SUBDURAL HEMATOMA
INTRACRANIAL
ADULTS
WILLIAM A. COX, M.D.
FORENSIC PATHOLOGIST/NEUROPATHOLOGIST

October 22, 2009

General Information

Subdural hemorrhage is bleeding below the dural membrane, whereas epidural hemorrhage, as previously discussed, is bleeding above the dura, i.e. between the dura and the overlying calvarium or base of the skull. The most common cause of a subdural hematoma is some form of direct impact trauma either as the result of an assault, fall or vehicular accident. Acute subdural hemorrhages can also result from sudden acceleration-deceleration of the head in which there is no injury to the head as would be the case in a whiplash-like injury such as occurs with a rear-end collision by a motor vehicle, blast injury or violent shaking during torture as reported by Pounder et al in 1997.

Although some forensic pathologists do not believe there is such an entity or 'spontaneous subdural hemorrhage,' others disagree. Leestma clearly states that 'spontaneous subdural hemorrhage' does occur, doing so primarily in infancy due to abnormalities of coagulation, either inherited (inherited platelet disorders) or acquired; during delivery; some inherited errors of metabolism (mitochondrial disorders such as Alpers disease and Menke disease and glutaric acidemia type 1 (GA-I); vascular anomalies and other conditions (benign extra-axial subarachnoid space enlargement in infancy, which can give rise to abnormal pericerebral fluid collections in either the subdural or subarachnoid space and stretching of the fragile cortical bridging veins giving rise to the potential for spontaneous rupture).

Subdural hemorrhage may occur at any age, however, they are especially prevalent in the two extremes of life, infancy and the elderly. In both infancy and the elderly there is a large subarachnoid space. In infancy the enlarged subarachnoid space is developmental, whereas in the elderly it is due to atrophy of the brain. In both cases the bridging veins must traverse a widen space, thus making them very vulnerable to shearing stresses.

In adults the most common cause of acute subdural hemorrhage is impact trauma as the result of a fall or assault in approximately 70% of cases. Acute subdural hematomas are seen in about 24% of motor vehicular accidents. What is interesting to note is if you look at those patients who have been unconscious in excess of 24 hours, who have no mass lesion, but do have diffuse brain damage, 89% are victims of MVA and only 10% are victims of a fall or an assault. In approximately 30% of cases there is no evidence of a preceding traumatic event.

It would appear the difference in the incidence of acute subdural hemorrhage in falls and assaults vs. that occurring in motor vehicular accidents is related to the rates of angular acceleration/deceleration. The impact trauma of falls and assaults is associated with higher rates of angular acceleration/deceleration as compared to the much slower rates in motor vehicular accidents. Another important factor is the direction of the angular acceleration/deceleration. The greatest shearing stress on the bridging veins is when the angular acceleration/deceleration is anterior-posterior or posterior-anterior rather than side-to-side (lateral) or top of the head to the base of the skull (superior-to-inferior).

The disruption of the bridging veins can occur in the space between the cortical surface, the arachnoid, and the dural sinuses or at the points of juncture with the arachnoid or the dural sinuses.

Although the major source of blood in subdural hemorrhages is the bridging veins, superficial cortical arteries can also serve as a source. Typically, rupture of bridging veins gives rise to subdural hemorrhage in the frontoparietal and parasagittal region, whereas arterial rupture occurs in the temproparietal region. From a pragmatic standpoint it is very difficult to determine the actual source of the bleeding. However, if the clinical onset of symptoms is rapid, the most likely source of bleeding is arterial.

There is another source of bleeding into the subdural space, which although uncommon, does occur and that is a ‘burst lobe.’ A burst lobe typically occurs in the temporal lobe, however they have also been reported in the frontal lobes. It consists of a focus of intraparenchymal hemorrhage associated with an overlying contusion, which ruptures through the contused cortical surface giving rise to subarachnoid and subdural hemorrhages.

Subdural hemorrhages can also occur due to prominent distention of the cortical veins leading to their rupture. This can be the result of a prominent increase in vascular volume or due to thrombosis of cortical veins or a dural sinus.

Some have reported that in their experience subdural hemorrhages in the interhemispheric fissure is suggestive of an underlying coagulopathy, either inherited (hemophilia) or due to anticoagulation therapy.

Most subdurals originate in the vertex region of the cerebral convexity, most likely due to prominent confluens of the cortical veins. Because the blood can spread freely throughout the subdural space, it will cover the entire hemisphere, draining downward giving rise to accumulation in the anterior and middle fossae, and into the posterior fossa, the latter due to drainage through the tentorial opening. It often can also be found on the superior surface of the tentorium cerebelli directly beneath the inferior surface of the occipital lobe.

Most believe that subdural hemorrhages of at least 50 cc can cause symptoms, with death occurring when the volume exceeds 100 cc. There are two potential exceptions to these quantities: The first is infratentorial subdurals, which require only a minimal quantity of blood to induce neurologic symptoms due primarily to the small anatomic space and the presence of the brainstem. The second is supratentorial subdurals in the elderly who have substantive brain atrophy, which has given rise to widening of the subarachnoid space. These patients may require up to 65 cc more of blood than 50 to 100 cc to induce neurologic symptoms.

Subdural hematomas are typically unilateral, although some have said they can be bilateral in up to 20% of cases. Most are associated with acute subarachnoid hemorrhage, coup/contracoup contusions, other cerebral injuries and epidural hemorrhages. Subdural hematomas can occur singularly in approximately 13% of cases.

Subdural hematomas are generally far more extensive than epidural hematomas due to the latter, in most cases, not being able to extend beyond suture margins due to the tight adhesion of the dura mater at these margins.

Although, acute subdural hemorrhages constitute a neurosurgical emergency, there is one particular location, which deserves special mention and that is those occurring within the posterior fossa, which has been mentioned briefly above. Acute subdural hemorrhages

occurring in this region are especially dangerous due to the small anatomic space and the presence of the brainstem. Thus, it requires only a small subdural accumulation of blood to exert mass effect with its consequent compression of the brainstem and resulting unconsciousness, respiratory depression and cardiorespiratory arrest. Consequently, a person who has fallen and struck the back of their head or who has been assaulted and has been struck on the back of their head are to be considered an absolute neurosurgical emergency and taken to a hospital, most especially one with a neurosurgical service, as soon as possible. It is best to approach these patients as if they have no leeway as far a time is concerned.

What must be understood is for the subdural hematoma to be considered as the underlying cause of death it must exert mass effect (midline shift as manifested by uncal grooving, tonsillar grooving, herniation of the cingulate gyrus on the side of the hematoma or herniation of the occipital lobe into the middle fossa or the temporal lobe into the frontal fossa). If the subdural consist of a thin rim of hemorrhage, being but a few millimeters in thickness, it cannot be used as the underlying cause of death due to the fact it did not produce any mass effect. This is not to say the underlying cause of death cannot be due to impact trauma to the head, which although only produced a thin rim of subdural hemorrhage, but did produce substantive diffuse axonal injury.

Historically neuropathologists have classified SDH into acute, subacute and chronic. Not everyone agrees with this classification believing that from a pragmatic standpoint there should only be two classes, acute and chronic. However, the more clinical approach through the utilization of scanning appears to support the more traditional neuropathology classification with acute represented by clotted blood, subacute a mixture of clotted and liquid and chronic being totally liquid.

Clinical presentation-Acute Subdural Hematomas

In 63% of cases (Leestma) these usually present within a few hours of the initiating event, with all substantive acute SDH manifesting themselves within 24 hours; some will extend this period out to 72 hours. Approximately 33% of these patients will have a lucid interval before coma develops, although most of the patients are drowsy to being in frank coma from the moment of the traumatic event. Typically these latter patients have associated intraparenchymal hemorrhage in addition to the evolving acute SDH. Should

the patient have a lucid interval they will manifest a unilateral headache and a slightly enlarged pupil on the same side as the evolving subdural hemorrhage. The clinical manifestation of stupor, coma, hemiparesis and unilateral papillary enlargement are signs of a large SDH. The association with intraparenchymal traumatic lesions (diffuse axonal injury) is associated with a protracted recovery course and the genesis of postconcussive syndromes.

For a diagnosis of postconcussive syndrome at least two of the following must have occurred: 1) a period of unconsciousness lasting more than 5 minutes, 2) a period of posttraumatic amnesia that lasts more than 12 hours of a traumatic brain injury, or 3) a new onset of seizures (or marked worsening of a preexisting seizure disorder) that occurs within the first six months after traumatic blunt injury (TBI). There must also be documented cognitive deficits in either attention (concentration, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information). Accompanying the cognitive disturbances, there must be three or more symptoms that are present for at least 3 months following the closed head injury. These include becoming fatigued easily; disordered sleep; headache; vertigo or dizziness; irritability or aggression with little or no provocation; anxiety, depression, or affective liability; apathy or lack of spontaneity and other changes in personality (e.g., social or sexual inappropriateness).

Mortality

Those acute subdural hematomas, which occur in serious TBI Glasgow coma scores of 3 to 5 have a mortality rate of 25% under the age of 30 and a mortality rate between 60 to 75% over the age of 60.

Macroscopic Appearance

When viewed at the time of autopsy, acute SDH appears as dark red congealed mass with some having a minor liquid component. It is important that the quantity and location of the acute SDH be documented. Following the removal of the SDH the appearance of the dural and leptomeninges must be noted, paying particular attention to color. If the dura and leptomeninges have a yellow color than this is evidence of a previous subdural. If only the leptomeninges have a yellow color than this is evidence of a previous subarachnoid hemorrhage without associated subdural.

Following noting the location of the SDH and or subarachnoid hemorrhage, the brain should be gently rinsed with cold water. Than inspect the cerebral vasculature looking for the source of the SDH. It is rare that you will identify the actual source. It is very important that the blood on the surface of the brain be rinsed away prior to formalin fixation. If you do not remove the blood on the surface of the brain prior to fixation it will cause the blood to congeal and adhere to the blood vessels. When you attempt to remove the congealed blood following formalin fixation to examine the vessels, they will tear thus obliterating any evidence of the source of the bleeding.

One of the consequences of an acute SDH is focal compression of the superficial cortical vessels, which in turn gives rise to focal areas of cortical ischemia and hemorrhages, thus simulating the appearance of an acute contusion suggesting a focus of traumatic impact. Unlike epidural hematomas, which give rise to flattening of the underlying gyri and narrowing of the sulci, acute SDH, although compressing the underlying cortex, does not give rise to the broad flattening of EDHs. It does however give rise to midline shifting of the brain, which manifest, by cingulate gyral and uncal herniation on the side of the SDH; this is accompanied by tonsillar grooving and often collapse of the ventricles on the side of the SDH with shifting of the collapsed ventricles across the midline.

Uncal herniation refers to impaction of the anterior medial temporal gyrus, which is called the uncus, into the anterior portion of the tentorial opening resulting in its compression by the edge of the tentorium. This movement of the uncus gives rise to compression of the third cranial nerve (oculomotor) on the same side as the displaced uncus causing dilatation of the pupil on the same side (ipsilateral). This is due to the compression of the parasympathetic fibers, which are peripherally located in the oculomotor nerve. Often these patients will be in coma, which is due to compression of the midbrain against the opposite tentorial edge by the displaced parahippocampal gyrus (remember, the uncus is at its most anterior end). If the shifting of the parahippocampal gyrus is severe this can also cause compression of the opposite cerebral peduncle, giving rise to Babinski sign and hemiparesis on the side of the SDH (Kernohan-Woltman sign). The actual compression of the opposite cerebral peduncle is called Kernohan's notch. To look at this in another fashion, Kernohan's notch is an ipsilateral condition, in that a left-sided primary lesion giving rise to a left-to-right shift will produce a Kernohan's notch on

the opposite side (right) and thus give rise to a clinical presentation of motor impairment (hemiparesis) or paralysis (hemiplegia) on the left side of the body. The reason the motor impairment is on the same side as the SDH is because the cortical fibers forming the right cerebral peduncle, which is compressed, eventually cross at the lower border of the medulla where they form the pyramidal decussation; thus, the motor fibers of Brodmann's area 4 of the right cerebral hemisphere supply the voluntary muscles of the left side of the body, hence, hemiparesis or hemiplegia appears on the left side, which is the same side as the SDH.

Stroking the lateral aspect of the sole of the foot brings about the Babinski sign. This is followed by an immediate slow tonic dorsiflexion of the great toe in addition to slight spreading of the other toes. What is most important is the movement of the great toe. If it extends it indicates a problem regardless of what the other toes do. The Babinski is not a muscle stretch reflex, but a plantar skin reflex. It is a strong indicator of a disorder of the pyramidal system (upper motor neuron lesion).

In addition to compressing the upper brainstem, the displacement of tissue can give rise to compression of either the anterior and or posterior cerebral arteries as they pass over the edge of the tentorium. This can give rise to infarction in the distribution of these arteries. The anterior cerebral supplies the medical surface of the frontal and parietal lobes including the paracentral lobule, parts of the lingulate gyrus and the precuneus, the anterior 4/5 (four fifths) of the corpus callosum, approximately 1 inch of the lateral surface of the frontal and parietal lobes next to the medial longitudinal fissure, anterior portions of the basal ganglia and the internal capsule and the olfactory bulb and tract.

The branches of the posterior cerebral artery are divided into two sets, ganglionic and cortical. The ganglionic branches supply much of the thalami and walls of the third ventricle, tela chorioidea of the third ventricle and the choroids plexus.

The cortical branches are divided into two main vessels, the posterior temporal (temporo-occipital) and the internal occipital artery. The posterior temporal supply the anterior portions of the inferior surface of the temporal lobe. The largest branches supply the lateral surface of the cerebral hemisphere immediately anterior to the preoccipital notch. The more posterior branches supply the occipitotemporal and lingual gyri. The internal occipital artery divides into the parieto-occipital artery and the calcarine artery, both of

which supply different regions on the medial aspect of the occipital lobe and portions of the splenum of the corpus callosum. Thus, these branches supply the medial and inferior surface of the occipital lobe and the inferior surface of the temporal lobe with the exception of the temporal pole. Branches of these arteries extend onto the lateral surface of the brain and supply the inferior temporal gyrus and variable portions of the lateral occipital region; some of the branches from the medial surface supply much of the superior parietal lobule. The calcarine branch supplies the primary visual cortex (area 17), thus occlusion of the posterior cerebral artery produces a contralateral homonymous hemianopsia, frequently sparing macular vision. It is believed that anastomoses between branches of the middle and posterior cerebral arteries in the region of the occipital pole probably account for preservation of macular vision.

Subacute Subdural Hematoma

From a clinical perspective most acute SDH manifest themselves in the first 24 hours and all by 72 hours, whereas subacute SDH manifest themselves between 3 to 14 days. What is important to remember is that all SDH begin as acute lesions. What appears to be the underlying factor as to whether a subdural hemorrhage manifest within the first 24 hours (acute) or over the next 3 to 14 days (subacute) is the rate of accumulation of the blood, the ability of the brain to compensate for the increasing mass effect and the coexistence of other cerebral lesions. Should the lesion continue to expand, albeit slowly, a point is reached in which the brain can no longer compensate to the ever increasing intracranial pressure (ICP), which leads to herniation, brainstem compression with or without circulatory compromise, respiratory depression and ultimately cardiac and respiratory arrest.

Macroscopic Appearance

The gross appearance of a subacute hematoma at the time of autopsy will depend on the length of time of survival since the initiation of the subdural, i.e. the proportion of clot vs. liquid blood. If the patient dies closer to the 2 to 3 days of the traumatic event the clot will have a very dark red appearance. However, as time progresses toward the 14day mark it will become blacker and then brown with fragments having an orange to yellow to yellow-green color. The physical change of the subdural is due to normal fibrinolysis, which occurs within a clot, and the migration of macrophages into the clot. The

macrophages begin to digest the lysed red blood cells (RBC) giving rise to hemosiderin, which is the first pigment to appear. As the quantity of hemosiderin increases the color of the clot takes on a more brown color. With continued phagocytosis the pigment hematoidin appears after approximately 7 days giving rise to the orange to yellow to yellow-green color of the clot. This process culminates into the entire clot becoming liquefied somewhere between 14 to 21 days.

Microscopic Appearance

The appearance of these clots microscopically first published by Munro & Merritt in 1934 and then subsequently slightly modified by Leestma and Grcevič and Knight. The following is the modified version:

Time after injury	**Clot**	**Dural side**	**Arachnoid side**
36 hours	fresh RBC	fibroblast activity at junction	fibrin
4 days	RBC lose sharp contour	2-4 cells thick fibroblastic layer	fibrin
5 days	same as above and variability in staining	3-5 fibroblastic layer	fibrin and pigmented macrophages
7-8 days	lysis of RBC, fibroblast enter clot	12-14 fibroblastic layer	fibrin and pigmented macrophages
11 days	RBC are broken into islands by fibroblast and early capillary formation	fibroblasts migrate around entire clot	pigmented macrophages noted on arachnoid side of clot
15-17 days	very few intact RBC remain, definite capillary formation	fibroblastic thickness ⅓ to ½ that of dura	fibroblastic membrane noted on arachnoid side. Clot may appear to be completely encased

From a pragmatic standpoint if the clot has a brown color with evidence of a discrete membrane you are between 7 and 14 days after the traumatic event.

From a clinical perspective these patients are far more difficult to diagnose due to the ever so gradual accumulation of the subdural hemorrhage. These lesions are not completely benign for they have a mortality rate of approximately 22%.

Chronic Subdural Hematoma

These lesions typically make their clinical appearance around 21 days. There is some overlap between late subacute SDH and chronic SDH. Generally it is stated that subacute SDH present between 3 to 14 days, however they may extend out to as long as 26 days. From a pragmatic standpoint it can be very difficult to draw a sharp demarcation between a late subacute SDH and an early phase of a chronic SDH.

Chronic SDH are more likely to occur in patients over the age of 50, which is probably due to an increase in the subarachnoid space secondary to a decrease in brain size (atrophy).

A significant proportion of chronic SDH are associated with head trauma, which can be quite minor, most especially in the elderly. As previously stated, this is due to atrophy with enlargement of the subarachnoid space, which means the bridging veins, which due to the aging process are increasingly more fragile, have a longer distance to travel before reaching the venous sinuses. What is of interest is the expanding subarachnoid space can accommodate a larger volume of fluid, some say as much as an additional 65 cc over and above the 50 to 100 cc, before neurologic symptoms arise. This may account for the slower evolution of symptoms in the elderly in acute, subacute and chronic SDHs.

In 25 to 50% of cases, there is no known head trauma. Having said that, approximately 50% of these patients have a history of alcoholism or epilepsy, thus often not remembering the trauma. Other risk factors are over drainage of ventriculoperitoneal shunts and bleeding disorders, including conditions relevant to anticoagulant medication, and cerebral atrophy. There also appears to be some racial variation, as an example, there is a higher incidence in Japanese. There is also a higher incidence in males.

Clinical Presentation

The smaller chronic SDH, those less than 100 cc, often are asymptomatic, however, those which are in the range of 100 to 150 cc often produce symptoms such as headache, personality changes, confusion, stroke-like features and dementia, which often are misinterpreted as to their underlying causation. If these lesions continue to expand they

can produce a mass effect that ultimately exceeds the brains ability to compensate at which point they develop the signs of herniation, which have been previously discussed.

Macroscopic Appearance

What distinguishes a chronic SDH is the formation of an organized membrane, which completely encases the fluid mass. The contents of the chronic SDH vary according to the age of the lesion. More recent hematomas have a tan to red-brown appearance and are quite viscous, often with a gelatinous appearance to the surface. It is not uncommon to have focal areas of a dark red color, which is due to rupture of the fragile capillaries giving rise to fresh bleeding. The propensity of these fragile capillaries to rupture is the underlying reason why these lesions can suddenly expand, and if large enough, create a mass effect. What is also important to remember is these lesions can persist for months with their contents taking on a dark brown very viscous appearance. The membrane encasing these lesions is very fibrous. If the lesion has a volume in excess of 100 cc, the underlying brain will have a flattened appearance. This flattened appearance differs from the preservation of the gyral and sulci contours of an acute subdural. The reason for the flattening of a chronic SDH has much to do with the thickness of the arachnoid side membrane, i.e. it has an analogous structure and function of the dura in an epidural hematoma giving rise to flattening of the gyri and narrowing of the sulci.

The underlying mechanism for the genesis of chronic SDH and why some continue to expand eventually giving rise to neurologic symptoms is repetitive bleeding from the fine fragile capillaries within the membrane encasing the contents of the chronic SDH The underlying leptomeninges often have a yellow brown color.

As stated, what defines a chronic SDH is the formation of a well-defined membrane, which completely encases the contents of the subdural. The length of time this takes is somewhat variable, but typically requires 2 to 3 weeks. Distinguishing an older subacute SDH from an early chronic SDH can be difficult; however, if you see a complete membrane encasing the subdural it is best to classify it as a chronic subdural.

Microscopic Appearance

Time after injury	Clot	Dural side	Arachnoid side
18-26 days	Completely liquefied	Membrane thickness same as dura	Membrane ½ as thick as dura. Pigmented macrophages throughout membrane
27-36 days	Large capillaries	Well-formed membrane	Well-formed membrane
1-3 months	Large giant capillaries with secondary hemorrhage	Hyalinization of membrane. Less cellular	Hyalinization of membrane
3-4 months	No original RBC. If RBC are seen due to rebleeding	Hyalinized membrane with possible focal calcification or ossification	Hyalinized membrane with possible focal calcification or ossifycation
1 year and beyond	No RBC	Dural appearance	Dural appearance

Treatment

Subdural producing neurologic symptoms due to significant mass effect, whether acute, subacute or chronic, should be evacuated. The main indication for surgery is the presence of symptomatic mass affect in the form of focal neurologic deficits, or seizures. The thick clotted blood of an acute subdural usually requires a craniotomy. The outcome following surgical evacuation will depend on the severity of the initial neurologic deficit and the interval between the traumatic injury and surgery.

The liquefied contents of a chronic SDH can often be removed through a series of burr holes.

In regard to treatment of a subacute SDH, that will depend on the stage of development. If it is early and still has substantive clot formation demonstrated by scans than a craniotomy may be the best approach. If however it were an older lesion, which is primarily liquefied than drainage via burr holes would be the best approach.

Reoperation for acute and chronic SDH occurs in about 15% of cases.

Subdural Hygroma

These lesions are represented by a subdural collection of clear, xanthochromic or blood stained fluid. One of the proposed mechanisms that underlie the formation of this lesion

is a valve-like tear in the arachnoid, which allows CSF into the subdural space. The reason the hygroma enlarges is that CSF continues to leak into the subdural space, but due to the valve-like action of the tear in the arachnoid it cannot escape. What separates these lesions from a chronic SDH is they typically have no membrane and there are no subdural veins present. The other mechanism is that a hygroma is nothing more than the residual cavity of a large, incompletely organized chronic subdural hematoma. Liquefaction of the hematoma transforms it into a fluid of low viscosity which, with the passage of time, turns from turbid dark brown to xanthochromic and finally to clear fluid. The capsule of collagenous tissue that had formed around the chronic subdural hematoma prevented it from collapsing during this entire process; this interpretation of hygromas is supported by the observation of hematomas in various stages of organization and transition to hygromas, and by microscopic evidence of old hemorrhage in the walls of hygromas.

Most of these lesions are small and clinically insignificant.

There are two types of subdural hygromas simple and complex. In the simple hygromas there is typically no history of trauma, and generally no associated lesions or if present are minor. They typically occur in infants and children. Also subdural veins are not present in contrast to that seen in chronic SDH and there is no evidence of brain atrophy. The complex hygromas are commonly seen in the elderly, typically after trauma, often associated with extracerebral hematomas or intraparenchymal injuries. They, however, have been reported in children following Haemophilus meningitis. They have also been reported following ventricular shunting due to the sudden decrease in ICP. This can lead to leakage of cerebral spinal fluid (CSF) into the subdural space especially in the elderly who have substantive brain atrophy. They can also occur following neurosurgical procedures and have been reported in dehydration of the elderly, and those with lymphomas and connective tissue diseases.

Treatment

The larger lesions, which produce mass effect, require drainage via burr holes, which if it completely removes the fluid will allow the cavity of the hygroma to completely collapse with its consequent apposition of membranes and closure of the valve-like defect, assuming that is the underlying mechanism in its formation.