Cranial Computed Tomography in the Abused Child with Head Injury

Cranial computed tomographic (CT) findings are described in 37 children with head injuries resulting from physical abuse. CT findings included subarachnoid hemorrhage (27 patients), cerebral edema (24), cerebral hemorrhage (11), and subdural hematoma (nine). Intravenous contrast material was administered in 10 children in whom there were neurologic symptoms or signs but no history or physical signs of trauma or abuse. In five of these children, increased vascularity was seen in areas that later showed infarction. Eight children were studied by both CT and sonography. Sonography detected 50% fewer abnormalities than did CT. It was found that children with skull fracture had the same range of injuries as children without fracture, and they had a higher incidence of subarachnoid hemorrhage. In acutely traumatized children, cranial CT should be the method of choice to provide the most accurate diagnosis and documentation of injury. Skull radiographs should also be obtained because they sometimes show fractures not recognized by CT.

Since the early descriptions by Caffey [1] and Silverman [2], the medical community has become increasingly aware of the manifestations of child abuse. Before the availability of computed tomography (CT), the diagnosis and documentation of intracranial injury depended on arteriography, surgery, and autopsy studies. In the past decade CT has become the primary diagnostic method for the evaluation of serious craniocerebral trauma. Previous reports of cranial CT of abused children have demonstrated a wide spectrum of abnormalities, but the examinations were performed primarily on early-generation scanners [3-5]. We have studied 37 abused children by cranial CT. In this selected group of patients we found a 95% incidence of intracranial abnormalities on scans obtained shortly after injury.

Materials and Methods

In the 6 year period from March 1978 through February 1984 there were 1439 reported cases of physical abuse in infants and children seen at CHMC. Ninety (6%) of these had serious clinical head injury. Cranial CT was performed in 40 of these 90 children. One CT scan was nondiagnostic because of motion artifact and two patients had only follow-up scans. The other 37 patients who were scanned at the time of presentation are the subject of this study. The scans were obtained for clinical indications and not for the purpose of retrospective review.

Nine patients were examined on an EMI 5005 and 28 scans were obtained on a GE 8800. CT scans, plain radiographs, other imaging studies, and hospital records were reviewed.

Results

The age range in the 37 children was 1 month to 8 years. Most patients (95%) were less than 1 year of age. Only two patients were older than 2 years. The mean age was 9 months. Twenty-two (59%) were boys and 15 (41%) were girls.
five patients demonstrated intracranial abnormalities. Only two were normal. All patients under 2 years had abnormal scans.

Subarachnoid Hemorrhage (SAH)
SAH occurred in 27 patients (73%). Eight of these patients had lumbar punctures and all had bloody cerebrospinal fluid consistent with SAH (fig. 1).

Cerebral Edema
Cerebral edema occurred in 24 patients (65%). Eighteen patients had focal areas of edema or unilateral hemispheric edema (fig. 1). Six patients had a generalized pattern of edema. In three of these patients the gray matter had lower attenuation than the white matter, a pattern we term the “reversal sign.” In these patients the thalamus, brainstem, and cerebellum were relatively spared, maintaining nearly normal density (figs. 2A and 2B).

Cerebral Hemorrhage
Eleven patients (30%) had cerebral hemorrhage, including three with brain lacerations. Five patients had frontal hemorrhage, three parietal, one temporal, one occipital, and one had multifocal areas of hemorrhage. All three patients with brain lacerations had fractures of the overlying calvaria. In two of the three patients, hemorrhage extended into the ventricles; these were the only two patients with intraventricular hemorrhage (fig. 3).

Subdural Hematoma
Nine patients (24%) had subdural hematomas. Five were acute, three were low-attenuation chronic collections, and one was both acute and chronic.

Discussion

Five patients had intravenous contrast medium on initial scans. Although not routine, contrast-enhanced scans were sometimes obtained when there were neurologic symptoms or signs but no history or physical signs of trauma or abuse. Five of these patients had increased vascularity in areas that later showed infarction (fig. 4).

Follow-up Examinations
Twenty-four patients had follow-up CT examinations 2 weeks to several years after head injury. Six patients developed mild to moderate generalized atrophy. Seventeen patients developed focal unilateral or asymmetric areas of cerebral atrophy (fig. 5). Only one patient, a 17-month-old with an acute anterior interhemisphere subdural hematoma, had a normal follow-up scan. Three patients developed chronic subdural collections. None of our patients developed obstructive hydrocephalus.

Cranial Sonography
Eight patients had cranial sonographic examinations within 5 days of the initial CT scan. Sonography detected 50% fewer (10/20) abnormalities than did CT (table 1). Follow-up sonography was useful in five cases in assessing ventricular dilatation and atrophy.

Skull Fractures
Eleven patients (30%) had skull fractures, none of which were depressed. Except for epidural hematoma and brain laceration, patients without fracture suffered the same gamut of injury as those with fracture, and they actually had a higher incidence of SAH (85% vs. 45%) (table 2).

Associated Skeletal Injuries
Radiographic skeletal surveys showed a variety of fractures in addition to the 11 skull fractures. Fractures of the ribs occurred in six patients, clavicle in four, femur in three, tibia in two, radius and ulna in two, humerus in two, and classical multiple metaphyseal corner fractures in only two.

Outcome
Four patients died. Autopsy examinations were performed by the coroner in all four. Eleven patients had severe impairments, some living in vegetative states in institutions. Nine had minor impairments including mild weakness, visual problems, and seizure disorders. To date, seven patients have no detectable abnormality. Insufficient data are available in six patients.

Hypervascularity
Ten patients received intravenous contrast medium on initial scans. Although not routine, contrast-enhanced scans were sometimes obtained when there were neurologic symptoms or signs but no history or physical signs of trauma or abuse. Five of these patients had increased vascularity in areas that later showed infarction (fig. 4).

Discussion
Child abuse is an important cause of morbidity and mortality. About 1.3 million cases of child abuse are reported each
year in the United States [6]. Of these, 45% (585,000) are cases of physical abuse. Most victims of child abuse with head injury are under 2 years of age. It is also a well known cause of mental retardation and neurologic impairment.

All of our patients under 2 years of age had abnormal cranial CT scans. The incidence of intracranial abnormalities by CT ranges from 7% of a large unselected group to 96% of a selected group of abused children [3, 7]. In our patients we found a very high incidence of intracranial abnormalities on both initial scans (95%) and follow-up scans (96%). The disparity in previous reports reflects differences in patient selection and referral patterns. Our patient group was also highly selected in that it consisted primarily of young infants who presented with neurologic signs or symptoms. Also, we included only officially reported cases of abuse and excluded suspected cases. The high incidence of intracranial abnormalities in child abuse is also attributed to the increased susceptibility of the infant brain to injury resulting from head trauma [8]. In addition, the higher resolution of current-generation scanners allows for improved detection of intracranial

Fig. 2.—4-month-old boy. Reversal sign. Generalized low attenuation but relative sparing of thalami (A) and brainstem and cerebellum (B). Follow-up examinations at 2 weeks (C) and 1 year (D) after injury. Progressive ventricular dilatation from severe generalized cortical injury. Brain damage from abuse is irreversible.

Fig. 3.—2-month-old boy 2 days after injury. Left parietal brain laceration (L) with both SAH and intraventricular hemorrhage, as well as herniation of brain through diastatic fracture (arrows). Diffuse brain edema.
The pathophysiology of brain injury in infants differs from that in adults. The softer calvaria, open sutures, and the degree of myelinization are some of the factors that influence the type of injuries that occur with craniocerebral trauma. The mechanism of injury in child abuse varies. Children with fractures or obvious scalp bruises have received impact injuries. Nonimpact injuries resulting from severe “shaking” or “whiplash” also occur, especially when there are no external signs of injury [9, 10]. Both impact and nonimpact trauma may cause intracranial injury.

After SAH (73%), cerebral edema (65%) was the most common abnormality found in our patients. The extent and severity of the cerebral edema depends on the interval from the traumatic event. If scanned early, the findings may be minimal or even absent. Scans obtained days later often show edema and increased mass effect (fig. 6). Patients with a generalized pattern of edema may have suffered diffuse brain damage secondary to hypoxia and/or hypotension. The vascular control mechanisms and blood-brain barrier may be altered by acute brain trauma, which then contributes to the development of edema, either focal or generalized [8, 11].

Three of our patients had what we termed the “reversal sign” where the gray matter had lower attenuation than the white matter. In these patients the thalamus, brainstem, and cerebellum were relatively spared. These findings have also been seen in infants with asphyxia or head trauma not resulting from abuse [12-14]. In our experience, all patients with the reversal sign have demonstrated severe brain damage on follow-up scans (figs. 2C and 2D). We have not encountered an instance in which a patient manifesting the reversal sign did not have irreversible brain damage.

Among the 11 patients with cerebral hemorrhage were three who suffered lacerations of the brain. All three had associated fractures of the overlying calvaria. Such linear hemorrhages may represent severe forms of what pathologists term contusional tears, which are seen in young infants with impact injuries with or without skull fracture [15]. Often these tears are microscopic and are located in the white matter near the gray-white junction. However, occasionally
they extend through the cortex and ventricular wall, as in two of our cases. One of these patients had a dural tear with associated herniation of brain parenchyma (fig. 3).

Interhemispheric hyperdensity in children may have several causes including normal falx, SAH, interhemispheric subdural hematomas, and diffuse cerebral edema. Zimmerman et al. [3] found a 58% incidence of posterior acute interhemispheric subdural hematomas (AIHSH) in 26 abused children studied by CT. The diagnosis was based on finding a thick, asymmetric area of increased attenuation about the posterior falx cerebri. In another article, Zimmerman et al. [16] noted that AIHSHs are often subtle and more difficult to diagnose in children. Useful criteria for diagnosing AIHSH are a flat medial border and a convex lateral border and/or extension over one tentorial surface. Some asymmetry of the posterior falx cerebri may be normal and, by itself, does not necessarily represent AIHSH.

The falx cerebri appears prominent when there is diffuse cerebral edema. Therefore, in the presence of severe edema, interhemispheric hyperdensity alone is not a reliable sign of SAH. Also, familiarity with normal variations in the appearance of the falx cerebri and the interhemispheric fissure in children is useful in the diagnosis of SAH.

We found it difficult to differentiate AIHSH from SAH and/or intrafalcine hemorrhage. We had one patient with a definite AIHSH. Three others had asymmetric blood collections along the posterior falx, and one of these patients died 2 days after injury (fig. 7). An autopsy revealed both subarachnoid and subdural blood but no localized interhemispheric subdural hematoma. In all four patients who died, autopsies demonstrated thin layers of subarachnoid blood and cerebral necrosis. All four had subdural blood as well, but only one had a subdural clot (5 mm thick) located over the convexity.

There is no doubt that AIHSH occurs in child abuse, but we did not find it as often as previously stated. We believe it is likely that increased subdural interhemispheric density may represent a combination of SAH and subdural hemorrhage and is often accentuated by adjacent brain edema.

All but one of 24 patients who had follow-up scans showed persistent abnormalities by CT. In general, the degree of brain damage demonstrated on CT correlates with the severity of neurologic impairment. Most patients had definite signs of parenchymal brain damage. Six had mild to moderate generalized atrophy without evidence of focal brain damage.

It must also be remembered that acute brain injury may be superimposed on abnormalities that have occurred from previous episodes of abuse that were unrecognized. This was evident in one of our patients with concurrent chronic and acute subdural hematomas.

Sonography detected only one-half of the abnormalities seen on CT and was less specific in diagnosis. Sonography is limited by the size of the fontanelle and, therefore, in many cases is impractical or ineffectual in the second year of life. Also peripheral lesions are more difficult to detect by sonography. Therefore, we believe that CT should be the method of choice in acutely traumatized children to provide the most accurate diagnosis and documentation of cranial injury. As long as the anterior fontanelle remains open, sonography may be used for follow-up examinations to assess ventricular dilatation and brain atrophy.

As noted in several previous studies, serious brain injury often occurs in the absence of a skull fracture, particularly in the "whiplash-shaken infant syndrome" [9]. Obviously, serious brain injury also occurs in the presence of skull fractures. Skull radiographs should be included in the skeletal survey for child abuse, because they sometimes show fractures not recognized on CT scans and because skull fracture may be the only clue to the diagnosis of abuse [17].

In many cases of child abuse there is no doubt that injury has been inflicted. In some cases the diagnosis is not clear

**TABLE 1: Comparison of CT and Sonographic Findings in Eight Abused Children with Head Injury**

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>No. Detected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular dilatation (mild)</td>
<td>3</td>
</tr>
<tr>
<td>Intraventricular hemorrhage</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
<td>3</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>7</td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral edema</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
</tr>
<tr>
<td>Sonography</td>
<td>10</td>
</tr>
</tbody>
</table>

* Widened interhemispheric fissure.

**TABLE 2: Comparison of Cerebral CT Findings in Abused Children with and without Fracture**

<table>
<thead>
<tr>
<th>Finding</th>
<th>With Fracture (n = 11)</th>
<th>Without Fracture (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidural hematoma</td>
<td>1 (9)</td>
<td>0</td>
</tr>
<tr>
<td>Acute subdural hematoma</td>
<td>1 (9)</td>
<td>5 (19)</td>
</tr>
<tr>
<td>Chronic subdural hematoma</td>
<td>2 (18)</td>
<td>1 (4)</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>5 (45)</td>
<td>22 (85)</td>
</tr>
<tr>
<td>Cerebral edema</td>
<td>8 (73)</td>
<td>16 (62)</td>
</tr>
<tr>
<td>Cerebral hemorrhage</td>
<td>5 (45)</td>
<td>7 (27)</td>
</tr>
<tr>
<td>Brain laceration</td>
<td>3 (27)</td>
<td>0</td>
</tr>
</tbody>
</table>
Fig. 6.—10-month-old boy. A, Shortly after injury. Low attenuation effacing left occipital horn. B, 5 days later. Pronounced left hemispheric edema with occipital predominance.

Fig. 7.—4-month-old boy shortly after injury. Increased attenuation along posterior falx cerebri proven at autopsy 2 days later to represent subarachnoid and subdural blood surrounding brain, but no localized interhemispheric subdural clot. Generalized low attenuation of brain substance is worse on left.

and the radiologist may be the first physician to suggest the diagnosis. Although a wide spectrum of injury occurs, the most common abnormalities we encountered were SAH and cerebral edema. Though not specific this constellation of findings in a young infant should suggest the possibility of child abuse even if there are no other signs of trauma.

ACKNOWLEDGMENTS

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REFERENCES

1. Caffey J. Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. AJR 1946;56:163–173
2. Silverman FN. The roentgen manifestations of unrecognized skeletal trauma in infants. AJR 1953;69:413–427