CHRONIC SUBDURAL HEMATOMA

*Historical Review and Analysis of 60 Cases**

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CHRONIC SUBDURAL HEMATOMA has been well described in the literature. Initial descriptions were published several centuries before 1857, when Virchow's paper,[1] now considered a classic, first presented a clear account of the histopathologic nature of the lesion and suggested an explanation of its origin. Putnam and Cushing[2] in 1925 discussed the neurosurgical aspects, and Gardner[3] in 1932 offered a plausible explanation of the delayed development of symptoms. The purpose of this report is to present a brief historical review together with an analysis of the cases of 60 patients seen and treated at the University Hospital in Copenhagen.

Etiologic Aspects

Trauma is believed generally to be the most important cause of a subdural hematoma. However, although demonstrable in nearly all acute cases,[4,5] in a high percentage of chronic cases there is no traceable history of injury. Virchow[1] recognized that the lesion sometimes was traumatic, but he believed that chronic subdural hematoma (which he called "pachymeningitis chronica hemorrhagica") was most often caused by chronic inflammation of the dura with extravasation of blood into the subdural space and formation of a film of fibrin over the inner surface of the dura. Sperling[6] in 1872 reported that whole blood injected into the subdural space resulted in formation of membranes that in many respects were similar to those of chronic subdural hematoma. Huegenin[7] in 1877 reported that he frequently found fatty degeneration, sometimes thrombosis, and occasionally rupture of the veins of the pia. Barrett[8] in 1902 stated that the pathologic findings did not appear to be inflammatory and that the formation of a film of fibrin over the inner surface of the dura (an important point in Virchow's theory) did not prove the presence of inflammation.

Kasemeyer[9] in 1911, and Henschen[10] in 1912 showed that delayed symptoms frequently occurred in traumatic cases of subdural hematoma. Trotter[11] in 1914 and Putnam and Cushing[2] in 1925, in their respective series, found that a definite history of trauma usually could be obtained. Putnam and Cushing stated that they had found no case of nontraumatic hematoma of the dura without some precedent disease. However, later authors [5,12] do not agree that there always is a precedent disease in cases of "nontraumatic" hematoma.

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Solely on the basis of the literature mentioned, it is difficult to understand why the viewpoint has changed so completely from that expressed by Virchow. Some of the reasons may be: (1) later authors [4,12] have substantiated the findings of Putnam and Cushing[2] that a history of trauma is obtainable in high percentages of cases; (2) it is not possible either from the clinical or from the pathologic evidence to distinguish between the traumatic and the "nontraumatic" cases; (3) most pathologists do not consider the lesion to be an inflammation; (4) the source of a possible inflammation has never been found; (5) the accumulation of blood behaves like other hematomas in preformed cavities; (6) the late development of symptoms is explainable on the basis of osmosis[3]; (7) most subdural hematomas can be cured by simple removal of the fluid through a burr hole and a cure would not be likely if there were an active inflammatory process.

In the present series of 60 patients with chronic subdural hematoma there were 51 males and 9 females, whose ages ranged from 6 to 75 years (average 47 years). No infants are included because they usually present a different clinical picture. All were treated in the Department of Neurosurgery of the University Hospital in Copenhagen. Only 32 of the 60 patients gave a history of moderate to severe cranial injury (such as that occasioned by a fall from a ladder, a fall from a bicycle, a car accident), and in only 12 of these was the trauma so severe that the patient became unconscious. In 13 of the remaining 28 patients, the injury was very slight (such as that caused by a fall from a chair without actually striking the head, striking the head against a trunk door while lifting something from a car, a blow on the cheek from a schoolteacher). In the remaining 15 patients (25 per cent) it was impossible to trace a history of trauma.

Other authors [4,12] cite a high frequency of unknown cause of chronic subdural hematoma; thus, if one assumes that almost all subdural hematomas are caused by trauma, it seems reasonable to say that in many cases the injury may be very slight, similar to that experienced by all of us several times during our lives. Why only a few people sustain this type of hematoma after slight injury is still open to question, as no single antecedent factor common to all cases has been found. Virchow noted that the lesions were more common in insane than in sane persons. In the present series none of the patients was insane prior to the development of the hematoma; however, 48 of the 60 patients had rather severe psychic disturbance at the time of admission to the hospital. Chronic alcoholism is also believed to be a causal factor in the development of the lesion.[2] Twelve of the 60 patients were known to be addicted to alcohol. The significance of this high percentage is not entirely clear; however, alcohol addicts as well as insane persons are prone to sustain cerebral injury through irresponsible actions.

Symptomatology and Diagnosis

A latent interval of perhaps months from the occurrence of trauma, and slow development of symptoms is characteristic of chronic subdural hematoma. The long delay of symptoms contrasts with the delay of perhaps only hours that

usually follows bleeding into other intrabody spaces. This slow development was useful to Virchow in his recognition of "spontaneous" pachymeningitis. He and later authors attributed the progressive development of symptoms to repeated slight bleedings from the capillaries on the inner surfaces of the membranes; but such evidence never was found at operation or at necropsy; usually the gross content of the hematoma was perfectly homogeneous. Trotter[11] suggested that the development of the clinical picture was due to repeated bleeding from the responsible vessel; this theory likewise has not been proved. Some authors [13,14] suggested that the lesion was a neoplastic process.

Gardner's[3] explanation of the slow development of symptoms of chronic subdural hematoma has received general acceptance. According to Gardner the mechanism of the delayed appearance of symptoms is dependent on osmosis. He believes that the subdural space is anatomically unique: there is no lymphatic drainage; thus, when a blood clot is formed in the subdural space, it cannot be absorbed and it becomes surrounded by a semipermeable membrane. The colloidal-osmotic pressure draws fluid into the membranous sac and the hematoma becomes enlarged. Gardner experimented with cellophane sacs containing whole canine blood and found that when they were immersed in spinal fluid or placed in the subdural space in dogs, the contents of each sac increased in weight up to about 100 per cent within a few days. Zollinger and Gross [15] in their studies showed that disintegration of blood produced a slow rise in osmotic pressure.

The duration of the latent interval perhaps is correlated with the size of the hematoma or with the extent of the possible senile atrophy of the brain. In those cases of the series considered here in which it was possible to determine the age of the hematoma, the variation of the latent interval was from two weeks to eight months (average, nine weeks).

The symptoms of chronic subdural hematoma may be slight for a long time or they may be misleading or, as stated by Pette,[16] "pachymeningitis is found where it is not diagnosed, and diagnosed where it is not found." Characteristically, in contrast to most other intracranial lesions, chronic subdural hematomas cause more generalized symptoms. For example, whereas most tumors of the hemisphere, epidural hematomas, and acute subdural hematomas produce localizing neurological symptoms such as paresis, aphasia, anisocoria and spasticity, chronic subdural hematomas are more likely to produce what must be presumed to be generalized cerebral symptoms such as mental confusion, drowsiness, and psychosis. In this series, as stated, 48 of the 60 patients had severe psychic disturbance (Table 1) and 10 of these patients before their admission to the neurosurgical service were admitted to the Department of Psychiatry and a diagnosis of neurosurgical lesions was subsequently made, usually because of the development of papilledema. It therefore is wise to hesitate before making a diagnosis of chronic subdural hematoma in the patient who is completely alert and oriented.

Frequently one suspects the diagnosis on first meeting the patient: his appearance shows neglect of personal grooming and suggests mental derangement. Occasionally other lesions, such as glioblastoma multiforme, cause

similar signs. A history of trauma may help to establish the correct diagnosis, yet it must be kept in mind that slight injury often aggravates the symptoms of an intracranial tumor, and the patient is then likely to attribute all of his complaints to the trauma, forgetting that he had been feeling not completely well before it occurred.

The incidence of various symptoms in patients of this series is tabulated in Table 1. Headache, the most frequent complaint of the patients, usually was not severe but rather like a dull pressure. However, the headache can be very severe and without objective symptoms, as it was in a patient recently admitted to the Cleveland Clinic. He was completely alert and his only complaint was a headache so severe that it simulated neuralgia. In 27 cases the spinal fluid was examined and was normal in 11 cases—protein content was elevated in 16; the spinal fluid was xanthochromic in 7. The Wassermann reaction was positive in 2 of 19 specimens tested.

TABLE 1

Symptoms in 60 Patients Having Chronic Subdural Hematoma

Symptoms	Incidence, No. of Patients
Psychic disturbances	48
Headache	43
Papilledema	32 (of 58)
Neurological symptoms, localizing (paresis, aphasia, convulsions)	12
Vomiting	9

The frequently misleading nature of the symptoms is exemplified by the fact that the tentative diagnosis was correct in only half of the patients of this series (Table 2).

TABLE 2

Tentative Diagnoses in 60 Cases of Chronic Subdural Hematoma

Tentative Diagnosis		No. of Cases
Correct		30
Incorrect		27
	Intracranial tumor 15	
	Cerebral contusion 5	
	Epidural hematoma 2	
	Other 5	
Not recorded		3

Although both encephalography and angiography often will aid in verifying a tentative diagnosis of subdural hematoma, and either of the procedures may show the lesion, bilateral angiography seems to be the procedure of choice if a subdural hematoma is suspected. The diagnosis of a hematoma is more certain when the vessels are seen pressed back from the skull in an A-P angiogram than it is when a dislocation of the ventricular system is found in an encephalogram. Also, if the lesion is bilateral, the latter dislocation may be slight. In some cases only an oblique angiogram will be diagnostic, as the hematoma may be located anteriorly or posteriorly. The preoperative differential diagnosis between tumor and subdural hematoma is important, because the hematoma can be treated with evacuation through a burr hole, which is a simpler procedure than turning a skull flap as for a tumor.

Trephine often is used as a diagnostic procedure in cases of suspected subdural hematomas; but if the findings are negative, another diagnostic procedure usually will be necessary. If a subdural hematoma is present, the dura in most cases appears greenish or dark bluish at trephination. However, in one of the patients in this series in whom an epidural hematoma was suspected, the color appeared to be normal; hence, the dura was not opened and the outcome was fatal. It therefore seems advisable to open the dura in all cases of explorative burr holes.

Treatment and Results

When a subdural hematoma has been diagnosed, the only adequate treatment is surgical removal of the lesion. It usually is preferable to remove the hematoma through one or more trephines, and in the Clinic in Copenhagen it has been routine practice to make anterior and posterior burr holes. Quite often the lesion has been discovered through one of the parietal burr holes made for the purpose of ventriculography, and then that specific opening has been used therapeutically. The content of the chronic subdural hematoma commonly is a dark fluid that is easily evacuated by suction and washing with saline solution. However, if the hematoma is of more recent onset, it may be somewhat solid, and complete removal through a small burr hole may be difficult. Suction through a soft rubber catheter introduced into the subdural space may be helpful in those cases. It has been routine procedure in the Clinic in Copenhagen to try to open the inner membrane and the arachnoid in an attempt to drain the hematoma to the subarachnoid space. In the Cleveland Clinic[17] it is believed to be advantageous to try to distend the compressed brain by injecting saline solution through a lumbar puncture. If this is done, the arachnoid at the site of the burr hole should not be opened, as the fluid may escape. Usually there is only slight bleeding on evacuation of a chronic subdural hematoma; however, as the removal of the hematoma may not always be complete, a rubber drain is left in each burr hole for about 48 hours.

In our series, if the correct diagnosis could not be established either by the clinical examination or by the roentgenographic studies, the preoperative

diagnosis often was intracranial tumor, and in those cases a skull flap was turned down and complete removal of the membranes often was possible. The amount of membrane removed does not seem to be a factor in the patient's recovery, the postoperative courses after minimal removal comparing favorably with those following removal of most of the membrane when a skull flap was turned down. The cerebral cortex frequently appeared dehydrated, as described by Christensen.[18] Other authors[5] found at autopsy that there appeared to be edema of the underlying cerebral tissue.

Of the 60 patients in this series, 12 died (9 during hospitalization and 3 following discharge from the hospital). At the time of follow-up, of the remaining 48 patients 30 were without complaints and doing very well; 5 had several minor complaints but the results were considered to be good; 10 had various degrees of incapacity (some could not do their former work); 1 was completely deranged mentally; and 2 could not be traced.

At the time of follow-up, the most common complaints were headache, impairment of memory, and fatigue. A few patients had slight paresis, which in some was homolateral with the hematoma.

A surprisingly long time elapsed after surgery until the patients in the two best groups were completely well—an average of seven months. The interval of time was especially long for the elderly patients.

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