Clinical History:

86-year-old female patient, found down, scalp haematoma, concern for seizure vs stroke.

Imaging Findings:

Examination demonstrates symmetric bilateral medial thalamic acute infarcts with positive diffusion signal abnormality and diffusion restriction. Punctate susceptibility signal loss is noticed on both medial thalamic lesions. There is preservation of signal flow void behaviour in both internal cerebral veins and vein of Galen.

Discussion:

Bilateral thalamic infarction (BTI) is a rare entity that represents 0.6% of the first cerebrovascular ischaemic event. [1, 2] Patients present with a variable clinical presentation that includes fluctuations in the state of consciousness, a "sleep-like coma " stage, confusion and disorientation. When the commitment is extended to the rostral mesencephalon, alterations in eye movements are included. Its diagnosis becomes a challenge for emergency services. [2]

The paramedian bilateral thalamic infarction has been known for its cardinal manifestation, the abrupt alteration of the state of consciousness, and is usually accompanied by paralysis of the vertical gaze or other alterations in the ocular movements. The coma develops as a result of the interruption of both activating systems, the ascending reticular formation ending in the intralaminar region and the thalamic midline nuclei, and the hypothalamic wake center. Associated with paralysis for vertical gaze and pupillary abnormalities as a result of the involvement of the intralaminar and dorsomedial thalamic nuclei, coexisting with lesions of the superior mesencephalon (interstitial rostral nuclei of the medial longitudinal fasciculus and the posterior commissure). Akinetic mutism can also occur when there is involvement of the posterior diencephalon and "loss of psychic self-activation." [3, 4, 5]

The main cause is the disease of small arteries originating in the rostral portion of the basilar artery independently of the topography of the lesions, and secondly, embolic sources (artery to artery embolism or cardioembolism). [6]
The existence of the Percheron artery is the anatomical substrate that explains bilateral thalamic infarcts. However, sometimes the irrigation of the rostral region of the mesencephalon depends on the peduncular paramedian artery, which can be born from the thalamic paramedian artery itself, or independently from the basilar artery. This variability is what explains the existence of thalamic infarcts without the mesencephalic involvement and the possible asymmetry of the mesencephalic extension of bilateral thalamic infarcts. [7]

Honig et al. Reinforce in their study the need to quickly diagnose of BTI for immediate intervention with IV-tPA or angiography. Computed tomography and CTA are not contrasted with the majority of cases, whereas the IRM-DRI has a much higher sensitivity for the diagnosis of cerebral infarction. Therefore, it suggests that resonance should be used urgently and early for the evaluation of comatose patients. [8]

Another imaging finding is a V-shaped hyperintense signal intensity on axial DWI and FLAIR images on the pial surface of the midbrain [9].

**Differential Diagnosis List:** Bilateral thalamic infarction, Top of the basilar artery syndrome, Bilateral internal cerebral vein thrombosis

**Final Diagnosis:** Bilateral thalamic infarction

**References:**


Description: The most common, where the thalamic perforating arteries leave each of the posterior cerebral arteries. Origin: HMC
Description: It is the least common variant, or called asymmetric. Where the thalamic perforating arteries come directly from the proximal segment of one of the posterior cerebral arteries. Origin: HMC
Description: The bilateral thalamic arteries come from a trunk called the artery of Percheron, which originates from the P1 segment of a posterior cerebral artery. Origin: HMC
Description: The variant of the arcade or bridge, where the small perforating arteries leave an arch or bridge that arises from both segments P1 of both posterior cerebral arteries. Origin: HMC
Figure 2

**Description:** Examination demonstrates symmetric bilateral medial thalamic signal abnormality. **Origin:** Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University
**Description:** Examination demonstrates symmetric bilateral medial thalamic signal abnormality. **Origin:** Augusta University.
**Description:** Examination demonstrates symmetric bilateral medial thalamic signal abnormality. **Origin:** Augusta University
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Punctate susceptibility signal loss is noticed on both medial thalamic lesions. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Punctate susceptibility signal loss is noticed on both medial thalamic lesions. Origin: Augusta University.
Description: Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Punctate susceptibility signal loss is noticed on both medial thalamic lesions. Origin: Augusta University
**Description:** Examination demonstrates symmetric bilateral medial thalamic signal abnormality. Punctate susceptibility signal loss is noticed on both medial thalamic lesions. **Origin:** Augusta University.
Figure 6

Description: Brain CT. Bilateral medical thalamic hypodensity. Origin: Augusta University.
**Description:** Brain CT. Bilateral medical thalamic hypodensity. **Origin:** Augusta University
**Description:** Brain CT. Bilateral medical thalamic hypodensity. **Origin:** Augusta University.
Description: Brain CT. Bilateral medical thalamic hypodensity. Origin: Augusta University.
Figure 7

a

Description: Symmetric bilateral medial thalamic acute infarcts with positive diffusion signal abnormality and diffusion restriction. **Origin:** Augusta University.

b

Description: Symmetric bilateral medial thalamic acute infarcts with positive diffusion signal abnormality and diffusion restriction. **Origin:** Augusta University.
Description: Symmetric bilateral medial thalamic acute infarcts with positive diffusion signal abnormality and diffusion restriction. Origin: Augusta University.
Description: There is preservation of signal flow void behaviour in both internal cerebral veins and vein of Galen. Origin: Augusta University.
**Description:** MR venography oblique. There is preservation of signal flow void behaviour in both internal cerebral veins and vein of Galen. **Origin:** Augusta University.
Description: MR venography axial.
There is preservation of signal flow void behaviour in both internal cerebral veins and vein of Galen.
Origin: Augusta University.
Description: MR venography sagittal.
There is preservation of signal flow void behaviour in both internal cerebral veins and vein of Galen.
Origin: Augusta University.