RESOLVING SUBDURAL COLLECTIONS

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SUBDURAL hematoma is one of the most common neurosurgical conditions. At the same time it is one of the most baffling. It is unpredictable in that the patient may make a remarkable recovery after surgical evacuation; on the other hand the patient may expire in spite of surgical therapy. For this reason it was decided to attempt to learn more about the natural history of patients with subdural collections. Patients with angiographically demonstrated subdural collections who had minimal neurological deficits were selected.

CASE REPORTS

Case 1. N.S., a 51-year-old man, fell and struck his head, sustaining a transient loss of consciousness shortly before admission.

Physical examination revealed a confused patient with a superficial abrasion of the left occiput. The left pupil was larger than the right and reacted sluggishly to light and in accommodation. There was no other neurological deficit. Roentgenograms of the skull did not demonstrate a fracture.

The patient gradually became alert but anisocoria remained. A lumbar puncture on the 10th hospital day disclosed xanthochromic fluid. A left carotid angiogram (Fig. 1) on the 19th hospital day revealed an acute subdural collection. The patient refused surgery. During the next 37 days the patient remained alert but ptosis of the left upper eyelid developed in addition to his anisocoria. In this period cerebral angiograms revealed marked diminution in the size of the subdural collection.

The patient finally consented to operation and on the 57th hospital day left parietal and temporal burr holes were performed. There was a subdural membrane present enclosing a small subdural hydroma.

Case 2. L.R., a 29-year-old male, was well until 2 weeks prior to admission when he first noted the onset of frontal headaches and low-back pain. Upon admission the patient was drowsy. He had a left central facial palsy and slight nuchal rigidity. The left upper extremity was slightly weak, the deep tendon reflexes were increased on the left, and the superficial abdominal reflexes were absent on this side. Lumbar puncture revealed xanthochromic fluid. On the 7th hospital day a left carotid angiogram (Fig. 2) demonstrated an acute subdural collection.

The following day a left parietal burr hole was inserted. A #20 gauge needle was inserted through the dura mater and 0.6 cc. of brownish-red material resembling old blood was aspirated. The benzidine test on this fluid was positive. There was no leakage through the dural puncture. A Gelfoam patty was placed over the dura mater and the wound was closed.

The patient then was followed with serial carotid angiograms. He became alert, coherent and oriented and his neurological deficit disappeared. Eight weeks later there was no evidence of subdural hematoma by angiography.

Case 3. D.C., a middle-aged man, was a known alcoholic of long duration. Upon admission to the hospital he was confused, disoriented and drowsy. No history could be obtained.

Physical and neurological findings were normal. After a bout of delirium tremens he was alert and oriented by the 4th hospital day. A lumbar puncture at this time revealed grossly bloody spinal fluid with a xanthochromic supernatant. During the following days the patient remained alert and exhibited only a "glove-and-stocking" sensory impairment of the limbs.

Ten weeks after admission a right carotid angiogram (Fig. 3) revealed a chronic subdural collection. Six days later a right parietal burr hole was performed and a #20 gauge needle was inserted through the dura mater. Six cc. of liquid, darkish-brown material were aspirated. After checking for leakage a Gelfoam patty was placed over the dura mater and the wound was closed.

The patient’s condition remained unchanged. He was followed with serial angiograms and the subdural hematoma was seen to disappear gradually. Twenty-one weeks after admission a pneumoencephalogram was normal. Twenty-two weeks after admission the burr hole was reopened.

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The skull defect was filled completely with granulation tissue which was contiguous with the dura mater. On opening the dura mater there was a thin (1 mm.), soft, dark-red membrane. This probably represented the inner membrane of a subdural hematoma. No fluid was present within it. Upon opening this membrane the cerebral cortex appeared normal but the surface of the brain was 4–5 mm. from the inner table of the skull. Later cerebral angiograms revealed re-expansion of the brain.

Case 4. H.A., a 47-year-old man, had been an alcoholic for the past 10 years. He was well until the morning of admission when he awoke and experienced difficulty in speaking and weakness of the left-sided extremities causing him to limp.

Physical examination revealed a confused, disoriented, slightly aphasic patient. There was flattening of the right nasolabial fold, and grasp and function of the biceps were decreased on the left. Lumbar puncture revealed increased pressure and xanthochromic fluid.

Following admission the patient had several generalized seizures. His condition improved steadily and on the 11th hospital day the spinal fluid was clear and the manometric pressure was normal.

On the 18th hospital day bilateral carotid angiograms revealed an acute subdural collection on the right (Fig. 4). Because of difficulty in getting into contact with the patient's next of kin, operation was delayed. During this period the patient was followed with serial angiograms and the subdural collection was seen to diminish gradually.

On the 40th hospital day biparietal burr holes were performed. At the right burr hole there was a moderately thick membrane intimately adherent to the dura mater and a thin inner membrane overlying the surface of the brain. There was no fluid collection between these two layers.

The patient was discharged only to return 3 months later with the same complaints. At this time bilateral carotid angiograms showed no evidence of a subdural collection on the right but did show an acute subdural collection on the left. The patient was treated conservatively and followed by angiograms. In 24 days the subdural collection had cleared.

**DISCUSSION**

Those patients in whom subdural collections develop and who expire within the first few days undoubtedly do so because of associated cerebral damage. However, what of those patients in whom subdural masses develop without associated cerebral damage? Many of these patients will recover from the initial episode and go through a period of relative normalcy except for headaches. Days or weeks later the patient may quickly manifest neurological signs and symptoms and pursue a rapid downhill course. Other patients present no symptoms and the subdural
collection is discovered at a later date during the course of work-up for some other condition.

The hypothesis of Gardner\(^3\) has seemed an attractive manner in which to explain the various phenomena encountered in patients with subdural collections. It was proposed that fluid was drawn into the sac by osmotic forces resulting from the breakdown of protein into smaller molecules in the enclosed collection. Increasing neurological dysfunction was felt to be associated with the enlargement of the mass of fluid.

The lack of neurological symptoms in the present patients would suggest that compression of the brain by an enlarging subdural mass is not the only factor to be considered. The slow disappearance of abnormal

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**FIG. 2, Case 2.**
electroencephalographic findings after the evacuation of subdural collections would also indicate that there are other factors.\(^4\)

Anatomical studies\(^1\) have demonstrated the presence of focal intracerebral edema underlying hemorrhagic collections on the surface of the brain. This edema occurred at any time from 4 hours to months after the initial injury. Pneumoencephalograms performed after the evacuation of extracerebral masses have demonstrated ventricular distortion for periods up to 1 month.\(^2,5\) This was felt to be the result of persistent intracerebral edema.

It would appear that the brain can accommodate for a moderate decrease in the available intracranial volume. However, with the onset of intracerebral edema at some indeterminate time after the head injury, the brain is compromised further and neuro-

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**Fig. 3. Case 3.**
logical symptoms appear. Thus the collection on the surface and intracerebral edema together constitute an intracranial “mass lesion” which causes cerebral distortion, displacement and transtentorial herniation. The time interval between the formation of the subdural collection and the appearance of intracerebral edema would account for the lucid period exhibited by many patients harboring collections on the surface of the brain. If the changes incident to distortion and displacement have not been too severe, recovery will occur. In patients in whom there is relative absence of edema of the hemisphere underling the lesion on the surface there may be a paucity of abnormal neurological signs. In this case the subdural collection may gradually undergo resolution. These would appear to be the circumstances in the patients considered in this report.

In sum then, in addition to the collection on the surface of the brain, the time of appearance and degree of intracerebral edema are the essential factors contributing to the abnormal clinical states encountered in patients with extracerebral collections.

We wish to emphasize that we are not advocating a new method for treating subdural hematomas. Prompt surgical evacuation remains the only method of treatment for this condition.

SUMMARY

Four cases of resolving subdural collections are presented. In 2 cases the lesion was proven by surgery to be a hematoma before it resolved. One patient had two consecutive subdural collections, both of which resolved. The cerebral changes occurring with subdural collections are discussed and the importance of intracerebral edema is emphasized.

REFERENCES