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Original Research Paper

Radio-Diagnosis

CHANGES IN THE BRAIN ON CROSS-SECTIONAL IMAGING IN A CASE OF NEAR SUICIDAL HANGING: A CASE REPORT.

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INTRODUCTION

Suicide is an alarming public health concern, which is on a rise in the 21st century. Unfortunately, hanging is the most common method of suicide accounting for 57.8% of suicidal deaths in India. **[1]**

The mechanism of Injury in hanging depends on various factors like the type of hanging: judicial or suicidal, body weight, the height of the drop, etc. Reports suggest that judicial hanging led to hangman's fracture (fracture of C2 vertebral arch leading to spinal cord injury), and hyoid fracture causing asphyxiation and death. However, in cases of suicide, a drop from a significant height is seldom observed and death mainly occurs due to pressure, indirectly dependent on the body weight, over the carotid artery and Juglar vein causing cerebral hypoxia.**[2]**

Patients who survive a suicide attempt, are often brought to the emergency department with different neurological symptoms of varying severity ranging from a state of full consciousness to a state of coma and complete unresponsiveness.**[3]**

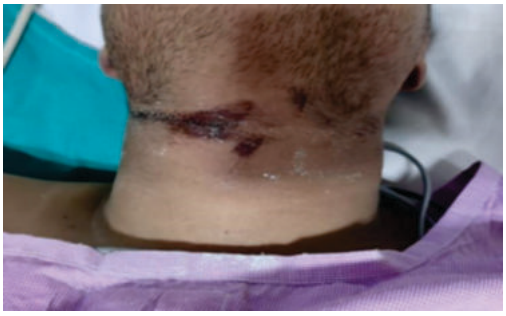
Radiological cross-sectional imaging of brain plays a vital role in detecting various changes occurring secondary to vascular compromise. To our knowledge, not many cases have been reported describing various CT and MRI changes in brain in an attempted case of suicide. We in this case report will be discussing the importance of cross-sectional imaging in the rapid detection of sequalae of attempted hanging in a male patient brought to the Emergency department in a semi-comatose state.

Case Report:

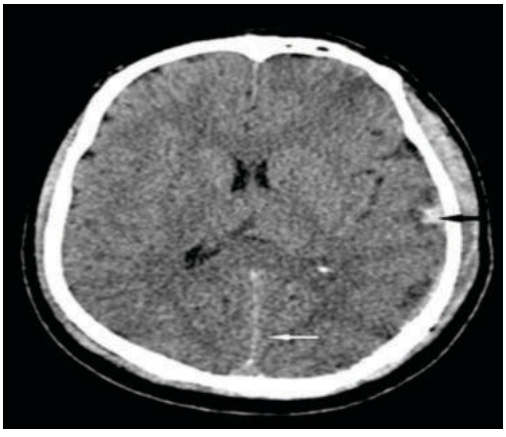
A 25-year-old male patient was brought to the emergency department in a state of altered sensorium with two episodes of vomiting after attempted suicide by hanging from a household ceiling fan. On admission his vitals were as follows: Blood pressure-126/80 mmHg, Pulse- 81/min, SPO2-99%. Local physical examination revealed a large asymmetric circumferential ligature mark over the anterior aspect, above the level of thyroid cartilage. (Figure 1)

Central nervous system examination showed disoriented and aggressive behavior with spontaneous speech and eye-opening and a Glasgow coma sore of 9/15. The patient had normal heart sounds, bilateral equal air entry on respiratory examination, and absence of any organomegaly on per abdomen examination. His pupil showed a normal response to light with an absence of papilledema. The patient was stabilized with a cervical collar and was intubated for risk of airway obstruction.

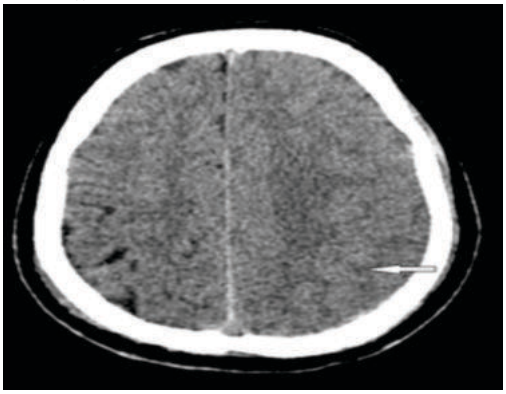
This was followed by a Brain and cervical spine CT which revealed: i) subdural hemorrhage (SDH) of 2.5 mm thickness in the left parietal region, along the falx and tentorium cerebelli (Figure 2). ii) Blood attenuation areas (HU-65) with surrounding oedema in the left frontal and temporal region, likely suggesting venous infarcts (Figure 2). iii) left sided cerebral edema seen in the form of effacement if ipsilateral sulcogyral spaces (Figure 3) and iv) Subarachnoid hemorrhage (SAH) in bilateral high parietal region (Figure 4). The cervical spine CT showed no abnormalities.



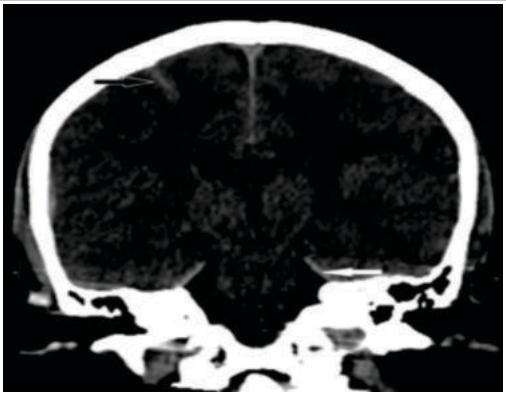
(Figure 1: Ligature mark seen in the supra-thyroid level)



(Figure 2: Axial non-contrast CT shows SDH along falx (white arrow) and hemorrhagic venous infarct in left temporal lobe (Black arrow))

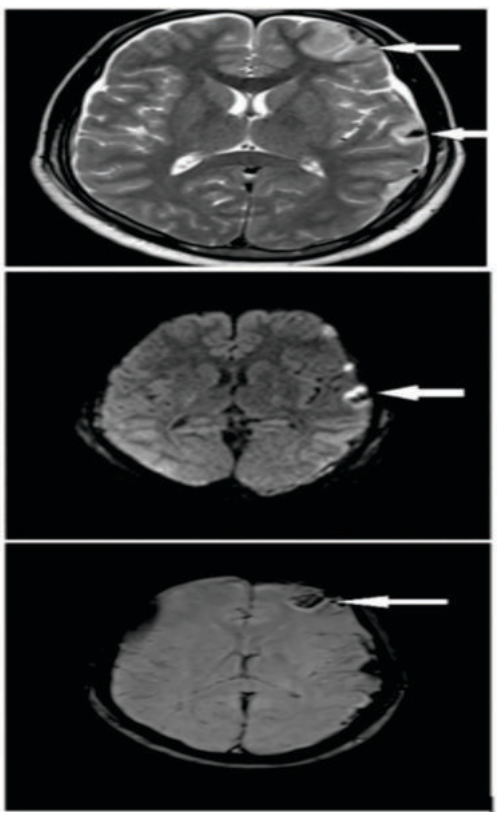


(Figure 3: Axial non-contrast CT shows left sided cerebral oedema (white arrow))

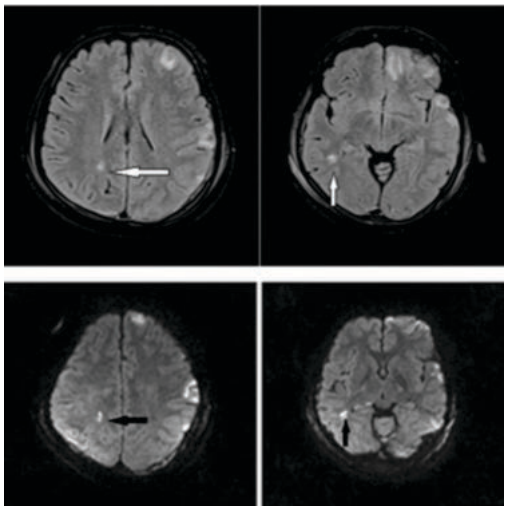


(Figure 4: Coronal non-contrast CT shows SAH in right high parietal region (Black arrow) and SDH along tentorium cerebelli (white arrow))

After 24 hours of admission, MRI brain was performed on a 1.5 Tesla machine, the sequences which were acquired were: T1WI, T2WI, FlAIR, SWI, Diffusion weighted images (DWI) and apparent diffusion coefficient (ADC)sequences. These sequences revealed:1) focal areas of cortical and subcortical gyral oedema appearing hyperintense on T2/TRIM and showing true restriction on DWI images noted in the left frontal, parietal and temporal region with few showing internal well-defined area of signal drop out on T2 and SWI sequences suggesting hemorrhagic component. These findings were suggestive of venous hemorrhagic infarcts (Figure 5). 2) A few patchy areas of altered signal intensity in the right peri trigonal region, centrum semiovale ,right temporal lobe, and right hippocampus appearing hyperintense on T2/TRIM and showing true restriction on DWI likely suggestive of Acute infarcts (Figure 6). 3) SDH along the left high parietal region, the falx, and bilateral folds of tentorium cerebelli .4) Linear T1 iso, FLAIR hyperintensity seen in bilateral(left>right) high parietal sulcal spaces, minimal blooming seen on SWI, likely suggesting SAH.



(Figure 5 (a: T2WI, b: DWI, c: SWI) shows multiple patchy areas of Acute venous (white arrow) infarcts in left frontal, parietal and temporal areas).



(Figure 6 a, b: Axial FLAIR sequences show patchy area of increased signal intensity (white arrow) in right centrum semiovale and right temporal region. 6 c, d: Axial DWI images showing true restriction (black arrow) in these areas suggesting Acute infarcts.

The patient was treated conservatively and given intravenous mannitol, pantoprazole, paracetamol and normal saline. The patient was monitored in Intensive care unit for five days and discharged on the 6th day after attaining normal mental status.

DISCUSSION:

Suicidal hanging is on the rise in developing countries especially India, which saw 1,53,052 reported suicide deaths in 2020.[4]

In a retrospective study conducted by Nagar N et al, on demographic details of hanging in North India, the mean age group of suicide was in the 3rd decade, with a very high male-to-female ratio (3.33:1). This demographic profile was consistent with our case of a male patient of 25 year of age.[1]

Hanging has been classified as 1) Judicial hanging, 2) suicidal hanging and 3) Homicidal hanging. Death in judicial hanging occurs due to transection of the pontomedullary junction causing instant death secondary to the 2nd and 3rd vertebral arch fracture (Hangman's fracture). Whereas death in suicide by hanging occurs secondary to neurological injuries and airway compromise. When the patient is bought to hospital alive the term near hanging is preferred as a consequence of hanging depends on the weight, method, and time duration of hanging. The sequential stages of hanging comprise of compression over the Juglar veins (needs a force of at least 2kg), followed by occlusion of carotid artery (needs force of at least 5 kgs) and finally compression of airway causing hypoxia and death.**[5]** Homicidal hanging is not been discussed as it is beyond the scope of this article.

Cross-sectional imaging like CT and MRI play an important role in detecting the various changes occurring secondary to vascular compromise in cases of near hanging. CT is also useful in detecting hyoid or vertebral fractures and soft tissue oedema seen in few cases. To our knowledge limited literature is available, that compiles the various changes in the brain in cases of near hanging on both CT and MRI.

In our case we found multiple, patchy areas of blood attenuation with surrounding oedema on CT in the left frontal, temporal, and parietal cortical and subcortical region. These were seen as areas of restricted diffusion, appearing

hyperintense on T2/TRIM with blood components showing signal drop out on SWI images suggesting venous hemorrhagic infarcts. Venous infarcts have been previously reported in cases of hanging **[6]** and occurs secondary to Juglar vein compression causing venous stasis, this is followed by raised venous pressure , finally the pressure exceeds the resistance of venous walls causing cortical hemorrhage.**[7]**

Subarachnoid hemorrhage is commonly seen in cases of trauma, homicide and aneurysmal bleed, however rarely seen in cases of suicidal hanging and only few authors have previously reported it **[6][8].** Theoretically, this occurs due to back pressure changes leading to vessel rupture. Our case also had some degree of subarachnoid bleed in the bilateral (left>right) high parietal region.

Prolonged Hanging causes ischemic damage to the brain cells due to compression of the carotid arteries. It is observed that the boundary zones (parieto occipital cortex) between the anterior, middle and posterior cerebral arteries, the hippocampus, purkinjee cells of the cerebellum, and basal ganglia are most sensitive to damage due to hypoxia. These areas that are one of the first areas to be affected when systemic blood pressure reduces**.[6]** In our case, patchy areas of altered signal intensity in the right peri trigonal region, centrum semiovale, temporal lobe, and hippocampus showing diffusion restriction, hyperintensity on FLAIR, and mild hypo intensity T1WI were observed suggesting ischemic brain damage.

We did not come across any publication where Subdural hemorrhage was associated with hanging. We in our case saw Subdural hemorrhage along the falx , bilateral folds of tentorium and along the left parietal convexity and this might probably be associated with the venous infarcts occurring secondary to Juglar vein compression. We did a literature search and found only 9 cases where subdural hematoma associated with cortical venous infarcts occurring secondary to venous sinus thrombosis was reported.[9] The hypothesis behind Subdural hematoma in these cases was possibly due to the rupture of bridging veins, resulting from high backpressure by the thrombosed vein. We presume that in our case of hanging SDH occurred due to backpressure changes by the compressed Juglar vein.

CONCLUSION-

Cases of near hanging or attempted suicide by hanging are not infrequently admitted to the Emergency department with varying presentation from conscious state to semiconscious/ comatose state. It is important for the treating physician to know the severity of brain damage; this is where MRI and CT imaging play a major role in detecting various types of changes occurring secondary to pressure effects over the carotid and Juglar vessels. Venous infarcts, cerebral oedema, and acute arterial infarcts/insults are commonly seen as changes having a typical pattern of involvement. However, radiologists and clinicians should be aware that a few rare cases may also show Subarachnoid and Subdural hemorrhage as seen in our case

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