Computerized tomography of contusional clefts in the white matter in infants

Report of two cases

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Blunt trauma to the brain in infancy may cause lacerations in the soft, incompletely myelinated white matter of the centrum semiovale. This characteristic entity was first recognized in autopsy material by Lindenberg, et al. Later, Lindenberg and Freytag described this clinicopathological entity in 16 infants with head injury.

The lesion has rarely been detected during life, but it can now be clearly visualized by computerized tomography (CT). Two instances, each the result of deliberate trauma, are presented. It appears that the clefts may become less apparent with the passage of time.

Case Reports

Case 1

At 2 months of age, this baby boy was brought to the hospital because of irritability and seizures. His birth had been uncomplicated, but his weight was low (2240 gm) for birth at term. On examination, he was pale and irritable, with opisthotonos and decerebrate posturing. He had generalized seizures and exhibited a left gaze preference. There were ecchymoses of the scalp and body. Radiographs revealed bilateral fractures of the skull. Bloody fluid was obtained from both sides by subdural taps. Computerized tomography (Fig. 1 upper) revealed bilateral fluid-containing clefts in the frontal white matter in addition to the subdural hematomas.

The subdural collections were treated with daily taps. The fluid persisted for 3 weeks. Finally, bilateral frontal burr holes were made. The subdural membranes were very thin, and the fluid was xanthochromic. The patient was discharged without focal neurological deficits.

Denver Developmental Test has shown a slight psychomotor delay. Follow-up CT 6 months after discharge was of interest because the left frontal cleft was no longer apparent (Fig. 1 lower). The right cleft extended from the subcortical white matter toward but not quite to the anterior horn of the right lateral ventricle. Both lateral ventricles were dilated, slightly more on the left side than the right.

The patient has continued to do well, and he has no signs of increased intracranial pressure.

Case 2

This baby boy was admitted to the hospital at 4½ months of age for the evaluation of multiple fractures and failure to thrive. The child weighed 3000 gm at birth after a pregnancy complicated by anemia and
Traumatic white matter clefts in infants

Fig. 1. Case 1. Upper: Computerized tomography scans on admission. In addition to the subdural fluid, there are bilateral clefts in the frontal white matter. Lower: After 9 months, the subdural fluid is gone. The cleft on the left side is not visible, and the other cleft and the lateral ventricles are enlarged.

Discussion

Toxicemia. Jaundice in the neonatal period required phototherapy. At 1 month of age, he was admitted to another hospital because of vomiting and weight loss. At that time, a fracture of the right tibia was identified. Films of the lungs, on subsequent review, revealed multiple rib fractures. He was referred to our care.

On admission, skeletal evaluation showed healing fractures of the third through seventh ribs on the right and sixth through eighth on the left. Healing of the tibial fracture was appreciated. Computerized tomography for evaluation of separation of cranial sutures revealed tears of the frontal white matter (Fig. 2). The child was transferred back to the referring hospital pending resolution of the difficult situation in the child's family.

Although only nine had a history of trauma, all had signs of bodily violence. Seven of the 16 infants had linear skull fractures, and 12 had an associated subdural hemorrhage. Fourteen of the infants died between the ages of 9 days and 4 months. The baby

Fig. 2. Case 2. Computerized tomography showing bilateral clefts in the frontal white matter.

Lindenberg and Freytag found subcortical white matter tears in the brains of 16 infants at autopsy.
who died at 12 months sustained his injury at the age of 5 months. No tears occurred after that age.

Macroscopically, the lesions consist of fairly symmetrical clefts in the subcortical white matter of the orbital frontal and temporal lobes. Fresh clefts may contain some blood. A second injury can also cause an accumulation of fresh blood in an old cleft. Occasionally, the tear extends to the cortical surface, or to the wall of the lateral ventricle. Tears have also been found in the parietal lobe, but not in the brain stem or cerebellar white matter.

Lindenberg and Freytag described three histological phases. In the first, which is found in infants who died soon after injury, generally prior to admission to the hospital, the smooth-walled white matter tear contains some blood but insufficient to be regarded as a hematoma. The nuclei of the glial cells bordering the surface of the defect may show pyknotic changes. The second phase starts at about 36 hours and lasts about 3 weeks. It is characterized by a marked astrocytic proliferation along the margin of the tear. In the third phase, a wall of gliotic tissue is formed around the white matter cleft. These authors ascribed the splits to the pliancy of the infantile skull, the soft consistency of the unmyelinated brain, the smoothness of the fossae, and the shallowness of the subarachnoid space; however, they pointed out that the tear may occur adjacent to or removed from the site of actual impact. Since the periventricular lesions may be cystic and traversed by glial septa or strands, they might be mistaken for polycystic encephalomalacia. The clinical sequela of white matter tears are not known.

In Case 1, the lesion on one side collapsed until it was not detectable in the follow-up CT scan. Diminution in the size of neonatally acquired cystic lesions of the cerebrum has not been reported. However, a similar collapse of cystic lesions induced in hemispherical white matter was found in neonatal kittens that were allowed to survive to young adulthood.

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References


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