Case Report

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Traumatic Subacute on Chronic Subdural Hematoma: An Autopsy-based Case Report

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ABSTRACT

A typical consequence of head trauma and a notable finding during such autopsies is subdural hematoma (SDH). Upon diagnosis, a variety of clinical manifestations, including the lethal acute SDH, subacute, or chronic SDH (CSDH), may be present. Sometimes, these traumas may be so trivial that the exact history of incidence may be difficult to elicit, especially in the cases of subacute and chronic SDH. The signs of subacute SDH are usually evident only after three days to 2 to 3 weeks and grossly appear during autopsy as dark red-black liquefied blood with blood clots. CSDH is a gelatinous membrane with outer and inner layers adherent to dura matter. The present case is of a 47-year-old man with histories of falls, at his residence, first two years no history of treatment was there and a recent fall, about two weeks and led to the initial presentation of hemiparesis and hospitalization with a diagnosis of SDH, the patient died after a week. This article discusses the findings observed in the case where the cause of death was due to the complication of SDH, which was found to be subacute on the chronic type. Such cases usually present late to the hospital due to delayed symptom onset, and high suspicion of such cases should be kept when patients have a history of minor trauma or bleeding disorders and especially when the patient is elderly. Such cases necessitate immediate diagnosis via imaging modalities of the intracranial hemorrhages and necessary intervention.

Introduction

An enclosed deposit of old blood between the dura and the arachnoid membrane is a chronic subdural hematoma (CSDH). In 1857, Virchow coined the term "pachymeningitis haemorrhagica interna" for such cases. The subdural hematoma (SDH) occurs due to the rupture of bridging veins. Direct head trauma is the source of CSDH in about 50% to 70% of the instances. Sometimes, the trauma is so minor which is been ignored. Headache, disorientation, weakness, and lethargy are symptoms that appear gradually after the trauma. CSDH is frequently caused by vascular abnormalities, coagulopathy, epilepsy, and falls that do not strike the head.

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Several less prevalent aetiologies, such as coughing, sneezing, straining due to constipation, weightlifting, and amusement park rides, have been identified in addition to trauma.[1] In neurosurgical practice, the management of SDH is one of the essential tasks. SDH is a common outcome of trauma and a significant finding during autopsies of such cases. Different clinical presentations may occur upon diagnosis, ranging from a fatal acute SDH to a more insidious CSDH. Small SDH is usually detected on computed tomography (CT) scans following head trauma or instances where there is no history of direct impact to the head. On average, three weeks after the initial trauma, between 1% and 6% of untreated acute SDHs convert into CSDHs. These two entities, however, exhibit very different dynamics and behaviors. First, the presence of blood in the subdural region causes inflammation. Later, an organized neo-membrane of granulation tissue forms and attains structural integrity inside the subdural space as stimulated fibroblasts and capillaries multiply. Internal septations and fluid-filled loculations are simultaneously created by dural adhesions. Persistent protein-rich CSDHs are reinforced by exudation from outer membranes and fresh bleeds, resulting in hemosiderin deposits due to these newly generated fragile structures.[2] In cases of secondary trauma, rebleeding results in a heterogeneous mixture of fresh blood and partially liquefied hematoma, often with a sediment level, and is a feature of chronicity of the SDH. The traditional classification of chronic, subacute & acute is straightforward but ineffective at presenting concise information. SDHs are isodense compared to brain parenchyma on CT scans during the subacute phase, which usually occurs 72 hours to 3 weeks after trauma to the brain.[2] The symptoms, such as headache, altered mental state, or focal neurological impairments, which can be signs of physical trauma, can be mimicked by ischaemic strokes and intracranial hemorrhages, which can also be non-traumatic. Furthermore, the elderly are at increased risk of hemorrhage even following trivial head trauma due to aging-related changes, such as increased dura adherence to the inner table of the cranium, atherosclerosis of the cerebral vessels that lead to delicate bridging veins, brain atrophy, and increased use of anticoagulants.[3] The medicolegal issue is whether proper medical intervention is being provided for the diagnosed condition and how its outcome influences the patient's health. We report a case of an older adult's death caused by subacute on chronic subdural hematoma.

Case Report

Case History: The deceased was a 47-year-old male with an alleged history of fall at his residence hall about 10 to 15 days back. He complained of bed wetting and hemiparesis, for which he was taken to a local hospital and then to a nursing home and was finally admitted to a tertiary care hospital for one week before death. As per history, the deceased allegedly also had a fall in the bathroom at his residence about two years back. As per clinical records of the nursing home, the deceased was diagnosed with a case of large subacute SDH on the CT brain. As per the Medical Certification of Cause of Death (MCCD) issued by the tertiary care hospital, the cause of death was bilateral chronic SDH with raised intracranial pressure as a consequence of the fall.

Autopsy Findings: On external examination, an anteroposteriorly placed hypo-pigmented scar of size 4 cm x 2.5 cm was present over the right temporal region of the scalp. The anterior end of the scar was 10 cm above the right mastoid process. A scar of size 3 cm x 2.5 cm was present over the posterior aspect of the right leg in the lower third part. The medial end of the scar was 4 cm behind and 2 cm above the medial malleolus. An obliquely placed 4.5 cm x 1 cm scar was present over the medial aspect of the right leg in the middle third part. These injuries correlated with the alleged history of the fall two years back. A blackish scab abrasion with a scab falling off from the margins of size 5.5 cm x 4 cm was present over the front of the left leg, just below the knee. This injury was correlating with the recent history of the fall of the deceased. On internal examination, dark reddish-colored fluid with blood clots filled in a encapsulated collection of size 15 cm x 8 cm x 2 cm and 14 cm x 7 cm x 1.5 cm with a gelatinous membranous adhesion to the inner layer of dura present over the frontal, parietal and temporal regions of the right and left cerebral hemispheres respectively (**Figure 1**). The outer membranous encapsulation adherent to the inner aspect of the dura was yellow-colored, and the inner membrane was reddish-black-colored **(Figure 2)**. The encapsulation caused compression symmetrically around the frontal, parietal, and temporal regions on both sides, corresponding to an overlying subdural collection. Atherosclerotic plaques were present in the left posterior cerebral, right posterior communicating, and middle cerebral arteries on both sides. Uncal herniation was present on the left side, and tonsillar herniation was on the right. The cause of death was opined as "cerebral damage consequent to subdural hemorrhage in an old case of fall at the same level." All the injuries were antemortem and caused by blunt force impact.

Figure 1: Shows encapsulated gelatinous yellow-colored membranous (black arrow) adhesion to the inner aspect of dura present over the frontal, parietal, and temporal regions of both the cerebral hemispheres.

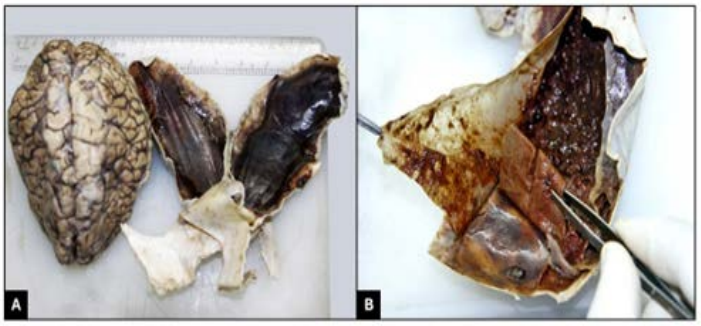

Figure 2: A. Shows the reddish-black discoloration of the inner membrane adherent to the dura. **B.** Shows the yellow-colored outer membrane of the encapsulation adherent to the inner aspect of the dura and the collection of dark reddish-colored fluid with blood clots inside.

Discussion

Damage to the communicating or bridging veins, which crosses the "subdural space," results in SDH and is one of the major causes of traumatic intracranial hemorrhage. In many cases, there may be no visible history or proof of any brain trauma, especially in the very young and very old. The shearing or rotating forces that move the brain inside the skull are the main culprits for injuries to cortical veins, as they stretch and rupture these fragile veins with thin walls.[4] This case demonstrates that SDH may be encased in gelatinous "membranes" and solidify into a firm, rubbery capsule. The elderly are most likely to have such old subdural blood collections because of their brain atrophy, which makes space for hematoma without showing any discernible clinical impact. Those who are more likely to fall, such as chronic alcoholics, also have CSDHs. The complications of SDHs include compression of the brain and internal herniation through the tentorium cerebelli due to its space-occupying lesion. The injuries to brain parenchyma due to trauma hastens the fatal outcome.[5]The study on spontaneous nontraumatic CSDH by Missori et al. observed that all such cases had raised cerebral venous pressure.[6] In our case, there was a history of trauma, and as per MCCD, raised intracranial pressure was one of the clinical findings that was associated with the cause of death. Upon concluding, in traumatic and non-traumatic cases, raised intracranial pressure, depending on the duration and severity of SDH, contributed to the cause of death in present case.The intracranial effect of aging is an important factor for intracranial hemorrhage, such as SDH, even in minor trauma.[7] In this case, the individual suffer falls in his bathroom and hall room. Similarly, Teo et al. observed that most SDHs were caused by falls at home or in a domestic setting.[8] The various intracranial effects of aging and an isolated SDH without skull fractures or injuries over the scalp or head constitute about one-third of all SDH cases.[9,10] In our case, there was scar present over the right temporal region of the scalp. As in this case, many SDH instances show no visible signs of soft tissue swelling, skull fractures, or scalp lacerations.[3] In the case of SDH, lumbar pain and headache & hemiparesis are common presenting clinical symptoms. In the present study, the deceased complained of hemiparesis. Similar presentations have been observed in other studies.[11-13]. In our case, the internal findings were missing after the initial CT was done two years back at the time of the fall, as the deceased was residing in a rural area. The old scar over the head represents the etiology and co-relation between the fall at ground level 2 years back and CSDH because the internal findings demonstrate the existence of typical membranes. The subacute changes within the CSHD also correlated with the fall history of 10 to 15 days back. Aoki et al. observed a similar interval for developing subacute SDH.[14] Subacute SDH at 2-3 weeks, the foci of red-black blood are seen, and the clot begins to liquefy similar to our case where dark reddish-colored fluid with blood clots collection was found inside the yellow colored membranes of CSDH adherent to the dura, inferring subacute on CSDH.[15]

Conclusions

This case of subacute on chronic subdural hematoma underlines that careful assessment for any intracranial pathology in general and cautious evaluation for trivial trauma, anticoagulant therapy, history of bleeding, and age-related risk factors such as elderly patients. Subacute and chronic SDHs, which progress slowly over time via the gradual onset of symptoms, can have delayed presentation as the nature of the present case, increasing the risk for complications if these conditions are left untreated. So, urgent imaging studies & prompt neurosurgical intervention, are required for better outcome. This case underlines the importance of timely diagnosis and treatment to avoid neurological deterioration with an improved overall prognosis.

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