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Tentorial Subdural Hematoma Following Hypertension – *A Case Study*

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Abstract:- Acute subdural hematomas (a SDH) is a common pathology in head trauma. In this case report, we study about left tentorial subdural hematoma, posterior interhemispheric SDH and small hemorrhagic foci in the left temporal lobe near the temporal horn of left lateral ventricle due to hypertension. A 85-year-old male patient was confirmed to have Left hypertensive tentorial bleed. The patients had been diagnosed with CVA one and half years back and MRI Brain, CT Plain Brain was taken on the day before admission. And finally, he was diagnosed with left hypertensive tentorial bleed which is manifested with weakness in both upper and lower limbs and inability to speak and walk. The patient was treated with anti-convulsant, antibiotics, anti-hypertensive agent, psychostimulant, nootropic and anti-hyperlipidemic agent. The treatment for hypertensive tentorial bleeding requires early diagnosis, proper management of blood pressure, therapeutic management and surgical intervention if required.

Keywords:- *Head Injury, Hypertension, Tentorial Subdural Hematoma.*

I. INTRODUCTION

The convexity of the brain is where acute subdural hematoma following head injury is most frequently found[1]. One of the layers that serves as protection for the brain tissue beneath the calvarium, the dura mater, is where a subdural hematoma develops. Without the protective layers, this network of neurons and neuronal connective tissue is vulnerable to damage, beginning with the scalp and the bone components of the skull[2]. Subdural hematomas are caused by about 3% of intracranial aneurysm ruptures. 70% percent of these aneurysms originate from the middle cerebral artery or the internal carotid artery[3]. A hazardous medical condition known as hypertensive crisis is characterized by severely increased blood pressure; often, the systolic and/or diastolic readings are above 180 and 120 mmHg, respectively. There are two types of hypertensive crises: hypertensive urgency and hypertensive emergency. While hypertensive emergency manifests with end-organ damage and calls for more aggressive blood-pressure lowering, hypertensive urgency is asymptomatic ischemic strokes, cerebral hemorrhage, subarachnoid hemorrhage, head trauma, and hypertension encephalopathy are common presentations for neurological end-organ failure in associated with hypertensive crisis.[4] Whether a subdural hematoma (SDH) is traumatic or nontraumatic in origin affects its natural course. The types include, 1. Parafalcine and Tentorial Acute Subdural hematoma. 2. Posterior Fossa Acute Subdural Hematoma. 3. Nontraumatic Acute Subdural Hematoma.[5] ICH has a number of risk factors and causes. The majority are trauma, high blood pressure/hypertension, aneurysm, arteriovenous malformations.[6] The symptoms include Sudden weakness, numbness, tingling, or paralysis of the face, arm or leg, especially on one side of the body, Headache, having trouble swallowing, Eyesight loss or vision problems, Stiff neck and light sensitivity, Slurred or abnormal speech, Reading, writing, or speech comprehension issues, Changes in alertness or level of awareness, a lack of energy, fatigue, or coma, breathing issues and an unusual heart rate (if bleed is located in brainstem).[7] The treatment for hypertensive tentorial subdural hematoma includes both surgical and non-surgical procedure. Non – surgical procedures compromise administration of clotting factor if the patient was on blood thinners, controlling blood pressure lowers the chance of further bleeding; monitoring and managing ICP (pressure on brain tissue due to clot). And Surgical procedure compromise, removal of clot, in order to remove the clot, a craniotomy entails exposing the brain and removing a portion of the skull bone. It is helpful when the clot is at the brain's surface or when it is linked to an underlying brain damage. And a clot that is deeply embedded in the brain can be removed using the minimally invasive procedure known as stereotactic clot aspiration. Neuro-navigation technology, which functions similarly to GPS in autos, makes this possible.[8-9]

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II. CASE DESCRIPTION

Mr. C, a male patient of 85 years presented to the Neurology department with the complaints of weakness in both upper & lower limb and no sense in both the limbs and inability to walk & speak. He had a complaint of slip and fall in the bathroom 4 days prior to presenting to the Hospital. He has a past medical history of T2DM and Hypertension for 10 years and had a CVA attack one and a half years back after which the patient used to walk with a walker and only speaks few words after that incident. The patient has a past medication history of Tablet. Telmisartan 40mg OD, Tablet. Atorvastatin 10mg OD, Tablet. Metformin hydrochloride prolonged-release and glimepiride 500mg+1mg OD. On admission the patient has increased blood pressure level (160/80mmHg) and complete blood glucose level (338mg/dl). The other vitals like pulse rate (78beats/min), respiratory rate (25breaths/min) and temperature (97.1ºF) are normal. The MRI Brain report shows possibility of left Hypertensive tentorial bleed with adjacent contusional hemorrhage extending into ipsilateral temporal horn of left lateral ventricle should be considered. And CT Brain Plain report shows presence of left tentorial SDH (max thickness 12mm), Posterior interhemispheric SDH, Small hemorrhagic foci in the left temporal lobe near the temporal horn of left lateral ventricle. And the blood report of the patient had decreased Na (130 mmol/L), potassium (3.3 mmol/L) and lymphocytes (07%) level and the WBC (18,000 mill/ml), neutrophil (86%) level was increased. From the patient's demographics and imaging study report the patient was diagnosed to have, Left hypertensive tentorial bleed. During hospital stay the patient was provided with Ryle's tube and Foley's Catheter. Then the patient was treated with the following drugs for 10 days: Injection. Levetiracetam 1.5g BD (anti- convulsant), Injection. Citicoline IV BD (psychostimulant), Injection. Piracetam IV BD (nootropic), Injection. Pantoprazole 40mg IV BD (proton pump inhibitor), Tablet. Atorvastatin 40mg R/T HS (HMG CoA reductase inhibitor), Tablet. Telmisartan 40mg R/T OD (angiotensin receptor blocker), Injection. Sulbactam + Cefoperazone 1.5g IV BD (anti-biotic), Injection. Mannitol 100ml IV OD (diuretic), Tablet. Cilnidipine 10mg IV BD (calcium channel blocker), Injection. Labetalol 10mg IV SOS (beta-blocker), Tablet. Tolvaptan 15mg R/T BD (vasopressin V_2 receptor antagonist), Injection. Clindamycin 600mg IV OD (antibiotic), Syrup. Potassium Chloride 15ml R/T. The patient condition is improved on the course of treatment and the patient was discharged in stable condition.

III. DISCUSSION

Acute subdural hematomas that have an arterial origin are not uncommon. A potential cause of delayed rebleeding with a very high fatality rate is traumatic brain aneurysms. CT imaging was used to determine the subdural hematoma diagnosis. The CT scan was able to rule out the differential diagnoses of epidural hematoma and subarachnoid haemorrhage, which were both possible possibilities. The traumatic presentation makes a brain mass less likely to be the diagnosis than other probable diagnoses like it. Moreover, CT imaging was able to rule out the possibility that this patient's symptoms were caused by a brain mass. Patients with suspected vascular damage following traumatic brain injury should be advised to have repeated vascular investigations, and the aneurysm should be blocked as soon as the diagnosis is made. Sometimes do patients stop taking their antihypertensive medications because their hypertension has gone away. Therapy termination most often signifies a failure to follow medical instructions. The increased risk of hematomas in these individuals highlights the significance of maintaining therapy, conducting adequate blood pressure checks, and promoting patient compliance in those with established hypertension. Adherence to treatment is crucial for patients with risk factors such as hypertension, diabetes, and dyslipidaemia to avoid the complication of subdural haemorrhage. Patients who have a head injury and are on anticoagulant drugs are at risk of developing traumatic subdural hematoma. Initial studies indicated higher fatality rates ranging from 40% to 90%; but in recent years, advancements in neuroanesthesia, specialized intensive care units, and surgical techniques have changed this picture. ASDH caries still have a significant morbidity and mortality rate, nonetheless. As a result, an adequate neurosurgical treatment depends on an accurate assessment of predictive outcome characteristics. Age-related brain atrophy, greater subdural space, stretching of bridging veins, and a higher risk for trauma due to gait or orthopedic issues all contribute to the prevalence of aSDH in older people. On the other hand, their aging-related brain shrinkage enhances the compliance to the growing hematoma. With the right and timely surgical surgery, these characteristics may lessen the basic parenchymal injury caused by the first incident and boost the brain's ability to be saved.

IV. CONCLUSION

We have presented a case of 85 years male patient diagnosed with Hypertensive tentorial bleed. The patient was treated with anti-convulsant, antibiotics, anti-hypertensive agent, psychostimulant, nootropic and anti-hyperlipidemic agent. The patient condition improved on subsequent days and was discharged.

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