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ORIGINAL ARTICLE

Reversible cerebral vasoconstriction syndrome following carotid artery revascularization: About three case reports and review of literature

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 Reversible cerebral
 vasoconstriction
 syndrome (RCVS) ;
 Posterior reversible
 encephalopathy
 syndrome (PRES)

Summary Complication after carotid artery revascularization is mainly represented by stroke. Reversible cerebral vasoconstriction syndrome triggering by carotid artery revascularization is exceptional but it is an unrecognized aetiology of stroke. It could be associated with brain edema and henceforth, a posterior reversible encephalopathy syndrome can be confused with post-carotid artery revascularization cerebral hyperperfusion syndrome. We reported three cases about reversible cerebral vasoconstriction syndrome following revascularization therapy whose one had also a posterior reversible encephalopathy syndrome. This complication occurred within two weeks after carotid artery revascularization. All took nimodipine and all had a functional improvement at 3-months follow-up. Reversible cerebral vasoconstriction syndrome with posterior reversible encephalopathy syndrome after carotid artery revascularization may mimic cerebral hyperperfusion syndrome. After a comprehensive review, nineteen cases of reversible cerebral vasoconstriction syndrome post-carotid artery revascularization have been reported in the literature mainly after symptoms. It occurs mainly in women around sixty years of age. The onset is expected four days after revascularization.

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Abbreviations

CAR	carotid artery revascularization
CAS	carotid artery stenting
CBF	cerebral blood flow
CEA	carotid endarterectomy
CHS	cerebral hyperperfusion syndrome
CT	computed tomography
CTA	computed tomography angiography
ICA	internal carotid artery
ICHD-3	International Classification of Headache Disorders
MCA	middle cerebral artery
MRI	magnetic resonance imaging
mRS	modified Rankin Scale
NASCET	North American Symptomatic Carotid Endarterectomy Trial
NIHSS	National Institutes of Health Stroke Scale
PRES	posterior reversible encephalopathy syndrome
RCVS	reversible cerebral vasoconstriction syndrome
SSRI	selective serotonin reuptake inhibitor
TICI	thrombolysis in cerebral infarction

Introduction

In Europe, stroke causes 1.1 million deaths annually [1] and it is the most common cause of worldwide disability. In nearly one third of cases, large-artery atherosclerosis is the etiology of stroke [2]. Carotid artery endarterectomy (CEA) is indicated for 50 to 99% symptomatic carotid artery stenosis evaluated by the NASCET scale [3–5]. However, an alternative option is carotid artery stenting (CAS) especially for patients with high-surgical risk or hostile neck [6]. Carotid artery revascularization (CAR) must be performed within fourteen days after symptoms onset to prevent stroke recurrence. In mostly cases, no complication occurs but a stroke might happen especially during CAS (10%) compare to CEA (6%) [7]. Herein, we report three cases of stroke during reversible cerebral vasoconstriction syndrome (RCVS) after CAR, discuss the differential diagnoses and describe the reported cases in literature.

Methods

We used the hospital database of three French hospitals to collect medical history, clinical data, radiological finding, treatment and outcome of patients.

We reviewed the medical literature on RCVS and CAR (from 1990 to October 2020) using these keywords ‘RCVS’, ‘Reversible Cerebral Vasoconstriction Syndrome’, ‘Vasospasm’, ‘endarterectomy’, ‘vasoconstriction syndrome’, ‘carotid surgery’, ‘carotid stenting’. For this, we conducted database searches (PubMed®) and cross-referenced the bibliographies of related articles [8–25]. Whenever possible, we collected information about patients’ feature at the time of diagnosis, type of intervention, laterality of intervention, trigger, diagnosis, initial treatment, additional treatment and patient outcome.

All cases fulfilled the ICHD-3 diagnosis criteria of RCVS [26]. In case 2, in the absence of validated diagnostic criteria of PRES, the diagnosis was made based on clinical and

neuroradiological evidence by experts in neurovascular field [27].

Results

Observation

Case No. 1: a 62-year-old woman was admitted with a left sudden and painless amaurosis fugax for 15 minutes. She was a smoker (40 pack per year) with a history of hypertension, hypercholesterolemia, family history of cardiovascular disease and depression. She took citalopram, simvastatin, spironolactone and altizide. A computed tomography (CT) angiography (CTA) showed a near occlusive internal carotid artery stenosis on left side without any abnormalities in brain (Fig. 1a). Additionally, a Doppler ultrasonography found an 80–90% carotid artery stenosis on North American Symptomatic Carotid Endarterectomy Trial (NASCET). She was treated with eversion CEA under general anesthesia without elective shunting nor intraoperative angiogram or monitoring. No early complication was reported. She was discharged on the 13th day following the procedure with aspirin, nicotine replacement therapy in addition to her previous medication. Six days later, she was brought to the hospital for recurrent thunderclap headaches and right hemiplegia (National Institutes of Health Stroke Scale [NIHSS] = 6). Her blood pressure was normal. A non-enhanced CT scan showed a left-sided brain infarction. An arteriography showed diffuse narrowing of cerebral arteries especially in the M2 segment of the left middle cerebral artery suggesting a RCVS (Fig. 1b). Only selective serotonin reuptake inhibitors (SSRI) and nicotine replacement therapy was discontinued and nimodipine was introduced. At 3-month follow-up, she had a modified Rankin scale (mRS) of 1, nimodipine was withdrawn and no significant vasospasm on her arteries (Fig. 1c) was showed. No further vascular event, myointimal hyperplasia or headaches recurrences were reported.

Case No. 2: a 72-year-old woman was admitted to the Emergency Room after recurrent right hemiparesis and aphasia. She had a history of arterial hypertension and depression. Her medications include clopidogrel, hydrochlorothiazide, valsartan and fenofibrate. Two weeks before her admission, an eversion CEA was successfully performed for an ulcerative carotid stenosis (NASCET 80%) associated with an ipsilateral silent parietal infarction. The procedure was performed without the use of a shunt or intraoperative angiographic control. The procedure was well tolerated and the patient showed no immediate postoperative complications. At the admission, her neurological examination found a bradycardia (NIHSS = 1) with mild headaches. A CT Angiography (CTA) of intra- and extracranial artery was unremarkable and cerebral CT scan revealed no new finding (Fig. 2a). She was discharged with the diagnosis of transient ischemic attack. Ten days after, she was readmitted for persistent right hemiparesis. A CTA showed a narrowing of A2 and M2 segment of the left cerebral artery (Fig. 2b) in favor of RCVS but few days after nimodipine onset and SSRI withdrawal, her condition worsened and she developed a right hemiplegia and aphasia during acute hypertension. An MRI showed a vasogenic edema on

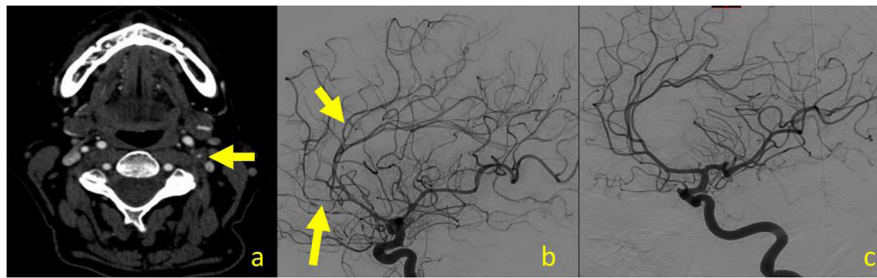


Figure 1 Case 1 imaging. a: enhanced brain computed tomography showed tight stenosis (yellow arrow); b: sagittal digital subtraction angiogram at acute stage showed diffuse vasospasms (yellow arrows); c: sagittal digital subtraction angiogram at three-month follow-up showed the reversibility (yellow arrows).

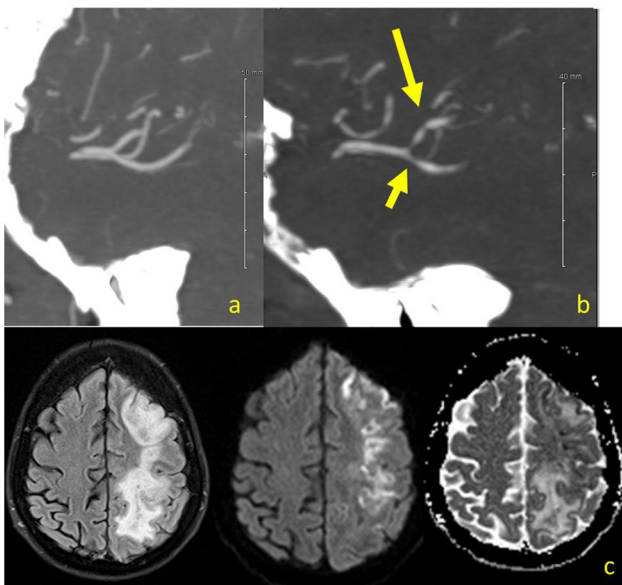


Figure 2 Case 2 imaging. a: enhanced brain computed tomography showed no abnormality; b: enhanced brain computed tomography showed vasospasm on MCA (yellow arrows); c: fluid-attenuated inversion recovery (FLAIR) and diffusion weighted sequences showed hyperintensities in left hemisphere with no diffusion restriction diffusion evocating an vasogenic edema.

the left hemisphere in favor of a posterior reversible encephalopathy syndrome (PRES) (Fig. 2c). After strict blood pressure control without hyper- or hypotension, the patient has slowly recovered. At 3-month follow-up, she still had a persistent right upper limb monoparesia (mRS=3) but MRI showed complete resolution of the vasospasm and the brain edema. She still took clopidogrel, hydrochlorothiazide, valsartan and fenofibrate without recurrence of vascular disease. No ultrasonography follow-up has been conducted.

Case No. 3: a 53-year-old woman, with a past medical history of current smoking (30 packs per year), a non-treated hypercholesterolemia and high blood pressure was admitted for a sudden right hemiplegia and aphasia (NIHSS = 10). At admission, her NIHSS improved to 3. An MRI reported an acute brain infarction in the left deep middle cerebral artery (MCA) territory with a tandem lesion (left internal carotid

occlusion and occlusion of the first segment of the MCA), and a subacute stroke was detected on the contralateral side. An endovascular treatment was performed: the occluded carotid was traversed using an aspiration catheter that achieved a full recanalization of the intracranial arteries by endovascular treatment. After confirming the persistence of 53% NASCET carotid stenosis (Fig. 3a), CAS was performed under local anesthesia. An angiogram showed successful stent implantation without residual stenosis. Her neurological condition quickly improved and she was discharged at home with NIHSS 1 with aspirin, clopidogrel, atorvastatin and nicotine replacement therapy. Two days after (seven days after endovascular therapy), she had spontaneous frontal acute headaches with no other symptoms nor hypertension during 4 hours when she was sleeping. A CT scan showed narrowing in the downstream of left carotid confirmed by MRI without any brain complication (Fig. 3b). Nimodipin was added to her treatment for 4 weeks and nicotine replacement therapy stopped. At 3-month follow-up, CTA confirmed the reversibility and the permeability of stent (Fig. 3c). She had an NIHSS 0 and mRS of 1. Only clopidogrel was withdrawn at this time. She denied any recurrence and her follow-up showed an excellent patency of the stent.

Review

We reviewed the previously published literature and identified 19 cases of RCVS following a CAR (Table 1). Among these 19 cases, 12 cases had a symptomatic carotid stenosis [8–11,16], 5 patients had been treated by a carotid stenting [14,20] and the other by CEA. Similar to the present cases, high-grade carotid stenosis was reported in almost all patients but different radiological modalities were used for the stenosis assessment. The median time of RCVS onset following revascularization is 4 days (range 0–30) with headaches as the main onset symptom. RCVS post-revascularization affects relatively young patients, with a mean age at onset of 62.7 (± 10.1) years, mainly female patients. Few patients had additional precipitant factors like SSRI uptake [14,16]. No PRES was reported. Treatment for RCVS included calcium channel blocker [9–17], corticotherapy [9] and hypertension management [10]. No recurrence has been reported in any of these cases. Condition of all patients improved after RCVS.

Table 1 Summary of the literature cases of RCVS after CAR.

Authors	Age	Sex	% stenosis	Side of stenosis	CAR indication	Intervention	Symptom onset	Postoperative symptoms	Treatment	Brain infarction	Outcome
Brick [8]	57	M	70%	Right	Symptomatic	CEA	8	Headache, confusion, agitation, paranoia	None	No	Improvement
Lopez-Valdes [9]	54	F	80–90%	Left	Symptomatic	CEA	8	Headache, nausea, dizziness	Verapamil	Yes	Improvement
Lopes-Valdes [9]	50	M	95%	Right	Symptomatic	CEA	7	Headache, loss of vision and transient numbness of left side	Corticoid	Yes	Improvement
Dagher [10]	62	M	70–99%	Left	Symptomatic	CEA	5	Right upper extremity weakness and expressive aphasia	Diltiazem	No	Improvement
Rosenbloom [11]	54	F	–	Left	Symptomatic	CEA	2	Headache	Nimodipine	Yes	Improvement
Bodenant [12]	74	M	90%	Right	Asymptomatic	CEA	4	Headache	Nicardipine	No	Improvement
Wu [13]	58	F	95%	Right	Asymptomatic	CEA	3	Headache, nausea, transient mild left-sided weakness and clumsiness	Nimodipine	Yes	Improvement
Aghaebrahim [14]	60	F	–	Right	–	CAS	30	Headache and left-sided weakness	Verapamil	–	Improvement
Causey [15]	69	M	80–90%	Left	Asymptomatic	CEA	4	Transient episode of right hand ataxia	Verapamil, nimodipine	Yes	Improvement
Fitas [16]	55	F	65–70%	Right	Symptomatic	CEA	3	Headache, apathy, somnolence	Nimodipine	Yes	Improvement
Asuzu [17]	54	F	80–99%	Right	Asymptomatic	CEA	3	Headache	Verapamil, nimodipine	Yes	Improvement
Judge [18]	65	F	–	Left	–	CEA	1	Headache	–	No	Improvement
Tunguturi [19]	61	F	90%	Left	Symptomatic	CEA	4	Headache	Verapamil	Yes	Improvement
Meybodi [20]	59	F	> 90%	Right	Symptomatic	CEA	4	Confusion	Corticoid	No	Improvement
Higashi [21]	73	M	85%	Left	Symptomatic	CAS	0	Disturbance of consciousness, Right hemiparesis	Hypertensive therapy	Yes	Improvement
Isikbay [22]	84	F	≥ 70%	Right	Asymptomatic	CEA	10	Left arm and leg weakness and numbness	Verapamil	Yes	Improvement
Shoskes [23]	74	F	Occlusion	Right	Symptomatic	CAS	5	Left hemiplegia with right gaze deviation	Verapamil	Yes	Improvement
Soltanolkotabi [24]	49	F	70–80%	Left	Symptomatic	CAS	0	Headache	Verapamil	Yes	Improvement
Kobayashi [25]	79	M	95%	Left	Symptomatic	CAS	0	Right hemiplegia, aphasia, and loss of consciousness	Corticoid	Yes	Improvement

F: female; M: male, CAR: carotid artery revascularization, CEA: carotid endarterectomy; CAS: carotid artery stenting.

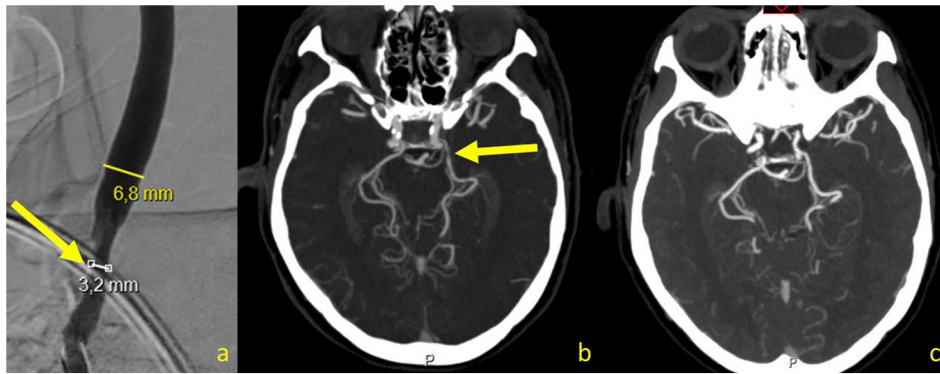


Figure 3 Case 3 imaging. a: digital subtraction angiography showed tight stenosis of the left internal carotid artery (yellow arrow) during the intervention; b: computed tomography angiography eight days after stenting showed vasospasms on left posterior cerebral artery (yellow arrow); c: computed tomography angiography at three-month follow-up showed vasospasm reversibility.

Discussion

We report three cases of RCVS after CAR. This case series highlights two points:

- the RCVS should be considered as curable etiology of neurological worsening after CAR;
- RCVS and its related disease like PRES share some radio-clinical features with another CAR complication: the cerebral hyperperfusion syndrome (CHS).

RCVS, a rare cause of neurological worsening after CAR

While stroke remains a major cause of neurological worsening after CAR, the underlying pathophysiological mechanism is relevant to prevent and manage this complication. Stroke from thromboembolism emerges as the predominant factor in more than half of cases [28] and can result from the disruption of the atheromatous plaque during the intervention or, unusually, from a focal dissection. Hemodynamic impairment characterized by cerebral hypoperfusion that may be induced by intravenous anesthetic drugs, injury of the pericarotid sympathetic baroreceptors and/or carotid occlusion by clumping or balloon dilatation can lead to junctional stroke. On the other, impaired cerebral autoregulation may provoke neurological worsening and stroke after CAR. Initially, CHS, due to acute cerebral perfusion raising, was described both in CEA and CAS but, recently, another impaired cerebral autoregulation disorder, RCVS had also been described after CAR [11] (Table 2).

According to the International Classification of Headache Disorders 3rd edition (ICHD-3) classification [26] (Table 2), key symptoms of RCVS are recurrent thunderclap headaches [29]. Nonetheless, the absence of typical thunderclap headache does not exclude the diagnosis of RCVS [30]. Multiple complications may appear during RCVS like seizure, intracranial haemorrhage (hematoma or subarachnoid haemorrhage) but also brain infarction and PRES respectively in up to 39 and 38% of cases [31,32]. Apart from common precipitating factors like vasoactive substances and peripartum, another factors have been described included CEA [11] but with a very low prevalence (Table 1). RCVS

is radiologically characterized by segmental and transient cerebral vasoconstriction on angiography. Calcium channel blockers are administered to relieve the vasospasm and eviction of precipitating factors should be considered. The outcome is usually good.

Similarities of RCVS with PRES and CHS, a common spectrum?

As previously described, some authors have described co-occurrence between PRES and RCVS [33] but the association of RCVS-PRES share some radio-clinical presentation with another impaired cerebral autoregulation disorder: the CHS.

Cerebral hyperperfusion syndrome (CHS) is characterized by transient cerebral hyperperfusion after carotid recanalization. CHS has a postoperative incidence of 1% and occurs within the first month after CAR. CHS predominates in men aged 70 to 78 years [34]. Patients may have headaches, seizures, focal neurological deficit, and acute encephalopathy. Most cases achieve a remarkable recovery, although intracranial hemorrhage occurs in 10% and death in 4% [35]. Cerebral perfusion imaging plays a central role in diagnosis.

According to Table 2, only perfusion imaging is required to make the diagnosis of CHS, but vessel analysis is not in the diagnostic criteria of CHS and this may lead to a measurement bias, since no study in the literature has described vessels during CHS [35–37]. Distinguishing RCVS-PRES and CHS can be challenging in clinical practice, since, both conditions share common neurological and radiological features except thunderclap headaches and vasospasms in RCVS. Early discrimination between these entities is important because of management differences. It is required to always assess the arterial vasculature after neurological symptoms following CAR from the carotid to the brain. On one hand, the treatment of CHS, like PRES, relies on hypertensive therapy, which may be deleterious in the case of RCVS due to hypoperfusion. On the other hand, as illustrated in case 2, in RCVS, the prescription of a calcium channel blocker – a vasodilator with a low antihypertensive effect – can increase cerebral blood flow and lead to cerebral oedema. We did not consider the value of perfusion imaging in case 2, given the common evidence of spasm and oedema, and

Table 2 Main characteristics of RCVS, PRES and CHS.

	PRES [39]	RCVS [33]	CHS [36]
Definition/diagnostic criteria	N/A	ICHD-3	N/A
Precipitating factors	Malignancy, pre-eclampsia, renal injury, autoimmune disorders, sepsis, acute hypertension, immunosuppressive and chemotherapeutic medications, idiopathic	Pregnancy and puerperium, vasoactive drugs and illicit substances Acute stress	Acute carotid reperfusion
Clinical presentation			
Headaches	Moderate to severe	Thunderclap headaches	Moderate to severe
Seizure	Common	Uncommon	Uncommon
Focal neurological deficit	Uncommon	Uncommon	Uncommon
Encephalopathy	Common	Rare	Uncommon
Visual loss	Common	Uncommon	Not reported
Radiological findings			
Usefulness of DSA	None	Yes	No
Oedema	Yes	No	Yes
Contrast enhancement	Uncommon	Rare	N/A
Hemorrhagic manifestations	Uncommon	Yes	Uncommon
Ischemic manifestations	Very rare	Uncommon	Rare
Vasoconstriction	Uncommon	Very frequent	N/A
Perfusion	Hyperperfusion	Hypoperfusion	Hyperperfusion
Criteria diagnosis	Suggested by Fischer et al. [27] Neurological symptoms of acute onset Focal vasogenic edema on neuroimaging Reversibility of clinical and/or radiological findings	According ICHD-3 [26] Thunderclap headaches and/or triggered by sexual activity, exertion, Valsalva maneuvers With or without focal deficits and/or seizures Angiography (with "string of beads" appearance) Disappearance of the headaches and arterial abnormalities within three months	Suggested by Van Mook et al. [36] Acute neurological symptoms after carotid reperfusion No abnormalities on CT scan Cerebral hyperperfusion on perfusion imaging Disappearance of symptoms after normalization of cerebral blood flow
Treatment	Conservative management Antihypertensive therapy	Conservative management Withdraw any vasoactive substance Calcium channel blocker	Conservative management Antihypertensive therapy

N/A: not available; CHS: cerebral hyperperfusion syndrome; RCVS: reversible cerebral vasoconstriction syndrome; PRES: posterior cerebral encephalopathy syndrome; ICHD-3: The International Classification of Headache Disorders 3rd edition; DSA: digital subtraction angiography.

the patient was managed with strict blood pressure control associated with nimodipine administration.

RCVS, PRES, CHS: an underlying common pathophysiology

CHS, PRES, and RCVS might share the same pathophysiological pathways, even if partly understood, leading to dysregulation of cerebral autoregulation [38]. Cerebral autoregulation refers to the capacity to maintain constant cerebral blood flow despite changes in blood pressure through arteriolar diameter adjustments. In PRES, acute

high blood pressure above the upper autoregulation range leads to cerebral hyperperfusion, which may cause blood brain barrier disruption and vasogenic edema. Endothelial dysfunction caused by cytotoxic injury may lead to vasospasm and increased vascular permeability leading to vasogenic edema [39]. In CHS, after a chronic brain hypoperfusion, hyperperfusion post-revascularization might overwhelm cerebral autoregulatory mechanisms leading to a blood brain barrier disruption. Moreover, release of oxygen-derived free radicals during carotid endarterectomy could alter endothelial function and induce cerebral regulation dysfunction leading to vasogenic edema [36]. In RCVS, impaired cerebrovascular autoregulation attributed to autonomic

dysregulation, oxidative stress, impaired endothelial function, and blood brain barrier disruption might impact in pathophysiology and lead to exaggerated vasoconstriction [33].

The description of these strictly unilateral abnormalities after revascularization reinforces the hypothesis of a common pathophysiology between RCVS-PRES and their resemblance to CHS among a cerebral autoregulation impairment disorder spectrum.

Conclusion

We report three cases of uncommon and curable complications following CAR that vascular physicians should be aware. The underlying pathophysiology of these syndromes involves a cerebral autoregulation impairment. According to common pathophysiology and clinical features, RCVS, PRES and CHS might be three faces of a one disorder.

Human and animal rights

The authors declare that the work described has not involved experimentation on humans or animals.

Informed consent and patient details

The authors declare that they obtained an informed consent from the patients included in the article (oral consent for patient 1, next of kin written consent for patient 2, written consent for patient 3). The authors also confirm that the personal details of the patients have been removed.

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Disclosure of interest

The authors declare that they have no competing interest.

Author contributions

All authors attest that they meet the current International Committee of Medical Journal Editors (ICMJE) criteria for Authorship.

Pacôme Constant dit Beauvils (PCDB): conceptualization, investigation, methodology, writing – original draft preparation, validation, rebuttal preparation.

Alderic Lecluse (AD): investigation, writing – review and editing.

Benoît Guillon (BG): investigation, writing – review and editing.

Godwin Tatah (GT): writing – original draft preparation, review.

Guillaume Marc (GM): writing – original draft preparation, review.

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