

Bilateral Thalamic Infarct in Two Unresponsive Octogenarians

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Authors' contributions

This work was carried out in collaboration between all authors. Authors LM, GL and FG designed the study and wrote the first draft of the manuscript with assistance from authors DAG and MDN. All authors read and approved the final manuscript.

Case Study

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ABSTRACT

Bilateral thalamic infarct (BTI) represents an uncommon stroke presentation. Pathophysiology recognizes the occlusion of an anatomic variant of the thalamic blood supply from perforating branches of posterior cerebral arteries. Presentation could be nonspecific and dramatic in the same time, being coma or stupor the possible clinical scenario encountered. Diagnosis is performed by neuroradiological imaging showing the typical bilateral paramedian thalamic infarcts. Literature lacks of evidence in very old patients, therefore we describe two cases of BTI occurred in octogenarians presenting unresponsive. BTI in very old patients presenting comatose should be taken in account as diagnostic possibility.

Keywords: Percheron syndrome; bilateral thalamic infarct; stroke, elderly.

1. BACKGROUND

Bilateral thalamic infarct (BTI) is a rare but well-described stroke manifestation [1]. Much recently, Jiménez Caballero found a 0.7% BTI incidence within a cohort of more than 1250 consecutive patients enrolled for ischemic stroke [2]. Presentation could be nonspecific and coma or stupor could be the main manifestation as described in a recent systematic review, making difficult and extremely wide the spectrum of differential diagnosis [3,4]. Literature lacks of evidence in very old patients 80 years-old and older, therefore we describe two cases of BTI occurred in octogenarian females presenting to our attention for coma and stupor respectively.

2. CASE REPORTS

A 84-years old female was referred to Emergency Department of our Hospital after having found comatose [Glasgow Coma Scale, GCS 7/15, E2, V1, M4) at the moment of wake up. Her history revealed arterial blood hypertension lasting from more than ten years and treated with ACE-inhibitors. First examination showed no abnormal pyramidal signs and no cranial nerves deficit. Fine examination of oculomotricity was impossible. There was a mild neck stiffness. Electrocardiography, blood arterial gas analysis (pH 7.41, paO₂ 76mmHg, paCO₂ 38mmHg, HCO₃ 26mEq/L), complete biochemical examinations and chest x-ray resulted within the normal range. A lumbar puncture was performed for cerebrospinal fluid (CSF) examination and gave normal results. Basal brain computer tomography (CT) was negative, whereas T1 and T2 weighted brain magnetic resonance (MR) images after six hours (Fig. 1) showed paramedian bilateral thalamic infarcts. Aspirin 300 mg once/daily was prescribed as secondary stroke prevention. Clinical course was favorable with complete recovery of vigilance after a period of hypersomnia lasting around two days.

The second case is dealing with a 86-years old female who came to our attention for stupor (GCS 11/15, E3, V3, M5). Neurological examination showed no abnormal pyramidal signs and no cranial nerves deficit. Spontaneous eye movements were preserved. Her history revealed arterial blood hypertension and diabetes lasting from around twenty years. Electrocardiography showed sinus rhythm. Basal brain computer tomography (CT) scan was negative. Carotid ultrasonography revealed non haemodynamic atherosclerosis. We started therapy with aspirin 300 mg once/daily, saline solution and enoxaparin 40 mg once/daily for venous thromboembolism prevention. No clinical improvement was noted. Two days later, a new CT scan revealed bilateral thalamic infarcts (Fig. 2A) and four days later another CT scan showed bilateral hemorrhagic transformation (Fig. 2B). After one week the patient presented sudden paroxysmal tachycardic atrial fibrillation. After ten days the patient was discharged from Hospital. Physical impairment and dependence were severe as demonstrated by Barthel Index of 10/100 and modified Rankin Scale of 5/6.

3. DISCUSSION

The thalami receive blood supply from perforating arteries both from anterior and posterior brain circulation [5]. Anterior circulation derives from the internal carotid arteries and supplies the antero-inferior portion of the thalami with perforating arteries arising from the posterior communicating arteries. The posterior circulation derives from the vertebro-basilar system and supplies the medial, lateral and superior portions of the thalami, respectively by branches arising from P1 segments (medial) and P2 segments (lateral and superior) of the posterior cerebral arteries [5]. Many anatomical variants have been described, the most

known of them is named Percheron artery [5]. Percheron in fact, for the first time in 1973 [6] and more accurately in 1976 [7-10], described three different types of anatomical variants of thalamic blood supply originating from P1 segments. In the second type of anatomical variant described from Percheron a common trunk arises from one of the two P1 segments of posterior cerebral arteries, providing alone for bilateral vascular distribution of medial portions of the thalami. Occlusion of this trunk results in paramedian BTI named as Percheron syndrome [5]. Small vessel disease, as occurred in case report 1, is an unusual form of atypical lacunar syndrome [11], whereas cardioembolism, as occurred in case report 2, may represent another plausible source for BTI [3]. Since midbrain (mesencephalus) receives the same blood supply from the thalami by anterior and posterior brain circulation and brain stem receives blood supply from P1 segments of posterior cerebral arteries, BTI is often associated with midbrain or brainstem infarct [5].

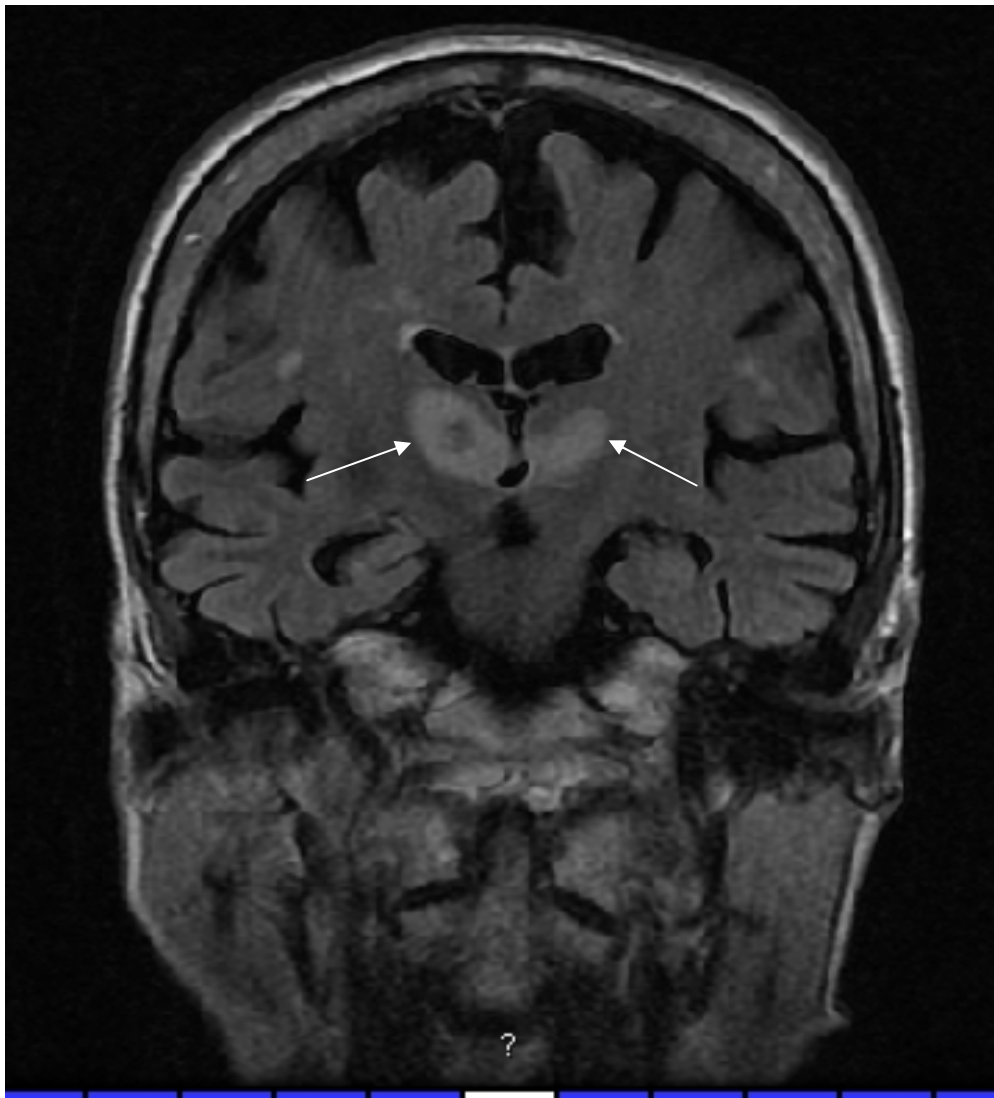


Fig. 1. Brain MR shows paramedian bilateral thalamic infarct

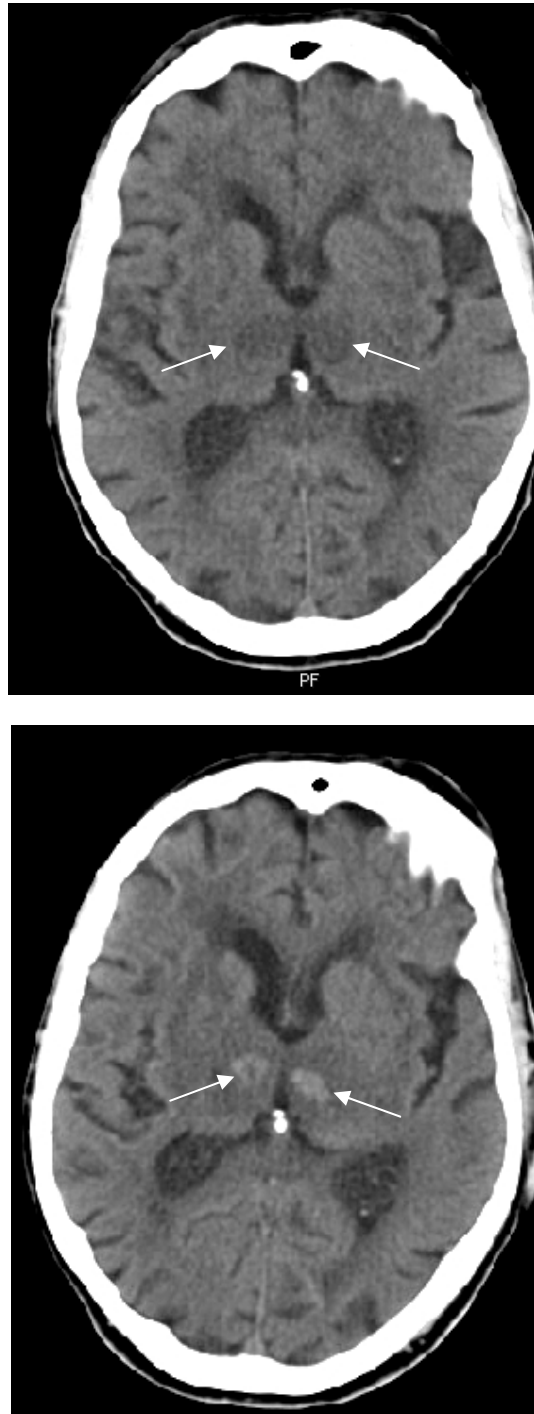


Fig. 2. Brain CT shows BTI after 48 hours from hospital arrival (A). Brain CT after four days with evidence of hemorrhagic transformation of paramedian bilateral thalamic infarct

Clinical suspicion of BTI is usually difficult because of nonspecific symptoms. A review performed by Monet P et al. based on literature evidence from 1985 to 2006 showed that main symptoms are represented by amnesia (63%), paralysis of third cranial nerve with various type of vertical gaze (61%), confusion (55%) and coma (47%) [12].

Neuroimaging such as brain CT scan and MR, this last one especially recommended when non-focal signs are present in patients with stupor or coma, is necessary for diagnosis, showing the characteristic bilateral ischemia [13]. The clinical course is variable; mortality accounts for around 10%, whereas complete recovery as occurred in one of the two case described has been shown in around 13-15% of patients. The main consequences are represented by memory lost, psychiatric and visual disturbances in more than one half of patients [12]. Akinetic mutism represents at the same time one of the main differential diagnosis and the consequence of BTI [14]. Contrarily to BTI in which antithrombotic drugs represent the choice treatment, dopaminergic drugs such as bromocriptine may represent a possible choice when the reduced level of consciousness is thought to be due to reduced dopaminergic outflow to the telencephalon such as in akinetic mutism [15].

The description of our cases represents examples of BTI befell octogenarians and jointing to a case with characteristics similar to our first case described by Wells and colleagues of a 84-years old female with history of Alzheimer disease presented unresponsive to their attention and whom only brain MR was able to detect BTI [16], we would stress the attention of physicians in keeping in mind that, despite uncommon in very old patients, BTI should be taken in account as diagnostic possibility in comatose subjects.

4. CONCLUSION

- Bilateral thalamic infarct represents an uncommon stroke presentation in very old patients.
- Physicians should think about bilateral thalamic infarct in unresponsive geriatric patients.
- Neuroimaging of bilateral thalamic infarct is mandatory for diagnosis.
- Bilateral thalamic infarct is an uncommon presentation of small vessel disease.

CONSENT

Not applicable.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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