Subdural Hematomas: Unusual Presentation and Pattern of Recovery that Differs from TIA and Stroke

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Received Date: August 07, 2019, Accepted Date: September 10, 2019, Published Date: September 17, 2019.
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Abstract
We report on an eighty year old man who presented with sudden onset of transient, self-limiting episodes, of left sided weakness due to subdural hematomas. Our case is unique due to unusual pattern of presentation and recovery which differed from that of transient ischemic attack (TIA) and stroke.

Eight episodes occurred over a period of three days, five of which took place following admission to hospital. The weakness was grade 0-5 on the left upper limb and grade 4-5 on the left lower limb and each episode lasted for 5 to 10 minutes followed by full recovery. No altered consciousness or seizure activity was noticed and no signs of increase intracranial pressure. The right side of the body was entirely normal.

CT brain confirmed bilateral subdural hematomas with greater size on the left when compared to the right side. MRI brain showed no diffusion weighted changes suggestive of TIA or stroke and the presence of subdural hematomas. The initial treatment included octaplex to reverse the action of rivaroxaban which the patient was on for atrial fibrillation. Two days following admission the episodes of weakness had completely resolved and no surgical intervention was required.

Although extremely rare it would seem that, transient, self-limiting episodes of hemiplegia can occur due to subdural hematomas. The presenting symptom of intermittent episodes of weakness may mimic TIA or stroke and resembling to that have been reported in seizure with ictal paroxysm and cortical spreading depression (CSD). Interestingly the pattern of recovery in our case differs from that of a TIA in that recovery was immediate rather than gradual and this may emphasize one of the pathological processes, that is, pressure on the cortex from the haematoma rather than ischaemia associated with vessel occlusion.

Keywords: Intermittent; self-limiting episodes; subdural hematomas; hemiplegia; weakness

Introduction
A subdural hematoma (SDH) refers to a collection of blood between the dura and the brain. It may be acute, subacute or chronic. SDH is more common in elderly age group, patients on anticoagulant and patients with bleeding diatheses [1,2]. The clinical presentation of a SDH depends on the size of the hematoma and the degree of any associated parenchymal brain injury. Presenting complaints will vary and include aphasia, hemiparesis and hemi sensory defects which can mimic stroke or transient ischemic attacks [3,4]. There may be a “lucid interval” of a few hours after the injury where the patient appears relatively well and normal but subsequently deteriorates and loses consciousness as the haematoma forms. Hemiparesis is found in many cases; weakness of the limbs is usually mild and involves the contralateral side with a few cases of ipsilateral weakness reported.

Intermittent weakness with subdural hematomas has been rarely reported in the literature, mostly due to other associated neurological conditions such as a TIA, subtype of seizures with ictal weakness and cortical spreading depression [5]. Direct intermittent pressure on the cerebral hemisphere would be the postulated underlying mechanism.[6,7].

Case Presentation
An eighty year right-handed man with a history of chronic myeloid leukaemia, atrial fibrillation on rivaroxaban, ischemic heart disease and hypertension. Presented with sudden onset of transient self-limiting episodes of left sided weakness. The episodes lasting between 5 to 10 minutes during which the limb “became dead” (flaccid weakness) as described by the patient and was followed by a rapid and full recovery. Five episodes of weakness were reported by medical team in the hospital while three similar episodes happened over a period of three days prior to admission. There was no history of trauma and no seizure activity reported.

On examination the weakness was grade 0-5 on the left upper limb and grade 4-5 on the left lower limb (according to motor score). There was no altered consciousness and no signs of raised intracranial pressure (emesis, pupil changes, and headache). The right side of the body was entirely normal.

An urgent CT brain confirmed acute and subacute bilateral subdural hematomas, the largest was on the right side measuring up to 21 mm in maximal depth compared to 7 mm on the left. Midline shift of approximately 4 mm with underlying mass effect of the right cerebral hemisphere was noted [Figure 1]. MRI brain revealed no diffusion weighted changes suggestive of TIA or stroke and confirmed bilateral subdural hematomas [Figure 2]. The patient was admitted to the acute stroke unit stroke unit for neuro-observation. Octaplex (25 IU/kg) was given to reverse the effect of rivaroxaban which the patient was taking for atrial fibrillation. Two days following admission the episodes of weakness had completely resolved and no surgical intervention was required.
Discussion

SDH results from bleeding between the dura and the arachnoid membranes. Age and neurologic status, as assessed with the Glasgow coma scale (GCS) are important prognostic indicators. Although extremely rare and has not been reported independently due to SDH, this unusual presentation with self-limiting episodes of weakness involving one side of the body should be differentiated from other neurologic conditions which may represent different pathological mechanism of such rare presentation particularly TIA/stroke, cortical spreading depression and sub-types of seizure with ictal paralysis [8].

In this case the episodes of weakness were recurrent over a period of five days and of note both onset and recovery were immediate which has not been preceded or followed by any symptoms/signs suggestive of seizure. Although EEG was not performed on this case however the clinical pattern of symptoms, rapid and complete resolved of the weakness over a short period of time, sustained and stable neurological status over extended period of follow up, the fact that no surgical intervention or antiepileptic medications required, all these together reduced possibility of seizure or CSD and raised possibility of acute SDH as distinctive underlying cause. Furthermore presence of acute and subacute SDH was confirmed radiologically.

Interestingly the hemiplegia in this case was ipsilateral to the side of the largest hematoma which would be infrequently reported in the literature. Acute symptomatic SDH is a neurologic emergency that may require surgical intervention to prevent complications like irreversible brain injury and death due to hematoma expansion, elevated intracranial pressure and brain herniation. The decision of operative versus non-operative management is taken on a case by case basis and is based on multiple factors. Our patient was considered initially for surgery based on his symptoms and CT findings and hence was given octaplex to reverse the effects of anticoagulant. However he was ultimately treated non-operatively due to several factors which included high risk of bleeding, advanced age, anticoagulant treatment and the history of chronic myeloid leukemia [9]. Our patient made a complete recovery and was discharged home after five days.

In our case Octaplex was used to reverse the effects of rivaroxaban. Although there is no data available at the moment to support the role of octaplex in the management of SDH it may help to stop the bleeding and prevent haematoma expansion, however more research is needed [10].

Conclusion

Although extremely rare it would seem that, transient, self-limiting episodes of hemiplegia can occur due to subdural hematomas. The presenting symptom of intermittent episodes of weakness may mimic TIA or stroke and resembling to that have been reported in seizure with ictal paralysis and cortical spreading depression (CSD). Interestingly the pattern of recovery in our case differs from that of a TIA in that recovery was immediate rather than gradual and the patient remained neurologically stable after a short period of complete recovery and this may represent one of the pathological process, that is, may be a direct intermittent pressure on the cortex from the haematoma rather than ischaemia associated with vessel occlusion. Even though EEG was not performed on this case, however diagnostic tests for epileptic seizures, such as an EEG and ictal SPECT should be consider ruling out possibilities.

Conflict of Interest

All the authors declared that they have no conflict of interest.

References


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Received Date: August 07, 2019, Accepted Date: September 10, 2019, Published Date: September 17, 2019.

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