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Simultaneous Bithalamic Infarctions: Diagnostic Challenges in Low-Resource Settings —A Six-Case Series

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Abstract

Introduction: Simultaneous bithalamic infarction is rare and is typically related to occlusion of a common trunk supplying both paramedian thalamic arteries, known as the artery of Percheron. However, a few cases of bithalamic infarction secondary to deep cerebral venous thrombosis have been reported. The clinical picture is heterogeneous, non-specific, and often misleading. **Methods**: We conducted a descriptive study of six consecutive patients hospitalised for management of bithalamic infarction between 23 April 2017 and 1 November 2024 in the neurology departments of Libreville University Hospital and Lomé University Hospital. **Results**: The mean age was 58 years with a female predominance (sex ratio M:F = 1:5). Clinical presentation was polymorphic, most commonly with impaired consciousness, followed by motor, oculomotor, and finally cognitive disturbances. Bilateral thalamic involvement was demonstrated in all patients on brain CT. One out of two patients underwent brain MR angiography or CT angiography. An artery of Percheron occlusion was diagnosed in four patients, and deep cerebral venous thrombosis in two. Most patients received anticoagulation with low-molecular-weight heparin followed by vitamin K antagonists. Overall outcomes were unfavourable: two deaths, one patient lost to follow-up, and moderate to severe sequelae in the remaining three. **Conclusion**: Because of its varied and non-specific presentation and the diversity of causes of bilateral thalamic lesions, bithalamic infarction poses genuine diagnostic challenges. These challenges are compounded in our setting, where access to brain and supra-aortic trunk MR angiography remains limited.

Keywords

Infarction, Bithalamic, Percheron Artery, Deep Cerebral Venous Thrombosis

1. Introduction

Simultaneous bithalamic infarction is a rare condition, accounting for 0.1% - 2% of ischaemic strokes (IS) [1]. It is linked to occlusion of the paramedian thalamic arteries that share a common trunk [2]. This anatomic variant of the thalamo-mesencephalic vascular network, present in 4% - 12% of individuals, was first described by Percheron in 1977 [3]. Because vascular territories vary anatomically and the extent of infarcted tissue differs, the clinical picture is highly polymorphic and may include impaired consciousness, memory disturbance, psychiatric symptoms, and oculomotor abnormalities [4]. However, some bithalamic infarctions have been reported secondary to occlusion of the internal cerebral veins or the straight sinus [5]. Their variable, non-specific, and often misleading semiology frequently leads to diagnostic delay or error [6]. We report six cases of bithalamic infarction that illustrate the challenges of clinical and aetiological diagnosis in our setting.

2. Methods

This study took place in Gabon and Togo, both of which are classified as developing countries (low- or middle-income countries) where limited access to healthcare, incomplete infrastructure, and unemployment create a low-resource settings. We conducted a descriptive study of six consecutive patients hospitalised for management of bithalamic infarction between 23 April 2017 and 1 November 2024 in the neurology departments of Libreville University Hospital and Lomé University Hospital.

2.1. Case 1

A 64-year-old woman with known hypertension presented to the emergency department with behavioural disturbances evolving over five days. On examination, the patient was drowsy, with a Glasgow Coma Scale score (GCS) of 13. There was a disorientation in time and space and Parinaud syndrome, giving a National Institutes of Health Stroke Scale (NIHSS) score of 4 (**Table 1**). Brain CT showed paramedian bithalamic hypodensities (**Figure 1(A)**). Brain Magnetic Resonance Imaging (MRI) revealed bilateral thalamic lesions that were hypointense on T1 and hyperintense on diffusion-weighted, T2 (**Figure 1(B)**), and Fluid-Attenuated Inversion Recovery (FLAIR) sequences (**Figure 1(C)**). Cerebral Magnetic Resonance Angiography (MRA) found no arterial occlusion or thrombosis of the internal cerebral veins. Carotid Doppler ultrasound demonstrated non-stenotic carotid atheroma, and the ECG was normal. A diagnosis of artery of Percheron oc-

clusion was made. The patient was treated with aspirin, atorvastatin, and thromboembolic prophylaxis using low-molecular-weight heparin. She was discharged after 10 days with persistent vertical gaze palsy and severe cognitive impairment as described in Table 2 (Montreal Cognitive Assessment, MoCA, score of 7). At six months, the outcome was favourable, with only mild cognitive impairment remaining (MoCA 20) as shown in Table 3.

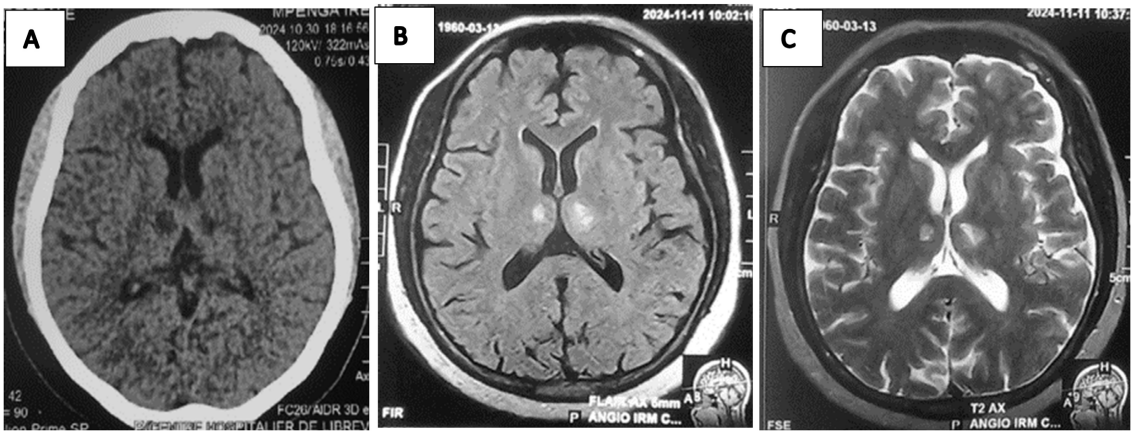

Figure 1. Day-5 brain CT showing paramedian bithalamic hypodensities (A). Axial brain MRI demonstrates bilateral thalamic lesions hyperintense on FLAIR (B) and T2 (C), consistent with an established bithalamic infarction.

Table 1. NIHSS patient score recorded at admission.

Item N°	Assessment area	Description	Score
1a	Level of consciousness	Responsiveness to voice, touch, or pain	1
1b	LOC questions	Ability to answer age and current month	2
1c	LOC commands	Ability to follow simple commands (open/close eyes, grip/release hand)	1
2	Best gaze	Voluntary horizontal eye movements	0
3	Visual fields	Testing for visual field loss	0
4	Facial palsy	Symmetry and movement of facial muscles	0
5a	Motor arm (Left)	Strength and drift of left arm	0
5b	Motor arm (Right)	Strength and drift of right arm	0
6a	Motor leg (Left)	Strength and drift of left leg	0
6b	Motor leg (Right)	Strength and drift of right leg	0
7	Limb ataxia	Coordination of arms and legs	0
8	Sensory	Ability to feel pinprick or touch	0
9	Best language	Ability to name objects, describe a picture, and read a sentence	0
10	Dysarthria	Clarity of speech	0
11	Extinction and inattention (Neglect)	Awareness of body and surroundings	0

Table 2. MoCA score on the 10th day of admission.

Item N°	Cognitive domain	Task description	Score
1	Visuospatial/Executive	Draw a clock (10 past 11) and copy a cube	2
2	Naming	Name three animals (lion, rhinoceros, camel)	2
3	Memory	Learn and recall 5 words (immediate and delayed recall)	0
4	Attention	Digit span (forward/backward), vigilance (tap on letter A), serial 7 subtractions	1
5	Language	Repeat two complex sentences and generate words starting with a given letter	0
6	Abstraction	Explain similarity between two pairs of items (train–bicycle, watch–ruler)	1
7	Delayed recall	Recall the 5 words from earlier without cues	1
8	Orientation	State date, month, year, day, place, and city	0

Table 3. Comprehensive MoCA result obtained at 6 months.

Item N°	Cognitive domain	Task description	Score
1	Visuospatial/Executive	Draw a clock (10 past 11) and copy a cube	4
2	Naming	Name three animals (lion, rhinoceros, camel)	2
3	Memory	Learn and recall 5 words (immediate and delayed recall)	0
4	Attention	Digit span (forward/backward), vigilance (tap on letter A), serial 7s subtraction	5
5	Language	Repeat two complex sentences and generate words starting with a given letter	2
6	Abstraction	Explain similarity between two pairs of items (train–bicycle, watch–ruler)	1
7	Delayed recall	Recall the 5 words from earlier without cues	2
8	Orientation	State date, month, year, day, place, and city	4

2.2. Case 2

A 42-year-old woman with hypertension and poorly controlled HIV presented with rapidly progressive impaired consciousness in a febrile context. Neurologic examination showed a stuporous with GCS of 9, an encephalitic syndrome, bilateral Babinski signs, and a left homonymous hemianopia; the NIHSS was 15. Brain CT performed 48 hours later revealed bithalamic hypodensities involving the anterior and paramedian territories (**Figure 2(A)**). There was a right temporo-occipital porencephalic cavity consistent with a chronic infarct in the superficial territory of the posterior cerebral artery, suggesting a recurrent ischaemic stroke (**Figure 2(B)**). 3D time-of-flight MRA was not performed. Because fever persisted, a lumbar puncture was obtained: the cerebrospinal fluid (CSF) was clear with a normal cell count; India-ink staining for cryptococcus, GeneXpert testing, and cul-

ture were all negative. The CD4 count was 485/μL. Stroke work-up revealed atrial fibrillation with complete irregularity on ECG. A diagnosis of bithalamic infarction due to artery of Percheron occlusion of presumed cardioembolic origin was made. The patient was treated with therapeutic-dose heparin bridged to vitamin K antagonists, along with empiric antibiotics and antiretroviral therapy. At one month she was discharged home but had hypersomnia, tetraparesis, and global aphasia, corresponding to a modified Rankin Scale score of 5.

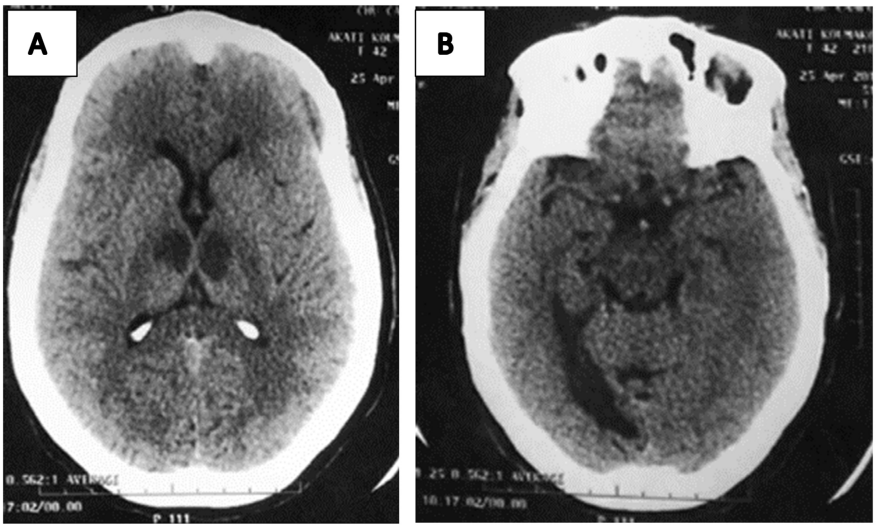

Figure 2. Brain CT at 48 hours showing bithalamic hypodensities involving the anterior and paramedian territories (A), and a right temporo-occipital porencephalic cavity (B) compatible with a chronic infarct in the superficial posterior cerebral artery (PCA) territory.

2.3. Case 3

A 54-year-old woman with previously undiagnosed hypertension was admitted to intensive care for sudden-onset coma. She had been found unresponsive by her son on awakening. Initial examination showed a GCS of 7, bilateral pyramidal weakness, left ptosis with a fixed dilated pupil, and a NIHSS of 26. Brain CT was normal at 2 hours after symptom onset (**Figure 3(A)**) but at 48 hours demonstrated bithalamic hypodensities in the anterior and paramedian territories (**Figure 3(B)**). Cerebral MRA revealed no arterial occlusion (**Figure 4(A)**) and no thrombotic lesion of the cerebral venous sinuses (**Figure 4(B)**, **Figure 4(C)**). The ECG showed rapid atrial flutter, and carotid Doppler identified irregular, prominent plaques of the right carotid bulb causing approximately 50% stenosis. A diagnosis of artery of Percheron occlusion was made. The patient received therapeutic-dose heparin bridged to vitamin K antagonists. Her impaired consciousness improved by day 10, allowing transfer to the neurology ward. She was discharged three weeks later with right hemiparesis, dysarthria, frontal-lobe syndrome, and anterograde memory impairment. At six months, the motor deficit had regressed, but severe neurocognitive impairment persisted with a MoCA score of 9.

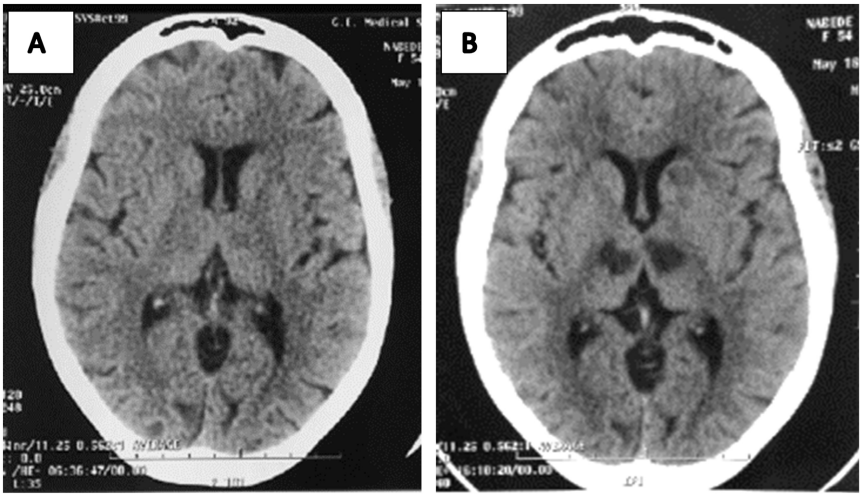

Figure 3. Brain CT normal at 2 hours (A), then at 48 hours showing anterior and paramedian bithalamic hypodensities (B).

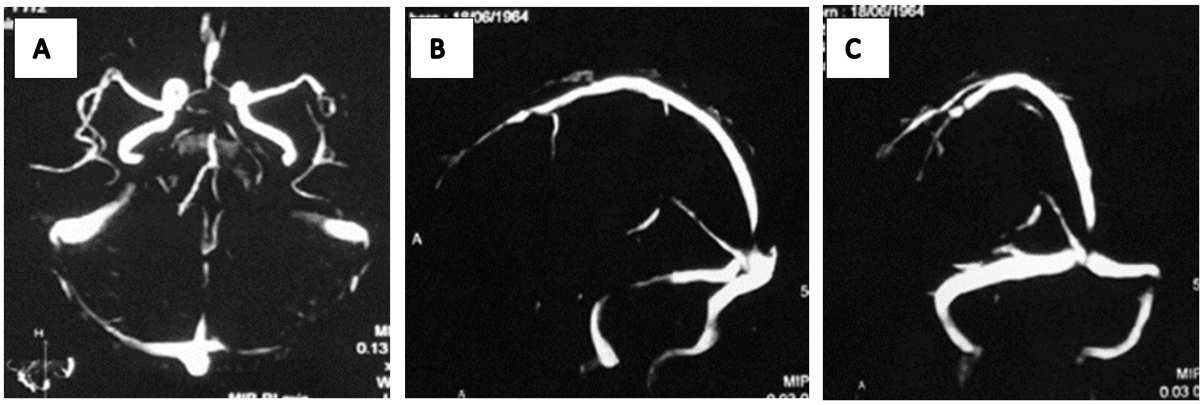

Figure 4. Axial time-of-flight (TOF) MRA with gadolinium shows no arterial occlusion (A) and no thrombosis of the venous sinuses (B, C).

2.4. Case 4

A 32-year-old man with no past medical history was admitted to the emergency department for sudden impairment of consciousness. Three hours earlier he had developed band-like holocranial headache with projectile vomiting, followed by a generalized tonic–clonic seizure and post-ictal coma. On examination: blood pressure 240/130 mmHg, temperature 38.5˚C, a stuporous with GCS 9, signs of raised intracranial pressure, and NIHSS 11. Laboratory tests showed impaired renal function and neutrophilic leukocytosis. Brain CT performed 24 hours later revealed bilateral, diffuse capsulo-thalamic hypodensities not respecting an arterial territory, with an oedematous appearance (**Figure 5(A)**, **Figure 5(B)**). Fundus examination showed papilloedema. MRA was not performed; however, the working diagnosis was bithalamic infarction secondary to deep cerebral venous thrombosis in a young, severely hypertensive patient. He received therapeutic anticoagulation with heparin (Calciparine), antihypertensive therapy, and broad-spectrum antibiotics. Clinical course was favourable within one week, though anterograde amnesia persisted. He

did not attend neurology follow-up and was lost to follow-up.

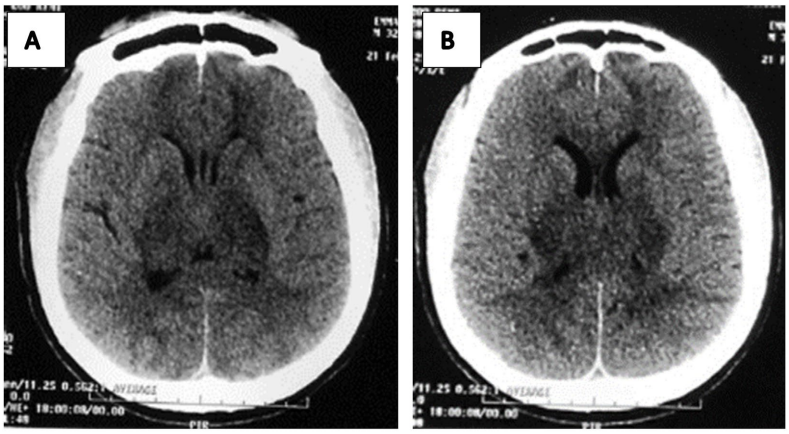

Figure 5. Brain CT at 24 hours showing bilateral, diffuse capsulo-thalamic hypodensities (A, B) with an oedematous appearance.

2.5. Case 5

An 81-year-old woman with poorly controlled hypertension was admitted to neurology for right-sided hemiparesis and language disturbance. On examination the patient was obtunded giving a GCS score of 12, with aphasia, right hemiplegia, and internuclear ophthalmoplegia consistent with a peduncular Foville syndrome; the NIHSS was 17. Brain CT performed 6 hours after admission showed paramedian bithalamic hypodensities (**Figure 6(A)**) and a left pontine lesion in the territory of basilar perforators (**Figure 6(B)**). Arterial-phase CT angiography showed no abnormality (**Figure 6(C)**). Occlusion of the artery of Percheron was suspected. Aetiologic work-up revealed dyslipidaemia and bilateral carotid bulb atheromatous plaques without stenosis. The standard ECG was normal. She was treated with an antiplatelet agent, a lipid-lowering drug, and prophylactic heparin. The course was marked by persistent confusion. Laboratory tests showed hypernatraemia and hypocalcaemia, which were corrected. Nevertheless, the patient died on day seven in the setting of worsening impaired consciousness and respiratory distress.

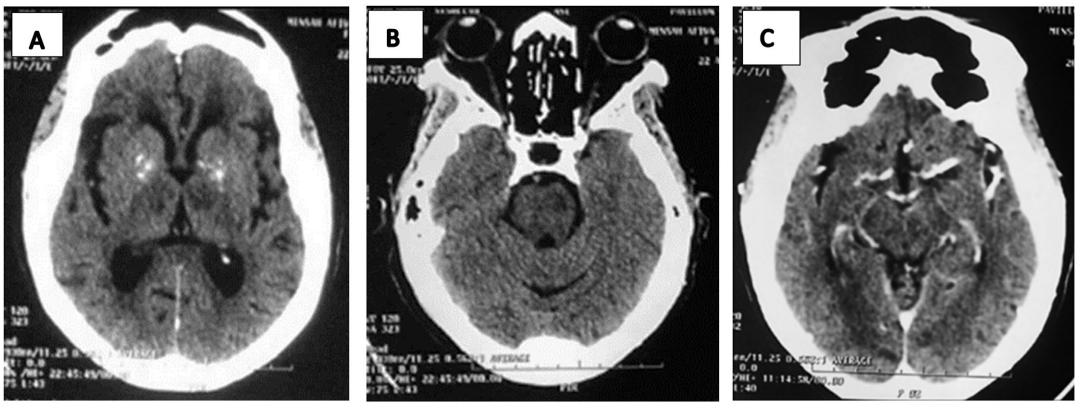

Figure 6. Brain CT at 6 hours demonstrating paramedian bithalamic (A) and left pontine (B) hypodensities in the territory of basilar perforators. Arterial-phase CT angiography (C) showed no abnormality.

2.6. Case 6

An 80-year-old woman was hospitalised in the neurology ward for management of a drowsiness. Examination found fever at 38.3˚C, a GCS score of 13, disorientation in time and space, and right hemiplegia involving the face, giving a NIHSS score of 16. Brain CT performed within 4 hours showed an asymmetric bithalamic hypodensity, predominant in the left thalamus (**Figure 7(A)**). A repeat CT obtained 3 days later after deterioration of consciousness (GCS 9) demonstrated bilateral capsulo-thalamic hypodensities (**Figure 7(B)**, **Figure 7(C)**). The ECG showed sinus tachycardia. Cervical and transcranial Doppler ultrasound revealed calcified atheromatous plaques and a floating thrombus in the right internal jugular vein at the level of the carotid bulb. A diagnosis of deep cerebral venous thrombosis was made. Chest radiography revealed infectious pneumonia. Immunologic and thrombophilia work-ups were not performed, and thoraco-abdomino-pelvic CT detected no neoplastic process. The patient received therapeutic-dose anticoagulation and broad-spectrum antibiotics. Despite treatment, she died on day eight in the context of a febrile coma.

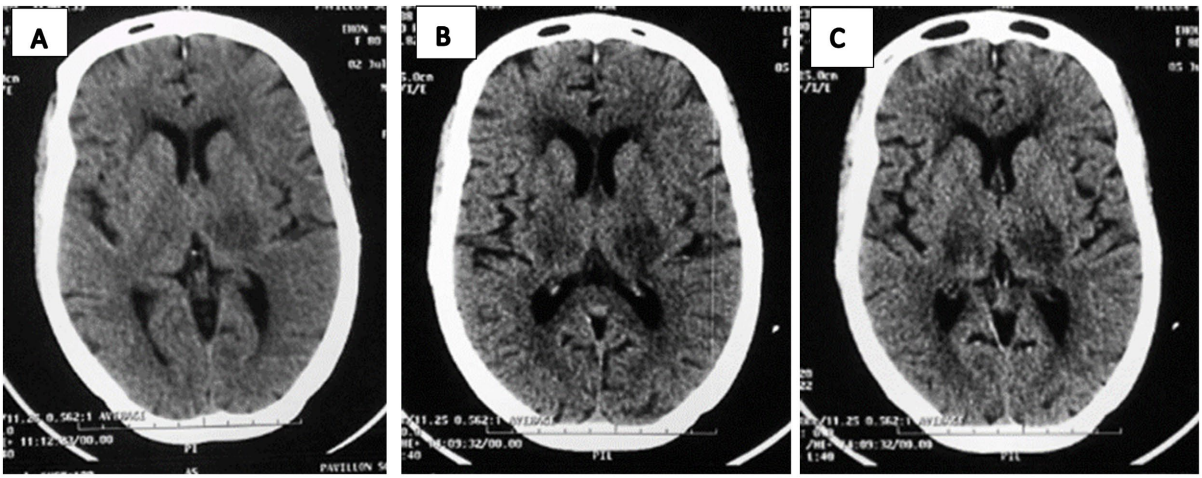

Figure 7. Brain CT showing an asymmetric bithalamic hypodensity at 4 hours (A) and, three days later, bilateral capsulo-thalamic hypodensities (B, C).

3. Comments

We describe cases of bithalamic infarction secondary to an artery of Percheron occlusion or deep cerebral venous thrombosis. These diagnoses were made without definitive imaging in several cases, which represents a primary limitation of this study, as they were presumptive based on the available clinical and radiological evidence.

The cases of bithalamic infarction we report illustrate the complexity of thalamic vascular supply, which comprises four arterial territories: anterior (tubero-thalamic), paramedian, inferolateral, and posterior choroidal. The paramedian territory depends on the thalamo-subthalamic paramedian arteries, also called thalamo-perforating arteries [7]. According to Percheron's anatomical descrip-

tions, there are four arterial variants in the thalamo-mesencephalic paramedian region [8]. Type IIb corresponds to a single common trunk arising from one P1 segment of the posterior cerebral artery and supplying the paramedian part of both thalami. Occlusion of this so-called artery of Percheron therefore produces a bilateral, paramedian thalamic infarct [9], as in our first and fifth cases. Two of our patients, however, had both paramedian and anterior bithalamic involvement. This can be explained by extension of the infarcted territory when the tuberothalamic artery is absent and the paramedian artery provides collateral supply in around one-third of cases [10].

Clinically, many presentations have been described, most of them atypical, but the most frequent picture includes vertical gaze palsy (65%), memory impairment (58%), confusion (53%), and coma (42%) [4]. In line with the literature, our patients each had at least two of these four major signs, and sometimes three were associated. Other, less common manifestations were also seen in our series, such as motor deficits in four cases and language disorders in two. Overall, the clinical picture was polymorphic, dominated by impaired consciousness, followed by motor, oculomotor, and finally cognitive disturbances. Likewise, Lamboley *et al.* [6] observed that no single sign—or combination of signs—is constant or specific for bithalamic infarction. On the contrary, this variety of non-systematised clinical signs may point to erroneous diagnoses such as vertebro-basilar stroke, subarachnoid haemorrhage, inflammatory or infectious encephalitis, opioid intoxication, or even Wernicke's encephalopathy [11]. Because of these varied and inconsistent manifestations, the same authors reported that making the diagnosis in the emergency setting is difficult [4] [6] [9].

Diagnostic wandering is also explained by the frequent absence of abnormalities on early brain CT in symptomatic, even comatose, patients [6]. In Lamboley's series [6], of four brain CTs performed 1 - 3 hours after symptom onset, only one showed thalamic involvement. In contrast, in our series, CT identified thalamic involvement in all patients, likely related to longer delays to imaging. Only one CT was performed 2 hours after symptom onset; in four others the delay ranged from 4 to 48 hours, and in one extreme case CT was done on day 5. Although in our study CT performed 2 hours to 5 days after onset revealed bithalamic hypodensity in all patients, it failed to detect lesions in the acute phase and required repeat imaging at 48 hours, as in our third case. This underlines the value of brain MRI, which is essential for establishing the diagnosis [6] and is currently the gold standard for bilateral paramedian thalamic infarction [2]. In our context, however, limited access and high cost often preclude urgent MRI. Only one in two patients underwent MR angiography or CT angiography in our study, and this was outside the acute phase. In most cases, patients receive CT, sometimes after long delays ranging from 11.0 ± 8.9 hours to 5.9 ± 6.5 days [12] [13]. Such delays may contribute to late diagnosis in this condition.

When a bithalamic hypodensity is seen on CT, in addition to artery of Percheron occlusion, deep cerebral venous thrombosis of the internal cerebral veins or

straight sinus should also be considered [6]. Deep venous thromboses are rare causes of cerebral ischaemia [14]. They cause behavioural, cognitive, and sometimes consciousness disorders up to coma, secondary to bilateral thalamic lesions. Diagnosing this rare, poor-prognosis form is particularly challenging [15]. MRI typically shows diffuse bithalamic oedema, sometimes involving other basal ganglia [6]—with lack of opacification of the deep venous system after contrast injection, but this examination is often unavailable. We also lack conventional catheter angiography which, although invasive, could confirm the diagnosis. Nevertheless, among our six cases we diagnosed deep cerebral venous thrombosis in two. In the first, the diagnosis was based on clinical and paraclinical arguments, a history of headache and a generalised seizure with a diffuse capsulo-thalamic pattern on CT, despite no venous MR angiography confirmation. In the second, the patient admitted for drowsiness in a febrile context with concomitant motor deficit had bilateral thalamic and internal capsular hypodensity and a floating thrombus in the right internal jugular vein.

In the remaining four patients, other differential diagnoses for bithalamic hypodensity were ruled out. The absence of fulminant hepatitis or decompensated cirrhosis argued against acute hepatic encephalopathy. There were no abnormal movements, tremor, or copper metabolism abnormalities to suggest Wilson disease. The profile did not match variant Creutzfeldt-Jakob disease, where patients are typically young with psychiatric symptoms, myoclonus, and dysaesthesias [16]. There was no history of chronic alcoholism, malnutrition, or ataxia suggestive of Wernicke encephalopathy. Ultimately, artery of Percheron occlusion was retained based on the bilateral, often symmetrical lesions confined to the paramedian and/or tuberothalamic territories, with a mechanism most likely embolic from the heart or proximal supra-aortic trunks [17]. Kostanian *et al.* showed that artery of Percheron occlusion can be visualised on superselective angiography [18]. In our series, none of the four presumed occlusions were confirmed by angiography or 3D TOF MRI; however, this does not invalidate the diagnosis, as failure to visualise the artery does not exclude its presence, particularly if occluded and given its small calibre [19].

For aetiological work-up, MR or CT angiography of the intracranial vessels and supra-aortic trunks is essential and also helps exclude basilar trunk obstruction [6], but these tests are not performed systematically, nor is prolonged Holter ECG monitoring, because of cost constraints. Consequently, work-ups are rarely exhaustive.

Therapeutically, although cases of intra-arterial and intravenous thrombolysis with favourable neurological outcomes have been reported [18] [20] [21], management is not standardised and no consensus exists. Antiplatelet therapy is generally initiated and continued long-term along with secondary prevention of cardiovascular risk factors. The superiority of effective anticoagulation in the acute phase has not been demonstrated, though some teams use it [22]. In our series, anticoagulation was required for the two deep cerebral venous thromboses and

for two patients with presumed Percheron artery occlusion in whom the work-up indicated a cardio-embolic cause, such as atrial fibrillation and atrial flutter. This anticoagulation was based on therapeutic-dose heparin bridged to vitamin K antagonists, which are still commonly used and have a lower cost than new oral anticoagulants, even though trends have evolved in recent years [23]. In the other cases where the presumed etiology of occlusion of the Percheron artery was an atheromatous embolism, patients received aspirin at a dose of 160 mg combined with lipid-lowering drug (atorvastatin 20 - 40 mg). Aspirin remains to date the reference treatment for secondary prevention of non-cardioembolic stroke, in profile of benefit-risk-cost balance [24].

According to the literature, disorders of consciousness often improve within hours or days [4]. However, two of our patients experienced worsening vigilance disorders and died during hospitalisation, which may be related to age, initial NIHSS score, and infectious or metabolic complications. In the other cases, moderate to severe motor and cognitive-behavioural sequelae persisted, potentially resulting in a dementia-like picture and constituting poor prognostic factors [11].

4. Conclusion

Because of its clinical polymorphism and the diversity of causes of bilateral thalamic involvement, bithalamic infarction poses genuine diagnostic challenges. These difficulties are compounded in our setting, where access to brain and supra-aortic trunk MR angiography remains limited, increasing the risk of diagnostic error and hindering optimal management. The high frequency of impaired consciousness in this condition complicates clinical assessment and mandates a diagnostic approach that correlates lesion topography with paraclinical findings. Thus, in similar settings, when confronted with an unexplained impaired consciousness, performing a control brain CT scan at 48 hours has great diagnostic value and is required when the initial scan is inconclusive.

Authors' Contributions

Chermine Mboumba Mboumba, Jennifer Nyangui Mapaga, Pupchen Gnigone and Grass Mambila Matsalou wrote this manuscript.

Nelly Diouf Mbourou, Keïla Ondimba Bassadila, Mael Ndao Eteno and Michel-Arnaud Saphou-Damon conducted a literature review.

Annick Nsounda and Ibrahima Camara translated the manuscript into English.

Kossivi Apetse, Agnon Koffi Balogou and Philomène Kouna Ndouongo made corrections.

Conflicts of Interest

The authors do not declare any conflict of interest.

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