CASE REPORT

REVISED Case Report: Thalamomesencephalic stroke in a patient with HIV [version 2; peer review: 1 approved with reservations]

Previously titled: 'Case Report: Thalamomesencephalic stroke due to vasculitis in a patient with HIV'

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Abstract

We present a 41-year-old HIV-positive female patient complaining of complete right palpebral ptosis, diplopia, and inability to balance herself. On examination, the right eye was able to move laterally and downwards. The motor exam showed left hemiparesis (4/5) on upper and lower limbs, bilateral Babinski sign with left hemiataxia without the sensory disorder.

CT scan and magnetic resonance imaging angiography demonstrated an ischemic infarct on the right paramedian branch of the posterior cerebral artery territory.

This patient did not present clinical manifestations of the thalamic lesion. To our knowledge, this is the first reported case of a young patient presenting a unilateral thalamomesencephalic ischemic stroke secondary to HIV vasculitis with bilateral Babinski signs and without thalamic signs in the medical literature.

Keywords

Thalamomesencephalic stroke, HIV, Vasculitis

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Any reports and responses or comments on the article can be found at the end of the article.

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REVISED Amendments from Version 1

In the previous version, we did not attach the necessary test results to prove this vasculitis's etiology and rule out systemic lupus erythematosus. In this version, we also include new images to prove vasculitis diagnosis apart from the previous explanation that I delivered before regarding this issue.

Any further responses from the reviewers can be found at the end of the article

Introduction

In 2018, Sato *et al.* reported a 62-year-old man presenting with a rare eye movement. This patient had a vertical one-and-a-half syndrome caused by unilateral thalamomesencephalic stroke (TMS)¹. Other eye movements' abnormalities, such as bilateral vertical gaze palsy, were previously reported due to a unilateral stroke of the rostral midbrain by other authors^{2,3}.

The anatomical circulation of the brain is complex and diverse, and the circle of Willis variations can include absence or fusion of components, incomplete process, fenestrations, fetal branches, and asymmetrical and duplication. From previous studies on autopsy and structural imaging scans, normal anatomical variations were detected in 48–58% of the general population, and even during fetal development^{4–8}.

An investigation done on human cadaveric brains have been demonstrated four major thalamic arterial territories, with notable blood variations. These areas receive blood supply by the polar, paramedian/thalamoperforating arteries, thalamo-oculomotor, and posterior choroidal arteries^{9–17}. The perforating arteries supply the medial the walls of the third ventricle, hypothalamus, and subthalamic-mesencephalic junctions. These areas include the oculomotor nucleus, red nucleus, subthalamic nucleus, substantia nigra, pretectum, trochlear nucleus, reticular formation of the midbrain, posterior part of the internal capsule, the rhomboid fossa, and also the rear part of the thalamus^{9–20}. Because the artery of Percheron occlusion can affect the thalamus and midbrain at the same time, here we have to mention that artery of Percheron is an uncommon vascular variant of the paramedian branches of the posterior cerebral artery, arising from one P1 segment, bifurcates, and bilateral supply to the bilateral paramedian thalami and the rostral midbrain^{10,11} but not unilaterally. Therefore, occlusion of Percheron arteriole causes an atypical pattern of bilateral infarct of the median thalami with or without mesencephalic damage.

From 2010 and 2017, several authors^{21–26} also reported a case series of ischemic stroke on the thalamus and different clinical manifestations. Other clinical presentations of TMS include see-saw nystagmus that shows intorsion and elevation of one eye, with synchronous extorsion and depression on the contralateral one, convergence-retraction nystagmus and contraversive ocular tilt reaction probable due to ischemic involvement of the interstitial nucleus of Cajal²⁷, anisocoria. Another author found vertical ocular motor disturbances in the vertical plane and eye movement synkinesis, hypersomnia, and coma as a

clinical manifestation of TMS²⁸. Others reported headaches, blurred vision, and diplopia as a particular variant of cerebral lacunae TMS²⁹. In 2012, Benjamin *et al.* established that HIV infection can cause TMS by opportunistic infections, secondary to a cardioembolic phenomenon, coagulopathy, and vascular diseases such as stenosis, acquired aneurysm, vasculitis, and direct/indirect effect of HIV infection and antiretroviral therapy³⁰. In our region, ischemic stroke due to infectious vasculitis is quite common. In 2017, the first case presenting bi-thalamic infarctions leading to acute vascular dementia associated with HIV infection was reported³¹.

Case presentation

A 41-year-old female presented with a 5-day history of inability to open the right eye associated with decreased vision of the right eye, which subsequently developed binocular diplopia. The patient also reported a failure to balance herself and could not walk independently. There was no history of trauma, excessive use of NSAIDs, contraceptives, use of vitamin supplements, or complaint of headaches. The patient did not smoke, drink alcohol, or use other recreation or illicit drugs. The patient has a background history of hypertension since her last pregnancy in 2016 and has been on treatment with hydrochlorothiazide (12.5 mg daily) and enalapril (5 mg daily). HIV-reactive with the latest CD4 (01/2020) count of 715 and viral load are lower than the detectable limit on treatment with a combination of tenofovir/emtricitabine/efavirenz TDF/FTC/EFV (300/200/600 mg daily).

On the nervous system examination, the patient was alert and well oriented with no meningeal signs. A cranial nerve exam revealed right cranial nerve 3rd palsy, right complete ptosis, right mydriatic pupil nonresponsive to light, and paralysis of the medial, superior, and inferior rectus plus inferior oblique (Figure 1).

Figure 1. Right, complete palpebral ptosis due to oculomotor nerve palsy.

The motor exam showed left hemiparesis (4/5) on upper and lower limbs, bilateral Babinski sign with left hemiataxia despite muscle weakness on the affected side, and no sensory disorder or extrapyramidal signs. The rest of the examination was within normal limits and there were no rashes noted.

The investigations done were as follows: Blood tests (on the day of admission) See [Table 1](#)

Computed tomography (CT) angiogram and MRI (done two days after admission) showed diffuse vasculitis with parenchymal changes seen in the right thalamus and midbrain and ischemic infarct in the area supplied by the right paramedian branch of the posterior cerebral artery due to vasculitis ([Figure 2–Figure 4](#)).

The cardiology team requested a cardiac review. A cardiac ultrasound (done the day after admission) showed an ejection fraction of 75%, No valvulopathy or effusion was present. The patient was admitted and started on the following treatment: Vitamin B12 supplementation (1000 µg IM daily for five days in the first week, then weekly for five weeks, aspirin (150 mg daily), enoxaparin (40 mg s/c daily), simvastatin (20 mg daily), pyridoxine (50 mg daily), thiamine (100 mg daily). The patient continued the chronic medication (hydrochlorothiazide 12.5 mg, enalapril 5mg and TDF/FTC/EFV 300/200/600 mg daily). Physiotherapy and occupational therapy are actively working with the patient.[TJ4] The patient received rehabilitation in our ward for two weeks. The right-sided hemiataxia did improve, but the power on the right side was still 4/5. She was referred to her base hospital to continue rehabilitation and a follow-up date with us in 1 month.

Discussion

Here we report a case of a 41-year-old woman with right-sided TMS. Unilateral TMS is uncommon, and its incidence remains unknown, but one study showed that it comprises about 0.6% to 1% of midbrain ischaemic strokes and often accompanied by other posterior circulation infarcts³².

Baran *et al.* conducted an observational study in 2018, which showed a male predominance. The study also showed that from an etiological point of view, the most common cause was extensive atherosclerosis, followed by cardio-embolism, apart from small vessel disease³³.

The main risk factors associated with extensive atherosclerosis are hypertension, diabetes, hyperlipidemia, smoking, and previous history of stroke. The main risk factors for cardio-embolism in these patients is atrial fibrillation³³. Patients who are suffering from peripheral vascular disease and coronary artery disease are at risk³⁴.

HIV is a risk factor for stroke³⁵ and is associated with advanced disease³⁶. There have been numerous mechanisms proposed to explain this. A systematic review done by Addallah *et al.*³⁷ reported that this could be due to HIV-associated opportunistic infections, HIV-induced coagulopathy, and chronic inflammatory processes that can accelerate atherosclerosis. Another systematic

review by Bogorodskaya *et al.*³⁸ also reported that some antiretrovirals (lopinavir, indinavir, and abacavir) were also associated with an increased risk of stroke.

Lesions of the midbrain can present as distinct syndromes. However, because of the structures' close organization, there can be considerable overlap of these syndromes. The neurological manifestation will depend on which area of the midbrain is affected and whether one half or both halves are involved and whether adjacent structures (thalamus, pons, cerebellum) are also involved. The symptoms may include but are not limited to, low equilibrium, weakness of one or both sides of the body, diplopia, and slurred speech³⁹. The most common examination findings include ataxia, limb weakness, dysarthria, sensory disturbance, oculomotor findings (3rd nerve palsy, internuclear ophthalmoplegia), and dysarthria⁴⁰. The exact pattern will depend on the area involved and whether surrounding structures are also involved (thalamus, pons, medulla, etc.). There are have been midbrain syndromes, which include, among others, Weber syndrome, Claude's syndrome, Nothnagel syndrome, and Benedikt's syndrome. Benedikt's syndrome presents with a contralateral rubral tremor, which she does not have.

Weber syndrome is a result of a lesion involving the ventromedial area of the midbrain. They present with ipsilateral 3rd nerve palsy with contralateral hemiplegia. Our patient has Claude's syndrome, which presents ipsilateral 3rd nerve palsy with contralateral cerebellar ataxia due to the dorsal tegmentum lesion, which involves the 3rd nerve nucleus/fibers and also involving either the red nucleus, superior cerebellar peduncle, or brachium conjunctivum³⁷. Benedikt's syndrome is due to a lesion involving the tegmentum. It presents with ipsilateral 3rd nerve palsy and contralateral ataxia, but there is also the involvement of the fibers of the corticospinal tract and will result in contralateral hemiparesis even⁴¹. When assessing these kinds of patients, it is essential to ascertain a good history and physical examination and check the National Institute for Health Stroke Score⁴². Imaging to confirm the diagnosis is mandatory. CT or MRI angiogram is usually requested to identify the stenosed vessels or identify other possible vascular problems. Blood workup for stroke is compulsory, which includes but is not limited to full blood count, renal function tests, international normalized ratio, lipid profile, HIV ELISA, and if young to include thrombophilia screen, Antinuclear antibodies, and glycosylated hemoglobin. ECG to rule out possible atrial fibrillation and transthoracic or even transesophageal echocardiography to identify cardiac causes.

The management approach depends on the etiology of the stroke. If the infarct is ischaemic, the reviewed literature recommends thrombolysis if posterior circulation strokes meet the established criteria⁴³. Mechanical thrombectomy benefits are not yet well established, but it can be done⁴⁴. Then after the acute period, it is crucial to managing the risk factors and causes. Then treat the risk factors such as arterial hypertension, diabetes mellitus, hyperlipidemia, and secondary prophylaxis. If there is a cardiac cause, then it should be treated. A multidisciplinary approach is vital for patients presenting

Table 1. Parameters measured in patient blood tests.

Variable	Patient value	Normal range
White cell count	7.10 x 10 ⁹ /L	3.9-12.6 x 10 ⁹ /L
Hb	10.4 g/dL	12-15 g/dl
Platelets	356 x 10 ⁹ /L	186-454/L
Sodium	140 mmol/L	136-145 mmol/L
Potassium	4.4 mmol/L	3.5-5.1 mmol/L
Chloride	102 mmol/L	98-105 mmol/L
Urea	7.1 mmol/L	2.1-7.1 mmol/L
Creatinine	68 µmol/L	48-90 µmol/L
Calcium	2.23 mmol/L,	2.15-2.5 mmol/L
Magnesium	0.83 mmol/L,	0.63-1.05 mmol/L
Phosphate	1.48 mmol/L	0.78-1.42 mmol/L
C-reactive protein	1 mg/L	<10 mg/L
Erythrocyte sedimentation rate	16 mm/h	0-10 mm/hr
Total protein	74 g/L	60-78 g/L
Total Bilirubin	<3 µmol/L	5-21 µmol/L
Alkaline phosphatase	92 U/L	42-98 U/L
Aspartate transaminase	23 U/L	13-35 U/L
Alanine transaminase	18 U/L	7-35 U/L
Total cholesterol	4.78 mmol/L	<4.5 mmol/L
HbA1C	5.1%	<7%
International normalized ratio	1.01	1
D-dimer	0.8 mg/L	0.00-0.25 mg/L
Rheumatoid factor	7 IU/ml	<20 IU/L
Vitamin B12	136 pmol/L	145-569 pmol/L
Thyroid stimulating hormone	0.78 mIU/L	0.27-4.2 Miu/l
Antocardiolipin antibody	negative	
Protein S	40 IU/dL	55-123 IU/dl
Protein C	100 IU/dl	70-130 IU/dL
Angiotensin converting enzyme	30 IU/L	8-53 IU/L
Anticardiolopin antibody	negative	
Anti-streptolysin O titre	88 IU/ml	<200 IU/L
Toxoplasmosis gondi IgG antibody	Positive	
Cytomegalovirus IgG antibody	Positive	
Rubella IgG antibody	Positive	
Rubella IgM antibody	Negative	
Cytomegalovirus IgM antibody	Negative	
C3	1.5g/L (0.9-1.8g/L)	
C4	0.4g/l (0.1-0.4g/L)	
Antinuclear antibody	Negative	
Anti-double strand DNA antibody	Negative	
Anti-RNP antibody	Negative

Figure 2. Magnetic resonance imaging of the brain. The axial view shows the right hyperdense lesion at the paramedian thalami caused by ischemic infarct secondary to HIV vasculitis.

Figure 4. Contrasted CT image. showing 'beading' of the paramedian blood vessels supplying the midbrain. (Suggestive of vasculitis).

Figure 3. Magnetic resonance imaging of the brain. The axial view shows a hyperdense lesion on the right midbrain caused by ischemic stroke due to HIV vasculitis.

TMS. The stroke team should include dieticians, physiotherapy, speech therapy, occupational therapy, and social workers apart from the medical specialists.

Risk factors for developing stroke in our patient were hypertension, HIV, and hyperlipidemia. We had done an extensive

workup to rule out other possible contributors to a stroke. The patient also had contralateral hemiparesis and hemiataxia with bilateral Babinski and hyperreflexia on both lower limbs. Her blood workup showed that she was virally suppressed and had hyperlipidemia. The MRI and CT angiograms showed evidence of an infarct involving the ventromedial midbrain and thalamus, and in the absence of other lesions. Cardiovascular investigations ruled out a cardiac source of the infarct. In this patient, hypertension, HIV infection, and hyperlipidemia predisposed her to the stroke. The patient is markedly younger than one would typically expect for a TMS (median age around 64 years)³². Of note in our patient is the presence of a bilateral Babinski sign, which never happens in a patient with Claude's syndrome.

Generally, the strokes involving posterior circulation have a higher mortality rate than those involving anterior circulation unless it involves the smaller blood vessels⁴¹, as happened in our case. The present case is unique, among other reasons, owing to the bilateral Babinski sign and the absence of thalamic manifestations without other lesions affecting different segments of the brainstem and the spinal cord. The patient's age (41 years) also makes this case uncommon. The patient's leading risk factor is HIV vasculitis, which has not been implicated for TMS from the literature reviewed. We did not find signs of middle longitudinal fascicle (MLF) typical syndrome. MLF syndrome, secondary to ischemic stroke affecting only the mesencephalon, is a rare occurrence^{45,46}. We would highlight that we could not find the cause of the bilateral Babinski signs in this case. This patient did not present thalamic characters despite the ischemic lesion in the right thalamus despite the midbrain's role over the thalamus. The modulation of

thalamic neurons is necessary to control adaptive behavior mediated by midbrain cholinergic transmission. The central cholinergic neurons in the mesencephalon are at the pedunculo-pontine nucleus and the laterodorsal tegmental nucleus, which provide dense innervation of the thalamus. Recently, Huerta-Ocampo *et al.* confirmed that midbrain cholinergic neurons could innervate all thalamic nucleus. They also found that these axons topographically are well organized and provide a segregated innervation of the thalamic nuclei⁴⁷. Therefore, we expected some evidence of thalamic dysfunction in this patient. However, these ischemic lesions on the midbrain did not cause abnormal behavior or thalamic manifestations in our patient, which is a novel finding.

To our knowledge, this is the first patient presenting with unilateral TMS secondary to HIV vasculitis with bilateral Babinski signs, and without thalamic manifestations to be reported in the medical literature. In young patients presenting with unilateral TMS, HIV vasculitis is one of the etiological diagnoses to be considered. Still, we recommend an extensive investigation based on a series of cases to support this postulate.

Data availability

All data underlying the results are available as part of the article, and no additional source data are required.

Consent

Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

Author contributions

All authors contributed equally to the elaboration of this manuscript. MT and SJ collected data and planning this report, JG and LIV wrote the first draft and reviewed bibliographically, TB and HFS wrote the final manuscript. All authors reviewed the final manuscript, made corrections, and agreed for publications

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Version 2

Reviewer Report 20 January 2021

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Jefferson V. Proano

Hospital de Especialidades, Centro Médico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Mexico City, Mexico

As the authors stated in their reply, the cause of the stroke in this case was not definitively established. However they assert in the footnotes of the images that the cause was HIV: "The axial view shows a hyperdense lesion on the right midbrain caused by ischemic stroke due to HIV vasculitis."

Secondly, no citation or reference is provided for the following statement: "In our region, ischemic stroke due to infectious vasculitis is quite common." If there is no literature supporting this affirmation the writing should be changed to a formulation similar to "in our clinical experience".

Finally, the only vascular imaging shown is a contrast enhanced CT of the brain, but angiographic imaging is mentioned ("The MRI and CT angiograms showed evidence of an infarct involving the ventromedial midbrain and thalamus, and in the absence of other lesions.") If such imaging is available, a representative image demonstrating vasculitis should be part of the case report.

If these modifications were carried out I would be in favor of indexing.

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: Neurology and Infection disease of the nervous system

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.

Version 1

Reviewer Report 30 October 2020

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Jefferson V. Proano

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The authors assume that the etiology of the strokes in the case they present, is due to vasculitis due to HIV, but it is completely necessary to exclude other causes of vasculitis, such as systemic lupus erythematosus, in addition to the fact that the patient has other risk factors for strokes. Furthermore, it is difficult to suppose that this patient having the HIV virus copy number parameter at levels of undetectable, however, develops vasculitis due to this cause.

Is the background of the case's history and progression described in sufficient detail?

Partly

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?

Partly

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?

Partly

Is the case presented with sufficient detail to be useful for other practitioners?

Partly

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: Neurology and Infection disease of the nervous system

I confirm that I have read this submission and believe that I have an appropriate level of expertise to state that I do not consider it to be of an acceptable scientific standard, for reasons outlined above.

Author Response 01 Nov 2020

Humberto Foyaca-Sibat, Walter Sisulu University/Nelson Mandela Academic Hospital, Mthatha, South Africa

Dear Reviewer,

We confirmed the diagnosis of vasculitis by MRI images, as you can see in the radiologist's report.

In this region, the commonest cause of vasculitis is an infectious disease caused by 1.- HIV/AIDS, 2.- TB, 3.- Neurocysticercosis, or 4.- Neurosyphilis.

Fortunately, we did not see SLE causing vasculitis at this shores. Because we could not rule out SLE, then we did not say HIV-vasculitis, then we titled this manuscript as....vasculitis in HIV patient.

Thanks for your kind attention and professional comments.

Regards,

Prof H Foyaca. MD Ph.D

Competing Interests: No competing interests were disclosed.

Author Response 08 Jan 2021

Humberto Foyaca-Sibat, Walter Sisulu University/Nelson Mandela Academic Hospital, Mthatha, South Africa

Dear Reviewer,

As you can see we made all the changes that you suggested. We are completely agreed and very happy with your suggestions.

Thanks a lot for your kind attention and professional support.

Regards,

Dr. Foyaca

Competing Interests: We declare that there is not competing interest in this publication

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