

# The importance of serial neurologic examination and repeat cranial tomography in acute evolving epidural hematoma

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**Computed tomography (CT) has revolutionized the diagnosis and management of head-injured patients, and its increasing availability has led to its liberal use. CT scanning provides excellent anatomic detail of the brain as fixed static images, but the dynamic nature of human physiology means that many injury patterns will evolve in time. We describe an 8-year-old child who had fallen 8 feet from a tree. He had a brief loss of consciousness but a normal neurologic evaluation on arrival to the emergency department (ED). He underwent expedited cranial CT scanning, which revealed no acute brain injury. Two and one half hours later, the patient had a mild depression in consciousness, prompting a second CT scan in the ED, which revealed an acute epidural hematoma. He had acute surgical evacuation of the hematoma and made a full neurologic recovery. This case illustrates that a single early CT examination may at times provide a false sense of security and underscores the importance of serial neurologic examinations.**

## INTRODUCTION

Computed tomography (CT) is a valuable investigation in the evaluation of acute head injury (1–3). The technology provides excellent anatomic detail of the brain and usually detects acute injury reliably. CT scanning provides static images in time, and thus physicians caring for head-injured patients must realize that early CT may reveal no injury or minimal nonoperative findings. We highlight the dangerous pitfall of an ultra-early CT scan, which can fail to detect an acute evolving epidural hematoma. In the following case, an 8-year-old boy who had fallen from an 8-foot tree suffered a brief loss of consciousness. On arrival to the emergency department (ED) he had a normal neurologic examination. He underwent expedited cranial CT scanning, which revealed no acute brain injury. Within 2 1/2 hours, he had a slight decline in mental status, prompting a second CT scan from the ED. This second scan showed an acute epidural hematoma. The patient underwent immediate surgical evacuation and made a full neurologic recovery. This case emphasizes the importance of serial neurologic examinations and the acute role of repeat CT scanning.

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## CASE

The patient was an 8-year-old male whose father carried him into the ED. The child had sustained an 8-foot fall from a ladder at about 10:50 AM. The child landed on his head and suffered a loss of consciousness for less than a few minutes. When the child regained consciousness, he began to cry. He then calmed after being placed in the family car. Enroute to the hospital, the child conversed normally and appeared to be well, according to the parents. Approximately 15 minutes after the accident, the patient began to complain of a headache with slight nausea and had an episode of emesis just outside the hospital entrance.

The boy arrived at 11:08 AM and was crying but oriented, had spontaneous eye opening, moved all extremities to command and had a Glasgow Coma Score (GCS) of 15. The patient's vital signs were as follows: blood pressure, 150/100 mm Hg; heart rate, 88 beats/minute; respiratory rate, 24/minute; temperature, 35.9°C. The child complained only of a headache without neck pain and pointed repeatedly to the left parietal area, where a small abrasion and ecchymosis were visualized. He had no pupillary abnormalities and no clinical evidence of a basilar skull fracture. The rest of the head and neck examination was normal, and the patient had normal chest, cardiac, and abdominal examinations. The child was nontender on examination of his spine but was placed in spinal precautions. Neurologically, examination results of the cranial, motor, sensory, and cerebellar nerves were normal. The patient was placed on a cardiac monitor, revealing normal sinus rhythm. An intravenous (IV) line, was started; bedside hematocrit was 37%, and pulse oximetry was 100% saturation at room air.

Thirty minutes post-injury, the patient was ready for cranial CT scanning and required sedation for mild agitation. Morphine (1 mg IV) was given, and the patient was transported to the helical CT scanner (Picker 2000S Spiral Scanner) at 11:24 AM. At the CT scanner, the patient required another dose of morphine (0.5 mg IV) for sedation. The study was completed at 11:39 AM, and the CT scan revealed no significant injury (Fig. 1A). His neurologic status remained unchanged, and radiographs of the cervical spine, chest, and pelvis were all negative. The second hematocrit was stable at 37%.

At 1:00 PM, the patient was noted to have a depressed level of consciousness with a GCS of 11. The patient was given 0.2 mg naloxone intravenously with re-evaluation showing a GCS of 14 with eye opening on verbal command. Because of this neurologic change, the patient underwent a second ED CT scan, which was completed at 2:08 PM. This CT revealed a left temporal epidural hematoma measuring 2 × 4 × 6 cm (Figs. 1C, 1D) and occipital contusions. Retrospective review of the initial CT scan showed an extremely small, undisplaced fracture in the left temporoparietal

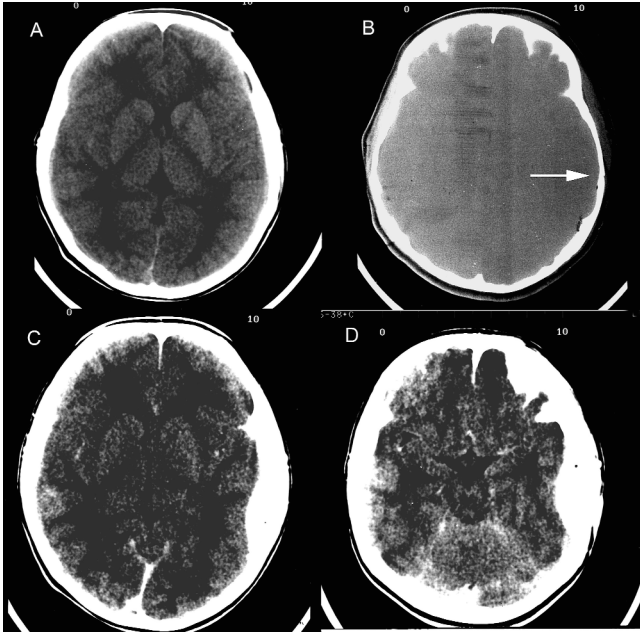


FIG. 1 A, Initial CT scan showing no significant intracerebral injury B, Bone window of initial scan showing a very small fracture in the left temporoparietal area. C, D, Repeat cranial tomography two and a half hours later revealing a moderately sized epidural hematoma and occipital contusions. There is no acute edema or midline shift.

area present on only one axial bone view (Fig. 1B). The patient was immediately taken to the operating room for evacuation of the epidural hematoma. Intraoperative inspection demonstrated a small, nondisplaced, left temporoparietal fracture and bleeding from an arterial vessel was noted and repaired. Postoperatively, the patient did well and there were no neurologic deficits at 3-month follow-up.

## DISCUSSION

Epidural hematoma is an infrequent injury in head trauma making up only about 3% of hospitalized head injured patients (4, 5) with delayed epidural hematomas comprising approximately 8 to 10% of all epidural hematomas (6–8). The diagnosis requires the absence of an epidural hematoma on an initial CT scan following the injury (9). A second scan that is often performed in-hospital or postoperatively is diagnostic of the delayed epidural hematoma. Most delayed epidural hematomas occur many hours after the initial injury, and these epidural hematomas are easily fatal or can be complicated with high neurologic morbidity if not appreciated in a timely manner. To our knowledge, our case, which occurred within 2 1/2 hours, is the first report of an initially neurologically intact patient requiring two CT scans for diagnosis during a single ED visit.

Improving image quality and speed and increased availability of CT scanning has helped make this technology more commonly used in trauma patients. The liberal use of the CT scan in head trauma has led to a decrease in the morbidity and mortality of many head-injured patients. One study showed a decrease in mortality from 29% to 12% after the advent of the CT and an improvement in outcome to good recovery from 39% to 68% (10). Nevertheless, there are limitations of this technology.

The pathophysiology of an epidural hematoma, as this case demonstrates, exposes the limitations, and potential pitfalls inherent in this diagnostic tool. Finding an epidural hematoma is dependent on the rate and type of bleed, extent of cerebral injury, loca-

tion, and the time of the CT scan post-trauma (11) and thus an ultra-early scan may miss an evolving bleed entirely (12).

Appreciation of comorbid risk factors and diligent serial neurologic assessment allow for anticipation of evolving epidural hematomas. A meningeal artery or branch laceration with an associated overlying skull fracture is a frequent source of delayed epidural bleeding (13). However, venous bleeding is also associated with delayed and chronic hematomas by nature of the low pressure and slow rate of bleeding (13). Other causes of low-tension bleeds are from small dural lesions, diploic source, or diffuse cerebral contusion sites (9, 13). Hypotensive shock may be protective initially in this injury until eventual hemodynamic resuscitation leads to reperfusion (14). This has the undesired effect of delaying the accumulation of the hematoma that may take hours. Depending on the rate of arterial or venous bleeding, signs of mass effect may take minutes to days. Another mechanism thought to contribute to delayed hematoma formation is brain edema (13). When brain edema is managed with hyperventilation and mannitol, it can release any tamponade effect that is present. Disseminated intravascular coagulation (15), ultra-low intracranial pressure, cerebrospinal fluid leak, contralateral hematomas, and mass lesions are also abnormalities thought to increase the risk of delayed hemorrhage (9). Other, later, causes of bleeding include post-traumatic pseudoaneurysms and arteriovenous fistulas (16). In an extensive review, Milo et al. found that a skull fracture under which the delayed hematoma accumulates was the rule (9). Patients with skull fractures are at high risk and warrant liberal CT scanning for changes in neurologic status. Indeed, on re-review, our patient had a small undisplaced fracture in the area of his delayed epidural hematoma, and a subtle change in mentation prompted his second ED CT scan. Our patient was at high risk for a skull fracture because of the mechanism of injury (fall) along with focal contusion and pain.

This period between neurologic symptoms versus signs is likely the onset of clinically significant mass effect of the epidural hematoma. Neurologic sequelae increase with depressed mental status or abnormal examination at time of the diagnosis of the epidural hematoma (17, 18). This is a short window of opportunity in which a decision to obtain a repeat cranial CT is critical, and close serial neurologic examinations will help determine which patients will benefit from repeat CT scanning. Concomitantly, it is important to consider reversible causes for decreased mentation including hypoxia, hypoglycemia, hypotension, and drug influences such as opiates, as was partially the case in our patient. The choice of sedative can be important, as demonstrated in our case. Opiates and benzodiazepines are acutely reversible and are available in short-acting forms. These medications allow us to differentiate medication-induced sedation from acute neurologic decline. Other common forms of sedation such as chloral hydrate, do not have these important properties.

## CONCLUSIONS

Early presentation post-injury with expedited computed tomography can reveal a normal CT that unfortunately provides a false sense of security. Finding an epidural hematoma is contingent on when the head scanned, because the bleeding may arise hours and even days after the initial injury despite an initial negative CT (19). Early repeat cranial tomography will be necessary for detection (20), and at times this will require a second CT scan ordered during the same ED visit. Close neurologic assessment is paramount (21) and will help identify high-risk cases. In addition, knowledge of predisposing conditions for delayed hematoma formation will help us make maximal use of this valuable technology.

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