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Atypical Finding of Subdural Hemorrhage

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Abstract— *Subdural Hematoma is a type of intracranial bleeding, localized to the area between the two outer meninges of the brain, Dura mater and Arachnoid mater. It is often crescent shaped. The initial presentation of SDH has a wide spectrum of manifestations, extending from headache and vomiting to Focal neurological deficits. One of the rarest forms of post-traumatic intracranial hemorrhage is ''Isolated interhemispheric subdural hematoma (ISH)'' where the bleeding occurs due torsional forces rupturing the large fixed bridging veins draining from the parieto-occipital cortex to the superior sagittal sinus. ISH has a peculiar presentation known as the Falx Syndrome. Our patient presented with a history of fall sustained a laceration over the occipital region without any focal neurological deficit and no sensory abnormalities, CT scan of the brain was done which revealed subdural hematoma in the left frontal region. However, a peculiarity of this case was the subdural hemorrhage located along the flax anteriorly on CT. A repeat CT scan was done the next day, which revealed an interval reduction in the SDH along the falx, which is a relatively uncommon type of SDH. Our patient lacked the characteristic findings of SDH which made her require a conservative treatment approach rather than surgery. Her course of stay was uneventful and hence was discharged with clinical and hemodynamic stability.*

Keywords— *Conservative management of Subdural hematoma: Falx syndrome: Isolated interhemispheric subdural hematoma (ISH): Neurology: rarest forms of post-traumatic intracranial hemorrhage*: *Subdural hematoma.*

I. INTRODUCTION

We are reporting a case of Subdural Hematoma, which is a crescent shaped intracranial bleed, occurring most commonly along the convexities of the cranial vault in the subdural space, and occurs due to the rupture of the bridging veins that run between the surface of the brain and Dural venous sinus. The peculiarity of our case is the interhemispheric location of the SDH, which is a relatively uncommon type of SDH, and is especially seen in patients with disturbances in blood clotting mechanisms (1).

II. CASE

A 90-year-old female was brought to the emergency room by her family within an hour after she slipped and had a fall in the bathroom while bathing. She sustained a laceration over the occipital region about 3cm*2cm in size associated with a scalp hematoma. There was no history of loss of consciousness, seizures, uprolling of the eyes, ear nose throat bleed, blurring of vision, focal neurological deficits, or paraplegia. Patient is known case of Pancytopenia, Hypertension, deforming Rheumatoid arthritis, Osteoarthritis of left shoulder. Status post previous spinal surgery and bilateral total knee replacement. She was allergic to Erythromycin, Penicillin, gentamicin and Amoxicillin clavulanate.

On arrival, the patient was alert and conscious, and was obeying commands. BP:156/90mmHg Pulse: 68bpm, SpO2: 99% on room air. On Neurological exam: pupils are bilaterally equal and reacting to light. Power was 5/5 on all 4 limbs. systemic examination was normal. Given the presence of a history of fall, CT scan of the brain (Figure 1) was done which revealed subdural hematoma in the left frontal region. However, a peculiarity of this case was the subdural hemorrhage located along the flax anteriorly as reported by 2 radiologists. In addition, chronic ischemic changes in bilateral periventricular area and frontoparietal white matter were seen, along with diffuse cerebral atrophy.

To look for a progression of the bleed a repeat CT scan was done the next day, which revealed an interval reduction in the SDH along the falx with a width of 8mm. However, no change was seen in the frontal subdural hematoma.

A neurosurgical consult was taken in the emergency within 45 minutes of the patient's arrival and CT scan. Since there was no midline shift, and the size of the bleed was less than 10mm, a conservative approach was taken. She was given a dose of levetiracetam prophylactically, along with acetaminophen to manage her pain and a shot of tetanus toxoid. She takes aspirin regularly, which is risk factor for the possibility of an expanding hemorrhage however the PT and INR values were normal.

Due to the SDH, the patient was shifted to the Neurology ICU for strict monitoring. During her ICU course, she was given Acetaminophen 4 th hourly for 2 days followed by Etoricoxib as needed for pain, a capsule containing a cocktail of Methylcobalamin, Niacinamide, Pyridoxine for 2 days, followed by a tablet containing Methylcobalamin (1500mcg) + Alpha Lipoic Acid (100mg) + Myo-Inositol (100mg) + Folic Acid (1.5mg) + Chromium Picolinate (200mcg) + Selenium

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(55mcg) + Benfotiamine (150mg), injection of levetiracetam 500mg twice a day to prophylactically prevent seizures for 2 days followed by an oral formulation of levetiracetam for a day. Tablet Pregabalin 75mg twice a day for 2 days, a steroid, methylprednisolone 20mg once a day, Calcium, Folic Acid, an oral Antibiotic (Ceftriaxone). She has been having hypertension for which she continued her regular medication. Patient was kept under observation for a period of 3 days. Her course of stay was uneventful and hence was discharged with clinical and hemodynamic stability.

III. DISCUSSION

SDH is a type of intracranial bleeding, localized to the area between the 2 outer meninges of the brain, Dura mater and Arachnoid mater. It is often crescent shaped because bleeding follows the contour of the overlying dura. The most common cause of SDH is trauma, with as many as 71% of cases occurring due to injury to head (2), as was seen in our case after a fall in the bathroom. SDH is seen in approximately 11% of mild-to-moderate injuries and 20% of high intensity traumatic brain injuries (3). Most common cause of injury leading to SDH varies with age, with traumatic injury being more common in adults of age 30-50 years, and fall being more common in the elderly (4). Second most common cause for SDH can be a drop in intracranial pressure due to ventriculostomy or CSF leak (5).

The most common risk factors for developing SDH include Cerebral atrophy, use of Antithrombotic therapy and other coagulopathies. Cerebral atrophy results in a larger space between the Dural membrane and cortical surface of the brain, which increases tension on bridging veins and increases the risk of their rupture even with a mild trauma (4).

Use of antithrombotic agents increases the risk of SD, as illustrated by a case-control study of 10,010 patients with a first-ever SDH, which showed that 47.3% of these patients were taking antithrombotic medications (6). Antithrombotic medications seem to play an even bigger role in the increasing risk of ISH, as shown by a study conducted on twenty-five patients with ISH and falx syndrome, of whom 23 (92% of the cohort) were taking anticoagulants or antiplatelet medications (7). This risk is increased with all antithrombotic medications, including Aspirin, Clopidogrel, Direct oral anticoagulants, and Vitamin K antagonists. This risk is lowest in case of Aspirin, with adjusted OR between cases and controls being 1.24, 95% CI 1.15-1.33. Our patient was at an increased risk for SDH, in view of her age (90-year-old) and her regular intake of Aspirin.

The initial presentation of SDH has a wide spectrum of manifestations, including headache, vomiting, signs and symptoms due to the mass effect like seizures, and site-specific focal neurologic signs. These can be hemiparesis and speech impairment (dominant hemisphere) or executive dysfunction (nondominant hemisphere) in frontal lobe SDH, speech impairment (dominant hemisphere) or sensory impairment (nondominant hemisphere) in parietal lobe SDH, anisocoria, dysphagia, cranial nerve palsies, nuchal rigidity, ataxia in posterior fossa SDH (8) (9) (10).

Focal deficits may be either ipsilateral or contralateral to the side of the SDH. Contralateral hemiparesis can occur due to direct compression of cortex underlying the hematoma, whereas ipsilateral hemiparesis can occur with lateral displacement of the midbrain caused by the mass effect of the hematoma. Such midbrain displacement results in compression of the contralateral cerebral peduncle against the free edge of the tentorium (11).

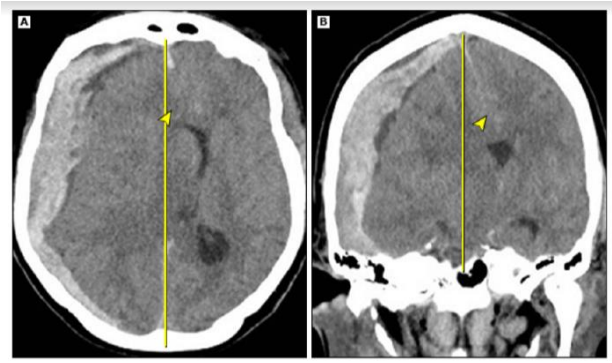

Figure 1: Computerized Tomography of the brain demonstrating interhemispheric subdural hematoma.

Isolated interhemispheric subdural hematoma (ISH) represents one of the rarest forms of post-traumatic intracranial hemorrhage. (12) ISH was first described at autopsy by Aring and Evans in 1940 (13). The bleeding occurs due torsional forces rupturing the large fixed bridging veins draining from the parieto-occipital cortex to the superior sagittal sinus (14).

When its mass is sufficiently large to compress the medial cerebral hemisphere, specific focal neurological abnormalities may occur (15). ISH has a peculiar presentation known as the Falx Syndrome, which includes paraparesis without facial weakness (16). Modality of diagnosis for ISH is computerized tomographic (CT) scan (14). The finding on CT includes interhemispheric hyperdensity, which was originally considered a sign of subarachnoid hemorrhage (SAH), the & "falx sign"; It has since been identified as a normal feature and has also been seen with interhemispheric subdural hemorrhage (17). A study was conducted to find the differential features of ISH, and CT findings were reviewed in 50 patients with SAH and 32 patients with ISH. SAH produced an anterior interhemispheric hyperdensity only, with the contour being zigzag, extending from the calvarium to the rostrum of the corpus callosum. On the other hand, ISH mostly produce unilateral crescentic hyperdensity, which is largest in the posterior superior part of the fissure (17).

Even though it has been suggested that these hematomas can resolve spontaneously and that a surgical intervention is rarely necessary (17), management of these hematomas largely depends on the size of the lesion. A small lesion which is asymptomatic may be managed conservatively, where a large lesion causing mass effects should be treated promptly (15).

This conservative approach can be rationalized in absence of clinical signs like paraparesis, dilated pupils, drowsiness, absence of GCS drop of more than 2 points, and no Cushing triad (bradycardia, hypertension and irregular respiration) on presentation. Patient should also lack the radiological features of severe disease including a clot thickness of >10mm, midline shift of >5mm, hydrocephalus, and brainstem compression (3).

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Also no progression of neurological symptoms, size of the hematoma < 15mm or volume <40ml, no features of a midline shift, paralysis of lower extremities or persistent raised intracranial pressure, thus a conservative approach was adopted (18). Our patient lacked the characteristic findings which made her require conservative approach in comparison to surgery.

SDH being a time bound emergency, may require an urgent surgical evacuation within 3-4 hours if the above indications are met. This intervention can reduce the chance of progression of the bleed and improve outcomes (30-40% mortality if performed within 3-4 hours in comparison with 75% mortality if delayed) (3).

In our patient who is a 90-year-old female with no clinically significant neurological deficits and radiological finding of a severe disease, a neurosurgery consultation was done, followed by a conservative approach including seizure prophylaxis and continuous monitoring in the ICU.

IV. CONCLUSION

Isolated interhemispheric subdural hematoma (ISH) represents one of the rarest forms of post-traumatic intracranial hemorrhage.

ISH has a peculiar presentation known as the Falx Syndrome, which includes paraparesis without facial weakness

SDH being a time bound emergency, can present ranging from headache to focal neurological deficits may require an urgent surgical evacuation within 3-4 hours if the indications are met for surgical approach.

When isolated interhemispheric subdural hematoma (ISH) presents as subdural hematoma in the left frontal region and a peculiar subdural hemorrhage located along the flax anteriorly with no clinically significant neurological deficits and no radiological finding of a severe disease, and does not meet the criteria for surgery a conservative approach is adopted for treatment.

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