Delayed apoplexy following head injury ("traumatische Spät-Apoplexie")

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The authors present their experience with three cases of delayed apoplecticiform reaction to head injury. Two cases were operated on successfully, and the findings are compared with those previously reported. Pathogenesis, diagnosis, and management are discussed.

Key Words: brain concussion - cerebral hemorrhage - head injury - intracranial pressure - subarachnoid hemorrhage

The sudden appearance of signs of serious intracerebral hemorrhage related to recent head injury in a previously asymptomatic individual is rare. We are reporting our experience with three such cases.

Case Reports

Case 1

A 3-year-old girl fell from a swing striking the back of her head and then was struck again in the same area by the swing as it came forward. She was unconscious approximately 30 minutes.

Examination. On admission to the hospital the patient was lethargic but could be awakened by calling her name. Her only neurological abnormality was the absence of deep tendon reflexes. Vital signs were normal. Skull x-rays were normal.

Course. The patient improved spontaneously over the first 24 hours, becoming much more alert and recovering normal reflexes. Forty-eight hours later, however, neck stiffness developed without other localizing signs. Lumbar puncture revealed grossly bloody spinal fluid. Diagnostic frontal, temporal, and parietal burr holes were made on the third hospital day and revealed only pinkish discoloration of the brain. Over the next several days she was out of bed, and played with other children on the ward. On the evening of the seventh postoperative day her vital signs were normal. On routine check of signs at 11:15 p.m., the patient was found dead in bed.

Postmortem Examination. Postmortem examination including dissection of the circle of Willis revealed no evidence of aneurysm or arteriovenous malformation. Extensive subarachnoid hemorrhage and hemorrhage into the 4th ventricle was present (Fig. 1). Section of the brain revealed early softening in the white matter around the occipital horn of the right lateral ventricle in the region of the trigone. Microscopic findings included some thickening of venous walls, perivascular round cell infiltration, and necrosis in subcortical areas with subarachnoid hemorrhage.

Case 2

This 8-year-old girl was struck by an automobile. She did not lose consciousness.

Examination. On hospitalization 2 hours later, the patient was sleeping intermittently
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but easily awakened. In other ways she was neurologically normal. There was a severe abrasion of the left forehead and cheek, a small laceration over the left eye, and a deep laceration of the distal left thigh. Admission blood count was normal. X-rays revealed an undisplaced intertrochanteric fracture of the left femur but no skull or facial fractures. Traction of the left leg was instituted, and on the 10th hospital day a Spica cast was applied. She remained neurologically normal, and on the 11th day she was discharged.

When seen as an outpatient 1 week later she was asymptomatic except for occasional headaches. Neurological and funduscopic examinations were normal.

Second Examination. The patient remained well until the 34th post-trauma day when she was readmitted following the sudden onset of severe frontal headache, grand mal seizures, and unresponsiveness. There had been no additional head trauma. The blood pressure was 170/120, pulse 60, and respiratory rate 20. The pupils were equal and reactive. There were no signs of head or neck trauma, except for marked subhyaloid hemorrhages bilaterally. The only response to deep pain was a minimal symmetrical facial grimace. The right Achilles reflex was hyperactive. A right sixth nerve palsy was present. Echoencephalogram showed a right-to-left shift of 7 mm. Decadron and Mannitol were started. An immediate carotid angiogram revealed an anterior right-to-left shift (Fig. 2). Improvement to the point where she could follow commands had occurred by completion of angiography, and a left hemiparesis could now be noted. Lumbar puncture showed bloody cerebral spinal fluid with elevated pressure.

Operations. Through a right frontal burr hole, 30 cc of intracerebral clot were aspirated. The patient initially improved but her continued lethargy prompted a right frontal craniotomy 4 days later with evacuation of a 40-cc intracerebral clot and a ventricular cast.

Postoperative Course. Recovery was complicated by a communicating hydrocephalus. Two weeks after craniotomy a ventriculoatrial shunt was installed; this was followed by marked improvement. The patient was discharged 3 weeks later, and when last seen had only minimal left hemiparesis.
Case 3

A 66-year-old man was in good health until he fell, striking his head. He was unconscious for approximately 15 minutes, but on awakening, except for headache and some grogginess, was neurologically normal. He was admitted to the hospital shortly after the injury.

Examination. On admission the patient complained of pain in the head and left shoulder in addition to his head injury. The neurological examination was normal. Skull and shoulder x-ray films were normal.

He was discharged 4 days later, and was free of symptoms for 5 days when he suddenly developed a left-sided headache and became unresponsive.

Second Examination. On readmission the patient was conscious, had a right hemiplegia, right Babinski sign, and showed a mixed motor and sensory aphasia. A carotid arteriogram revealed a left temporal lobe lesion (Fig. 3).

Operation. A left subtemporal decompression was performed and a large intracerebral hematoma evacuated from the temporal lobe.

Postoperative Course. Recovery was uneventful. The patient was discharged from the hospital on the 14th postoperative day showing much improvement in his neurological status.

Fig. 3. Case 3. Lateral carotid angiogram showing elevated middle cerebral artery and a large avascular area beneath it.

Discussion

Bollinger in 1891 described four cases of apoplectiform death following head trauma by days or weeks. He named this “traumatische Spät-Apoplexie” and cited the following criteria for diagnosis: the absence of pre-existing vascular disease, a definite history of trauma, an asymptomatic interval, and a subsequent apoplectiform episode. His study was based on four postmortem examinations. His first case was a 7-year-old boy who fell, did not lose consciousness, but 3 days later developed a right hemiplegia, aphasia, and died 52 days later. At autopsy, a hemorrhagic focus was found extending from the wall of the 4th ventricle on the left through the pons. The second case was a 13-year-old girl who fell while skating. Three weeks later she died suddenly, and was found to have a large hemorrhage in the posterior right hemisphere with ventricular extension. The third case, a 26-year-old man, was struck by an axe. Twenty-one days later he developed headache, and on the 26th day a hemiplegia. Death followed on the 32nd day. At autopsy, hemorrhages were found in the meninges and 4th ventricle. The fourth case was a 39-year-old woman who sustained a forehead bruise and complained of headache for 24 hours. Two weeks later a sudden seizure was followed by death. Autopsy revealed a blood clot in the 4th ventricle.

Bailey in 1904 distinguished three types of traumatic apoplexy: an immediate form coincident with the injury, apoplexy developing days or weeks after the injury, and a type that developed many months later. Naffziger and Jones in 1928 felt that the maximum time for traumatic hemorrhage to develop was 8 weeks, and that hemorrhage after that period was not related to trauma.

Recent publications show a diversity of time intervals between the injury and delayed apoplexy. Anttinen and Hillbom presented five patients who developed apoplectic paralytic conditions years after a brain injury received during World War II. The earliest onset of post-injury symptoms was 32 months and the longest was 11 years; at the time of the onset of these symptoms the patients were under 40 years of age. Austarheim felt that delayed intracerebral hemorrhage did not occur until many weeks or...
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months after the initial injury, and reported a patient who died 23 months after head trauma.

We have reviewed the literature for those cases that most resemble ours and conform with Bollinger's original description. In all of the cases1,2,5,11-13,22,27,30,31,33,35 the asymptomatic interval was less than 120 days (Table 1). This selection resulted in the inclusion of most of the cases of delayed intracerebral hemorrhage reported in the literature.

The severity of the head trauma in most of these cases was minimal and occasionally trivial. Approximately one-half of the patients had a brief period of unconsciousness, lasting from minutes up to 1 hour. There was usually no fracture. It should be stressed that most of these patients would have met the criteria for the diagnosis of cerebral concussion. Other aspects reviewed were the asymptomatic interval, age range, neurological presentation, surgical and autopsy findings. Most asymptomatic intervals were less than 30 days (Table 2). We have not interpreted this period as one which is necessarily symptom-free, but rather one in which if symptoms were present they might be considered as being of the relatively mild, post-concussive type. Certainly, no patient had symptoms that would suggest a need for hospitalization or neuroradiological studies.

The incidence by age (Table 3) is of significance since "Spät-Apoplexie" seems to occur in an age group in which arteriosclerotic cerebrovascular disease would not seem to be a prominent factor. Few cases occurred after the fourth decade.

The clinical manifestations of this condition are reviewed in Table 4. Sudden hemiplegia, an uncommon entity in a young age group, was the prominent finding.

At surgery or autopsy, intracerebral hematoma not involving the basal ganglia was the most common lesion in the 24 patients (Table 5).

Delayed post-traumatic intracerebral hemorrhage has been accorded brief mention in only a few neurological texts; 6,15,25,32 in contrast, traumatic intracerebral hemorrhage has been reviewed by many authors. 7,8,10,26,31,33, 24,26,29 The most controversial aspect of this entity is its pathogenesis. Duret 14 found that blows on the head and intracranial injections of fluid in experimental animals produced laceration and foci of softening in the region of the lateral ventricles, the aqueduct of Sylvius, and the 4th ventricle. Based on this, Bollinger 6 felt that cranial trauma produced cerebral softening, which occasionally involved the wall of an artery, with eventual necrosis, rupture of the vessel, and hemorrhage.

The histological features of the brain following head trauma as described by Scheinker, 34 and Evans and Scheinker, 16,17 suggested a theory of vasoparalysis. They postulated that head trauma initiated vasoparalysis deep in the hemisphere with subsequent dilatation and congestion of capillaries, and then decreased blood flow. The static condition allowed the accumulation of local metabolites to toxic levels with resultant increase in capillary permeability and ensuing perivascular edema and hemorrhage. They believed that traumatic intracerebral hemorrhage was due to a coalescence of multiple areas of diapedetic hemorrhage.

The importance of distinguishing pathologically between spontaneous intracerebral hemorrhage and traumatic intracerebral hemorrhage was emphasized by Courville, 8 and Gradwohl. 18 The former stated that the medicolegal problem lies in determining the cause of gross intracerebral hemorrhage into the brain of an individual who has had a history of recent fairly severe injury to the head, and who may have died under equivocal circumstances. Courville 8,10 found that

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traumatic intracerebral hemorrhage predominantly involves the subcortical layers of the frontal, temporal, or temporal occipital lobes. The location of the pathological findings in spastiaplexie are similar.

The differential diagnosis of delayed traumatic apoplexy is that of a hemorrhagic intracranial mass lesion. Without accurate history, it may be almost impossible to differentiate it from spontaneous intracerebral hemorrhage. Common entities to be considered are aneurysm, vascular malformation, hemorrhage into a tumor, and sudden decompensation in a subdural hematoma. While the pathogenesis remains speculative, Bollinger's entity of delayed intracerebral hemorrhage remains a clinical possibility. The medicolegal implications of traumatic late apoplexy are obvious. Modern diagnostic methods should help to clarify the clinical picture and identify those cases in which trauma and hemorrhage are coincidental unrelated events. The routine follow-up care of head injury should take cognizance of the possibility of spastiaplexie occurring as a delayed complication.

**Summary**

Bollinger in 1891 described four cases of apoplectiform death following head trauma by days or weeks (traumatische Spät-Apoplexie) characterized by absence of pre-existing vascular disease, a history of head trauma, and a relative asymptomatic interval between the trauma and apoplectic episode. We have reported three such cases and reviewed other reports. Most patients were under 40 years of age and the asymptomatic interval less than 30 days, culminating in sudden hemiplegia. Since the treatment is usually surgical evacuation of the hematoma, clinical awareness of this diagnostic possibility is important.

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**References**

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