

Non-traumatic cerebral microhaemorrhages after by partial hanging

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Section: Neuroradiology

Area of Interest: CNS Emergency Neuroradiology brain

Imaging Technique: MR-Diffusion/Perfusion

Special Focus: Haemorrhage Case Type: Clinical Cases

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Patient: 19 years, female

Clinical History:

A 19-year-old female was admitted to the emergency medicine department with altered sensorium. She hung herself for about 20 minutes and her feet were in contact with the ground, according to a witness statement. She was immediately brought to the hospital upon being found out. On admission, her vitals were: blood pressure - 114/63 mmHg; heart rate - 118 beats/minute; respiratory rate - 20 breaths/min; body temperature of 36°C. She was in a semi-comatose state, with a score of 6/15 on the Glasgow Coma Scale. Her pupils were normal and equal in size and reacted to light promptly. No other neurological abnormalities were revealed in examinations. The only outer, visible sign of asphyxia was the incomplete ligature mark around her neck

Imaging Findings:

NCCT of the brain and cervical spine were unremarkable. No fractures or dislocations of cervical vertebrae seen. MRI of the brain with TOF angiography performed on the day of admission were normal. MRI of the brain was repeated on day 12 as she persisted to be in semi-comatose state. Repeat MRI showed symmetrical T2/ FLAIR hyperintensities (Figure 1) in bilateral centrum semiovale, deep white matter of bilateral frontal, parietal and temporal lobes. On SWI, numerous punctate microhaemorrhages (Figure 2) were present in deep and subcortical white matter. These areas were showing diffusion restriction (Figure 3-C) with low values on corresponding ADC maps (Figure 3-D). MR angiography and MR venography showed no arterial or venous sinus occlusion.

Discussion:

Background

Hanging is the second most common method of suicide among adolescent age group. Hanging injuries are associated with a high overall mortality rate of 70 %, with the Glasgow Coma Score at the time of admission being the best independent predictor of outcome [1]. Hangings are referred to as complete or incomplete depending on whether or not the feet are in contact with the ground [2].

Clinical perspective

Neurological injury occurs due to compression of the neck. The jugular veins get compressed at force of 2 kg. While the carotid arteries and tracheal compression require greater force of 5 kg and 15 kg, respectively. Carotid compression leads to cerebral oedema and hypoxic brain injury. Compression of the airways needs greater force (15 kg) which can lead to severe hypoxia and death [3].

Imaging perspective

There are only a few reports of brain CT and MRI of hanging victims. The most common abnormality described was diffuse cerebral oedema. Uncommon radiological and clinical findings include dissection of carotid and vertebral arteries [4], posterior reversible encephalopathy syndrome [5], subarachnoid haemorrhage [2], hyperthermia [6], amnesia of the event [7] and Korsakoff's psychosis [8].

The primary mechanism of brain damage in hanging is hypoxic injury due to arterial compression.

In complete hanging, the rapid and higher compression force, combined with gravitational pressure on the neck constricts both the arteries and the veins. Arterial inflow cessation leads to rapid ischemia.

In incomplete hanging, less gravitational pressure is exerted on the neck. Hence partial or complete venous compression occurs, while arteries are spared. Jugular vein compression causes venous hypertension and facial congestion, cyanosis and superficial haemorrhages. Continued venous stasis may lead to dural sinus thrombosis[3].

Microhaemorrhages are an uncommon and atypical imaging finding in cases of incomplete hanging. Probable underlying mechanism is spontaneous rupture of venules and capillaries within a high-pressure venous system created by venous outflow obstruction, and continued arterial inflow. Relief of hanging force and subsequent reperfusion also contributes to this appearance. Reperfusion injury is also seen with the same underlying mechanism in cases of post-treatment stroke.

Outcome

In the current case, the patient did not have a history of head trauma. Venogram showed no cerebral venous thrombosis. This case shows that spontaneous microhaemorrhages in cerebral white matter can develop due to a sudden elevation of intracranial venous pressure and subsequent reperfusion injury after release of ligature as occurs with survivors of hanging.

Take home message

Neuroimaging findings in cases of suicidal asphyxia are not limited to ischemic changes and may show atypical overlapping features with other clinical entities.

Written informed patient or patient guardian consent for publication has been obtained as the patient remained semi-comatose.

Differential Diagnosis List: Ischemic changes and cerebral microhaemorrhages – secondary to venous hypertension and reperfusion injury, Diffuse axonal injury, Cerebral vasculitis, Cavernous malformations, Fat embolism syndrome

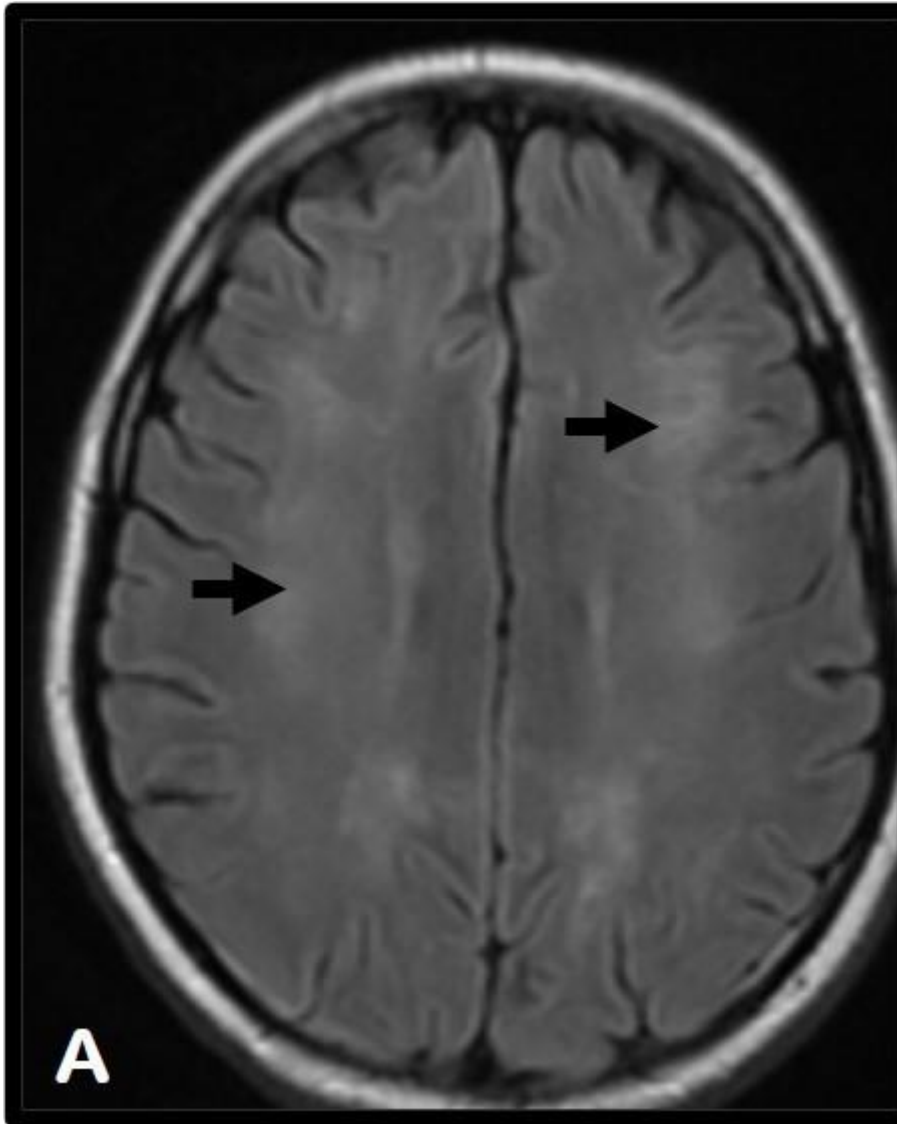
Final Diagnosis: Ischemic changes and cerebral microhaemorrhages – secondary to venous hypertension and reperfusion injury

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Figure 1

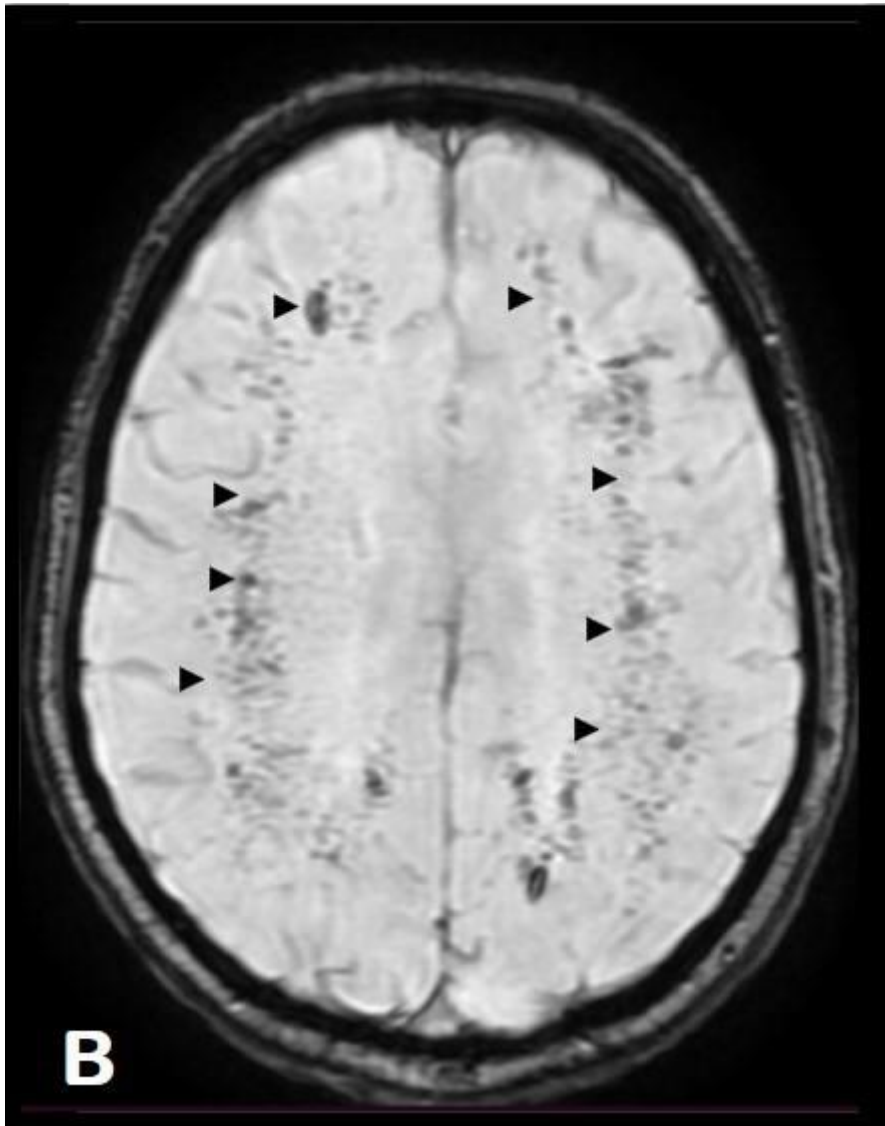
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Description: Axial FLAIR image at the level of centrum semiovale showing symmetrical hyperintensity (arrows) in subcortical, deep and periventricular white matter **Origin:** © Department of diagnostic and interventional radiology, AIIMS, Rishikesh

Figure 2

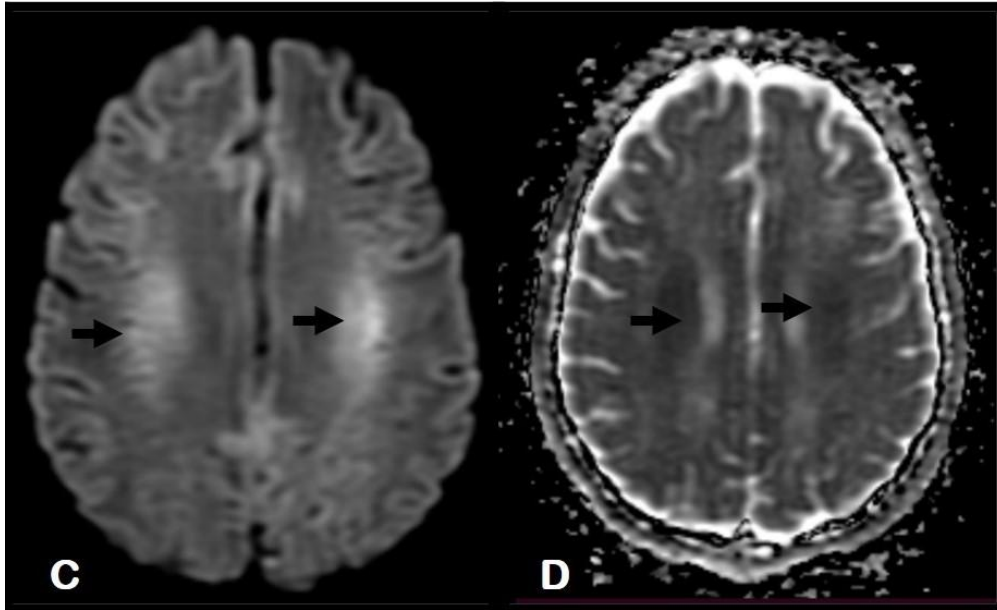
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Description: Axial SWI image showing numerous microbleeds (arrowheads) in bilateral cerebral hemispheres at subcortical and deep white matter **Origin:** © Department of diagnostic and interventional radiology, AIIMS, Rishikesh

Figure 3

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Description: Axial DWI image (C) showing confluent areas of diffusion restriction in bilateral centrum semiovale white matter (arrows) with low values on ADC map (D) **Origin:** © Department of diagnostic and interventional radiology, AIIMS, Rishikesh