SUMMARY

Two cases of laminar fractures of the cervical vertebrae, occurring near the intervertebral foramina and complicated by root compression were reported. The close resemblance of the clinical picture to that of a ruptured cervical disk was emphasized. Diagnosis, differential diagnosis and treatment were outlined.

REFERENCES


TWO CASES OF ACUTE SUBDURAL HYGROMA SIMULATING MASSIVE INTRACRANIAL HEMORRHAGE

EVERETT H. DICKINSON, M.D., AND BERNARD H. PASTOR, M.D.

Neurosurgical Service, Philadelphia General Hospital, Philadelphia, Pennsylvania

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The occurrence of subdural collections of fluid was mentioned by Cushing2 in 1908. In his recent review, Wycis9 recorded a total of 99 cases of subdural hygroma, including 7 of his own. Naffziger8 discussed subdural collections of fluid, and observed that they could occur in the first few hours after injury as well as in the later days. Most of the cases in the literature have been of the more chronic variety. Cohen1 recorded an interesting case in which operation was performed 6 weeks after the initial injury, and during the succeeding 4 months, the subdural space was emptied 9 times, yielding a total volume of 2,300 cc. of fluid. Such recurrences appear to be rare. Scott9 recorded 8 cases in which prolonged stupor, varying from 23 to 28 days, was the outstanding symptom, and McConnell7 reported 6 cases with post-traumatic amnesia of 6 to 66 days' duration.

Of the acute variety which appears to occur less frequently, Walsh and Shelden11 and Scott10 each reported a similar case in which rapid onset of symptoms following head injury led to a pre-operative diagnosis of epidural hemorrhage. A total of 5 such acute cases were reported by Lanigan4 and Haynes.3 In all of these, clear fluid was found in the subdural space at operation. In a more extensive report, McConnell7 recorded 7 cases in which the patient was deteriorating rapidly, usually within 48 hours of injury, in which subdural collections of fluid were found, as well as 92 cases in which the course was more chronic. All of his cases, he felt, were clinically indistinguishable from massive intracranial hemorrhage as it is commonly observed. Da Costa and Adson3 had a considerable series of 19 cases, and they pointed out that symptoms may come on immediately, or may be delayed a period varying from hours to weeks. Three of Wycis12 7 cases were of the acute variety.

We have recently observed, within a short period of time, 2 cases in which, following a head injury, there was more or less acute onset of symptoms suggesting massive intracranial hemorrhage. In both cases, exploration revealed large collections of xanthochromic fluid in the subdural space.

CASE REPORTS

Case 1. H. C., a 66-year-old colored male, was admitted to the service of Dr. S. Gilpin at the Philadelphia General Hospital, at 7:15 P.M. on June 11, 1946. Earlier in the afternoon, the patient had been thrown from a wagon, knocked unconscious, and found lying on the highway by the police. When seen in the ward, he had regained consciousness, but was some-
what confused and disoriented. Bloody spinal fluid was leaking from his right ear. Pupils were equal and reacted to light, and examination of the fundi revealed only vascular changes. The only abnormal neurological findings were a positive Babinski sign on the right, and marked deviation of the tongue to the right. General physical examination was negative except for an apical systolic murmur. His temperature was 101°F., pulse 70, respiration 20, and blood pressure 140/80.

An hour after admission, during a period of about 20 minutes, the patient became rapidly more confused and lapsed into unconsciousness. He then had 3 generalized convulsions at approximately 5-minute intervals. The right pupil became dilated, and reacted sluggishly to light. A left hemiparesis developed. The patient’s respiration became deep, slow, stertorous, and later of the Cheyne-Stokes variety. When operation was decided on at 8:30 p.m. he was deeply comatose. A diagnosis of epidural hemorrhage was made.

Exploratory trephining was done on the right over the middle meningeal artery. No epidural bleeding was found, but the dura, although normal in color, was tense and did not show the usual pulsation. Upon incising the dura, there was a gush of slightly blood-tinged straw-colored fluid under considerable pressure. An estimated 200 cc. of fluid were evacuated, and the intact arachnoid could then be seen lying deep to the dura and pulsating. At this time, the patient awoke and began to struggle and protest loudly. It was necessary to use intravenous sodium pentothal anesthesia to complete the operation. The patient left the operating room conscious and in good condition. Postoperatively his temperature was 100°F., pulse 80/min., respirations 20/min., and blood pressure 130/70. At 12:00 midnight he was fully conscious.

The following day (June 12) his condition was excellent, he was fully conscious, and fairly well oriented. By June 14, he was out of bed in a wheel chair. On June 18, there was still slight nuchal rigidity, but this was attributed to concomitant subarachnoid hemorrhage. Spinal fluid culture was negative. On June 21, the spinal fluid was still bloody. It was xanthochromic on June 23, and the neck was no longer stiff. On July 7, crystal-clear spinal fluid was obtained and studied. There were no abnormal findings. At no time was there any regression in the mental picture, and the patient was discharged asymptomatic on July 4, 1946. When seen in the follow-up clinic a month later, the tongue still deviated markedly to the right.

Comment. This case represents one in which rapidly progressive signs of increasing intracranial pressure led to a diagnosis of epidural hemorrhage. The dramatic recovery immediately after evacuation of a large subdural effusion leaves little room for doubt that it was producing the symptoms.

Case 2. H. R., a 67-year-old colored male, was admitted to the service of Dr. W. McConnell at 11:00 p.m. on July 25, 1946. He had fallen from a 9-foot-high scaffolding at 2:00 p.m., was unconscious for a few minutes, and subsequently behaved in a confused manner all afternoon, and up to the time of admission. When first seen, he was confused, imperfectly oriented, and insistent that he wanted to go home at once. His pulse was 100/min., respirations 20/min., blood pressure 170/100, and temperature normal. There was a soft, tender swelling in the right parietal region, and a scalp abrasion on the occiput. Pupils were normal and reacted to light and accommodation. No papilloedema was noted. General physical examination was essentially negative except for the hypertension, an apical systolic murmur, and a loud "tambour" aortic second sound. Neurological examination at this time was negative. A diagnosis of cerebral concussion and hypertensive cardiovascular disease was made.

The patient was apparently well during the next 48 hours, except for the persistence of mild confusion. He was quiet and alert, and very cooperative. Skull x-rays taken on admission showed no evidence of fracture.

On July 28, at 3:00 a.m., the patient was noted to be having Jacksonian convulsions involving the left arm, leg and face. He appeared to be conscious, but was more confused. The convulsions were almost continuous, but during a momentary cessation of convulsions, there were definite left hemiparesis and left facial weakness. The pulse was 120/min., respiration rapid and shallow with periods of Cheyne-Stokes, and blood pressure 170/100. Lumbar punc-