosyntropin and 8 mcg/dL after 60 minutes had elapsed, again consistent with adrenal insufficiency. Only trace amounts of cortisol were detected in the 24-hour urine collection. The patient remained on steroid supplementation at his discharge from the hospital to a long-term facility 10 days after his initial injuries.
Bilateral Adrenal Hemorrhage

**Discussion**

The incidence of adrenal hemorrhage in trauma patients is unknown because some adrenal injuries are asymptomatic and may remain undiagnosed. Retrospective evaluations of CT scans performed on patients with traumatic torso injuries showed an incidence of adrenal injuries of 1-3%, with the right adrenal gland injured more often.\(^2\) In a series of 1,120 patients who underwent CT for blunt abdominal trauma, adrenal hemorrhage was found in 20 patients (2%). The injuries were unilateral in 17 patients (12 right-sided and five left-sided) and bilateral in three patients (23 total adrenal injuries).

Rana et al.\(^5\) evaluated 2,692 trauma patients who underwent CT. Adrenal hematoma was detected in 51 patients (1.9%), with bilateral lesions occurring in three patients. In the pediatric trauma population, Sivit et al.\(^6\) reviewed 1,155 CT examinations and found adrenal hemorrhage in 34 patients (3%) with the majority of injuries to the right adrenal gland.

Diagnosing adrenal insufficiency in a patient who has suffered multiple injuries is difficult because the signs and symptoms are often nonspecific and coincide with what is seen with other traumatic injuries such as hypovolemia, closed head injuries, and spinal shock. The patient often presents with altered mental status, fever or hypothermia, and progressive hypotension that is often refractory to vasopressors.

The diagnosis of adrenal insufficiency is confirmed with laboratory testing. In normal subjects, serum cortisol concentrations are highest in the early morning, ranging from 10 to 20 mcg/dL. An early morning serum cortisol concentration less than 10 mcg/dL is suggestive of adrenal insufficiency. Adrenal insufficiency is diagnosed most often with a cosyntropin stimulation test. There are differing opinions on how to perform the ACTH stimulation test and the amount of ACTH to administer. To perform the standard high dose ACTH stimulation test, a baseline serum cortisol level is determined prior to the administration of 250 mcg of cosyntropin intravenously. A serum cortisol level is checked 30 minutes and 60 minutes after cosyntropin administration. A normal response would produce a cortisol level
greater or equal to 25 mcg/dL for critically ill, post-traumatic patients. Those who fail to demonstrate the appropriate response are diagnosed with adrenal insufficiency.

The imaging study used to diagnose adrenal hemorrhage is CT. Usually, adrenal hemorrhage is an incidental finding when a CT is ordered for other reasons. In the study by Burks et al., nineteen of the adrenal injuries (83%) appeared as discrete round to oval hematomas expanding the adrenal gland, while two (9%) appeared as diffuse irregular hemorrhage obliterating the gland, and two (9%) appeared as uniform swelling of the adrenal gland. Associated CT findings included “stranding” of the peri-adrenal fat caused by blood in 14 cases (61%) and posterior pararenal hemorrhage mimicking a thickened diaphragmatic crus in nine cases (39%).

In the study by Sivet et al., all of the adrenal hemorrhages had decreased attenuation relative to the liver and spleen on contrast-enhanced CT and ipsilateral diaphragmatic crural thickening was a frequent (61%) associated finding. In a study by Sinelnikov et al., the CT findings of adrenal trauma were: focal hematoma (30%), indistinct (27%) or enlarged (18%) adrenal gland, gross (15%) or focal (7%) adrenal hemorrhage, and adrenal mass (11%). Associated CT findings included peri-adrenal fat stranding (93%), retro-peritoneal hemorrhage (22%), and thickened diaphragmatic crura (10%).

The primary treatment of adrenal insufficiency is steroid replacement with mineralocorticoid and glucocorticoid coverage. Hydrocortisone 100mg, intravenous, is administered every 6-8 hours and is adjusted to the stress level of the patient. It also is recommended to administer fludrocortisone for mineralocorticoid supplementation. The amount of steroid administered can be tapered as the patient responds and may be changed to an oral form when the patient is tolerating enteral intake. The likelihood of regaining adrenal function after bilateral adrenal hemorrhage is uncertain. There are at least two reports of patients who have regained adrenal function after experiencing adrenocortical insufficiency following bilateral adrenal trauma. However, many patients require lifelong steroid replacement.

Our patient had not regained adrenal function 10 days after his traumatic injury and was discharged to a long-term care facility on steroid supplementation. It is uncertain if patients with bilateral adrenal hemorrhage will regain adrenal function, but testing for the return of adrenal function should be done so that patients do not remain on unnecessary, lifelong steroid use.

**Conclusion**

Although traumatic bilateral adrenal hemorrhage resulting in adrenal insufficiency is rare, it can be a significant cause of morbidity and mortality in critically injured patients. Evaluation for adrenal insufficiency should be undertaken when a critically injured patient remains hemodynamically unstable after the more common etiologies of hemodynamic instability have been excluded. Administration of steroids for adrenal insufficiency can be life-saving.

**References**

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Keywords: adrenal insufficiency, adrenal glands, hemorrhage, blunt injuries