POST-TRAUMATIC INTRACRANIAL HEMATOMAS IN PATIENTS WITH ARRESTED HYDROCEPHALUS

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The deceptively benign clinical course that hydrocephalic patients may have with post-traumatic intracranial hematoma has not been mentioned in the literature. Two adult patients with long-standing “arrested” hydrocephalus who received head injuries have been treated in this department. Each of them had a course immediately after injury that did not suggest the gravity of the condition. A third case of hydrocephalus has been seen in an adult who had an epidural hematoma develop as a complication of a ventriculogram. This patient also experienced an atypical course. In this paper, these 3 cases are reported, the literature is reviewed for pertinent related cases, and a few conclusions are drawn concerning the care of the hydrocephalic patient who has sustained a head injury or has had intracranial surgery.

The occurrence of a subdural hematoma following surgical treatment for hydrocephalus has been recognized in children; and in reviewing the literature, it is seen that a similar apparently benign clinical course may be found in these cases also. Anderson¹ has reported this development in 3 out of 24 patients operated upon. The first patient had a spinoureteral anastomosis. Symptoms of increased intracranial pressure developed 10 days postoperatively. After three unsuccessful attempts to relieve it by repeated shunts, a diagnosis of large bilateral subdural hematoma was made at autopsy. The second patient had a right choroid plexectomy. After a satisfactory postoperative course for 6 weeks, a diagnosis of an existing left subdural hematoma was made only after craniotomy, which was done preparatory to performing a left plexectomy. The third patient had a resection of an intraventricular cyst and a choroid plexectomy. Signs of increased intracranial pressure developed on the third postoperative day. Because of experience with the first 2 cases, the diagnosis of subdural hematoma was suspected and promptly confirmed by subdural taps. Davidoff and Feiring² have reported the development of subdural hematomas in 3 cases subsequent to surgical treatment of 85 hydrocephalic children. Their first patient had a bilateral choroid plexectomy and had a satisfactory course until nearly 2 years later when he suffered a head injury and a subdural hematoma developed over the left cerebral hemisphere. The hematoma was removed and the patient had a satisfactory postoperative course. Eighteen months later, * Present address: University of Mississippi Medical Center, Jackson, Mississippi.

590
he had convulsive seizures but his neurological findings were within normal limits. A subdural puncture on the left side yielded brown fluid demonstrating a persistence of the hematoma. Their second patient had a subarachnoid-peritoneal anastomosis which was followed by the development of convulsive seizures, headaches and vomiting 1 to 2 months after operation. Bilateral subdural hematomas were removed. The hematomas persisted and became symptomatic again 3 months later. Their third patient had a bilateral plexectomy. She was well for 3 years and then sustained a minor head injury which did not render her unconscious or alter the neurological findings. Two months after the injury, she became listless, vomited and had headaches. A large subdural hematoma was found over the right cerebral hemisphere.

These cases show the unusually long intervals that may occur between the injury and development of the hematoma and the appearance of symptoms. The following cases would suggest that the same course may be seen after head injuries or intracranial surgery in adults with hydrocephalus.

**CASE REPORTS**

*Case 1.* L.S., a 34-year-old white male, fell from a hayrake 2\(\frac{1}{2}\) months prior to admission to the University Hospital in September 1951. He was alone and it is not known if he were unconscious. Neurological findings were normal and roentgenograms of the skull did not reveal any fractures. Convalescence was uneventful except for vomiting twice on the day of injury and headaches for a week.

His vacation ended 2 weeks after the injury and he returned to his job as an automobile assembly worker. He performed his work satisfactorily until 2 weeks before admission to the hospital when he began to have difficulty with coordination, stumbled frequently, became quarrelsome, and was noted to be putting the automobile door hinges on upside down.

Past history revealed that he had been a healthy, normal child except for an enlarged head since early childhood.

*Examination.* His calvarium was unusually large in proportion to his face. The gait was slightly unsteady and coordination was impaired throughout. A left lower facial paresis was present. Vital signs and speech were normal. Reflexes were hyperactive and both plantar responses were extensor in type.

A diagnosis of “decompensation” of arrested hydrocephalus with stenosis of the aqueduct of Sylvius was considered as well as one of communicating hydrocephalus with subdural hematoma.

*Operation.* The patient was prepared for ventriculography. A trephination at Keen’s point on the right was made and the dura mater was noted to have a bluish-white color. On opening the dura mater there was a gush of dark blood and about 700 cc. were removed. A small craniectomy was performed and the membranes of a subdural hematoma were identified. The subdural membranes extended from the frontal to the occipital pole. They were removed entirely except for points of attachment along the sagittal sinus. The brain was depressed 2 inches from the inner table of the skull and did not re-expand after removal of the membranes. A cannula was placed in the lateral ventricle and 280 cc. of Ringer’s solution were injected. The brain then expanded to within 0.5 cm. of the inner table of the skull. The cerebral
tissue was about 2 cm. thick. After careful hemostasis of all bleeding points, a Penrose drain was placed from the subdural space to the exterior and the dura mater, bone and skin were closed in layers.

Postoperative Course. He became more confused and stuporous and, on the 4th postoperative day, right coronal and posterior parietal burr holes were placed and a small recurrent subdural hematoma was removed. The cortex was then seen to approximate the bone at both sites. Following the second operation, there was rapid clearing of the sensorium; but he had focal seizures in the left side of the face and left arm, and forced deviation of the eyes to the left. These seizures were controlled with Dilantin 0.1 gm., 3 times daily. A left hemiparesis was present after operation but this cleared completely. He has had no further difficulty.

Case 2. H.H., a 25-year-old farm worker, was admitted to St. Joseph Mercy Hospital on Nov. 17, 1956, 2 hours after he was found in a semiconscious state at the bottom of a 12-foot ladder. He was hemorrhaging from the nose and vomiting blood.

He was said to have had a normal birth and development except for a large head. Also, he had had several episodes of fainting during the previous 2 years.

Examination. He was a stuporous white male with a strikingly large calvarium who lay quietly but could be aroused to give his name and address, or to obey simple commands. Fresh blood was present in both nares. Blood pressure was 118/84 mm. of mercury, pulse rate 64, and respiratory rate 20 per min. The pupils were equal and reacted to light. A coarse nystagmus was present on either side but was more marked to the right. The left lower facial movements were slightly decreased. Deep reflexes were symmetrically hyperactive throughout and plantar responses were weakly positive bilaterally.

Roentgenograms of the skull revealed a very faint fracture line in the left frontal and parietal bones and these were compatible with the clinical impression of a disproportion of size between the skull and face.

Course. In view of his gradual recovery of consciousness and the stable vital signs during the 1 hour in the x-ray department and the emergency room, it was elected to admit him to the surgical floor for observation. In recalling the experience of one of the authors with Case 1, it was recognized that he might have, or there might develop later, an intracranial hematoma without the usual signs being manifested.

He was observed for 5 days. The sensorium cleared rapidly to the dull level that his family regarded as normal. Despite his right plantar response reverting to normal and his vital signs remaining normal, the lower left facial paresis increased, and the right pupil became equivocally larger than the left. He complained of a mild headache on several occasions.

Operation. Because of the experience with Case 1, multiple trephinations were performed. A small subdural hygroma was evacuated from the area of Keen's point on the right. Right coronal and temporal trephine openings were made and a fresh hematoma was noted to cover the entire anterior part of the hemisphere. It was estimated to be 200 cc. in volume, and was completely removed. Under the left temporal burr hole an epidural hematoma, 175 cc. in volume, underlying the horizontal fracture line, was completely removed. Penrose drains were placed on either side and all incisions were closed in a single layer.

Postoperative Course. All abnormal neurological signs cleared and he was free of headaches. Hearing was slightly diminished on the left side. A serous otitis was
diagnosed by an otological consultant who did a myringotomy. The diagnosis of "arrested" communicating hydrocephalus was confirmed by ventriculography (Fig. 1) and recovery of dye in the lumbar space 20 minutes after injection into the ventricles. No further operative treatment was advised. He was discharged on Dilantin 100 mg., 3 times daily, and went back to work as a farm laborer.

**Subsequent Course.** One month after discharge from the hospital, the patient was readmitted in status epilepticus of sudden onset, with pulse rate of 150 per min., rapid respirations and temperature 106°F. (R). Lumbar puncture revealed cerebrospinal fluid which was cloudy with leucocytes and *Staphylococcus aureus*. Despite heavy antibiotic therapy and sterilization of the cerebrospinal fluid on culture, a progressively downhill course persisted and respirations ceased on the 14th day of hospitalization.

**Autopsy** revealed a large abscess in the left temporoparietal area which had ruptured into the lateral ventricle. No definite connection of the abscess with the petrous ridge could be established. The lateral ventricles were abnormally large (Fig. 2) and the diagnosis of hydrocephalus was confirmed.

**Case 3.** No. 890876. G.T., a 16-year-old boy, was admitted to University Hospital on Jan. 15, 1958, with a history of a large head since birth, retarded intellectual development, and a recent onset of severe headaches and frequent episodes of vomiting.

**Examination.** The boy's head was obviously large, measuring 26 inches in circumference. He had very poor mental development and a short span of attention. Positive neurological findings were 4 diopters of papilledema and small retinal hemor-
rhages O.U., moderate nystagmus of lateral gaze, and slow pupillary response to light.

The admitting diagnosis was hydrocephalus and in view of the experience with the above cases, it was realized that he could have a subdural hematoma as a basis for his recent complaints.

Operation. Bilateral coronal and left Keen's-point trephinations were accomplished without any evidence of an epidural or subdural hematoma. An opening at Keen's point on the right was not utilized in order that this area might be available as a fresh site for a future ventriculocaval shunt. Ventriculography was performed with the exchange of 300 cc. of ventricular fluid for air. A Scott cannula was then placed in the area of Keen's point and connected to a Bering bottle set at 200 mm. of water.

Course. Ventriculograms revealed a massive hydrocephalus and the findings were compatible with an aqueductal stenosis (Fig. 3). He was thought to be a suitable candidate for a ventriculojugular shunt and was returned to the ward to await absorption of the ventricular air. The patient was awake, and his condition was unchanged from his preoperative status. He remained this way until 48 hours later when, over a period of 4 hours, he became restless and was drowsy. He would become very agitated with mild stimulation and later exhibited signs of decerebrate rigidity. Body temperature went up to 105°F, with the development of these new neurological signs.

The patient was given a large dose of cortisone and 100 cc. of 50 per cent glucose intravenously. The trephines were re-explored. No evidence of a significant subdural or epidural hemorrhage was seen. The brain was pulsating normally and the cortex was immediately adjacent to the dura mater. The ventricular drainage was continued. The patient's condition continued to deteriorate and there developed fixed pupils, marked decerebrate rigidity, irregular respirations and positive Babinski's
response bilaterally. He lived for 6 more days without improvement in conscious level or change in vital signs except for irregularity of respiration. The blood pressure and pulse rate were normal until the respirations ceased. No changes of the vital signs like those usually accompanying increased intracranial pressure were seen.

Autopsy revealed an obstruction of the third ventricle with massive dilation of the lateral ventricles and marked thinning of the cerebrum. In addition to the changes of hydrocephalus, there was an epidural hematoma, 920 cc. in volume, over the posterior portion of the right cerebral hemisphere (Fig. 4).

Fig. 3. Case 3. Ventriculograms revealing huge lateral ventricles and small posterior fossa.

Fig. 4. Case 3. Autopsy specimen showing large epidural hematoma over the right hemisphere.
Undoubtedly, in Case 1 the patient’s subdural hematoma resulted from his fall from the hayrake 2 1/2 months prior to entering the hospital. It is believed that he did not show signs earlier because the large hematoma was accommodated through a process of “internal decompression” by collapsing the huge lateral ventricles. The operative findings of the thin cerebral substance which did not re-expand until the ventricle was inflated with saline would suggest that this assumption was correct.

Case 2 presented a picture of a rapidly clearing sensorium, stabilization of vital signs at normal levels and only minor headaches immediately after the injury. The symptoms and signs of the bilateral hematoma and the right subdural hygroma were masked through a process of “internal decompression,” similar to the situation in Case 1. Although the patient in Case 1 had a higher level of intelligence than the patient in Case 2, he also had a thinner cerebral substance. Cerebral thickness was estimated to be 2 cm. in Case 1 and more than 3 cm. in Case 2. It is doubtful that the ventricles in Case 2 (Fig. 1) could have collapsed enough to permit the nearly symptomless accommodation of such a large hematoma as in Case 1.

In Case 3, the tube for ventricular drainage allowed the ventricles to collapse to a marked degree and thus gave a large “internal decompression.” Undoubtedly, the epidural hematoma originated from the right coronal trephination. It would seem probable that its development to such a large size was abetted by the weak resistance of the patient’s thin cerebral hemisphere to distortion. It is thought that the internal decompression plus the ease of distortion accounted for the delay of onset of new neurological symptoms for the 48 hours immediately after ventriculography.

Since the collapsing brain offered little resistance, the blood tended to pool in the most dependent area over the posterior half of the hemisphere and did not remain in the area of its origin (right coronal trephination) as would be the usual case. The lack of significant subdural or epidural hematoma in this area, plus the normal pulsation of the brain, caused an incorrect conclusion of no significant intracranial hematoma to be made at the second operation. At this point, it must be noted that if the usual three trephine openings had been placed in the coronal, temporal, and Keen’s-point sites, the epidural hematoma would have been found at the second operation. The lack of any signs of increased intracranial pressure can be explained by the continued use of ventricular drainage during the postoperative course.

CONCLUSIONS

In patients whose disproportionately large calvariums suggest the possibility of hydrocephalus, the history of a head injury must be evaluated with an unusually high index of suspicion for intracranial hematoma. The old injury may have an innocent-sounding history and the acute injury may present a deceptively benign clinical course. Experiences with the above
HEMATOMAS IN HYDROCEPHALIC PATIENTS  

Patients would suggest that caution must be exercised in accepting their apparently straightforward recovery. In cases in which doubt exists, and probably in any case in which there is a period of unconsciousness, contrast studies or multiple burr holes should be utilized to rule out a still silent intracranial hemorrhage. As demonstrated in Case 3, it is particularly important to place a trephination in the dependent portion of the suspected area when searching for an acute or chronic intracranial hematoma in a patient whose brain may be easily collapsed. It is not germane to this discussion, but nevertheless should be noted, that this case demonstrates again the well known fact that the advanced adult hydrocephalic does not easily tolerate a large exchange of air and ventricular fluid during ventriculography.

Several cases collected from the literature would indicate that the same degree of caution and close observation must be exercised in the care of postoperative hydrocephalic children.

SUMMARY

The literature is reviewed concerning the development of subdural hematomas in patients with hydrocephalus who had had operative treatment or suffered a head injury. Two cases of post-traumatic and 1 case of postoperative intracranial hematoma in patients with “arrested” hydrocephalus are presented. Their meager symptoms and signs are discussed relative to their deceptively benign course. Suggestions as to the method of treatment of such patients are presented.

REFERENCES