osttraumatic adrenal hemorrhage is an uncommon finding on CT. In a series of 1120 adult patients who underwent CT for blunt abdominal trauma, Burks et al. [1] found adrenal hemorrhage in 20 patients (2%). In the pediatric trauma population, Sivit et al. [2] reviewed 1155 CT examinations and found adrenal hemorrhage in 34 patients (3%). Approximately 85% of trauma-related adrenal hemorrhages occur in the right adrenal gland [1–4]. In several cases found in the literature, inferior vena cava thrombosis was seen concurrently with adrenal hemorrhage [1, 5]. However, a cause-and-effect relationship has not been clearly established. It was suggested that the inferior vena cava thrombus and adrenal hemorrhage both result from direct compression by adjacent organs [1, 5]. In contrast, we present a case in which inferior vena cava thrombus was detected in the suprarenal inferior vena cava before an adrenal hemorrhage. Our case suggests that outflow obstruction caused by traumatic inferior vena cava thrombosis may be a mechanism of post-traumatic adrenal hemorrhage.

Case Report

A 45-year-old woman, who was an unrestrained passenger involved in a collision with another vehicle, was brought to the emergency department. She was ejected from her car and lost consciousness at the scene. Her vital signs were blood pressure, 130 over 80 mm Hg; pulse, 100; respiration rate, 25/min; and O₂ saturation of room air, 85–90%. The initial trauma series included a portable chest radiograph, and lateral cervical spine and pelvic radiographs. The chest radiograph revealed bilateral pneumothoraces and a left scapular fracture. After insertion of chest tubes bilaterally, the patient underwent CT scans of the head, cervical spine, abdomen, and pelvis. All CT examinations were performed on a Hi-Speed CT/i scanner (General Electric Medical Systems, Milwaukee, WI). Body scanning was performed using 5-mm collimation and a power injection of nonionic contrast medium (150 mL, 300 mg/mL, 1.5 mL/sec). The initial abdominopelvic CT revealed bilateral pleural effusions, a filling defect in the inferior vena cava (Fig. 1A), and pelvic fractures.

On further review of the trauma series, the radiologist identified left first and second rib fractures. CT of the chest was then performed to evaluate possible intrathoracic injury. This scanning process was completed approximately 1 hr after the initial abdominopelvic CT. The images obtained through the upper abdomen again revealed the filling defect in the inferior vena cava (Fig. 1B) and a new large round fluid collection in the right suprarenal area consistent with interval formation of adrenal hemorrhage. The patient remained hemodynamically stable throughout her hospitalization. No further workup was done for the adrenal hemorrhage and inferior vena cava thrombus. The patient responded well to treatment and was transferred to a nursing care facility for rehabilitation.

Discussion

CT characteristics of adrenal hemorrhage have been well described in the literature [1–4]. Adrenal hemorrhage on CT usually appears as a round or oval well-delineated mass, but occasionally the hemorrhage may be diffuse and irregular. Associated findings include infiltration of the periadrenal fat and thickening of the diaphragmatic crus. Adrenal hemorrhage is usually, but not always, associated with other ipsilateral thoracoabdominal injuries such as rib and spine fractures; pneumothoraces; lung contusions; and splenic, hepatic, and renal injuries [5].

There are several hypotheses for the mechanism of adrenal hemorrhage resulting from blunt abdominal trauma. One theory is that the injury is caused by direct compression of the adrenal gland by the adjacent vertebra and viscera [1–6]. Another proposal is that sudden deceleration forces result in the rupture of the small adrenal vessels, causing hemorrhage [2, 3]. A third explanation is that compression of the inferior vena cava from trauma causes an acute rise in intraadrenal venous pressure, re-
sulting in hemorrhage. This latter theory may account for the much higher frequency of right adrenal hemorrhage because the right adrenal vein drains directly into the inferior vena cava and is more susceptible to sudden fluctuations in inferior vena cava pressure [1–6].

A vascular cause may also be responsible for nontraumatic adrenal hemorrhage in pediatric patients. The left adrenal gland is most frequently affected in this population. It has been postulated that thrombosis of the left renal vein, with secondary thrombosis of the adrenal vein, leads to outflow obstruction and adrenal hemorrhage. Sonography has supported this theory by showing echogenic inferior vena cava and renal vein thrombus in some of these patients [7–9]. Right and bilateral adrenal hemorrhages are uncommon in these patients unless the thrombus extends into the inferior vena cava, in which it can obstruct outflow from the right adrenal vein. We postulate a similar mechanism of adrenal hemorrhage from outflow obstruction by inferior vena cava thrombus in our patient.

Evidence for outflow tract obstruction causing adrenal infarction may also be seen in patients undergoing adrenal venous hormone sampling. Catheter placement in the small adrenal veins may result in stasis and venous thrombosis, which has reportedly led to adrenal infarction. As a result, systemic heparinization is administered to avoid adrenal vein thrombosis [10].

In summary, we have discussed a case of posttraumatic adrenal hemorrhage in which an initial CT scan showed an inferior vena cava thrombus with normal-appearing adrenal glands. Approximately 1 hr later, a repeated study showed right adrenal hemorrhage. This case suggests that outflow obstruction from thrombus within the inferior vena cava may cause post-traumatic adrenal hemorrhage.

References