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CASE REPORT

Ischemic stroke as initial manifestation of Takayasu arteritis: A case series

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ABSTRACT

Takayasu arteritis (TAK) is a rare, chronic inflammatory disease involving large blood vessels, particularly the aorta and its branches. This case report is noteworthy due to its unusual presentation with ischemic stroke in a young adult, highlighting the importance of considering this diagnosis in similar clinical scenarios to improve patient outcomes. In Case 1, a 38-year-old male with right-sided hemiparesis and a history of leg pain underwent imaging, revealing ischemic stroke, subsequently confirmed as TAK. Case 2 involved an 18-year-old female with recurrent strokes, cramps, and leg pain, leading to the diagnosis of TAK. Both patients received corticosteroids, antiplatelets, and other therapies, resulting in symptomatic relief. This case series emphasized the need for healthcare professionals to consider TAK in young stroke patients for timely diagnosis and multidisciplinary management. The absence of specific treatment guidelines highlights the necessity for larger prospective studies to guide effective treatment strategies in TAK patients with stroke.

Keywords: Diagnosis, ischemic stroke, stroke, Takayasu arteritis.

Takayasu arteritis (TAK), also referred to as "pulseless disease," "thromboartropathy," and "Martorell syndrome," is a chronic inflammatory condition characterized by granulomatous vasculitis involving medium- and large-sized arteries, particularly the aorta and its branches. This leads to thickening of blood vessel walls, fibrosis, stenosis, and thrombosis. The clinical presentation of TAK varies widely based on the organs affected by ischemia.[1,2] Over 50% of patients with TAK may exhibit neurological symptoms such as headaches, vision disturbances, seizures, transient ischemic attacks (TIAs), cerebral infarctions, intracerebral hemorrhage, and even orthostatic syncopal episodes.[2]

A meta-analysis revealed that 15.8% of TAK patients experienced ischemic stroke or TIA due to vasculitis or embolism, serving as an early indication of the disease.[3] Although cerebral ischemia is not a common occurrence, this complication is associated with a significant morbidity and mortality rate.[3] As a result, appropriate and precise management is crucial to reduce complications, disabilities, and mortality. This study presented two cases diagnosed with TAK that initially presented with symptoms of ischemic stroke. Through this case series, we aimed to contribute to the understanding of this disease, ultimately improving patient outcomes.

CASE REPORT

Case 1– A 38-year-old male was admitted to the Emergency Department of Dr. Cipto Mangunkusumo Hospital (RSCM) with sudden-onset right-sided body weakness 12 h before admission. The right arm and leg could not be lifted or moved. The patient also had trouble speaking or articulating words and had trouble

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understanding commands or words. There was no history of previous stroke, hypertension, diabetes mellitus, heart disorders, or vascular abnormalities in the patient's medical history. According to the family, the patient frequently complained of pain in both legs, particularly when walking or standing for a long time, and the discomfort improved with rest. In the family history, it was noted that the patient's father had a history of stroke. The patient had a 10-year history of smoking one pack of cigarettes per day. Written informed consent was obtained from the patient.

On physical examination, the patient was alert and oriented, with a blood pressure of 180/100 mmHg in both the right and left arms (measured at the brachial artery). The blood pressure in both the right and left legs was 170/90 mmHg, heart rate was 68 beats per minute and regular, respiratory rate was 18 breaths per minute, body temperature was 36.8°C, and oxygen saturation was 99%. No temporal, ophthalmic, carotid, or bilateral vertebral bruits were detected in the general examination. Heart and lung examinations were normal. Acral extremities were warm to touch, no edema was detected, and the dorsalis pedis artery was weakly palpable in both legs. Neurological examination revealed a Glasgow coma scale (GCS) score of E4M6V aphasia, impression of central right-sided 7th and 12th cranial nerve paresis, accompanied by right hemiparesis. Fundoscopy showed bilateral hypertensive retinopathy. The National Institute of Health Stroke Scale (NIHSS) score upon initial admission was 18. Blood laboratory tests and chest X-ray results were normal. Electrocardiogram showed sinus bradycardia at 56 beats per minute and T-wave inversion in leads I, aVL, V5, and V6, with an impression of left ventricular hypertrophy. Noncontrast brain computed tomography (CT) revealed multiple infarcts in the right hemisphere and basal ganglia, as well as infarcts in the left frontal and parietal lobes, and left internal capsule, accompanied by sulcus and gyrus narrowing on the left side with a positive dense middle cerebral artery (MCA) sign, indicating extensive ischemia in the territory of the left MCA.

Based on the examination results, the patient was diagnosed with aphasia, central right-sided 7th and 12th cranial nerve paresis, right hemiparesis due to ischemic stroke in the MCA territory on the first day, accompanied by anterior ischemia on the heart, and a history of sinus bradycardia. The patient was managed with 30° head elevation, 500 mL of saline infusion every 12 h, 250 mL of mannitol followed by four doses of 125 mL intravenous drip, oral acetylsalicylic acid (ASA) 320 mg once daily followed by 80 mg once daily, oral simvastatin 20 mg once daily, oral folic acid 5 mg twice daily, and oral vitamin B6 and B12 administered twice daily.

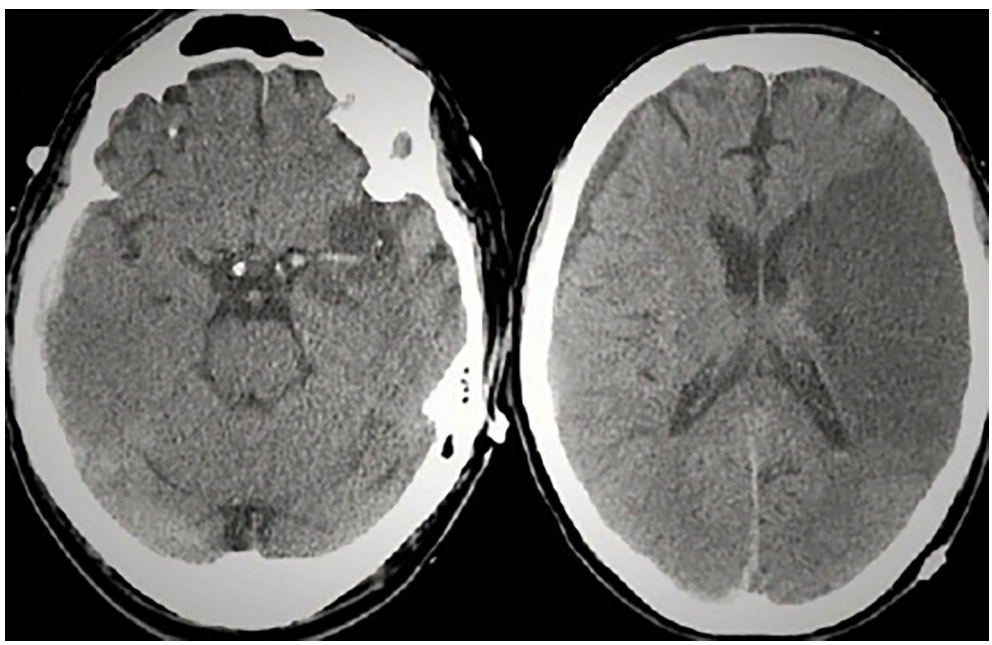

Figure 1. Results of the brain computed tomography.

The patient was admitted to the stroke unit for further diagnostic examination and management. An echocardiogram was performed with normal results, showing no thrombus or emboli. Carotid Doppler (CD) examination revealed a total occlusion of the left internal carotid artery (Figure 2a), with high peripheral resistance in the left common carotid artery (Figure 2b), and the presence of a nonstenotic hard plaque in the right carotid bulb and distal right common carotid artery. The patient was scheduled for digital subtraction angiography (DSA).

During treatment, the patient's clinical condition improved, with stable hemodynamics, no decrease in consciousness, and no new neurological deficits. Aphasia symptoms showed improvement, and the patient began to speak one to two words. In the DSA examination, total occlusion was observed in the left internal carotid artery, severe stenosis in the vertebrobasilar artery, and panarteritis of cerebral blood vessels (Figure 3). Further diagnostic examinations included blood tests for immunological markers and arteriography for the entire vascular system. Immunological test results showed positive antinuclear antibodies (ANA), with a titer of 1/320, negative antineutrophil cytoplasmic antibodies, and negative anticentromere antibodies immunoglobulin (Ig) M and IgG. Double-stranded DNA was within normal limits (6.3), accompanied by an elevated erythrocyte sedimentation rate (ESR) of 53 mm/h and C-reactive protein (CRP) level of 25.2. The patient's diagnosis was confirmed as ischemic stroke secondary to TAK. Subsequently, the patient underwent additional diagnostic examinations to identify abnormalities in other large arteries. The patient received additional therapy from the Allergy Immunology Division, including intravenous methylprednisolone 62.5 mg once daily, oral calcium and vitamin D supplements three times daily, and oral folic acid 400 µg once daily.

The patient underwent renal ultrasonography, which revealed bilateral small renal kidneys. Additionally, the patient underwent coronary angiography, aortography, and arteriography. Coronary angiography showed 30% stenosis in the mid-distal and 50% stenosis in the D1 branch of the left anterior descending artery, 70-80% stenosis in the mid-distal left circumflex artery, and 40% stenosis distally and 80% stenosis proximally in the right coronary artery. Aortography revealed 50% stenosis in the ostial right external iliac artery, 60-70% stenosis in the left internal iliac artery, and 80% stenosis in the mid-left internal iliac artery. Left extremity arteriography showed 30-40% stenosis in the superficial femoral artery, 60-70% stenosis in the proximal posterior tibial artery (PTA), and 60% stenosis in the mid PTA. On the right extremity arteriography, 40-50% stenosis was found in the superficial femoral artery to the popliteal artery, 60% stenosis in the ostial anterior tibial artery, 70% stenosis in the proximal PTA, and 70% stenosis in the mid PTA. The conclusion from these examinations was that the patient had two-vessel coronary artery disease and peripheral artery disease in both lower limbs.

By the fourth week of treatment, the patient's medical condition stabilized, showing notable progress in aphasia, and the patient

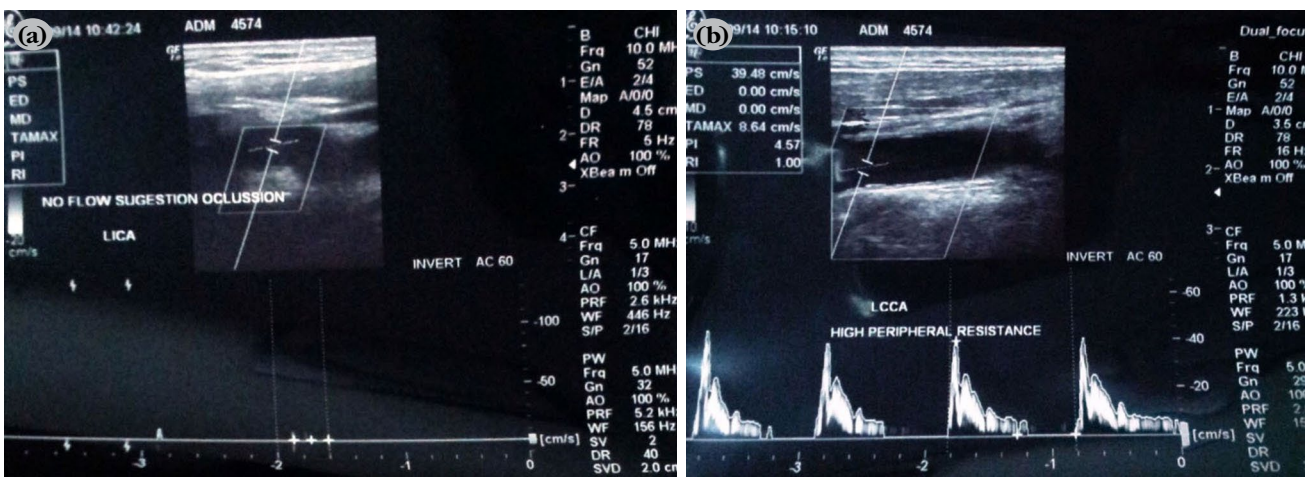

Figure 2. Results of the carotid Doppler examination. **(a)** Total occlusion of the left internal carotid artery, and **(b)** high peripheral resistance in the left common carotid artery.

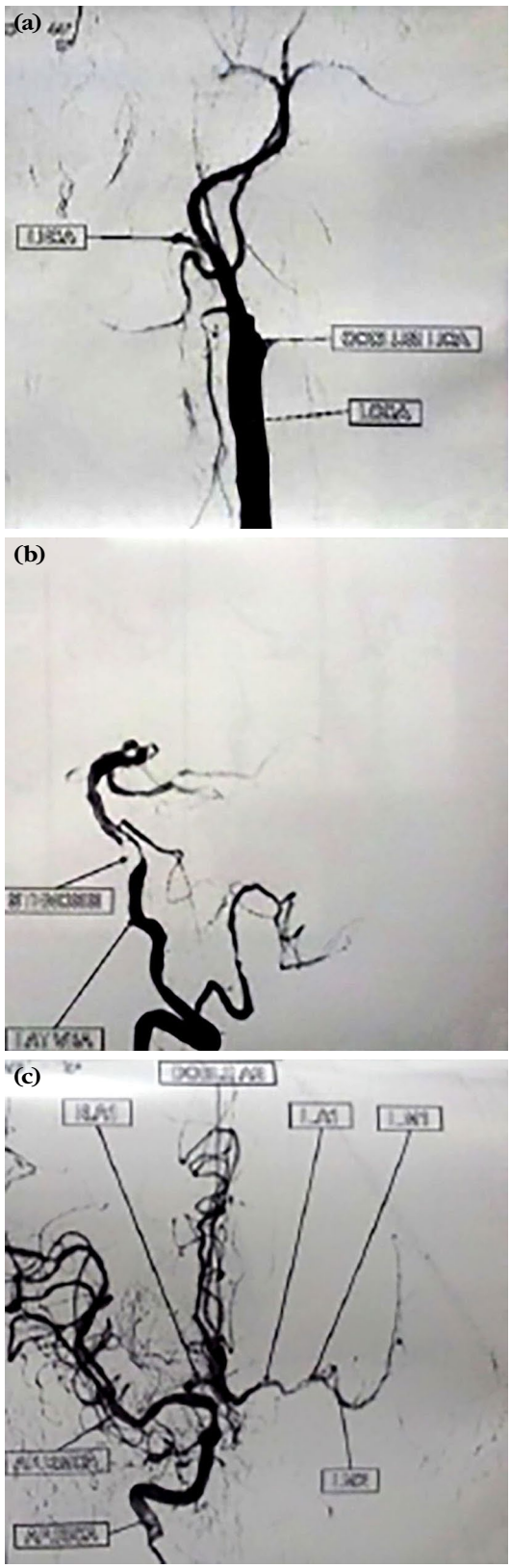

Figure 3. Results from digital subtraction angiography. **(a)** Total occlusion in the left internal carotid artery, **(b)** severe stenosis in the vertebrobasilar artery, and **(c)** panarteritis of cerebral blood vessels.

was able to articulate sentences. The dose of methylprednisolone was reduced and converted to the oral form for outpatient preparation. The morning cortisol level was 5.6 µg/dL (reference range: 6.2-19.4 µg/dL). Therefore, the oral methylprednisolone dose and regimen was adjusted to 16 mg in the morning, 16 mg in the afternoon, and 8 mg in the evening for the next seven days. The patient was discharged with an improved level of consciousness, indicated by a GCS score of E4M6Vaphasia. His functional status at discharge was assessed with a modified Rankin scale (mRS) score of 4. The discharge therapy included oral ASA 80 mg once daily, oral simvastatin 20 mg once daily, oral methylprednisolone (16 mg, 16 mg, and 8 mg), oral valsartan 80 mg once daily, oral amlodipine 10 mg once daily, oral folic acid 400 µg once daily, oral vitamin B6 and B12 twice daily, and oral calcium and vitamin D supplement three times daily.

Case 2– An 18-year-old female was admitted to the Neuroscience Clinic at the RSCM Kencana with a complaint of sudden-onset left-sided body weakness, which had been present for one month prior to admission. Three years earlier, the patient experienced her first stroke-like episode, characterized by weakness on the right side of her body, slurred speech, and an inability to speak, which lasted for 2 h. The patient gradually recovered and could speak clearly again. The patient was taken to the first hospital, where a noncontrast brain CT revealed a thrombotic stroke. Subsequently, the patient was hospitalized for five days and discharged at her own request.

One month later after the first episode, the patient experienced a seizure. The pre-ictal symptoms were unknown; however, during the seizure, her head turned to the left, mouth deviated to the left, and the right arm and leg appeared stiff. The seizure lasted for 3 min and occurred twice a day. After the seizure, the patient regained consciousness but had difficulty speaking, which improved within 2 to 3 h. Additionally, the weakness in the right arm and leg became more pronounced. The patient was then referred to the second hospital, where a noncontrast brain CT revealed a recurrent stroke. The patient was hospitalized for nine days, and after treatment, the symptoms improved, leaving residual weakness on the right side of the body. After treatment, the patient complained of frequent cramps and pain in both legs when walking or standing for a long time.

One month before admission to RSCM, the patient complained of left-sided weakness, difficulty holding objects, and dragging when walking. The inability to speak reoccurred but improved within 3 h. The patient was then referred to RSCM Kencana, with a NIHSS score of 10, where she underwent a nine-day treatment, diagnosed with TAK, and discharged with hemiparesis sequela. Written informed consent was obtained from the patient.

At the Neuroscience Clinic of the RSCM Kencana, the patient was alert and oriented, had a blood pressure of 160/100 mmHg in the right and left arm, pulse rate of 100 beats per minute, respiratory rate of 16 breaths per minute, and a body temperature of 36°C. The physical examination revealed a normal general status, and the neurological examination showed a GCS score of E4M6V5 and duplex hemiparesis. The motor strength, assessed from distal to proximal, was 4/4 in the right limbs, and 4+/4+ in the left limbs. Physiological reflexes were normal; no pathological reflexes were elicited. Fundoscopy of both eyes revealed normal findings in the right and left eyes. Laboratory tests showed anemia, with a hemoglobin level of 10.1 g/dL, and an elevated ESR of 22 mm/h. Kidney function, liver function, blood sugar, cholesterol, and hemostasis were within normal limits. Immunological tests revealed a decrease in protein S, with a result of 47.0, and the ANA test showed a positive result with a granular or speckled nucleoplasm pattern. The patient presented the results of a noncontrast brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) from the second hospital (Figure 4) on the second day of stroke treatment at the Neuroscience Clinic. The findings indicated subacute infarction in most of the left hemisphere, with minimal involvement in the right frontoparietal region. The MRA showed poor visualization of the left carotid artery and left MCA, while the right carotid and basilar arteries were patent.

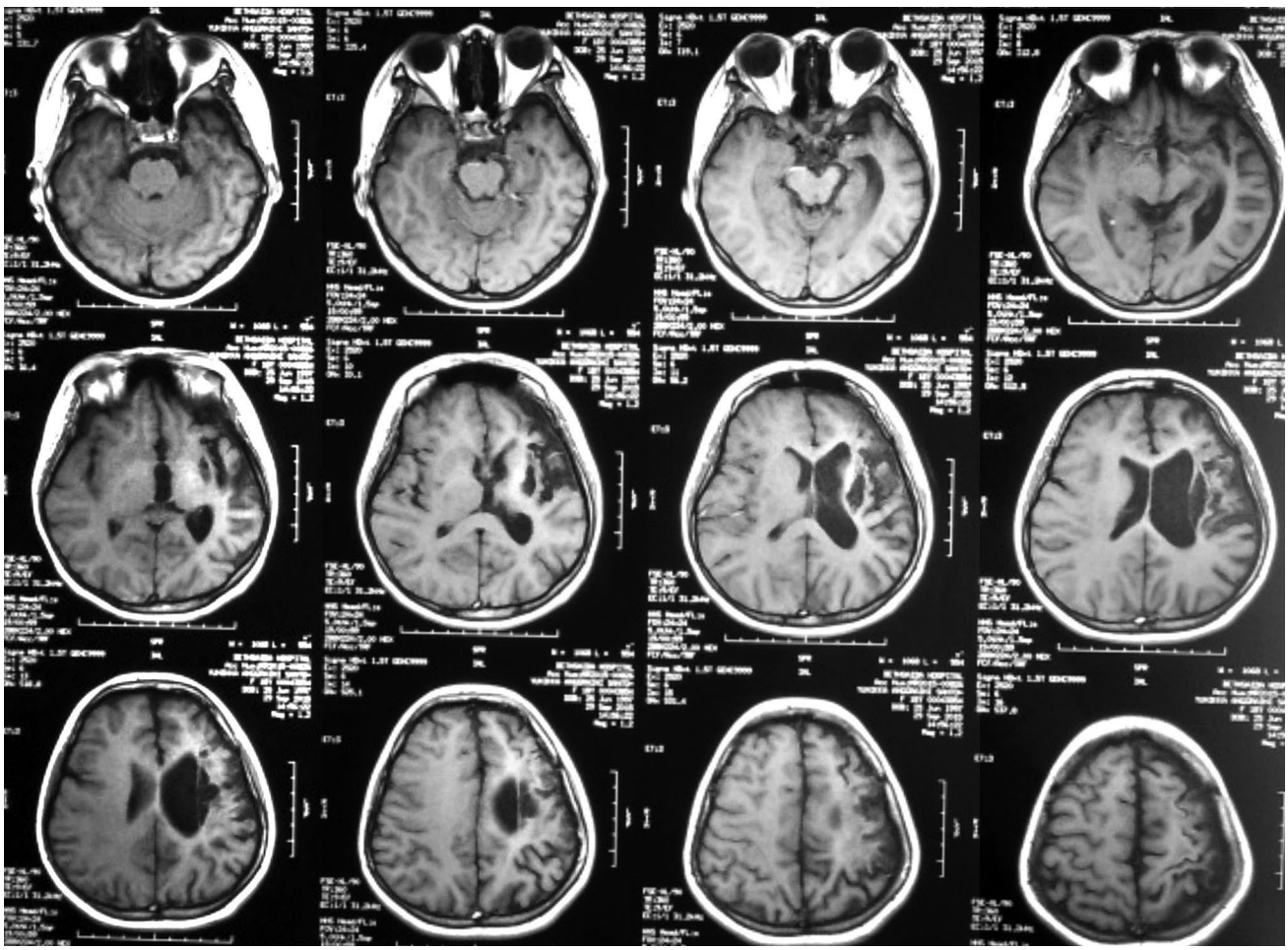

Figure 4. Axial T1-weighted brain magnetic resonance imaging.

The patient underwent an echocardiogram, revealing an ejection fraction of 70%, normal valves, and good systolic and diastolic functions of the left and right ventricles. The CD examination suggested high-grade stenosis or near occlusion at bilateral common carotid arteries with suspected arteritis. The transcranial Doppler (TCD) examination showed a normal TCD of accessible intracranial arteries with increased flow at posterior circulation. Subsequently, a multislice CT angiography was performed (Figure 5), revealing severe stenosis to near occlusion (90-100% stenosis) in bilateral common carotid arteries from the origin to the distal pre-bifurcation, suspected arteritis, and hypoplasia of the right vertebral artery. A repeat noncontrast MRI brain examination showed infarction in the right basal ganglia and encephalomalacia in the corona radiata and left frontoparietal lobe, with signs of cerebral atrophy. The brain MRA revealed hypoplasia of the right A1 segment of the anterior cerebral artery. An electroencephalogram was conducted to evaluate seizures. The results showed an alpha wave background with a frequency of 10-11 Hz and medium amplitude, responding to opening and closing of the eyes. Beta waves were observed with a frequency of 18-24 Hz and low amplitude, and photic driving was identified during photic stimulation. Finally, a CT angiography of the lower extremities was performed (Figure 6), revealing good caliber of the right and left common femoral arteries. There was evidence of narrowing in the right superficial femoral artery with approximately 42% stenosis of the popliteal artery, posterior tibial artery, peroneal artery, and dorsalis pedis artery.

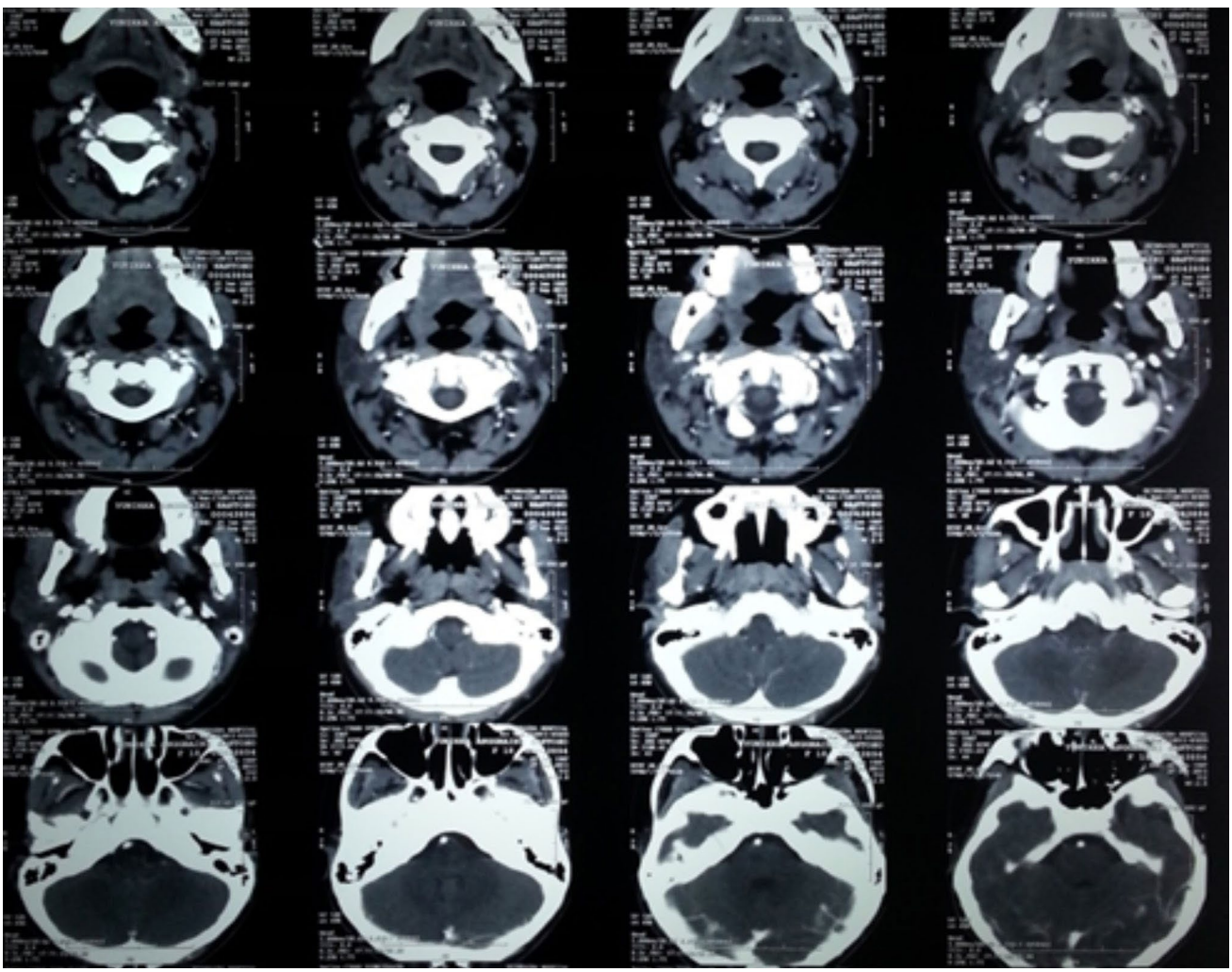

Figure 5. Multislice computed tomography angiography results.

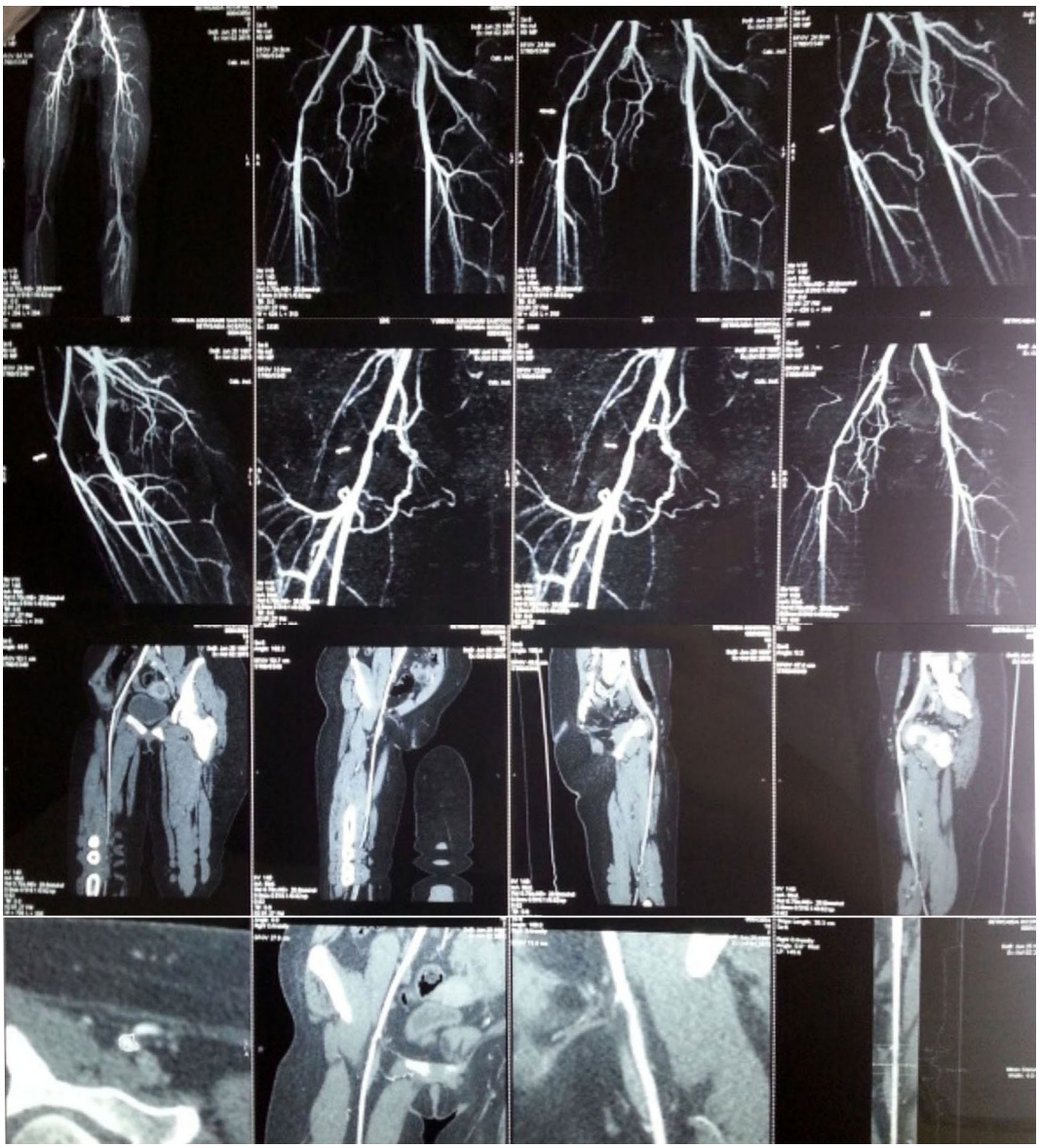

Figure 6. Lower extremity computed tomography angiography.

Based on the medical history, physical examination, and diagnostic tests conducted, the patient was diagnosed with a history of improving aphasia and hemiparesis, consistent with recurrent ischemic stroke in TAK. Patients' functional status remained stable, with an mRS score of 4, before and after treatment. At the most recent follow-up, the patient was receiving therapy consisting of oral ASA 80 mg once daily, simvastatin 20 mg once daily, oral methylprednisolone (16 mg, 16 mg, and 8 mg), oral valsartan 80 mg once daily, oral amlodipine 10 mg once daily, oral folic acid 5 mg twice daily, and oral vitamin B6 and B12 twice daily.

DISCUSSION

Takayasu arteritis is an autoimmune disorder with an unknown cause, characterized by persistent inflammation in the large vessels, particularly affecting the aorta and its major branches.[4,5] It primarily affects females (80-90%), typically manifesting between the ages of 10 and 40.[6] The symptoms vary depending on the location and extent of vessel inflammation, and in severe cases, patients may not have a detectable pulse, leading to the condition being referred to as "pulseless disease."[7] The disease progresses through two phases. Phase I is

systemic and asymptomatic disease, while Phase II involves vascular inflammation.[8] Involvement of the carotid and vertebral arteries can result in reduced cerebral blood flow, leading to symptoms such as strokes, dizziness, syncope, headaches, and seizures. Although the incidence of stroke in TAK is generally 10 to 20%, it is unusual for stroke to be the initial presentation in young patients.[9] Nevertheless, stroke is a significant and severe complication in TAK, with various contributing mechanisms, including large vessel occlusion or stenosis due to arterial thrombosis and vasculitis, or embolism from aortic regurgitation.[10,11] Therefore, it is crucial to consider TAK when evaluating young adults with symptoms suggestive of stroke.

The first diagnostic criteria for TAK were proposed by Ishikawa[12] in 1988, requiring an age of less than 40 years at diagnosis or the onset of characteristic symptoms or signs as a mandatory criterion. In 1990, the American College of Rheumatology (ACR) introduced classification criteria for TAK.[13] Later, in 1995, Sharma et al.[14] developed the modified Ishikawa criteria, which improved sensitivity and specificity by removing the age requirement (<40 years) as an obligatory diagnostic criterion. Based on these criteria, the first patient met four out of six ACR classification criteria and fulfilled one major plus four minor criteria from the modified Ishikawa criteria. Similarly, the second patient satisfied three out of six ACR classification criteria and met one major plus four minor criteria from the modified Ishikawa criteria.

In 2022, the ACR/European Alliance of Associations for Rheumatology (EULAR) introduced updated classification criteria for TAK, comprising two absolute requirements and 10 additional clinical and imaging criteria. These new criteria demonstrated high diagnostic accuracy, with a sensitivity of 92.0% and specificity of 93.2% in Asia, and a sensitivity of 90.5% and specificity of 94.4% in Europe and North America.[15] Both patients in this case series met the 2022 ACR/EULAR classification criteria for TAK.

For early diagnosis, noninvasive imaging techniques should be the first-line approach. Traditionally, the diagnosis of TAK has relied on angiography, which remains the gold standard for assessing vascular inflammation patterns and classifying TAK phenotypes.[16,17] Recent research showed that the most common patterns of vascular involvement in stroke patients with TAK were type 4 (61.5%) and type 1 (30.8%).[8,18] In contrast, a Brazilian cohort study reported a higher prevalence of type 1 over type 4.[19] Type 4 involves generalized inflammation across all aortic segments, whereas type 1 is restricted to the arch branches.[17]

In addition to CT angiography, MRA, and Doppler ultrasonography, fluorodeoxyglucose positron-emission tomography-CT (FDG PET-CT) is another noninvasive imaging modality that serves as a valuable diagnostic tool.[20] It is particularly useful in the early, pre-pulseless phase of TAK, when angiography may not yet reveal significant vascular abnormalities.[21] Due to its excellent sensitivity and specificity, clinicians widely use FDG PET-CT as an effective imaging technique for diagnosing large-vessel vasculitis, including TAK.[22]

Laboratory tests lack specificity and do not play a significant role in the diagnosis of TAK. Common findings include anemia, occasional increases in white blood cell and platelet counts, and elevated levels of ESR and CRP. In some cases, organ-specific autoimmune diseases and autoantibodies may be present in patients with TAK.[23] Stojanovic et al.[24] found that 13 out of 24 patient with TAK in the cohort had positive antibodies, with ANA being the most prevalent, although no significant difference was observed compared to the healthy group (p=0.12). On the other hand, a study by Choe et al.[25] revealed that contrast-enhanced MRI findings were consistent with laboratory results in most patients with TAK. Hence, laboratory tests could be useful in illustrating the patient's condition, particularly in settings with limited health facilities, assisting clinicians in determining the next steps in care. Furthermore, the ACR and the Vasculitis Foundation strongly recommend ongoing long-term clinical monitoring for patients with TAK who appear to be in clinical remission, rather than discontinuing monitoring altogether.[26] According to the cases presented, our patients exhibited elevated ESR and positive ANA before undergoing additional imaging examinations.

Given that TAK is responsive to corticosteroids, individuals with TAK experiencing stroke during active disease are typically treated with corticosteroids and subsequently prescribed disease-modifying antirheumatic drugs as an immunosuppressant.[27-29] This approach is considered effective, given the associated side effects and the frequent progression or relapse observed with corticosteroid monotherapy.[30] In a study by Pektezel et al.,[31] patients with

TAK and neurological involvement did not show impaired cerebral blood flow regulation compared to healthy individuals. This could be due to the protective effects of their immunosuppressive treatment or other factors. However, there is limited studies on the use of immunosuppressive treatments in patients with TAK with stroke. A study by Ahn et al.[32] did not find a statistically significant benefit of immunosuppressant use in reducing the risk of stroke in patients with TAK.[33] Nevertheless, the study faced limitations due to a small number of participants, and further research is essential to determine the effectiveness of immunosuppressants in preventing stroke in patients with TAK. Additionally, other medications that were proven useful in TAK include statins, which, when used concurrently with immunosuppressants, were found to reduce relapse rates. Antiplatelet medications are also associated with a lower frequency of ischemic events in TAK.[30] Both of our patients received corticosteroids and antiplatelets as part of their maintenance treatment.

In conclusion, TAK presents a broad range of early and late manifestations that are often overlooked by healthcare professionals. This case series emphasized the importance of considering TAK in young adult patients experiencing stroke, given the associated high morbidity and mortality rates. Managing patients with TAK requires a multidisciplinary approach due to the unpredictable nature of the disease. Currently, there is no specific guideline for the treatment of TAK with stroke. Larger prospective studies in the future will be essential to determine effective treatment strategies for patients with TAK with stroke.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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