CT demonstration of fat-embolism-associated hemorrhage in the anterior commissure.

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Abbreviated Reports

CT Demonstration of Fat-Embolism-Associated Hemorrhage in the Anterior Commissure

The anterior commissure is routinely visible on MR images [1] but not on CT scans. We present a case in which the posterior crus of the anterior commissure was visible as a high-density structure because of hemorrhage related to fat embolism.

Case Report

Upon admission, our patient, a 47-year-old alcoholic man who had been struck by a motor vehicle, was comatose. Radiographs showed bilateral fibular fractures. CT findings included increased density consistent with blood in the posterior crus of the anterior commissure (Fig. 1) and in the globus pallidus bilaterally. The prothrombin and partial thromboplastin times were elevated, and the platelet count and fibrinogen levels were diminished. After admission, the prothrombin and partial thromboplastin times returned to normal, but 1 week after admission, the fibrinogen level was elevated and fibrin split products were present. The patient died 11 days after admission. At autopsy, hematomas were found in the left frontal lobe and the splenium of the corpus callosum. Bilateral temporal contusions were present. The brainstem was mildly edematous. No gross hemorrhage was visible in the globi pallidi or the anterior commissure.

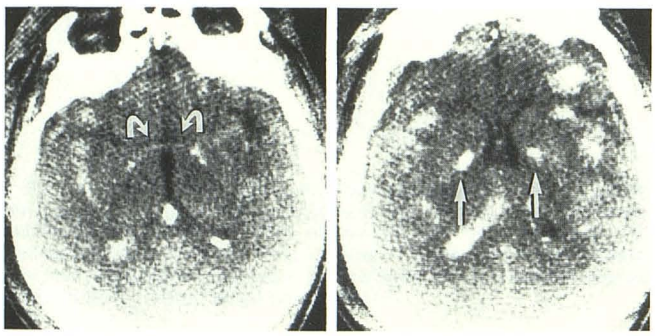

Fig. 1.—CT scans obtained at time of admission.

***A*, CT scan shows curvilinear increased density crossing midline corresponding to posterior crus of anterior commissure (*curved arrows*). Subarachnoid and intraventricular blood is visible.**

***B*, CT scan shows presence of blood in globus pallidus bilaterally (*arrows*). Left frontal hematoma and subarachnoid and intraventricular blood are visible also.**

Microscopically, fat globules were visible in several vessels throughout the anterior commissure and the globi pallidi. Microscopic perivascular hemorrhage was associated conspicuously with the fat embolism in the anterior commissure and the globi pallidi. No fibrin deposits were noted. No macroscopic or microscopic evidence of calcification in the globi pallidi was seen. Evidence of fat embolism was sought elsewhere in the brain, but none was found. No pulmonary fat emboli were seen, though fibrin deposits in the pulmonary vasculature were present.

Discussion

The anterior commissure crosses the midline in the anterior wall of the third ventricle [1], with its posterior crus continuing through the inferior and lateral portions of the globi pallidi to the middle and inferior temporal gyri [1]. Although postmortem evidence of diffuse axonal injury in the anterior commissure has been described [2, 3], it seldom is referred to in the literature on head trauma. In our case, the globi pallidi and the posterior crura of the anterior commissure showed increased density on CT because of the presence of microscopic hemorrhage. Previous reports of cerebral fat embolism have described areas of low attenuation [4], abnormal gyral contrast enhancement [5], generalized cerebral edema [5, 6], and absence of abnormalities on CT [5] but not increased attenuation without use of IV contrast material.

The pathogenesis of fat embolism is not clear. Originally, it was hypothesized that marrow fat entered the venous system at the site of injury and reached the brain after passing through the pulmonary circulation or cardiac shunts [5–8]. In keeping with this hypothesis is the frequent association of fat embolism with fractures and injuries to subcutaneous adipose tissue (both of which were present in our patient) and the occasional appearance [7] of marrow elements with fat emboli (not seen in our patient). A second set of hypotheses has been advanced to explain the occurrence of fat embolism in patients without fractures. These include the suggestions that posttraumatic changes in the composition of the blood [5–8] or vascular stasis [8] allow for coalescence of blood lipids. In our case, the fat embolism

may have resulted from vascular stasis brought on by shearing injury to the anterior commissure.

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CT Findings in Cystic Intramedullary Oligodendroglioma

Various cases of intramedullary tumors with a cystic component, which are basically astrocytomas and ependymomas, have been described [1–6]. However, no description of an intramedullary cystic oligodendroglioma has been reported. We present such a case, which was studied by metrizamide-enhanced CT.

Case Report

A 20-year-old woman had noticed an overall loss of strength in her right upper extremity and resultant thinning over a 2-year period. During the 6 months before admission, she experienced worsening symptoms and thinning of her right lower extremity. Examination showed an overall loss of strength in the right upper extremity, with muscular atrophy from the deltoid muscle to the interosseous muscles. Atrophy of the quadriceps and gemellus muscles was seen in the right lower extremity. Reflexes were absent in the right upper extremity. No alteration in the vibratory or positional sensitivity was observed, although the abdominal cutaneous response was absent. A CT examination was performed 2 hr after an intrathecal injection of metrizamide. Sections 10 mm thick were made consecutively from the cranial base as far as T2. From C3 downward, enlargement of the cord with obliteration of the subarachnoid space as far as C7 was observed. Contrast material filled a cavity with irregular contours and localized peripherally in the interior of the cord from the inferior portion of C4 as far as C7 (Fig. 1).

Laminectomy was carried out from C7 to C4. The neoplasm seemed to occupy the right half of the intradural space. Overall, the tumor could be readily differentiated from the medulla. The lesion was removed completely by microsurgery. Microscopic sections showed areas of nervous tissue, formed by white matter that appeared to be invaded by tumor cells with clearly delimited contours, clear cytoplasm, and hyperchromatic central nucleus corresponding to oligodendrocytes. Reactive astrocytosis was detected throughout. These results established a diagnosis of oligodendroglioma with areas of astrocytic proliferation.

Discussion

As defined by Barnett and Newcastle [7], syringomyelia is characterized by cavitation of the spinal cord and gliosis; one form is marked by a cavity in the spinal cord that is delimited partly by tumor cells. This definition helps clarify the different designations in the literature that refer to a cystic tumor or to a tumor associated with syringomyelia, which are similar clinical entities.

Syringomyelia can be distinguished from cystic intramedullary tumors [6] by three characteristics: (1) It appears at an earlier age (mean 24 vs 34 years); (2) the symptoms last longer (10 vs 5 years);

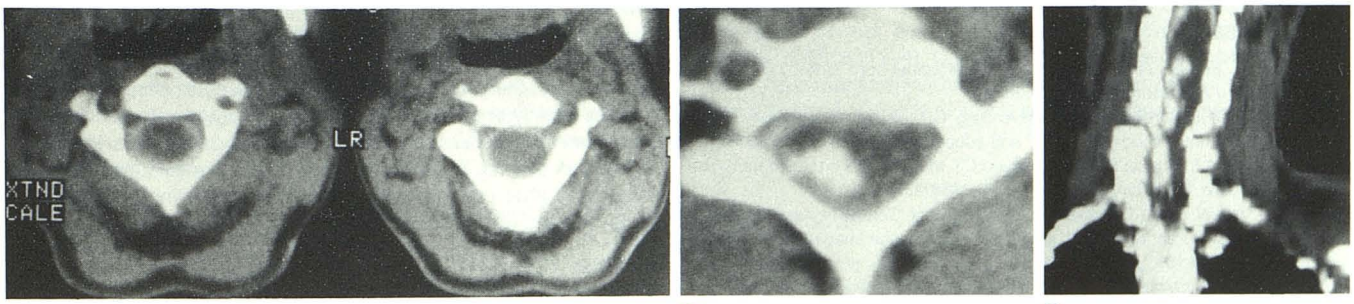

Fig. 1.—Contrast-enhanced CT scans of cystic intramedullary oligodendroglioma.
***A*, Scan at C3 level shows enlargement of cord with partial obliteration of subarachnoid space. No cyst is seen.**
***B*, Scan at C4 and C5 levels shows metrizamide filling of a cavity with irregular contours and peripheral localization in cord.**
***C*, Reformated coronal scan at C3–C7 level shows enlargement of spinal cord from C3 to C7 with cavity from C4 to C7.**