Delayed traumatic intracerebral hematomas: "Spät-Apoplexie"

Report of two cases

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Two patients who suffered a head trauma experienced sudden clinical deterioration more than 1 week after their injury. The initial computerized tomography (CT) scan demonstrated a small hematoma underlying a depressed skull fracture in one patient and a small interhemispheric subdural hematoma in the other. Both patients had made a complete recovery and follow-up CT scans were normal when clinical deterioration suddenly occurred. Both patients developed a large intracerebral hematoma and underwent emergency evacuation of the mass. The first patient recovered with the exception of a moderate hemiparesis and dysphasia, but the second patient died.

Delayed traumatic intracerebral hematomas have been described in the literature. Since the advent of CT scanning, the incidence of this phenomenon has been estimated as between 1.7% and 7.4% of closed head injuries. In 1891, Otto Bollinger described four patients who suffered head injury, followed days to weeks later by death from an apoplectic event. His criteria for diagnosis of "traumatische Spät-Apoplexie" included the absence of preexisting vascular disease, a definite history of trauma, an asymptomatic interval of at least several days, and an apoplectic episode. These two cases reemphasize the existence of Spät-Apoplexie as a rare clinical condition. In the presence of CT findings of even small traumatic intracerebral or extracerebral hematomas, the possibility of this late complication should be kept in mind.

KEY WORDS ▪ Spät-Apoplexie ▪ delayed traumatic hematoma ▪ hematoma

Since the development of computerized tomography (CT), the occurrence of delayed traumatic intracerebral hematoma has been well documented. Many of these hematomas occur in a subacute fashion 24 to 48 hours after the initial trauma. "Spät-Apoplexie" was described in 1891 by Otto Bollinger. He reported four cases of delayed traumatic intracerebral and intraventricular hematomas which occurred 2 to 4 weeks after mild head injury and resulted in death. In each case, the patients were recovering well and experienced a sudden apoplectic episode followed by death, hence the term "spät" (or late) apoplexy.

At the University of Maryland Hospital and the Maryland Institute for Emergency Medical Services System (MIEMSS) approximately 800 patients are evaluated by the Neurosurgery Service for closed head injury each year. Approximately 150 of these patients have a CT finding of intracranial pathology. In the past year, two patients experienced apoplectic events while recovering from head injury, a phenomenon consistent with Bollinger's initial description of "Spät-Apoplexie." A review of the clinical course of these two patients as well as an appraisal of the pertinent literature on this entity form the subject of this report.

Case Reports

Case 1

This 29-year-old man suffered a left frontal depressed skull fracture with underlying cerebral contusion after being struck in the head with a blunt object. On admission he was awake and alert but aphasic. He had no motor deficit. He underwent operative elevation of the fracture and closure of the dural laceration. Follow-up CT scans confirmed the presence of the small contusion underlying the craniectomy area. Ten days after the
event he was found in his room unresponsive. An
emergency CT scan demonstrated a large intracerebral
hematoma in the area of the previously noted contu-
sion. Preoperative studies ruled out the presence of
cogaulopathy and he underwent emergency evacuation
of the hematoma via a left frontotemporal craniotomy.
At discharge, a fair recovery characterized by the return
of some motor power had already begun. The aphasia
remained dense.

Case 2

This 15-year-old girl had been involved in a motor-
vehicle accident. At the time of admission, she was
combative but did not show any evidence of focal
deficit; CT scanning (Fig. 1 left) demonstrated an
interhemispheric subdural hematoma. Angiography several
days later demonstrated no vascular lesion. She was
treated conservatively and made a complete recovery.
On the 14th day after injury, the day of her planned
discharge from the hospital, she experienced a seizure
and rapidly lapsed into a coma with extensor posturing
and a unilateral fixed and dilated pupil; CT scanning
(Fig. 1 right) demonstrated the presence of bilateral
intracerebral and intraventricular hemorrhage. Coagu-
lation studies were within normal limits. The patient un-
derwent craniotomy and evacuation of the hematoma
but developed severe diffuse brain edema. She never
regained neurological function and died 2 days later.

Discussion

The four cases of death after a delayed apoplectiform
episode following head trauma described by Bollin-
ger met the following criteria: 1) a definite history of
trauma; 2) an asymptomatic interval; 3) an apoplectic
event; and 4) absence of vascular disease. Based on
experimental data previously gathered by Duret, Boll-
linger suggested that cranial trauma caused softening
of the brain involving the wall of an artery with event-
tual rupture of the vessel, necrosis, and hemorrhage.
Scheinker suggested the role of posttraumatic vaso-
paralysis as an alternative initial factor in the genesis
of the hematoma. Vasoparalysis accompanied by dilata-
tion of the capillaries with accumulation of toxic
metabolites would precipitate an increase in capillary
permeability, perivascular edema, and hemorrhage.
Coalescence of multiple areas of diapedetic hemorrhage
would create large traumatic intracerebral hemato-
tomas. After the original description, many authors
reported several cases meeting Bollinger's criteria.

This subject was reviewed in 1970 by Morin and
Pitts. They included a review of patients with an
apoplectic episode occurring after an asymptomatic
period of less than 120 days. In total, they collected 24
cases from the literature and added three of their own.
This review pointed out the following facts: 1) severity
of the injury was in most cases minor, usually without
fracture and with only a brief period of unconscious-
ness; 2) the asymptomatic interval was usually less than
30 days; 3) “Spät-Apoplexie” seemed to affect predomin-
antly younger people in whom arteriosclerotic disease
did not appear to be a factor, although a few cases
occurred after the fourth decade; 4) sudden hemiplegia
(an uncommon presentation in the young age group)
appeared to be the prominent presentation in these
patients; and 5) pathologically, the posttraumatic intra-
cerebral hematoma most commonly affected structures
other than the basal ganglia, as was demonstrated by
Courville and Blomquist. Morin and Pitts also stressed
the need to differentiate delayed posttraumatic hema-
tomas from those due to the unrecognized presence of
a vascular malformation or tumor.

Challenging the previous authors, Baratham and Den-
nyson maintained that Bollinger's original description
was unsatisfactory because apoplexy is a term of dubi-
ous validity, not denoting a specific pathological entity.
They questioned the relevance of trauma when the lucid
interval is prolonged, inferring that it may have been
purely fortuitous. They also explained the relatively
high incidence of “Spät-Apoplexie” in the young by

Fig. 1. Case 2. Left Pair: Nonenhanced computerized tomography (CT) scans obtained on admission showing an interhemispheric subdural hematoma. Right Pair: Nonenhanced CT scans obtained on the 14th day after injury showing bilateral intracerebral hemorrhage.
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stating that this group is more prone to injury than elderly patients. However, 14 of their 21 patients in fact had an asymptomatic interval of less than 48 hours and the majority of patients were in the seventh decade, a group quite different from Bollinger’s cases.

Since CT has been available, the management of head trauma patients and the understanding of the pathology of head injuries have been greatly improved by the recognition of different types of posttraumatic hematomas. From among 656 cases, Diaz, et al., presented nine patients with delayed hematomas in whom the interval from cranial injury to the development of the hematoma varied from 8 hours to 13 days. However, all of their cases differed from Bollinger’s classic description in that the patients who had initial negative CT scans did not have an asymptomatic interval. The 25 cases described in 1984 by Ninchoji, et al., differed from the classic traumatic “Spät-Apoplexie” syndrome because they lacked a symptom-free interval and an apoplectic episode. The patients who “talk and deteriorate” after severe closed head injury usually do so within the first 48 hours and should not be included in the category discussed here. Fukamachi, et al., reviewed 84 cases and described four subtypes of posttraumatic intracerebral hematomas, none of which matched the entity originally described by Bollinger.

We report two cases that seem to fall into the group of true “Spät-Apoplexie” cases. Both patients were young and had a definite asymptomatic interval. The onset of their symptoms was precipitous, in fact apoplectic, and no coagulation abnormality was detected (as has been noted in some delayed hematomas reported in the literature) or preexisting vascular abnormality found. Their clinical course was characterized by complete neurological recovery and by resolution of the initial lesion seen on CT prior to the development of the apoplectic event, which occurred in both cases more than a week after the original trauma.

Many years after its original description, we are still at odds in trying to explain the pathogenesis of this entity, but we believe that traumatic “Spät-Apoplexie” should remain a clinical entity. Although we cannot provide any clues to help neurosurgeons in detecting these patients in advance, we recommend that the existence of this condition be kept in mind, especially in view of its potential medicolegal implications.

References

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