Chronic subdural hematoma presenting as spontaneous subarachnoid hemorrhage

Report of six cases

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Six cases of chronic subdural hematoma presenting with the clinical findings of acute subarachnoid hemorrhage are reported. No systemic or focal cause for the bleeding was found, and possible mechanisms are discussed.

Key Words • subarachnoid hemorrhage • subdural hematoma

Subarachnoid hemorrhage (SAH) is sometimes associated with nonvascular diseases of the central nervous system. It occurs as an initial symptom in some cases of intracranial tumor, both primary or metastatic, infectious disease, or cerebral venous occlusion.

In this paper, we report six cases of chronic subdural hematoma that presented as SAH. Bleeding occurred a considerable period of time after head injury, but the trauma did not appear to be a factor in producing the SAH.

Summary of Cases

Between 1968 and 1982, six chronic subdural hematoma patients were admitted to our department with the presenting diagnosis of SAH. All of them were admitted 2 to 7 days after the onset of their acute illness. There were four men and two women, ranging in age from 24 to 39 years, and all had a history of injury which occurred 15 days to 3 months before admission.

Four of the patients presented with a sudden loss of consciousness. Neurological examination, performed immediately after the onset of illness, revealed a hemiparesis in two cases, a facial paralysis in one case, and no neurological deficit in one case. The other two patients presented with the sudden onset of severe headache and vomiting. Neurological examination revealed a third nerve palsy in one and aphasia with a right-sided hemiparesis in the other. All six patients had a stiff neck with a positive Kernig's sign. Lumbar puncture revealed SAH in all six cases.

Clinical and biochemical examinations did not demonstrate any diseases that could be responsible for the SAH, and four-vessel cerebral arteriograms, performed in all cases, showed no vascular abnormalities. However, the arteriograms did reveal unilateral extracerebral avascular areas characteristic of subdural hematomas.

All patients underwent removal of the hematomas through a burr hole, followed by drainage of the subdural space for 24 hours. In each case the hematoma was surrounded by a well developed capsule. Within 24 hours following surgery, the patients' focal neurological symptoms returned to normal. Two to 15 years later, the patients were well, with no neurological deficits and no recurrent SAH. They had all returned to work.

Discussion

Delayed apoplexy following head trauma is very rare. Bailey distinguished three types of traumatic apoplexy developing days or weeks after head trauma. The commonest form, "traumatische Spät-Apoplexie," usually consists of intracerebral hemorrhage.

In our cases, SAH occurred 2 weeks to 3 months after head trauma. Clinical tests did not reveal any vascular abnormalities or systemic disease that might have produced the hemorrhage. At operation a well developed hematoma capsule was found in each case. According to most authors, the development of a hematoma capsule requires at least 3 weeks. Our patients were operated on within 2 to 7 days after the trauma.
symptoms of SAH appeared, and we believe that the hematomas existed before the manifestation of their illness.

Three mechanisms may be considered as the cause of the SAH in these cases: 1) SAH was a result of vascular abnormalities that were not revealed on the arteriograms; 2) SAH was a manifestation of Spät-Apoplexie, and the presence of hematoma was coincidental; or 3) SAH was due to the chronic subdural hematomas. Four-vasl vessel arteriography in our cases did not show any vascular anomalies, and follow-up examination 2 to 15 years after surgery revealed no further vascular incident in any of the patients. In Spät-Apoplexie, bleeding is usually within the cerebral tissue producing intracerebral hematomas. Clearing of the focal neurological deficits within 24 hours after surgery suggests that the SAH was related to the chronic subdural hematoma.

Distortion and displacement of vessels by tumor growth are two of the accepted explanations of SAH associated with intracranial tumors. That same mechanism may also apply to chronic subdural hematoma. Another possibility is that pressure of the hematoma on the cerebrum caused degeneration in the wall of a vessel leading to rupture. In all six of our cases the wall of the hematoma capsule was well formed. Hemorrhage from proliferating vessels in or adjacent to the capsule may have been the source of bleeding, especially since the patients were young and meningeal reaction to blood in the subdural space can be considered. Another possible cause of SAH is a sudden increase of intracapsular pressure, leading to rupture of the arachnoid with bleeding from the hematoma into the subarachnoid space. We cannot say which mechanism was responsible for the subarachnoid bleeding in our cases. As far as we know this is the first report of chronic subdural hematomas causally related to spontaneous SAH.

References


