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Original Article

Takayasu Arteritis with Neurological Manifestations: Clinical and Neurovascular Imaging Characteristics

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Abstract

Background: Takayasu arteritis (TA) is a large vessel vasculitis that may be accompanied by neurological involvement in 57%-80% of the patients. The aim of this work is to describe the clinical and neurovascular imaging abnormalities in TA patients with neurological manifestations.

Methods: Thirty-nine TA patients with neurological manifestations and available brain magnetic resonance imaging (MRI), MR angiography (MRA), and MR venography of the intracranial and cervical vessels were studied. Patients with thrombophilia or hyperlipidemia were excluded.

Results: The mean age at TA diagnosis was 30.56 ± 8.74 years, and 33 (84.62%) of the patients were women. The most frequent neurological manifestations included headaches (64.1%), syncope (12.8%), and visual loss (10.26%). According to the angiographic classification, 37 (94.9%) of the patients had involvement of branches from the aortic arch (Type I), and only 2 (5.1%) had diffuse aortic involvement (Type V). Steno-occlusive lesions of the medium-sized intracranial arteries were observed in 11 (28.21%) of the patients. Features suggestive of small vessel disease were found among 16 (41%); 13 had abnormal MRI findings and 3 other patients had normal MRI and increased collateral circulation on MRA without associated intracranial arterial stenosis. Opening of the collateral circulation was seen in 14 (35.9%) patients. Venous involvement (affecting the cerebral venous sinuses and jugular veins) was found in 12 (30.77%) patients.

Conclusion: Patients with TA and neurological manifestations exhibit a spectrum of extracranial and intracranial vascular abnormalities including large, medium, and small blood vessels, as well as the cerebral venous sinuses and jugular veins.

Keywords: Takayasu arteritis, large vessel vasculitis, neurological manifestations, neurovascular imaging

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Introduction

Takayasu arteritis (TA) is a large vessel chronic granulomatous vasculitis that mainly affects the aorta and its major branches. The pulmonary and coronary arteries are also frequently involved. Thickening of the arterial wall, together with stenoses, occlusions, and less commonly aneurysms, can be seen on vascular imaging. Vascular damage leads to end-organ ischemia with serious sequelae including stroke, limb claudication, pulmonary hypertension, aortic valve insufficiency, myocardial infarction, and mesenteric ischemia.[1,2]

Neurologic manifestations may affect 57 up to 80% of the patients with TA.[3,4] Neurologic symptoms are usually subtle and examination may be normal, thus requiring a high index of suspicion. These are usually in the form of headache, fatigue, dizziness or lightheadedness, syncope, visual loss, and subclavian steal syndrome (shifting of blood from the vertebral artery to the upper limb due to proximal stenosis of the subclavian artery). The well-known cerebral complications of TA have included transient ischemic attacks (TIAs), stroke, intracranial hemorrhage, and seizures.[5-7] Aneurysms leading to cerebral hemorrhage have been reported. Neurologic manifestations in TA can be explained by extracranial and/or intracranial vascular involvement.[8,9] Most patients with central nervous system ischemia have stenoses of the extracranial arteries of the neck, such as the common carotid, vertebral, and subclavian arteries.[3-6] Intracranial arterial involvement in TA could be due to intracranial stenoses and uncommonly aneurysms. Intracranial stenoses in TA could be secondary to inflammatory vasculitis or the result of prior embolization; the presence of multifocal arterial stenoses suggests an inflammatory vasculitis etiology.[5] A literature review identified 300 patients with TA and available description of the intracranial vessels, and of those, 74 (24.7%) demonstrated intracranial vascular involvement.[7] The literature on the spectrum of neurovascular involvement in TA is sparse. The aim of this study is to describe the clinical and neurovascular imaging abnormalities in TA patients with neurological manifestations.

Materials and Methods

This is a single-center, cross-sectional, descriptive study including consecutive TA patients suffering from neurological manifestations. The patients were seen at the Rheumatology and Immunology unit of the Rheumatology and Rehabilitation department, Kasr Al Ainy hospital, Cairo University between 2015 and 2023. The protocol for this study was approved by the I Kasr Al Ainy Faculty of Medicine Ethics Committee (Approval no: 430, Date: 2023), and informed consent was obtained from the patients who agreed to take part in the study.. Patients were diagnosed as TA according to the American College of Rheumatology (ACR) 1990 criteria for the classification of TA,[10] and their records were reviewed for the 2022 ACR/EULAR classification criteria for TA.[2] All patients were subjected to brain magnetic resonance imaging (MRI), intracranial, and cervical vascular imaging.

The exclusion criteria were as follows:

age older than 40 years at the start of the TA manifestations;

thrombophilia including the antiphospholipid syndrome or the presence of antiphospholipid antibodies (lupus anticoagulant, anticardiolipin IgG or IgM, or B2 glycoprotein I antibodies); deficiency of protein C, protein S, antithrombin III, or factor V Leiden;

Behçet's disease and other autoimmune rheumatological diseases;

hyperlipidemia;

atherosclerosis evidenced by the presence of atheromatous plaques on imaging; and

reversible cerebral vasoconstriction syndrome.

Main Points

- Takayasu arteritis patients with neurologic involvement most commonly present with headache, syncope, and visual loss.
- The medium-sized intracranial arteries and small blood vessels are frequently involved.
- Although infrequently reported in previous research, the cerebral venous sinuses and jugular veins may be involved in a considerable number of patients.

The following demographic data were collected for every patient: sex, age at the disease onset, and the delay in diagnosis (calculated as the time lapse between the onset of symptoms and the diagnosis of TA). The duration of the disease was calculated from the date of onset of symptoms till the date of clinical assessment.The neurologic symptoms included headache, vertigo, lightheadedness, syncope, hemiplegia, hemisensory loss, stroke, TIAs, visual loss, epilepsy, cognitive disorders, extrapyramidal manifestations, respiratory distress, and psychosis (panic attacks, depression, self-injurious behavior, eating disorders, and suicide attempts).Information on other clinical manifestations including fever, limb claudication, limb sensory affection, blue extremities, arthritis, arthralgia, myalgia, mesenteric ischemia, angina, hypertension, and diabetes mellitus was recorded.Results of erythrocyte sedimentation rate and C-reactive protein were recorded when available.The National Institutes of Health criteria[3] and the Indian Takayasu Clinical Activity Score 2010 (ITAS2010)[11] were applied to identify patients with active disease. Patients who fulfilled 1 or both criteria were considered to have active disease.The renal arteries were studied by Doppler ultrasonography in all patients.Brain MRIs were studied for the evidence of infarcts, hemorrhages or white matter disease. Intracranial and cervical vascular imaging modalities included magnetic resonance angiography (MRA) and magnetic resonance venography for all patients. Computed tomography angiography for the aorta and central nervous system was available for only 12 (30.77%) patients. The images were evaluated for the presence of occlusion, stenosis, and aneurysm. Arterial involvement was classified according to the angiographic classification of TA proposed by Moriwaki et al.[12]Statistical analysis: Descriptive statistics were used. The frequency of demographic, clinical characteristics, and imaging abnormalities was expressed as number and percentage. For continuous variables, the mean ± SD and the range (minimum to maximum) were used.Results

Thirty-nine TA patients with neurological complaints were studied. Their mean age at TA diagnosis was 30.56 ± 8.74 years, and 33 (84.62%) were women. Neurological symptoms were present in 11 (28.21%) patients at the time of diagnosis and developed in 28 (71.79%) patients during follow-up.

All patients had normal renal functions and none of them had diabetes or hyperlipidemia.

Three female patients had hypertension; they had affected subclavian and brachial arteries and normal Doppler studies of the renal arteries. One male patient had hypotension with permanent narrowing of the subclavian and brachial arteries. Patient demographic data and clinical manifestations are summarized in Table 1.

According to the angiographic classification, 37 (94.9%) of the patients had involvement of branches from the aortic arch (Type I) and only 2 (5.1%) had diffuse aortic involvement (Type V).

The cervical arteries and/or subclavian arteries were involved in all patients while the medium sized intracranial arteries were affected in 11/39 (28.2%). Features suggestive of small vessel disease were found among 16 (41%) of the studied patients. Among those, 13 had abnormal MRI findings and 3 other patients had normal MRI and an increase in the collateral circulation on MRA without associated intracranial stenosis. Opening of the collateral circulation was seen in 14 (35.9%) patients.

Venous involvement was found in 12 (30.77%) patients. The intracranial and cervical vascular imaging abnormalities of all patients are shown in Table 2.

We had 2 patients with pediatric TA. They were 1 male and 1 female aged 11 and 12 years, respectively. The clinical and imaging characteristics of some of the studied patients are shown in Figures 1, 2, and 3.

Discussion

In the present study, the authors describe the clinical manifestations and the spectrum of intracranial and cervical vascular involvement in TA patients complaining from neurological symptoms. Takayasu arteritis is predominantly a disease of young adults in the second and third decades of life. The onset of illness may

Table 1. Demographic Data, Neurological and General Manifestations of Takayasu Arteritis Patients

Demographic Data	No. of Patients = 39 (%)
Sex	
Female	33 (84.62)
Male	6 (15.39)
Age at TA diagnosis mean ± SD (range, years)	30.56 ± 8.74 (12-46)
Delay in diagnosis mean ± SD (range, years)	3.37 ± 3.94 (0.5-18)
Duration mean ± SD (range, years)	3.67 ± 4.03 (1-20)
Neurological symptoms (%)	39 (100)
Headache	25 (64.1)
Vertigo	1 (2.56)
Lightheadedness	1 (2.56)
Syncope	5 (12.8)
Hemiplegia	1 (2.56)
Hemisensory loss	0
Paresthesia	3 (7.92)
Stroke	1 (2.56)
Transient ischemic attacks	3 (7.92)
Visual loss	4 (10.26)
Extrapyramidal manifestations	2 (5.13)
Epilepsy	1 (2.56)
Respiratory distress	1 (2.56)
Neuropsychiatric manifestations (%)	8 (20.5)
Cognitive dysfunction	2 (5.13)
Panic attacks	2 (5.13)
Depression	2 (5.13)
Self-injurious behavior	1 (2.56)
Eating disorder	2 (5.13)
Suicide attempts	2 (5.13)
Other symptoms (%)	
Fever	6 (15.38)
Arthritis, arthralgia	28 (71.80)
Myalgia	23 (58.97)
Limb claudication	15 (38.46)
Limb sensory affection	16 (42.11)
Blue extremities	2 (5.13)
Hypertension	3
Hypotension	1
Elevated ESR and/or CRP (n = 16)	12 (75)
Active disease	35 (89.7)

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; TA, Takayasu arteritis.

Table 2. Intracranial and Cervical Vascular Imaging of 39 Takayasu Arteritis Patients

	Number (%)	
Intracranial MR imaging findings		
Ischemic foci, white matter lesions	10 (25.6)	
Cerebellar atrophy	2 (5.13)	
Increased intracranial tension	1 (2.56)	
Intracranial vascular findings		
Open collaterals	14 (35.89)	
	Occlusion	Stenosis
Right anterior cerebral artery	3 (7.70)	1 (2.56)
Left anterior cerebral artery	1 (2.6)	0
Right middle cerebral artery	0	2 (5.13)
Left middle cerebral artery	0	0
Right posterior cerebral artery	0	2 (5.13)
Left posterior cerebral artery	0	1 (2.56)
Left posterior communicating artery	1 (2.56)	0
Venous sinuses		
Cavernous sinus	1 (2.56)	0
Transverse sinus	4 (10.26)	5 (12.782)
Sigmoid sinus	2 (5.13)	3 (7.7)
Internal jugular vein	0	3 (7.7)
Cervical vascular findings		
Right common carotid artery	0	5 (12.82)
Left common carotid artery	0	4 (10.3)
Right internal carotid artery	1 (2.56)	5 (12.82)
Left internal carotid artery	0	1 (2.56)
Right vertebral artery	1 (2.56)	7 (17.9)

be earlier in childhood[3,13] but rarely in infancy.[14] Two of the studied TA patients had their disease onset during childhood. Some differences may exist between children and adults with TA. Pediatric patients may have a longer delay in diagnosis compared to adults with fever, musculoskeletal complaints, arthritis and biological features of inflammation being more

Left vertebral artery	0	5 (12.8)
Basilar artery	0	1 (2.56)
Other large vessels involved		
Abdominal aorta	0	2 (5.13)
Right subclavian	0	8 (20.5)
Left subclavian	0	3 (7.7)
Both common iliac arteries	0	1 (2.56)

common.[3] Also, TA in children is more severe and associated with higher mortality.[15] Studies comparing pediatric and adult-onset TA are limited. It would be interesting to study the clinical presentation, imaging characteristics and outcome in pediatric and adult-onset TA groups prospectively, to verify if actual differences exist between both groups.

The most frequent neurological manifestations in the studied patients included headaches (64.1%), syncope (12.8%), and visual loss (10.26%), while stroke was less common affecting 2.56%. Other investigators also reported that among patients with neurological manifestations, headache was the most common symptom affecting 32-81 %.[5-7,16] Syncope was reported with a frequency of 6.3-14%[5,14] and visual disturbances/loss in 12.7-50%,[4,5,7,16] while stroke was more frequently reported in other studies affecting 12.7-60%.[4-7,16] The Diagnostic and Classification Criteria in Vasculitis (DCVAS) study, a large, worldwide, multicenter study included 630 TA patients; 38(6%) were found to have cerebrovascular events TIA or strokes. The lower frequency of strokes (3.5%) was explained by the demographic reasons due to the large contribution from Asian countries.[17]

Various neuropsychiatric symptoms were reported by 20.5% of the authors' patients, and all of them had clinically active disease. Other authors reported clinically significant neuropsychiatric manifestations in a few TA patients including anxiety,[18] depression[9,19], and cognitive decline.[20] Limited studies have investigated the mental health in TA patients as compared to healthy controls;[21,22] their pooled results revealed significantly higher depression scores among TA than healthy controls while anxiety levels did not reach statistical significance.[23] The association between disease activity and higher anxiety and depression scores is controversial[21,24] implying that the development of neuropsychiatric symptoms could not be entirely explained by disease activity. In this regard, the possible contribution of corticosteroids should be considered. Corticosteroids can cause a wide range of neuropsychiatric manifestations including euphoria, irritability, anxiety, depression, mania, and cognitive changes. These usually occur at doses ≥40 mg/day with most cases occurring during the first week of treatment.[25] An important limitation of the present work is the absence of information on treatment to control disease activity and its effect on neurological complaints. The spectrum of neuropsychiatric manifestations in TA in relation to neurovascular imaging abnormalities, disease activity and response to treatment warrants to be studied.

The neurological manifestations of TA have been thought to result mainly from decreased blood flow caused by steno-occlusive lesions involving the carotid and vertebral arteries and/or shifting of the blood flow (steal). Other factors of neurological manifestations in TA are hypertension and thromboembolism leading to stroke.[4] While all patients in the present study had involvement of the cervical and/or subclavian arteries, the intracranial arteries were involved in 11 (28.2%) of the patients. Involvement of the medium-sized intracranial arteries, previously thought to be uncommon in TA, was described by several investigators. Evidence of intracranial stenosis or occlusion was reported among 13-41% of TA patients with available neurovascular imaging[5,7, 9,16] and 4.5% had an intracranial aneurysm.[5]

Small vessel involvement in TA: Although TA has been classified as a large vessel vasculitis, microvascular disease has been described in several organs such as the small retinal vessels,[26] myocardium (presenting as cytotoxic T-cell-mediated myocarditis)[27] and the skin in the form of cutaneous necrotizing vasculitis.[28] In the brain, small vessel disease has been described as T2 hyperintense white matter lesions on MRI affecting 34.7% of the patients in 1 study.[5] Kim et al[20] reported the case of a 39-year-old male patient with TA complaining from severe headache and fever as a part of the initial presentation. Brain MRI showed multiple T2 high signal intensities and T1 contrast-enhanced lesions involving the brain parenchyma without stenotic or occlusive lesions in the intracranial and extracranial arteries, suggesting direct CNS involvement in TA, possibly due to small vessel disease. In the present study, the presence of small vessel disease was considered in 16 (41%) of the patients; 13 had ischemic foci or white matter lesions on MRI, and 3 other patients had normal brain MRI with opening of the collateral circulation on MRA. None of those 16 patients had intracranial arterial stenosis on MRA.

The collateral blood flow may be relatively well developed in patients with TA as shown by positron emission tomography in patients with established stroke.[29] Another study reported that in the majority of their patients who had normal brain MRI or CT scans and no evidence of intracranial stenosis on MRA, transcranial Doppler studies revealed elevated, non-circumscribed anterior circulation velocities, most likely presenting Willisian collateralization.[6] The results of the present study come in line with those observations as nearly 36% of the authors' patients (with or without intracranial vascular involvement) had open collateral circulation.

Venous involvement in TA: Venous involvement affecting the cerebral venous sinuses and the internal jugular veins was found in 30.77% of the patients. None of the patients had antiphospholipid antibodies or deficiency

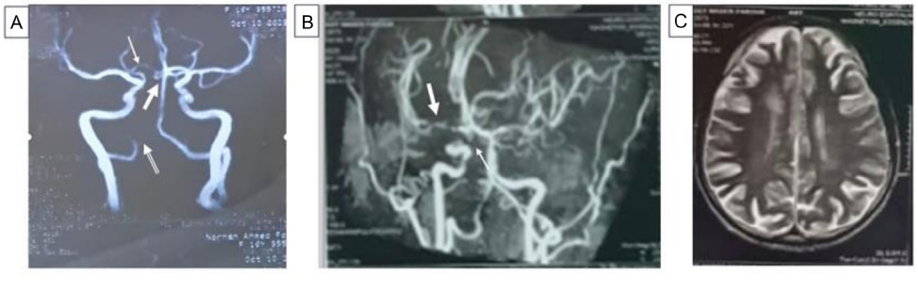

Figure 1 . (A) Female patient, 21 years old, complaining from headache, vertigo, depression, TIAs, self-injuries, and suicidal attacks. MRA; occluded right anterior cerebral artery (thin arrow), attenuated right posterior cerebral artery (thick arrow), and right vertebral artery (compound arrow). (B) Female patient, 38 years old. Neurologic manifestations included headache, paresthesia and TIAs. MRA; occluded right internal carotid artery (thin arrow), beaded right middle cerebral artery (thick arrow) with abundant collaterals. (C) Male patient, 11 years old, complaining from numbness, headache, depression, and loss of consciousness. MRI; white matter lesions, cerebral vasculitis, small vessel disease. MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; TIA, transient ischemic attack.

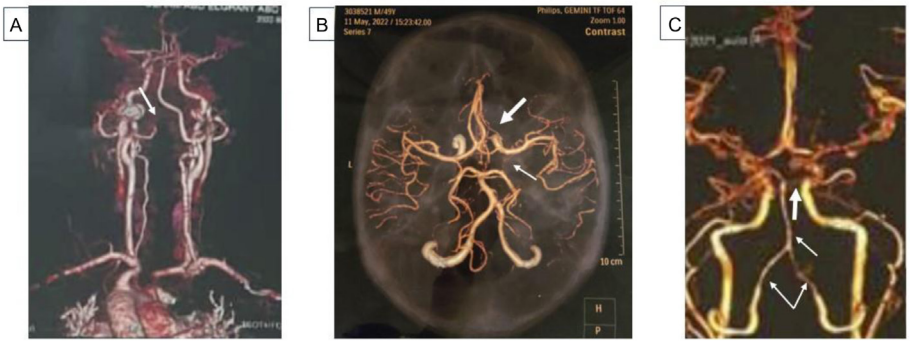

Figure 2 . (A) Female patient, 42 years old, suffering from rapidly progressive extra-pyramidal manifestations over 2 months. MRA; attenuated left vertebral artery (arrow). (B) Male patient, 43 years old. Neurologic manifestations: numbness, affected cognitive functions, headache, lightheadedness, recent onset extrapyramidal manifestation, loss of morning erection. CTA; occluded left posterior communicating artery (supplying red nucleus in brain), thin arrow. Attenuated right anterior cerebral artery (thick arrow). (C) Male patient, 22 years old. Neurologic manifestations: severe headache of 6 years duration, TIAs, loss of consciousness and progressive respiratory distress over 2 months with fatal outcome. CTA; attenuated both vertebral arteries, basilar artery (thin arrow), and right posterior cerebral artery (thick arrow) with open collaterals. CTA, computed tomography angiography; MRA, magnetic resonance angiography; TIA, transient ischemic attack.

of protein C, protein S, antithrombin III, or factor V Leiden. There is marked paucity of literature regarding venous involvement in TA and only a few reports describe such an occurrence. The notable ones detail the involvement of superior vena cava,[30] retinal vein occlusion,[31] pulmonary veins[32], and segmental narrowing of the internal jugular veins.[19] A few case reports have described the involvement of the cerebral venous sinuses[18,19,33] and involvement of the medium-to-small cortical veins with a beaded-string appearance.[19] All of those patients suffered from severe headaches; other manifestations included papilledema, anxiety, or depression.[18,19,33] It has been suggested that the involvement of the venous system in TA can either be the result of inflammatory changes or an abnormality in platelet aggregation and the coagulation system.[19]

In conclusion, patients with TA and neurological symptoms exhibit a spectrum of extracranial and intracranial vascular abnormalities including large, medium, and small blood vessels, as well as the cerebral venous sinuses and jugular veins. Due to the under-recognized significant involvement of large and medium-sized veins among TA patients, it would be useful, for a comprehensive assessment of neurological complaints, to specifically assess the veins by the recommended imaging modalities in addition to MRI and MRA.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of University of Kasr Al Ainy Faculty of Medicine (Approval no: 430, Date: 2023).

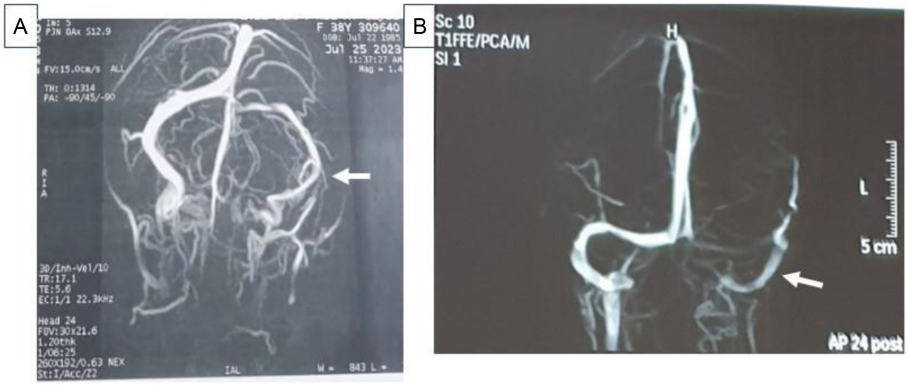

Figure 3 . (A) Female patient, 38 years old, complaining from persistent headache. MRV, attenuated transverse and sigmoid sinus (arrow). (B) Female patient, 43 years old, complaining from severe persistent headache. MRV, markedly attenuated left transverse and sigmoid sinus (arrow). MRV, magnetic resonance venography.

Informed Consent: Informed consent was obtained from the patients who agreed to take part in the study (or their parents in pediatric cases).

Peer-review: Externally peer reviewed.

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Declaration of Interests: The authors have no conflict of interest to declare.

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