A CASE REPORT ON SUB DURAL HEMATOMA

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ABSTRACT
Subdural Hematoma is one of the most common Neurological condition which involves the accumulation of blood between the dura mater and the subarachnoid mater. It most often occurs from rupture of veins which cross the surface convexities of the cerebral hemispheres resulting from a severe injury to the head or skull. As a subdural hematoma expands in the subdural space, it raises the Intracranial pressure and deforms the brain. Subdural Hematoma can be seen in all age groups. In this case, the patient is suffering from subdural hematoma along with coexisting illness such as Hypertension and Type 2 Diabetes Mellitus. Appropriate treatment was provided to the patient.

KEYWORDS: Subdural Hematoma, Dura Mater, Subarachnoid Mater, Cerebrovenous Pressure, Intracranial Pressure, Frontoparietal Region.

INTRODUCTION
Subdural hematoma is a condition which involves the accumulation of blood between the dura mater and the subarachnoid mater.¹,¹⁸ Which are the protective layers of the brain.² The blood seeps between the dura mater and the arachnoid mater, collecting inside the dura mater (brain’s tough outer lining).³ It most often occurs from rupture of veins which cross the surface convexities of the cerebral hemispheres¹,³ resulting from a severe injury to the head or skull.³,⁴,⁵,⁶

The blood may press against the brain increasing the intracranial pressure which damages the delicate tissue of the brain.³,⁸ As a subdural hematoma expands in the subdural space, it raises the Intracranial pressure and deforms the brain.⁶ Repeated bleeding and organisation causes progressive increase in size and shows laminated appearance.¹⁸ Subdural Hematoma can be seen in all age groups. In Infants the cause may be a non-accidental injury, motor vehicle accidents in young adults and fall (due to trauma or any other reason) in older persons (7). Possible causes for Subdural Hematoma³ are as follows.

1) Head injury [most common among younger people].
2) Brain shrinking (atrophy) [most common among older adults].
3) Being on medicines to prevent blood clots, such as warfarin, aspirin, and other blood thinners.

Fig. 1: Diagrammatic representation of Subdural Hematoma.¹⁸
Acute subdural hematomas frequently arise from the tearing of bridging veins within the dural border cell layer which results in blood flowing into a potential space within the dura mater (6, 10, 11). Bleeding continues via a positive feedback mechanism that causes the Cerebrovenous Pressure to increase as the Intracranial Pressure elevates. As the Hematoma enlarges, continued dissection of the border cell layer is seen. This continues until blood begins to coagulate, stopping the border cell layer dissection, and pressure within the Acute Subdural Hematoma cavity rises to equal that in the torn bridging vein or veins.\(^\text{[11]}\)

**Sub – Acute Subdural Hematoma**

Sub – Acute Subdural Hematoma develops when the clotted blood in the brain liquefies. The Clinical manifestations appear between 4-21 days after injury.\(^\text{[8]}\)

Causes of sub – acute subdural hematoma involves coagulopathies and ruptured intracranial aneurysms. Subdural hematomas have even been reported to be caused by intracranial tumors.\(^\text{[6]}\)

**Chronic Subdural Hematoma**

Chronic Subdural Hematoma develops mainly with brain atrophy and less likely following trauma. It is composed of liquid blood.\(^\text{[1]}\) The Clinical manifestations appear after 21 days of injury.\(^\text{[8]}\)

Causes of chronic subdural hematoma involve traumatic injury which causes the tearing of the bridging veins traversing from the brain to the draining dural-venous sinuses. This results in the accumulation of venous blood within the subdural space over time.\(^\text{[14]}\) Chronic subdural hematomas may also evolve from the liquefaction of an acute subdural hematoma. Liquefaction usually occurs after 1-3 weeks of.\(^\text{[6]}\)

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**Fig. 2:** A CT scan of a brain showing a subdural hematoma [noted by the red arrows].\(^\text{[12]}\)

**Fig. 3:** Intracranial Pressure due to Subdural Hematoma.\(^\text{[18]}\)

**Subdural Hematomas** can be classified into the following groups.\(^\text{[1,8,9,18]}\) They are

1. Acute Subdural Hematoma
2. Sub – Acute Subdural Hematoma
3. Chronic Subdural Hematoma

**Acute Subdural Hematoma:** Acute Subdural Hematoma develops following trauma and consists of clotted blood in the frontoparietal region of the Brain.\(^\text{[1]}\)

It is the most common type of Subdural Hematoma accounting for 24% cases of severe head injuries and has the highest mortality rate.\(^\text{[13]}\) The Clinical Manifestations appear during the first 3 days after injury.\(^\text{[8]}\)

The usual mechanism in an Acute Subdural Hematoma is a high-speed impact to the skull.\(^\text{[6]}\) This sudden impact can strain the blood vessels within the dura, causing them to rip and bleed\(^\text{[3,6]}\) resulting in brain injury and even death.\(^\text{[4]}\)

**Fig. 4:** Diagram Summarizing the cascade of events leading to the accumulation and expansion of an Acute Subdural Hematoma.\(^\text{[16]}\)

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**Fig. 5:** Diagram summarizing the pathophysiological processes involved in the formation of a Chronic Subdural Hematoma.\(^\text{[17]}\)
Sign and Symptoms\(^{3,4,5}\)

**Adults**
1) Slurred Speech
2) Difficulty in walking
3) Headache
4) Confusion
5) Seizures
6) Loss of consciousness
7) Nausea and vomiting
8) Weakness or numbness
9) Vision problems
10) Dizziness
11) Sleepiness
12) Coma

**Infants**
1) Bulging Fontanelles (The soft spots of the baby's skull).
2) Separated Sutures (The areas where growing skull bones join).
3) Feeding problems.
4) Seizures.
5) High-pitched cry.
6) Irritability.
7) Increased head size (circumference).
8) Increased sleepiness.
9) Persistent vomiting.

Risk Factors\(^{3,4,6}\)
1) Chronic alcoholism.
2) Epilepsy.
3) Coagulopathy.
4) Arachnoid cysts.
5) Anticoagulant therapy.
6) Cardiovascular disease.
7) Thrombocytopenia.
8) Diabetes mellitus.
9) Very young or very old age.
10) Head injury, such as from car crashes, falls etc.
11) Playing high-impact sports.
12) Previous brain injury.
13) Cerebrospinal fluid leak.

CASE REPORT
A 60yrs old male patient was admitted to the Neurology department of hospital complaining of fall at home followed by giddiness and difficulty in walking.

Past History: Known case of Hypertension, type 2 Diabetes Mellitus.

Past Medication History
Hypertension – tab. Telista 40 mg.
Type 2 Diabetes Mellitus – tab. Trivolib 1, H. Mixtard 50/50 15 Units.

Social History: Non – Smoker, Non – Alcoholic, decreased sleep and appetite.

Presentation of Case: Patient presented with complaints of fall at home, followed by giddiness and difficulty in walking.

Investigations: On 1\(^{st}\) Day: Complete Blood Picture was done and levels were within normal limits. Vitals were normal. Complete Urine Analysis was done which shows an increase in the Sugar and Albumin levels. Radiological Tests (Carotid Doppler, Electrocardiogram [ECG], 2D – Echo) were performed and the impression was Normal. MRI (Magnetic Resonance Imaging) Scan of the Brain was performed which confirms the presence of Anterior inter hemispheric Subdural Hemorrhage (Acute). On 2\(^{nd}\) Day: Blood Pressure and Pulse Rate were normal.


DISCUSSION
Subdural hematoma is a condition which involves the accumulation of blood between the dura mater and the subarachnoid mater\(^{2,16}\) which are the protective layers of the brain.\(^{2}2\) Subdural Hematoma can be seen in all age groups.\(^{7}\) In this case patient was diagnosed with Subdural Hematoma, Hypertension and Type 2 Diabetes Mellitus. Treatment for current condition includes inj. Cognistar 60mg iv BD prescribed for dementia like symptoms, inj. Lantus 12units s/c OD prescribed for Type 2 Diabetes Mellitus, a Cognition Enhancer (Piracetam) inj. Cognitam 15ml iv TID is given which acts by decreasing viscosity and improving microcirculation\(^{19}\), for prevention of anemic conditions or blood disorders inj. Optineuron 2amp OD was given, to reduce Gastric Acid Secretion, a proton pump inhibitor (Pantoprazole) inj. Pantodac 40mg iv OD is given, tab. Rosuvas 20mg p/o OD was given to prevent Hyperlipidemia, tab. Telma 40mg p/o OD was given for Hypertension, it acts by preventing angiotensin II from binding to angiotensin II receptors on the muscles surrounding blood vessels and decreases the effect of angiotensin II\(^{20}\) and it directly blocks the angiotensin II type I receptor that mediates the effects of angiotensin II\(^{21}\), inj. Vitamin K 10mg iv OD is given, tab. Vertin 16mg p/o TID is given to prevent nausea and vomiting and it has a long duration of action [24hrs]\(^{21}\), tab. Spinfree 1tab p/o SoS is given for severe giddiness and vertigo, It acts by inhibiting influx of calcium from endolymph into the vestibular sensory cells which mediates labyrinth reflexes.\(^{22}\)

CONCLUSION
Information must be provided to the patient regarding his current medical condition alongwith a proper explanation of drugs. Eradication of infection with amelioration of
signs and symptoms. As the patient is hypertensive, dietary sodium intake must be restricted to 1.5 g/day. Reduce Sugar intake to control Diabetes Mellitus.

REFERENCES