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Delayed Acute Subdural Haematoma: A Trivial Fall Causing a Rare, Life Threatening Complication.

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ABSTRACT

A 65-year-old man presented to us with giddiness and fall. His examination including a detailed neurological assessment was normal. CT brain revealed only age related cerebral atrophy and the patient was discharged after observation for 24 hours. 34 days later, he was brought to the emergency room in altered sensorium and a repeat CT revealed a large acute and subacute subdural haematoma with intracranial cerebral herniation needing emergency drainage by burrhole craniotomy. The patient recovered well following the procedure. Delayed acute post traumatic subdural haematoma is a rare condition that may occur following even the most trivial of falls and is often associated with poor outcome. It brings to light that a CT brain done immediately after the trauma is insufficient to predict delayed deterioration and calls for high level of vigilance in cases of mild head trauma and a low threshold for repeating a CT brain.

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INTRODUCTION

Traumatic brain injury is one of the most frequently encountered problems in the emergency room. While the management protocols for severe injuries are well defined, there exists a lack of consensus regarding the management of mild injuries defined as GCS of 13-15 with a loss of consciousness lasting not more than 30 minutes and post traumatic amnesia lasting not greater than 24 hours [1]. After, in the absence of any significant findings on Computed Tomography (CT) brain performed soon after the injury, most of these patients are believed to be out of harms way. This can at times be fraught with danger because though rarely, some of these cases may develop symptoms at a later date and the lack of suspicion can cause a delay in the diagnosis and in turn lead to a poor outcome. Delayed post traumatic acute subdural haematoma (SDH) is one such condition which may present days to weeks after the trauma and although existing literature is limited, it is suggestive of a poor outcome [2,3]. As it is impossible to monitor patients with mild head trauma in hospital over longer time periods perhaps early recognition of symptoms and a low threshold for repeating CT scan seem like the few strategies that may help make a difference. We report a case of an acute subdural haematoma presenting 34 days after the history of head trauma. This is one of the most delayed presentations of a post traumatic acute SDH reported in literature. Prompt neurosurgical intervention proved crucial in achieving a favourable outcome.

CASE REPORT

A 65-year-old man with no pre-morbidities presented to the emergency room with giddiness followed by a fall. There was no history of altered sensorium or loss of consciousness following the fall. On examination, the patient was conscious and oriented. His vitals were stable and systemic examination including a detailed neurological examination was normal. Routine laboratory investigations were normal and CT Brain revealed age related cerebral atrophy. The patient was observed for 24 hours and was discharged. 34 days later he was again brought to the emergency, this time in altered sensorium. The family reported irrelevant speech and drowsiness few hours prior to the admission. Preceding trauma, febrile illness or intoxication were ruled out on history. His vitals were stable and blood glucose was normal. On examination, the patient was drowsy, disoriented with a Glasgow Coma Scale score of 10/15. CT Brain was done urgently and revealed acute and sub-acute subdural haematomas with mid line shift and mass effect.

At the time of first admission, routine laboratory investigations were normal and CT brain (figure 1) was suggestive of age related cerebral atrophy. The second CT brain (figure 2) done 34 days after the first one revealed a subacute subdural haematoma along the left cerebral convexity. Another similar but small acute subdural haematoma along the right frontal convexity was noted with intracranial cerebral herniation. Investigations including bleeding time, clotting time, PT, aPTT and platelets were all normal. Holter monitoring and EEG were done to look for the possible cause of giddiness and were normal.

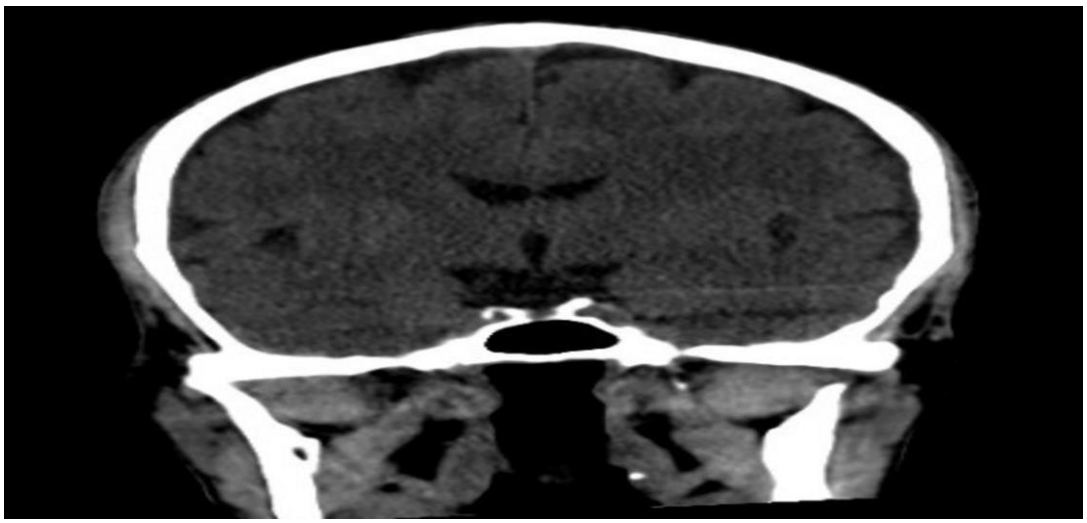

Figure 1: CT brain immediately following the trauma showing essentially normal brain with age related cerebral atrophy.

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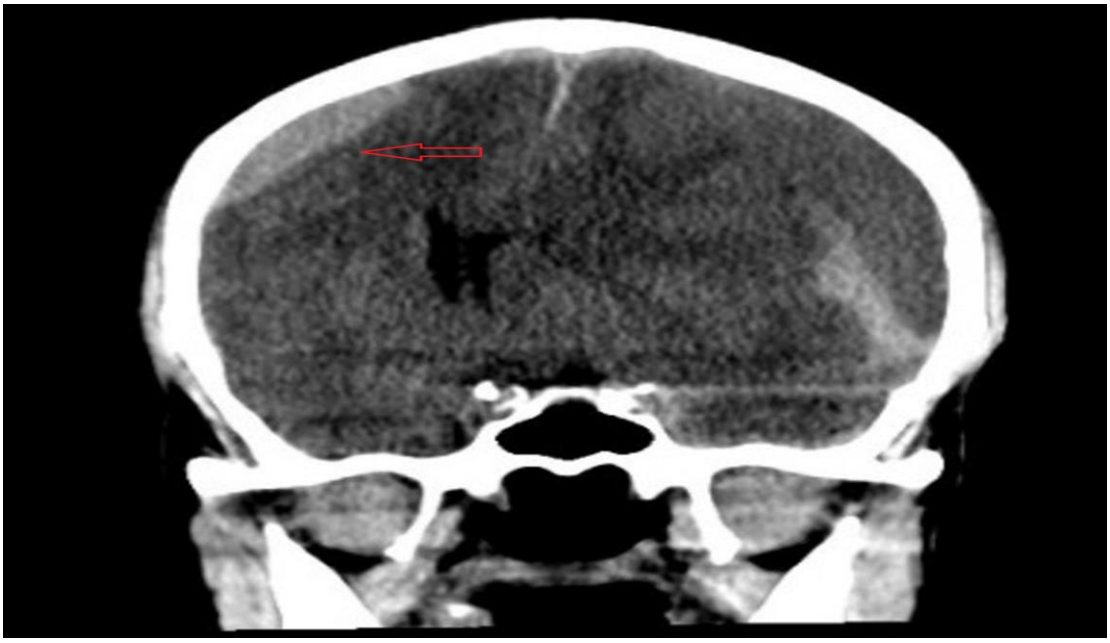

Figure 2: CT brain 34 days after the trauma showing subacute subdural hematoma along the left cerebral convexity and an acute subdural hematoma on the right side causing mass effect

In view of the large sized bilateral SDH and intracranial cerebral herniation, the family was given a guarded prognosis and the patient was taken up for emergency evacuation by burrhole craniotomy.

Patient recovered well after the surgery. His sensorium improved and he was stable at the time of discharge. Neurological examination on followup after 3 months was essentially normal.

DISCUSSION

'Traumatische Spat Apoplexie' or delayed post traumatic acute subdural haematoma was first described by Bollinger in 1891 as an acute SDH that occurred 2-4 weeks after mild head trauma [2]. In most cases of delayed acute SDH a CT brain done soon after the trauma shows no evidence of bleed but the patient may deteriorate at a later date when a repeat CT reveals an acute SDH. While delayed SAH, EDH and ICH are well known entities, delayed post traumatic acute SDH is rarely mentioned in literature and even less frequently described in detail [4-7].

The development of an acute SDH is attributed to the rupture of bridging veins as they traverse the subdural space to drain into the dural venous sinuses. The sinuses are strongly adherent to the inner table of the skull while the bridging veins lack support as they cross the subdural space, making them prone to injury [3]. However, the pathophysiology of injury in case of a delayed acute SDH is believed to be more complex and includes a combination of abnormal autoregulation, vasoparalysis, vasospasm and free radical injury [8].

Autoregulation of cerebral blood flow is intrinsic to preventing secondary insults to the brain following an injury. What is of interest here is that abnormalities in autoregulation are neither determined by the severity of the injury nor are they bound by time. They can present soon after the injury or gradually over time and can be short lived or persistent irrespective of whether the injury is mild, moderate or severe [8]. Abnormalities of auto-regulation in turn lead to cerebral hyperperfusion and vasoparalysis with consecutive rise in intracranial pressure which may form coalescent small perivascular hemorrhages. Cerebral vasospasm is also believed to contribute to the pathophysiology of delayed SDH. It may develop between the 2nd and the 15th day after the trauma and hypoperfusion may occur in about 50% of the patients who develop vasospasm [9]. Vasospasm in turn causes local tissue ischemia, tissue necrosis and vascular rupture. Free radical injury that occurs locally is associated with development of inflammation and early and late apoptosis of both vascular and brain tissue and are hypothesized to contribute to the pathophysiology [10].

Old age is an important risk factor for delayed acute SDH [3]. With age, as the brain atrophies, the bridging veins have to traverse a greater distance in the subdural space and hence are more prone to trauma. Moreover, the atrophied brain has a reduced tamponade effect as it shrinks away from the skull. Oral anticoagulation is a significant risk factor even in case of minor trauma and activities that may cause sudden rise in intra-cranial tension are also known to contribute to the injury [2,3].

We undertook an extensive literature search and came across 10 well documented cases of delayed acute SDH following mild head trauma (Table 1). The time of presentation of the delayed SDH varied between 6 hours to 8 month. Although, Nafziger and Jones claimed that a post traumatic delayed haematoma should not be considered a direct effect of the trauma if it occurs beyond 8 weeks after the injury [11]. Our patient presented 34 days after the initial history of trauma hence making it one of the most delayed presentations of a delayed acute SDH. CT brain which were done soon after the trauma were normal in most cases. Some scans including that of our patient revealed cerebral atrophy which could have played a significant role in the development of the delayed acute SDH. Presence of cerebral atrophy as well as the preceding history of trauma rules out the possibility of a spontaneous acute SDH in our patient. A few cases also had skull fractures. All the cases reported by Eyal et al in 2006 had an added risk of bleeding as they were on anti-coagulation. Case reported by Matsuda et al occurred following an attempt to fix a bolt, an event that can cause acute increase in intracranial tension. Outcome was mostly poor (4 deaths, 2 vegetative state, 2 poor GCS) with only 2 of the 10 cases showing good recovery. In our patient, timely neuro-surgical intervention helped the patient achieve complete recovery over the course of hospital stay.

Table 1: Review of Literature

CASE		1st CT	INTERVAL	2nd CT	OUTCOME	RISK FACTOR
Cassin and Spitz(12)	19/F	Skull fracture	15hr	ASDH	Death	
Lesoin (13)	40/M	Skull fracture	30d	ASDH	Recovered	
Doherty(14)	24/M	Brain edema	8mo	ASDH	VS*	Brain atrophy
Aoki (15)	75/M	Skull fracture	29d	ASDH	VS	
Koumtchev(16)	70/M	Normal	1d	ASDH	Death	
Matsuda(2)	18/M	Normal	6d	ASDH	Recovered	
Itshayek (3)						
Case 1	86/M	Brain atrophy	3d	ASDH	Death	Brain atrophy
Case 2	69/M	Brain atrophy	12hr	ASDH	Death	and
Case 3	65/F	Normal	1d	ASDH	GCS 3	anticoagulation
Case 4	72/F	Normal	1d	ASDH	GCS 4	
Present case	65/M	Brain atrophy	34d	ASDH	Recovered	Brain atrophy

*VS: vegetative state

An increasing number of patients going to the emergency room with mild head injury undergo CT scan and if the scan is normal are deemed fit to follow up on out-patient basis. This practice heavily relies on the ability of the initial CT scan to detect any finding that could suggest imminent or delayed deterioration and though useful in a large number of cases would fail to be effective in cases like the ones mentioned above. However, the presentation of delayed acute SDH may be delayed by weeks, making inpatient monitoring impractical. It would thus be useful to counsel the patient and the family about the small but potentially dangerous possibility of such delayed deterioration especially in the presence of risk factors. The family must be explained about the need for early contact with the emergency room team in case of any symptoms. A low threshold for suspecting delayed acute SDH and an early repeat CT may prove to be lifesaving [12-16].

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