Chronic subdural hematomas: a review

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Present knowledge of the still controversial pathogenetic, ultrastructural, diagnostic, and treatment aspects of chronic subdural hematomas is reviewed.

KEY WORDS • subdural hematoma • neomembrane • cerebrospinal fluid • experimental hematoma • ultrastructural study • computerized tomography

Since the detailed description of pachymeningitis hemorrhagica interna by Virchow in 1857, the pathophysiological, ultrastructural, diagnostic, and management aspects of chronic subdural hematomas (SDH's) have remained controversial. The aim of this article is to elaborate the present state of knowledge of chronic SDH in order to remind the practicing neurosurgeon of the principles that are generally accepted today.

Physiopathogenesis of Chronic SDH

Origin of SDH

Chronic SDH's are clearly delineated fluid collections located between the dura mater and the arachnoid. Long-standing SDH's are enclosed within a hematoma capsule. The initiating factor in chronic SDH is either subdural bleeding or parainfectious effusion. In the late clinical stages, these are indistinguishable physiopathologically, and, to some extent, clinically and diagnostically. Virchow's hypothesis that chronic SDH's result from a generalized inflammatory disease of the dura mater, called "pachymeningitis hemorrhagica interna," is no longer accepted; he did not take into consideration the fact that the dura reacts entirely nonspecifically to blood, fibrin, or fibrin degradation products with formation of a well vascularized hematoma capsule.

The origin of blood accumulation within the subdural space is usually traumatic, caused either by direct or indirect trauma to the cranium, such as acceleration injuries with tearing of the parasagittal bridging veins or Mittenzweig's vessels, by movement of the brain in relation to its coverings. Subdural hemorrhage may also occur in combined lesions of the cortex and the arachnoid, in fractures of the skull with tearing of the adjacent dura as well as lacerations of the venous sinuses, and in traumatized arachnoid cysts. Nontraumatic origins of subdural blood or fibrin accumulation are convexity arteriovenous malformations and aneurysms, other cerebrovascular lesions, hemorrhagic diathesis, infectious diseases, brain tumors, especially convexity meningiomas, and meningeal carcinomatosis or sarcomatosis.

Factors Promoting SDH

In addition to traumatic or nontraumatic causes of SDH, various other factors may increase the vulnerability of the bridging veins or Mittenzweig's vessels, the patient's tendency to bleed, and the hematoma size. Mechanical factors include low intracranial pressure (ICP), cerebral atrophy, and excessive deformation of the cranial vault in infancy or during delivery. Low ICP may occur in disease-related or traumatic cerebrospinal fluid (CSF) fistulas as a result of lumbar puncture, due to iatrogenic or disease-induced dehydration after implantation of a CSF shunt or spontaneously. Low ICP promotes excessive blood congestion of the bridging veins with consequent dilatation and increased tension of the vessels, which are further stretched by a downward displacement of the brain and consequently more vulnerable to the movement of the brain within its coverings due to decreased cerebral volume. Minor subdural bleeding is not immediately stopped because of the reduced counter-pressure of the cerebral hemispheres. The same principles are valid in cases of cerebral atrophy and external hydrocephalus, where CSF is present in excessive amounts.
amounts on the surface of the cerebral hemispheres. Susceptibility of the infant skull to deformation is an additional mechanical factor promoting the development of SDH.

Hematogenic factors promoting SDH are coagulopathies, such as hemophilia, thrombopathies, hepatogenic coagulopathies, and anticoagulant therapy. An interesting phenomenon of which the etiology is not yet clear is the elevated urinary estriol and estradiol levels in males with chronic SDH. The ectatic capillaries in the innermost layer of the dura mater in chronic SDH are believed to be similar to the vascular network of the skin in alcoholic hepatopathies with elevated estrogen levels. Consequently, those abnormal vessels would be responsible for repeated rebleeding into the subdural space. However, in the majority of chronic SDH cases, more than one of the previously mentioned contributing factors are present, and they have a cumulative effect. This explains the fact that chronic SDH is principally a disease of older age, in which physiological brain atrophy, frequent head trauma, and coagulation disorders due to therapeutic or prophylactic anticoagulation or alcoholic hepatopathy play a cumulative role.

**Development of Chronic SDH**

The inner layer of the dura mater has a very high reaction potential for cellular organization, and contains a very fine network of interconnected capillaries. It can be argued that, depending on cerebral counterpressure, accumulation of blood, fibrin, or fibrin degradation products within the subdural space may lead either to cellular organization and resorption of the subdural collection or to the development of a gradually enlarging chronic SDH. Often SDH's that are diagnosed in the acute stage are combined with edema of the adjacent hemisphere due to a concomitant cerebral lesion, such as brain contusion. In those cases, the subdural bleeding is stopped by the edema-induced counterpressure of the brain, and may be resorbed and organized if the subdural collection is not too large. This explains the greater incidence of chronic SDH associated with minor cranioencephalic injuries without concomitant brain swelling and also in the older age group with its reduced cerebral counter-pressure due to physiological brain atrophy.

The reasons for the development of a chronic SDH, and especially its increase in size, are still not fully understood. The lack of complete cellular organization and resorption of subdural blood or fibrin accumulation may be due to an already present latent coagulation disorder as well as excessive fibrinolytic activity in the cells of the neomembrane. Ito, et al. Yamamoto, et al. stressed the importance of local hyperfibrinolysis, which causes liquefaction of the subdural blood clot and continuous hemorrhage from the sinusoidal vessels of the neomembrane, in the development and enlargement of a chronic SDH. A gradual increase of fibrinogen degradation product levels from acute to chronic SDH suggests that fibrinolysis takes place in SDH and leads to intermittent hemorrhage, and therefore to an increase in size of the chronic SDH.

The phenomenon of recurrent bleeding from the hematoma capsule as an etiological factor for the development of chronic SDH was assumed by Putnam and Cushing in 1932, and later by Dandy; their hypothesis was supported by the experimental studies of Apfelbaum, et al. in contrast to the osmotic gradient theory proposed by Gardner in 1932 and supported by Zollinger and Gross and Munro and Merritt. According to the latter theory, a raised osmotic gradient causes the transport of CSF into the subdural sac after encapsulation of the original hematoma and breakdown of its cellular constituents. When the pressures on either side of the sac were equal, the sac ceased to enlarge and the subdural fluid was slowly resorbed. Gardner supported his thesis by serial measurements of the protein content of the fluid which gradually decreased, and by demonstrating that a cellophane bag containing subdural fluid increased 59% in volume when immersed in a container of CSF for 18 hours. When the same experiment was repeated with human subdural membrane instead of the cellophane container, the results were less convincing: the increase in volume amounted to only 2.9%. This osmotic theory has recently been questioned on the following four grounds: 1) there was no significant increase in volume in his model when hematoma membranes were used; 2) fresh erythrocytes were always present in the hematoma fluid on repeated tapping; 3) it was not possible to demonstrate that the arachnoid acts as a permeable membrane for CSF; and 4) it has been shown that albumin, the most osmotically active protein, cannot emerge from destroyed red blood cells but is derived from plasma.

Rabe, et al. noted that the amount of total protein available from erythrocytes in the subdural space would only be one-hundredth of the amount of albumin available from transcapillary turnover. Weir compared the osmolality of subdural hematoma fluid, venous blood, and CSF, and found no significant difference between them. The hemoglobin breakdown products were shown to migrate with the alpha II and beta globulins. Thus, albumins moving from the bloodstream to the subdural space do so against the osmotic gradient postulated by Gardner. Recently, Weir compared the onotic pressure of fluid from SDH's and hygromas to that of simultaneously drawn venous blood in 20 patients. He did not find a significant difference in the colloid osmotic pressure of fluid from SDH and venous blood, whereas the onotic pressure of fluid from subdural hygromas was significantly less than that of blood. Weir's findings failed to support the modification by Zollinger and Gross of Gardner's theory that chronic SDH's grow and produce symptoms after a latent interval by attracting fluid from the blood via dural vessels. In 1970,
Suzuki and Takaku were able to achieve resolution or marked reduction of the hematoma fluid in 22 of their 23 patients by repeated intravenous administration of 20% mannitol. Even if the effectiveness of such therapy could not be reproduced, some shift of fluid out of the hematoma may theoretically take place if mannitol does not cross the neovascular external subdural membrane. The latter theory is supported by the fact that the administration of a hyperosmolar substance causes the serum to have transient hyperosmolarity with respect to CSF.

**Experimental Studies in Chronic SDH**

Attempts to produce chronic SDH in experimental animals were not successful until 1972, when Watanabe, et al., produced a clinical form of chronic SDH by inoculating a clot of blood mixed with CSF into the subdural space of dogs and monkeys. They stressed that a peculiar type of fibrin, which differs greatly from regular fibrin in its physical, chemical, and morphological characteristics, is essential to induce the capsule formation of the expanding type of hematoma. Apfelbaum, et al., repeated Watanabe’s studies, and their findings yielded no support for the latter authors’ contention that CSF is necessary for the formation of membranes, or that it retards blood clot absorption and speeds the increase in clot size. Apfelbaum, et al., found the same results when they inoculated blood/CSF mixtures, blood alone, or blood mixed with artificial CSF or saline into the subdural space of experimental animals.

Labadie and Glover compared the histological and biochemical aspects of subcutaneous hematoma in rats and subdural hematoma in man, and found a remarkable similarity. They stated that inflammatory mechanisms appeared to be essential and that CSF played no discernible role in the process of chronic hematoma formation. By treating their rats with intramuscular dexamethasone, they inhibited neomembrane formation, a finding that offered some support to the theory that the neomembrane development and subsequent enlargement of the SDH is due to inflammatory reactions of tissues in contact with large blood clots.

**Ultrastructure of Chronic SDH**

In 1975, Sato and Suzuki published their ultrastructural observations of the capsule of chronic SDH in various clinical stages. Their article summarized today’s knowledge of the ultrastructural aspects of SDH from the time that pachymeningitis hemorrhagica interna was described by Virchow in 1857 and reconsidered later by Melnikow-Raswedenk, Jones and Laurent, and others.

The dura mater has two different components: the inner periosteum (or endosteme) of the neurocranium, and the actual hard (dural) meninges which contain within their inner layer a very fine inter-connected capillary network with ampulla-like dilatation of the capillaries at their contact points. In contrast to this, the arachnoid has no significant vascularization. The capillary network of the inner dural sheath has a very high reaction potential which is entirely nonspecific, for it leads to the formation of a neomembrane when it comes in contact with blood, fibrin, or fibrin degradation products. The external and internal membranes of chronic SDH so formed correspond precisely to what was previously known as Virchow’s pachymeningitis hemorrhagica. Corresponding to the low reaction potential of the avascular arachnoid, the inner capsule of chronic SDH presents no significant vascularization. The external membrane of chronic SDH is very rich in blood vessels and contains giant capillaries with a lumen 80 µ or more in diameter.

These vessels resemble veins, but they do not have a complete investment of the pericyte layer or of smooth-muscle cells as is usually seen in veins. Sato and Suzuki observed these giant capillaries, which have also been described as resembling the vascular network in hepatic disease, and are very similar to healing or granulation tissue found elsewhere in the body. The growing capillaries showed pseudopod-like protrusions of endothelial cells without any or with a very incomplete basement membrane, leading to an abnormally high vascular permeability. The transport of substances and migration of blood corpuscles occur easily through open gaps between adjacent endothelial cells, as is the case at the intercellular gaps of vessels in inflamed tissue. According to Sato and Suzuki, the permeability of the capillary walls decreased in patients treated by osmotherapy. Well developed capillaries present with pinocytotic vesicles of yet undetermined significance. As reported in different circumstances such as inflammation or vascular injuries, so-called “clear cells,” which probably represent the process of vascular degeneration, have been found among normal endothelial cells within the capsule of chronic SDH's. It is concluded that bleeding must occur easily and repeatedly from these partially degenerated capillaries, and contributes to the causes of growth of chronic SDH.

Apart from the abundant neovasculature described, the external membrane contains a large number of collagen fibrils and migrating cells similar to those seen in granulation tissue, which increase in vascular structures with the advancing age of the hematoma. Sato and Suzuki observed a decrease of the content of endoplasmic reticulum and collagen in fibroblasts with advancing age of the chronic SDH or following osmotherapy, resulting in a hematoma membrane that consisted almost entirely of fibrous material similar to the dura mater.

**Neuroradiological Diagnosis of Chronic SDH**

Apart from bilateral isodense chronic SDH, where carotid angiography and scintigraphy are the impor-
tant diagnostic procedures, there is no doubt that computerized tomography (CT) of the skull is the most effective and harmless diagnostic tool for detecting chronic SDH. There is general agreement among neuroradiologists and neurosurgeons that SDH may present on CT scans in at least three different forms: hyperdense SDH, with an attenuation coefficient higher than the surrounding brain (35 to 45 EMI units); isodense SDH of the same density as brain (14 to 24 EMI units); and hypodense SDH, with a lower attenuation than brain (4 to 14 EMI units). According to Scotti, SDH's were found to be hyperdense in 100% of acutely ill patients; isodense in 70% of the subacute group, and hypodense in 70% of the chronic group. Scotti, and others stated that CT scanning is probably the most accurate method of determining the age of a chronic SDH, especially in comparison with clinical or angiographic criteria.

Isodense chronic SDH's, particularly bilateral ones, may cause some problems in CT diagnosis. Kim, et al., reported visible displacement of cortical sulci in 100% of their seven cases with isodense chronic SDH, and shifting of cortical veins away from the inner table of the skull was diagnosed in 45% on infusion scans. Moeller and Ericson stressed the importance of the characteristic ventricular deformity, leading to the correct CT diagnosis of isodense chronic SDH in 100% of their cases. According to Tsai, et al., contrast-enhanced CT scans are essential for the evaluation of isodenuating chronic SDH; linear areas of contrast enhancement along the medial boundary of the collection are characteristic, and occur frequently enough so that angiography may be avoided. Protrusions of cortical vessels seen within the avascular subdural space on carotid angiography are indicative of adhesions and chamber formation in chronic membranous SDH. The diagnostic accuracy of nuclear medicine imaging (brain scan) in chronic SDH has been demonstrated by Hurwitz, et al. Scintiscanning was accurate in predicting chronic SDH in 93% of their cases, while echoencephalography was accurate in only 44%. Even if radionuclide brain scanning is a very useful screening method in detecting chronic SDH, CT scanning or cerebral angiography are usually required to give additional information on the intracranial conditions in anticipation of operative intervention.

Treatment of Chronic SDH

Untreated chronic SDH leads to death in most instances, either by hematoma-induced cerebral decompensation or as a result of concomitant infections, such as pneumonia, and worsening of the patient's general condition. Spontaneous resolution of the subdural collection is rare, and untreated patients, who survive, mostly children, may present with calcified or osified chronic SDH. The neurological literature presents a wide variety of approaches to the treatment of chronic SDH, and its operative indication is almost universally accepted. The fact that spontaneous resolution of chronic SDH has been observed has encouraged some clinicians to treat patients with bed rest alone or dehydrating agents; however, at least one clinical trial did not advocate this conservative method, mostly because the SDH did not decrease in size within a reasonably short period of time. Ultrastructural studies have shown considerable fibrosis of the hematoma capsule after osmotherapy. Prevede achieved regression of chronic SDH's by irradiation in three cases, but this form of conservative therapy cannot be supported.

Emptying a chronic SDH through fontanel tap is a method of treatment only possible in children. Loew and Kivelitz cured 51 of 71 infants with chronic SDH by this method. Internal drainage of the subdural fluid into a body cavity such as the pleura or the circulatory system has improved treatment possibilities in infants and small children; this method has an advantage over external drainage or repeated tapping in that no fluid, electrolyte, or protein losses occur. Because of frequent valve malfunctions due to the high protein content of the hematoma fluid, and because of high infection rates associated with subdural circulatory shunts, peritoneal drainage of chronic SDH has become the therapeutic method of choice in small children. Closure of the peritoneal shunt has been found to occur within several weeks, an interval usually adequate for complete resolution of the hematoma. The fear that brain development might be hindered if capsulectomy is not performed in infantile membranous chronic SDH does not appear to be justified, for Collins and Pucci demonstrated by repeated capsule biopsies at varying intervals that capsule thickness and vascularization rapidly decrease in cases with properly functioning peritoneal drainage. The latter finding has been confirmed in other series. Perret and Graf proposed subdural galeal shunting in chronic SDH or hygromas. They believed that the latter operation was simpler to perform and led to results as good as those associated with the subdural peritoneal shunting procedure.

With the present knowledge of the pathophysiology of chronic SDH, the twist-drill or burr-hole craniostomy and slow continuous external drainage of the subdural collection seems to be generally accepted today as the rational approach to this lesion in children beyond the infant period and in adults. Tabaddor and Shulman compared the results of twist-drill craniostomy with closed-system drainage (21 cases), multiple burr holes with drainage (22
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cases), and primary craniotomy (28 cases) in 71 unselected patients with chronic SDH. Whereas in the twist-drill craniostomy group none of the patients deteriorated and there were only two deaths, 10 in the multiple burr-hole group and 14 in the craniotomy group deteriorated or died.

Craniotomy with removal of the membranes encapsulating the SDH was once thought to be required in all cases, but the latter procedure is presently deemed of less importance than adequate drainage of the liquefied hematoma. Swien and Gelety's comparative study of capsulectomy versus simple burr-hole evacuation demonstrated that the latter procedure produced better results. At discharge from the hospital, 78% of their 50 patients who underwent initial burr-hole evacuation for chronic membranous SDH were classified as being essentially normal or in good condition, whereas only 50% of the initial craniotomy-membranectomy group of 19 patients achieved that status. Tyson, et al. recently reevaluated the role of craniectomy in the treatment of chronic SDH. They achieved clinical improvement in six of seven patients; these patients had undergone an unsuccessful sequence of conventional surgical procedures, ranging from burr-hole drainage to craniotomy and membranectomy, before the cranial vault overlying the SDH was finally resected. Hubschmann reported results similar to those of Tabaddor and Shulman in his 22 patients treated by twist-drill craniostomy and closed-system drainage. This procedure, originally proposed as a temporary emergency measure while preparing for more definitive treatment, allows steady, slow, and more complete evacuation of the subdural collection and a more gradual reexpansion of the compressed brain.

Direct perforation of the skull with the cutting edge of a No. 18 spinal needle has been shown to be an effective means of evacuating a chronic SDH in five of seven patients so treated. However, slow, continuous drainage through a catheter inserted into the subdural space seems to be more effective, and is only possible through a twist-drill or burr-hole trephination. The subdural suction drainage of hematomas proposed by Matrical may have some advantages in draining partially clotted subacute SDH, but usually the closed-system drainage proposed by Tabaddor and Shulman will be sufficient in draining the subdural collection by gravitational effects. It is generally accepted that craniotomy is reserved for those instances in which: 1) the subdural collection reaccumulates; 2) there is solid hematoma; or 3) the brain fails to expand and obliterate the subdural space.

Some additional measures to facilitate rapid cerebral expansion following removal of a chronic SDH should be mentioned. Adequate hydration or even mild hyperhydration of the patient, with normalization of eventually disturbed electrolyte and protein balance, are the most important additional measures, especially in infants. The results of intraventricular injection of physiological saline solution have not been statistically significant, for the brain may relax again a few hours later. Varying results have been achieved by filling the CSF spaces with fluid from a lumbar tap; however, this is generally considered useless, and may be dangerous. Postoperative positioning of the patient's head in a 30° Trendelenburg position for 3 to 5 days in order to increase the CSF pressure and facilitate brain reexpansion has been proposed by Svien and Gelety, but up to now no statistical evidence of the effectiveness of this method has been offered. Attempts to increase intra- and postoperative brain expansion by creating cerebral swelling by clipping cortical veins of the hemisphere adjacent to the SDH have been discontinued because of unsatisfactory results.

Conclusions

It is no longer justified to postulate that a late increase in the size of a chronic SDH occurs either because of an osmotic or oncotic mechanism. Rather, it represents the result of repeated microhemorrhages from the neocapillary network in the outer membrane, with additional aggravation by the fibrinolytic activity of fibrinogen degradation products. Ultrastructural studies of the neovasculature of subdural membranes support today's pathophysiological concepts by providing evidence of an abnormally high vascular permeability. Computerized tomography should undoubtedly be considered the most accurate method for detecting chronic SDH and planning operative intervention. In treating chronic SDH, the twist-drill craniostomy and closed-system drainage of the subdural collection seem to be today's most rational approach to this lesion in children beyond the infant period and in adults. Craniotomy, membranectomy, and craniectomy should be reserved for those instances in which the subdural collection reaccumulates, the brain fails to expand, or there is solid hematoma.

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