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Long-term successfully endovascular treatment of a complicated Takayasu's arteritis with thrombectomy and stenting: a case report

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Abstract

Background Takayasu arteritis is a rare form of large-vessel vasculitis primarily affecting the aorta and its major branches, with a higher prevalence in young women. This inflammatory disease can lead to significant complications, including ischemic stroke and transient ischemic attacks, though large-vessel occlusion stroke is an uncommon initial manifestation. Diagnosis relies heavily on vascular imaging, and treatment typically involves high-dose glucocorticoids, revascularization procedures, and close monitoring for restenosis, which occurs frequently after interventions.

Case presentation This case report describes a 38-year-old North African woman who presented with acute left-sided hemiparesis and visual disturbances due to large-vessel occlusion involving the common carotid artery and middle cerebral artery. Mechanical thrombectomy and stenting were performed successfully, resulting in complete recanalization without complications. The patient was later diagnosed with Takayasu arteritis, and this was managed with high-dose corticosteroids. During a 2-year follow-up, no complications such as restenosis or re-occlusion were observed, and the patient remained in good health.

Conclusion This case is notable for the successful use of dual mechanical thrombectomy and stenting in the acute management of stroke in Takayasu arteritis without short- or long-term complications, a rare outcome not commonly reported in the literature. It highlights the potential efficacy of this approach in carefully selected patients, suggesting that postoperative immunosuppressive therapy may reduce the incidence of restenosis.

Keywords Takayasu arteritis, Vasculitis, CTA, NIHSS, TIA, Stroke, Mechanical thrombectomy, Stenting, Case report

Background

Takayasu arteritis (TAK) is a large-vessel vasculitis (LVV) of unknown etiology, characterized by granulomatous inflammation that primarily affects the aorta and its major branches, particularly the subclavian, common carotid, and internal carotid arteries [1, 2].

Its incidence is variable, ranging from 2.6 per million in Europe and North America to 40 per million in Asia [3]. It is more common among young women, accounting for 80–90% of TAK cases, with age of onset usually ranging between 10 and 40 years [4], although it may also be seen in older patients.

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TAK is categorized into five types on the basis of the specific arteries affected, with clinical manifestations varying accordingly [2]. Types I, IIa, and IIb are classified depending on the involvement of the ascending or descending aorta, including the aortic arch and its branches. Types III, IV, and V are defined by the involvement of the abdominal aorta and the presence of additional features [5].

Its clinical manifestation includes constitutional symptoms, limb claudication, angina, hypertension, and absent peripheral pulses, and 10–20% of people with TAK exhibit neurological manifestations. Involvement of the common carotid and vertebral artery causes progressive neuro-ischemic symptoms such as visual disturbance, headache, vertigo, syncope, and transient ischemic attacks. However, acute stroke with large-vessel occlusion as a first manifestation of TAK is rare, although it may be observed in some cases [6–10]. Stroke and transient ischemic attacks (TIAs) are the major cause of morbidity. Cerebrovascular and cardiovascular events are the major cause of mortality in TAK.

Currently, there are no universally accepted diagnostic criteria for TAK. Rather, there are classification criteria, originally defined to classify patients who were already diagnosed with vasculitis, for including these cases in certain clinical trials. The historical American College of Rheumatology (ACR) classification criteria were defined in 1990 [11], but in 2022, on the behalf of the Diagnostic and Classification Criteria for Vasculitis (DCVAS) Study Group, a new set of classification criteria for TAK were published and validated [12]. The diagnosis of TAK is primarily based on clinical assessment but must be confirmed through vascular imaging, mostly magnetic resonance angiography (MRA) and computed tomography angiography (CTA).

In 2018 European Alliance of Associations for Rheumatology (EULAR) recommendations for the management of large-vessel vasculitis were updated with specific advice regarding TAK disease [13]. The cornerstone of treatment involves the use of high-dose glucocorticoids (GC), which are effective in inducing remission. Revascularization, through surgical procedures or endovascular interventions, such as balloon angioplasty, stenting, and stent graft replacement, may also be beneficial [14].

Mechanical thrombectomy may be considered for eligible patients with large-vessel occlusion stroke due to TAK, depending on the clot burden and the potential for medical futility. High restenosis rates following vascular interventions, typically between 37% and 62%, generally discourage prophylactic procedures. Patients who undergo urgent extracranial reconstruction with stenting should be closely monitored to ensure long-term vessel patency.

Corticosteroid dose and active disease at the time of intervention are important risk factors for restenosis or re-occlusion after revascularization procedure [4].

We report a case of a patient with acute stroke due to vasculitis causing occlusion of the common carotid artery (CCA) and middle cerebral artery (MCA), which was successfully treated with mechanical thrombectomy and endovascular stent placement.

In the literature, only a few cases of endovascular intervention using thrombectomy and stenting as a dual acute approach have been reported. To our knowledge, our case is the first in which no complications occurred during the subsequent 2-year follow-up.

Case presentation

A 38-year-old North African woman with no history of disease presented to our emergency room (12:29 a.m.) because of a sudden inability to move her left limbs and reflex incontinence.

She worked as an office clerk. Her medical history revealed full-term pregnancies, no miscarriages, a regular menstrual cycle, varied diet, no alcohol consumption, no smoking ever, constipation, and normal urinary and diuretic functions. She was not on any medication and had no known family health conditions.

Our patient reported that she had woken up at 4:00 a.m. with visual blurring, and at 11:30 a.m., she had developed conjugate gaze-head palsy and left hemiparesis.

On examination, she was afebrile and eupnoic. Her blood pressure was 80/50 mm Hg with 68 beats per minute (bpm) and a 100% O_2 saturation in ambient air. No abnormality was detected on chest and abdominal examination. Laboratory tests performed at admission are summarized in Table 1.

At 12:45 a.m., a neurological evaluation was performed. The patient was found to be awake, alert, and cooperative, but she presented with left oral deviation and left hemiparesis. Sensitivity was intact.

The initial National Institutes of Health Stroke Scale (NIHSS) score was 11; the Modified Rankin Scale (mRS) was 0.

A non-contrast brain computed tomography (NCCT) was performed, and the scan showed a hyperdensity in the right medium cerebral artery.

Subsequently, a neck CT angiography (CTA) was conducted that showed thrombosis of the right common carotid artery (CCA) extending up to the right middle cerebral artery (MCA; Fig. 1).

There was also a tight stenosis of the proximal section of the left CCA and an occlusion of the left subclavian artery. There was an evident predominance of the right vertebral artery.

Table 1 The laboratory tests performed at admission with their reference values

Analysis	Result	Unit measurements	Reference ranges
Leukocytes	10.06	10^9/L	4.30–11.00
Erythrocytes	4.48	10^{12}/L	4.00–5.50
Hemoglobin	11.8*	g/dL	12.0–16.5
Hematocrit	33.8*	%	38.0–48.0
MCV	75.4*	fL	77.0–102.0
RDW	13.2	%	12.0–15.0
MCH	26.3*	fL	27.0–32.0
MCHC	34.9	g/dL	32.0–36.0
Platelets	386	10^9/L	150–450
MPV	10.5	fL	9.6–12.9
INR	1.15	#	1.0–1.20
PT ratio	1.15	#	0.70–1.20
APTT ratio	1.02	#	0.80–1.20
CRP	48.4*	mg/L	0.0–5.0
Glucose	88	mg/dL	74–109
Urea	24	mg/dL	17–48
Creatinine	0.63	mg/dL	0.51–0.95
AST	14	U/L	11–34
ALT	15	U/L	8–41
Total bilirubin	0.39	mg/dL	0.10–1.20
Total alpha-amylase	73	U/L	31–107
CK	33	U/L	< 145
LDH	107*	U/L	125–220
Sodium	136	mmol/L	3.50–5.50
Potassium	4.03	mmol/L	8.8–10.2
Total proteins	5.3*	g/dL	6.6–8.7
Albumin	2.4*	g/dL	3.57–5.49
Alpha 1 globulins	0.45*	g/dL	0.19–0.41
Alpha 2 globulins	0.78	g/dL	0.45–0.98
Beta 1 globulins	0.38	g/dL	0.30–0.60
Beta 2 globulins	0.34	g/dL	0.20–0.54
Gamma globulins	0.94	g/dL	0.71–1.56
ANA	Positive	#	> 1:80

ALT, alanine aminotransferase; ANA, antinuclear antibodies; APTT, activated partial thromboplastin time; AST, aspartate aminotransferase; CK, creatine kinase; CRP, C-reactive protein; INR, international normalized ratio; LDH, lactate dehydrogenase; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; MPV, mean platelet volume; PT, prothrombin time; RDW, red cell distribution width;

* Value out of reference range

Our patient was intubated, and a thorax CTA was performed, which highlighted a significant concentric thickening of left CCA, conditioning reduction of the artery lumen until having a thread-like appearance.

Occlusion of the left subclavian artery was confirmed up to the axillary artery.

Wall thickening of the brachiocephalic artery, of the aortic arch, and of the descending thoracic aorta was also observed.

Additionally, there was significant wall contrast enhancement in the venous phase.

Takayasu arthritis was suspected, with a calculated score of 9 based on 2022 ACR/EULAR criteria [12].

Our patient was not eligible for intravenous thrombolysis with recombinant tissue plasminogen activator (r-tPA).

After a multidisciplinary assessment, an endovascular approach was chosen.

Under general anesthesia, a 6Fr introducer sheath (Radifocus introducer, Terumo) was positioned through femoral access. A preliminar digital subtraction angiography (DSA) run confirmed the occlusion of the left subclavian artery (SA), left vertebral artery (VA), right CCA, and right internal carotid artery (ICA); right arrhythmogenic cardiopyopathy (ACM); and left CCA stenosis with regular evidence of the downstream vessels.

Right VA and basilar artery (BA) were not occluded.

A mechanical thrombectomy was performed through guiding a catheter Envoy DA 6Fr × 95 cm (Cerenovus, J&J), with repeated aspiration attempts (three times) using the aspiration catheter Sofia 5 Fr × 125 cm (Microvention) until reaching the pre-ophthalmic segment of right ICA from right CCA, overcoming the occlusion with a J-shape guide-wire (Radifocus, Terumo).

A second mechanical thrombectomy was performed on the right MCA occlusion through a microcatheter Rebar 18 (ev3 neurovascular, USA) and a Synchro 0.014 microwire (Stryker Neurovascular, USA) using a stent-retriever Catch Maxi 5.5 × 40 mm (Balt), obtaining revascularization of the occluded tract (Fig. 2).

A following contrast run showed the persistence of stenosis of the right CCA, so, after an extensive consultation with the neurological team department, we proceeded to the deployment of a balloon-expandable stainless-steel vascular stent Genesis 7 × 29 mm (Cordis) over a guiding catheter Destination 7 × 90 cm (Terumo) while intravenously infusing 500 mg of acetylsalicylic acid (ASA).

Complete recanalization was achieved with grade 3 thrombolysis in cerebral infarction score (TICI) in 150 minutes from puncture (Fig. 3).

At the end of the procedure, a cone beam computed tomography (CT) showed no hemorrhagic complications.

A CT scan 12 hours later showed no hemorrhages, so a double-antiplatelet therapy (DAPT) with ASA 250 mg and clopidogrel 75 mg was started.

After the procedure, the patient was transferred to the neurosurgical intensive care unit of our hospital still sedated and intubated. High-dose steroid therapy was initiated with methylprednisolone 40 mg/day.

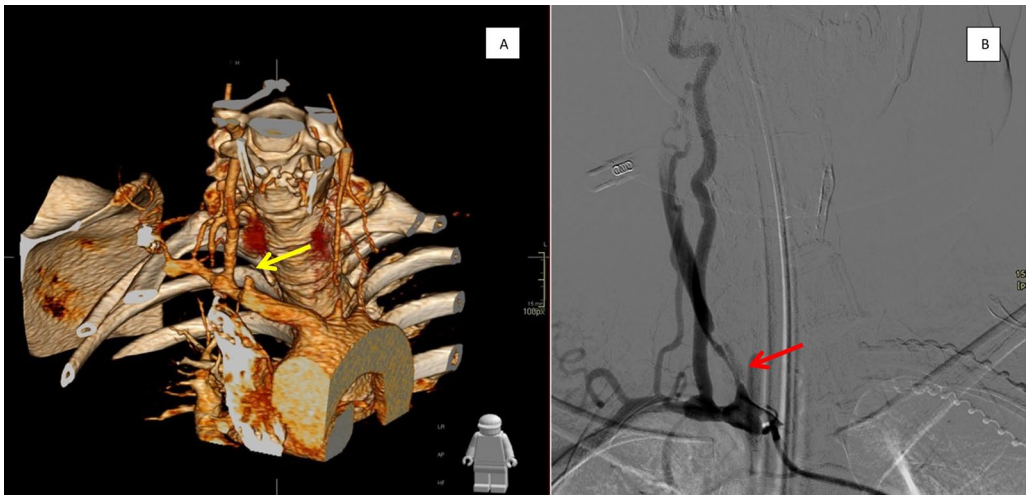

Fig. 1 **A** A three-dimensional volume rendering (3D-VR) showing occlusion of the right common carotid artery (CCA; yellow arrow). **B** A digital subtraction angiography (DSA) highlights and confirms the occlusion with minimal residual lumen (red arrow)

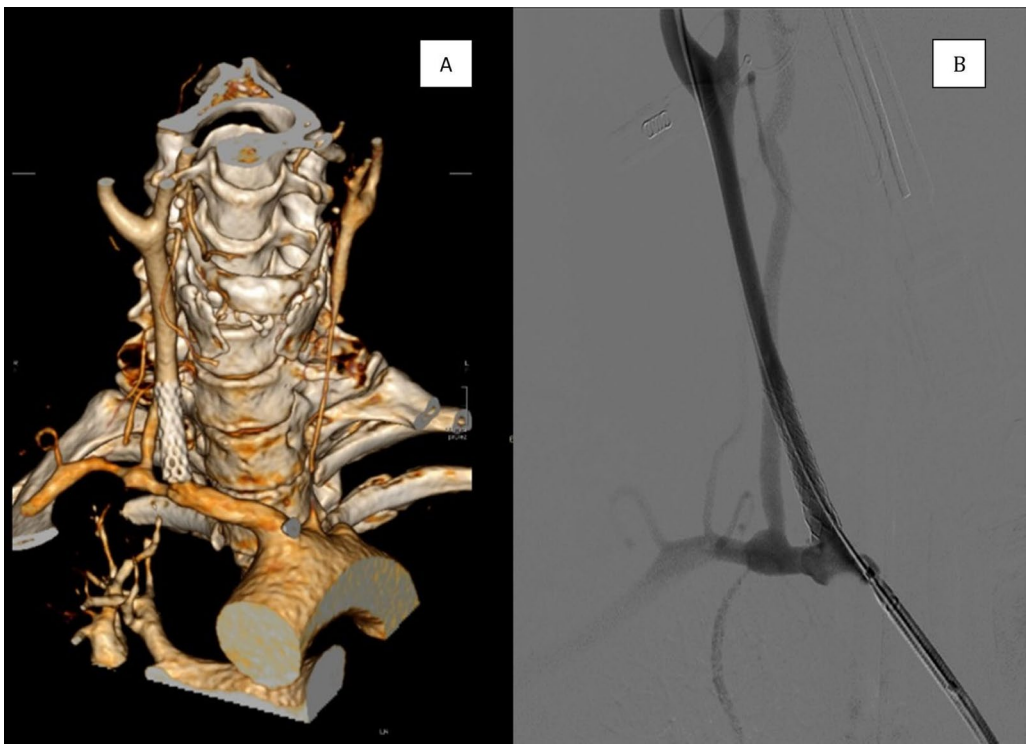

Fig. 2 **A** The 3D-VR and **B** the DSA showing a complete recanalization of the right common carotid artery after the vascular stent deployment

The next day she was extubated, and during the recovery, the patient was conscious and cooperative, following simple orders with slight improving weakness of the left hemisome.

A day after, her CT scan showed a little hypodensity in the right caudate–lenticular nucleus.

A few days after the endovascular procedure, the steroid therapy was adjusted by the rheumatologist to prednisolone 1 mg/kg (60 mg), dividing the dose as two-thirds at 8:00 a.m. and one-third at 3:00 p.m., with gastroprotection and blood pressure monitoring.

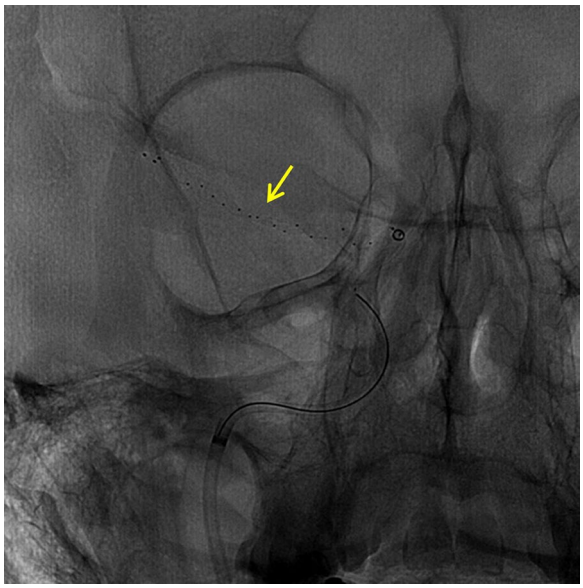

Fig. 3 A DSA showing the stent-retriever (yellow arrow) in the right MCA

Following the procedure, no complications were observed. The patient was monitored with regular check-ups every 6 months at the rheumatology department of our center.

The steroid therapy was gradually tapered over 6 months, reaching a dose of prednisolone 7.5 mg, divided into 5 mg at 8:00 a.m. and 2.5 mg at 3:00 p.m., a regimen the patient is still currently following.

The 2-year follow-up after the procedure revealed no complications or new neurological symptoms. At the time of writing this case report, the patient was in excellent overall health.

Discussion and conclusions

TAK is an inflammatory vascular disorder that involves large vessels, predominantly the aorta and its main branches. The entire course of TAK involves two overlapping phases. Phase I is a systemic and asymptomatic phase. Phase II refers to vascular inflammation and/or sequelae such as stenosis.

Current studies indicate that TAK pathology involves all arterial layers with varying degrees of inflammatory infiltrate. Acute, chronic, and granulomatous inflammation predominantly affects the media and adventitia, while hyperplasia and neovascularization are observed in the intimal layer. During active inflammation, the process begins in the adventitia, progresses to the intima, and can result in stenosis, occlusion, dilation, or aneurysm formation [15].

Intervention is frequently difficult due to the complexity of the lesions and the high rates of restenosis. About 20% of patients do not respond to any form of medical treatment. This often necessitates endovascular or surgical interventions, which are typically recommended during periods of quiescent TAK [16].

In fact, an actively inflamed arterial wall often responds poorly to endovascular intervention, leading to early restenosis or other complications. Therefore, interventions should ideally be conducted only after achieving stable control of inflammation, except in emergency situations such as severe ischemia or neurological issues such as aneurysm, dissection, or stroke [15].

The incidence of ischemic stroke in patients with TAK is reported to be 10–20% in Europe and the USA. In Japan, Watanabe *et al.* [17] reported an incidence of 13.2%, with embolism observed in 3.7% of these patients. The pathogenesis of cerebral infarction in this disease is thought to involve: blood flow failure due to vascular stenosis, embolism originating from thrombus related to vascular-wall irregularities or turbulence, and vascular occlusion associated with media dissection [18].

Regarding acute-phase mechanical thrombectomy and stenting placement for major cerebral artery occlusion related to TAK, only a few case reports have been published [6, 18–23].

Our patient with TAK had obvious indications for emergency treatment due to her severe neurological deficits related to occlusion of the CCA and MCA. After careful consideration, two consequent mechanical thrombectomies were performed, obtaining revascularization of the occluded tract; then, given the persistence of the right CCA stenosis, a balloon-expandable stainless-steel vascular stent was deployed.

We followed the patient with a 2-year follow-up, during which no complications, such as restenosis, thrombosis, or stroke—usually reported in the literature—were observed.

To our knowledge, this is the first case of treating stroke in Takayasu arteritis with thrombectomy and stenting that has not reported complications in the short or long term.

Often, technical failures followed by dissection of the vessels are reported as main complications in literature after endovascular treatment. Other complications include cerebral hyperperfusion syndrome, stent graft thrombosis, pseudoaneurysm, puncture site bleeding, stroke, restenosis, and renal failure. A 10-year retrospective study reported a complication rate of 8.2% in an endovascular intervention group [15].

Generally, when comparing the endovascular and the surgical approaches, the rates of early complications (within 30 days) are similar in terms of restenosis,

thrombosis, and stroke, according to Saadoun [24]. However, in terms of late complications (> 30 days), endovascular procedures have slightly higher rates of restenosis (64.5% compared with 46.1%), while surgical procedures have higher rates of thrombosis (15.4% versus 3.2%) [16].

In conclusion, unlike what the literature suggests, our case shows that a combined acute approach with thrombectomy and stenting may be a valid and effective treatment option in patients with ischemic complication due to TAK.

Postoperative immunosuppressive therapy, as in our case, may help reduce inflammation and therefore the incidence of restenosis, as suggested also in previous studies [18, 25].

Abbreviations

TAK	Takayasu arteritis
LVV	Large-vessel vasculitis
TIA	Transient ischemic attack
ACR	American College of Rheumatology
DCVAS	Diagnostic and Classification Criteria for Vasculitis
MRA	Magnetic resonance angiography
CTA	Computed tomography angiography
EULAR	European Alliance of Associations for Rheumatology
GC	Glucocorticoids
CCA	Common carotid artery
MCA	Middle cerebral artery
NIHSS	National Institutes of Health Stroke Scale
mRS	Modified Rankin Scale
NCCT	Non-contrast brain computed tomography
r-tPA	Recombinant tissue plasminogen activator
DSA	Digital subtraction angiography
SA	Subclavian artery
VA	Vertebral artery
ICA	Internal carotid artery
BA	Basilar artery
ASA	Acetylsalicylic acid
TICI scale	Thrombolysis in cerebral infarction
DAPT	Double-antiplatelet therapy

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Author contributions
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Availability of data and materials
Data and materials are contained within the article.

Declarations

Ethics approval and consent to participate
The procedure performed in studies involving human participants was in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards. Consent to participate was obtained from all subjects involved in the study.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare no conflict of interest.

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