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Novel Insights from Research Practice

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Coincidence of Takayasu Arteritis and Multiple Sclerosis: Narrative Review and Case Report

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Established Facts

- Takayasu arteritis (TA) and multiple sclerosis (MS) are both immune-mediated diseases with overlapping immune mechanisms, including T-cell dysregulation and cytokine release.
- Three cases of TA and MS have been described in literature; all of them were treated with steroids.
- Hypertensive retinopathy can be the first presentation of TA and be mistaken for optic neuritis.
- Interferon-beta therapy in MS has been described once as a potential trigger of TA.

Novel Insights

- This review contains the first case report where TA occurred in an MS patient and was not treated with steroids.
- The vessel wall inflammation of TA remitted after discontinuation of interferon beta.
- The case contributes to the limited number of documented instances of TA and MS co-occurrence and highlights the importance of differentiating between hypertensive retinopathy and optic neuritis as misdiagnosis can lead to inappropriate treatments.

Keywords
Hypertensive retinopathy · Multiple sclerosis · Optic neuritis · Interferon · Takayasu arteritis

Abstract
Introduction: Takayasu arteritis (TA) and multiple sclerosis (MS) are both immune-mediated diseases that can coexist, with TA causing vascular inflammation and MS involving demyelination driven by aberrant T-cell activity. The overlap of these conditions highlights shared immune mechanisms, such as T-cell dysregulation and cytokine

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release, underscoring the need for a nuanced understanding of their interplay, which is explored in a narrative review of reported cases. ***Case Presentation:*** We narrate all reported cases of TA in patients with MS and report the case of a 57-year-old woman with MS with suspected bilateral optic neuritis and typical contrast-medium enhancement in both optic nerves. Because of normal visual acuity on both eyes, malignant hypertension, and fundoscopic findings indicative of hypertensive retinopathy, we diagnosed hypertensive retinopathy with secondary contrast-medium enhancement of the optic nerves. We established antihypertensive medication and searched for secondary causes of hypertension and highly elevated erythrocyte sedimentation rate, which led to the finding of large vessel wall inflammation and the diagnosis of TA. ***Conclusion:*** TA can present with a variety of ocular symptoms, including hypertensive retinopathy, retinal ischemia, and anterior ischemic optic neuropathy, often mimicking other diseases. While rare, the coexistence of TA and MS, including cases associated with interferon-beta therapy, suggests shared immune mechanisms and underscores the need for careful monitoring of patients with MS receiving immunomodulatory treatments. The broad spectrum of potential causes for optic nerve abnormalities necessitates a thorough evaluation to avoid misdiagnosis and ensure appropriate treatment.

Plain Language Summary

Takayasu arteritis (TA) and multiple sclerosis (MS) are both diseases caused by the immune system attacking the body. TA leads to inflammation of large blood vessels, while MS causes damage to the protective coating of nerve cells. Although rare, these two conditions can occur together, possibly due to shared immune system issues. We reviewed all reported cases of TA in people with MS and described a case of a 57-year-old woman with MS who was thought to have optic neuritis, a condition affecting the optic nerves. However, further testing revealed that her symptoms were caused by hypertensive retinopathy (eye damage from high blood pressure) due to TA. Treating her high blood pressure and investigating further uncovered inflammation in her large blood vessels, leading to the diagnosis of TA. This case highlights the importance of carefully evaluating symptoms as TA can mimic many other conditions. It also emphasizes the need to monitor MS patients closely, especially those on immunomodulatory treatments, as these may be linked to the development of TA.

Introduction

Takayasu arteritis (TA) is an autoimmune inflammatory disease affecting the vessel wall of the aorta and its closest branches mainly in young women. It can lead to arterial stenosis, occlusion, dilation, aneurysms, ischemia, claudication, or hypertension. Occult hypertension with hypertensive retinopathy can be the first manifestation of TA [1–4].

TA involves vascular inflammation, granuloma formation, and fibrosis, ultimately leading to stenosis, occlusions, and aneurysms in large- and medium-sized arteries due to abnormal vascular dendritic cells and overexpression of Toll-like receptors, leading to cytokine release and T-cell infiltration into the arterial wall [5]. The development of multiple sclerosis (MS) involves a complex interplay of genetic, environmental, and immune factors with the involvement of CD4+ T-helper cells, genetic predisposition, and a strong association with the HLA-DRB1*15:01 allele [6].

The annual incidence of optic neuritis is 5 per 100,000 [7]. While optic neuritis only refers to inflammation of the optic nerve, it is commonly used as a synonym for idiopathic demyelinating optic neuritis, which is associated with MS. Yet 10% of patients initially diagnosed with idiopathic demyelinating optic neuritis are finally diagnosed differently. The most common differentials are tumors, hypertensive retinopathy, retinochoroidal disorders, and neuromyelitis optica [8]. Causes can be hereditary, infectious, autoimmune (also vasculitis such as giant cell arteritis), (para-)neoplastic, vascular, toxic, or traumatic [9]. It is important to distinguish the cause correctly because treatment with glucocorticoids might aggravate other conditions such as hypertensive retinopathy and the correct diagnosis affects long-term treatment.

Case Report

A 57-year-old woman was referred to our department of neurology from the ophthalmologists with suspected optic neuritis.

Admission and History

The patient complained of itching in both eyes for 10 days, which reminded her of allergic rhino-conjunctivitis and presented without redness, tenderness, or pain on bulbar movement. The time of onset coincided with the habitual onset of allergic symptoms in March. On inquiry, she specified a slight deterioration of visual acuity

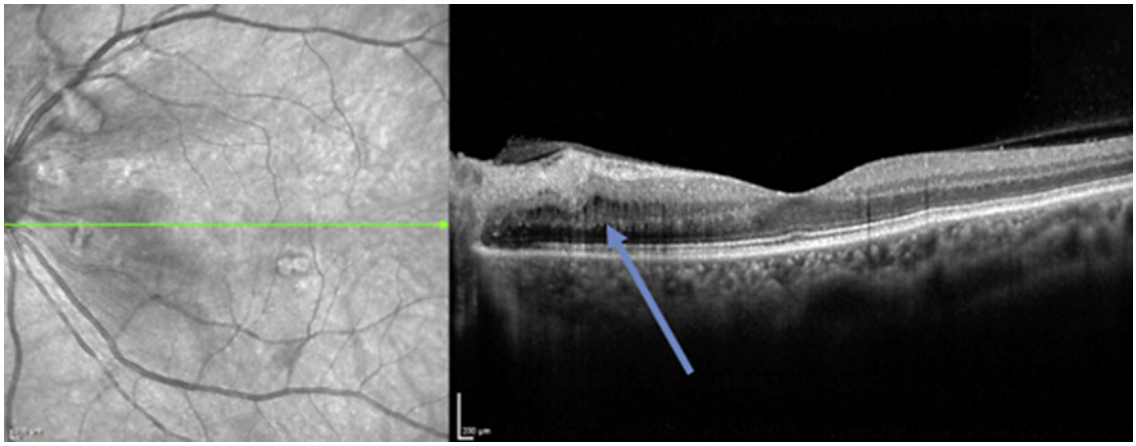

Fig. 1. Optical coherence tomography showing exudate (blue arrow).

for 1.5 months with a slow onset. Three months ago, she had new glasses made and it was only several weeks later that her sight subjectively diminished.

The patient is otherwise known for De Quervain's thyroiditis, migraine with visual aura, alcohol abuse 5 years ago after the sudden death of her husband with consecutive paracetamol intoxication in suicidal intention with temporary liver failure. She suffered from MS since the 4th year of her life with about 20 disease episodes (one optic neuritis on the left side, all other episodes with paresthesia). The diagnosis of MS was based on magnetic resonance imaging (MRI) findings typical of demyelination, positive oligoclonal bands (OCBs) in cerebrospinal fluid, and laboratory exclusion of other autoimmune diseases. She had a stable course with interferon (IFN) for 14 years. The patient was an active smoker and had stopped drinking alcohol. Family anamnesis was negative for MS. Systemic anamnesis showed no abnormalities, especially no B symptoms.

Examinations upon Admission

The patient initially presented with a hypertension of 242/124 mm Hg, a tetrahyperreflexia, increased muscle tone in the left ankle joint, a hypoesthesia for all qualities in the right anterior leg, positive Babinski sign on both sides without paresis. Ophthalmological examination showed a visual acuity of 100% (for distance vision) on both eyes, normal intraocular pressure, bilateral papillitis with vasculitis, focal bleedings, cotton wool spots, and peripapillary exudates (Fig. 1). MRI showed bilateral prechiasmatic enhancements of both optic nerves (right more than left), typical for demyelinating optic neuritis in MS and static periventricular and juxtacortical MS lesions over the course of 14 years (Fig. 2).

Course of the Disease

The patient declined a lumbar puncture to exclude infectious causes of bilateral optic neuritis decidedly. Differentials such as neuromyelitis optica, myelin oligodendrocyte glycoprotein-associated disease, and ANA-associated collagenosis were serologically excluded.

We found thrombocytopenia (97 g/L) and anemia (119 g/L) that had been preexisting 14 months ago in the general practitioner's records, but both cell lines had further decreased. An elevated lactate dehydrogenase (LDH) and D-dimers, a diminished haptoglobin, and C3 complement and positive antibodies against red blood cells indicated hemolysis. We found neither splenomegaly nor signs of thrombotic thrombocytopenic purpura due to slow progression and normal ADAMTS-13 activity.

We established antihypertensive treatment with amlodipine 5 mg, whereupon the patient showed average systolic blood pressures of 160 mm Hg. Renal artery stenosis, a tumor of the suprarenal gland, or elevated aldosterone were not found while cortisol and methoxytyramine were slightly elevated.

We could discharge the patient after less than 48 h in our ward with an improved hypertension. Because of the bicytopenia, we stopped IFN treatment (because hemolytic cytopenia is described as a side effect) after 14 years of remission under the treatment with reevaluation after some weeks.

The general practitioner called us 2 weeks later wishing further diagnostic measures due to an erythrocyte sedimentation rate (ESR) of 70 mm/h and proteinuria. He had started with metoprolol because of continuing hypertension. Otherwise, thrombocytes had normalized, which confirmed our hypothesis of an IFN-induced thrombocytopenia "ex juvantibus." We also saw decreased

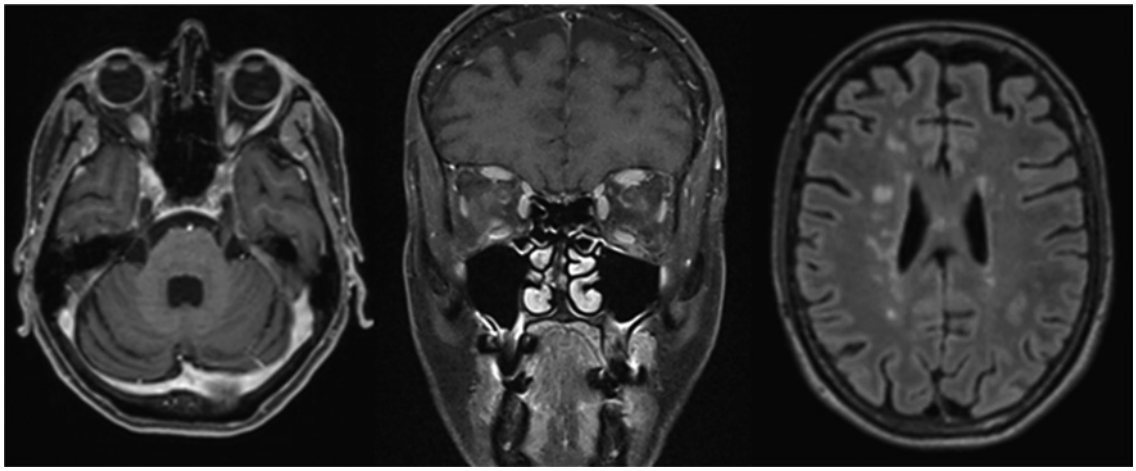

Fig. 2. Three MRI T1-weighted images (axial, frontal, axial) showing bilateral prechiasmatic enhancement of the optic nerve, pronounced on the right side. Left optic nerve is smaller, possibly due to a post-inflammatory atrophy. Right image showing typical periventricular MS lesions.

gadolinium enhancement of the optic nerves, and a clear ophthalmoscopic improvement of hypertensive retinopathy. Yet anemia persevered and the increasing erythrocyte sedimentation rate of 91 mm/h was unexplained. Since thrombocytes, complement factor, and haptoglobin had normalized, a lupus erythematosus seemed very unlikely. Overall, 24-h urine collection showed decreasing hyperproteinuria. Renal retention parameters were stable. We evaluated the most common causes of an extreme elevation of erythrocyte sedimentation rate. Multiple myeloma was unlikely due to normal protein electrophoresis with normal light chain kappa to lambda ratio. De Quervain's thyroiditis was improbable due to lacking local and systemic symptoms, although this disease was in the patient's history. Metastasizing neoplasm was unlikely due to missing B symptoms, normal abdominal ultrasound, and chest X-ray and differential blood samples had not yielded hints at leukemia. A rheumatological ultrasound found extensive inflammation of the common carotid and axillary arteries with thickening vessel walls up to 4.5 mm especially of the adventitia and acute and chronic components (Fig. 3). An MRI of the aorta found an aortitis of third degree and its closest branches matching TA (Figs. 4, 5). Since the patient was oligosymptomatic, no blood pressure differences between the extremities were measured, and there were hints in literature at an aggravation of TA under IFN [10] that we had already stopped, we evaluated if steroids were necessary. We were aware that it is unusual not to give steroids, but steroids could have aggravated the hypertension and led to a relapse of hypertensive retinopathy. Moreover, the marked vessel wall inflammation was eccentric and not hemodynamically relevant, so we agreed on a close follow-up and withheld the steroids for a potential aggravation.

We changed amlodipine to lercanidipine because of ankle edema, increased metoprolol which the general practitioner had started, and discharged the patient again. The vessel wall changes on ultrasound were stable after 2 weeks and had disappeared after 3 months without treatment.

Review of Other Reported Cases of TA and MS

Methods

A search was conducted on January 1, 2025, across PubMed, Scopus, and Google Scholar for literature on the coexistence of TA and MS. The search revealed a total of 31 results (11 from PubMed, 18 from Scopus, and 2 from Google Scholar). Despite the broad search, only 3 case reports explicitly describing the coincidence of TA and MS were identified (see Table 1). Additionally, a recent review discussing large vessel vasculitis in MS patients mentioned 2 cases but did not provide details, likely because 1 case was published later in fall 2024 [11, 12].

Results

All reported cases involved female patients and are depicted in Table 1. The sequence of diagnosis varied: MS was diagnosed before TA in 2 cases, whereas TA preceded MS in two others. In two MS patients, symptoms of TA emerged or worsened during IFN-beta treatment, but discontinuation of IFN led to symptom improvement

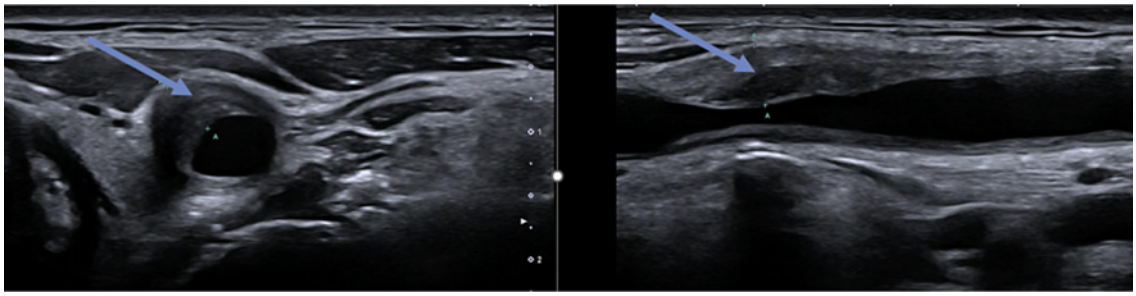

Fig. 3. Ultrasound of left carotid artery with thickening of vessel walls up to a maximum of 4.45 mm.

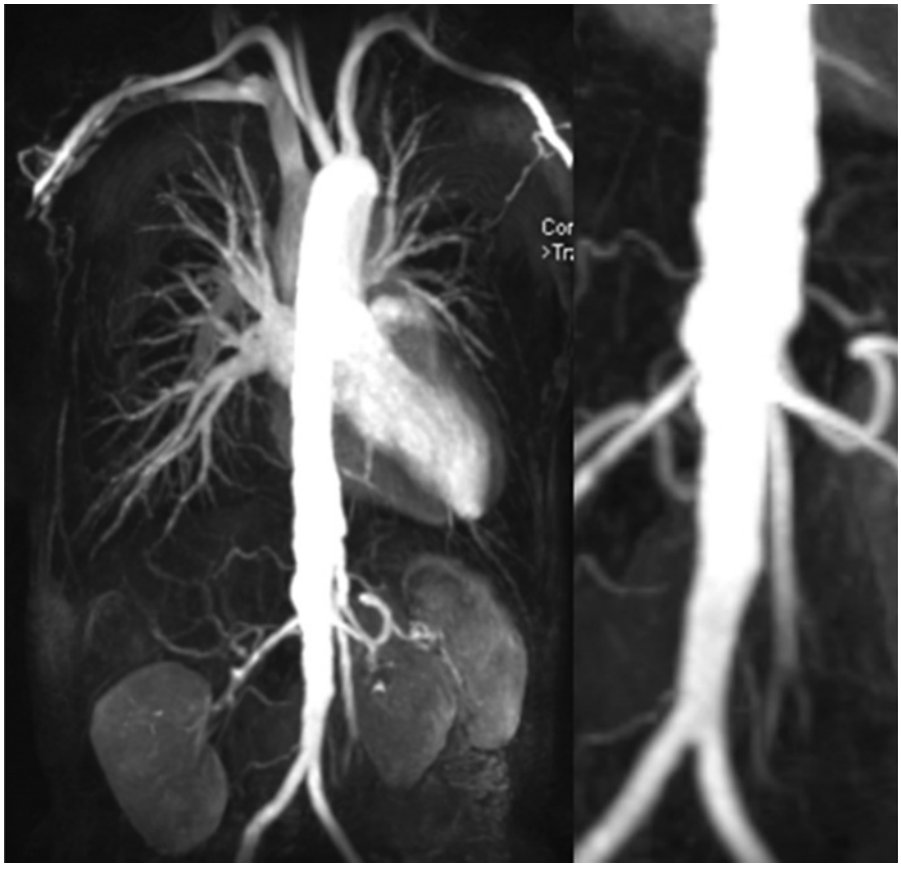

Fig. 4. MRI angiography of aorta showing only stenoses of the descending aorta (probably due to atherosclerosis).

without further relapses. Three of the patients were treated with prednisone, with one succumbing to severe vasculitis-related infarctions.

The diagnosis of MS in case 3 was mainly based on the presence of OCBs in cerebrospinal fluid, a finding that is uncommon in TA. OCBs are observed in approximately 25% of cases of primary central nervous system vasculitis [13]; however, intracranial lesions and associated OCBs are rare in TA [14]. On the other hand, OCBs can be positive in up to 5% of healthy controls [15]. Case 3 does not report a clear relapse. Both bilateral loss of vision and diffusion restrictions of white matter lesions are rare in MS [16]. This highlights the diagnostic complexity when distinguishing between MS and TA.

Discussion

We herein reported a case of a patient according to the case report guideline (CARE, online suppl. material; for all online suppl. material, see https://doi.org/10.1159/000545237) who was referred to us by ophthalmologists

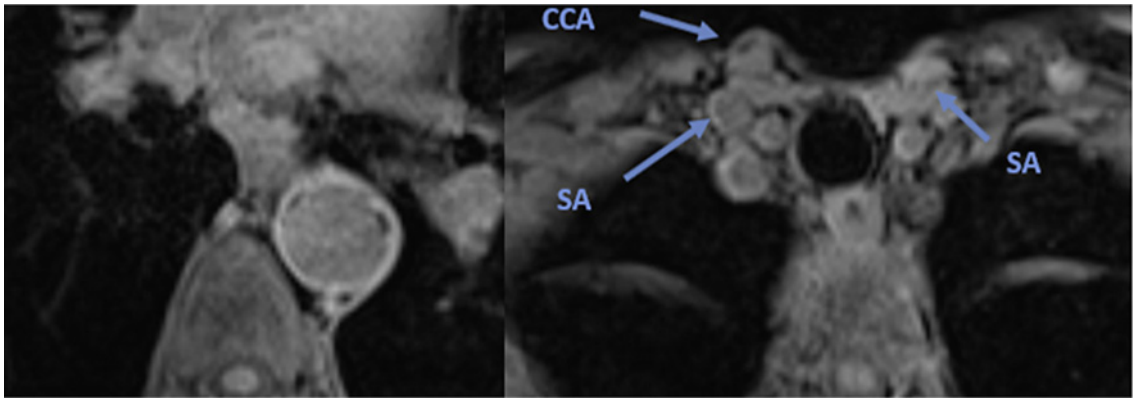

Fig. 5. MRI showing gadolinium enhancement of vessel wall: descending aorta (left), subclavian artery (SA) on both sides (right picture), and common carotid artery (CCA).

with suspected optic neuritis. Because of known MS and a contrast-medium enhancement of the optic nerves, the obvious association seemed to be an optic neuritis in the framework of MS. On closer examination, a slow progression over weeks, the lack of pain on eye movement, the bilateral affection of both optic nerves, the normal visual acuity, and the typical fundoscopic findings were indicative of hypertensive retinopathy. Literature research indicated that 5% of papillitis are due to hypertensive retinopathy [17, 18] and posterior optic neuropathy is associated with hypertension [19].

Several cases of TA presenting with hypertensive retinopathy were reported, emphasizing the importance of considering this diagnosis in patients with unexplained retinal findings [1, 2]. Case studies have documented various ocular manifestations of TA, including bilateral Takayasu retinopathy and retinal ischemia, highlighting the disease's complex and often severe impact on ocular health [11, 12].

TA can mimic many other diseases through its involvement of various organ systems, but it mostly mimics vascular phenomena such as intramural hematoma, infective endocarditis, Kawasaki syndrome, or aortic dissection [20–23]. It can also compromise the optic nerves, mainly by affecting retinal arteries, often on both sides [24]. Overall, 8–68% of the patients with TA present with ocular symptoms such as decreased vision (30%), ischemic retinopathy (15%), hypertensive retinopathy (16%), ocular ischemic syndrome (7%) and 23% of treated patients develop steroid-induced cataract (23%) [1]. The preservation of visual acuity, no hints at arteritis of the retinal vessels on fundoscopy or MRI and the vast improvement of retinal alterations after only 2 weeks of antihypertensives militate against a direct arteritic retinopathy or optic neuritis in our patient.

When erythrocyte sedimentation rate (ESR) is extensively elevated, it makes sense to distinguish patients with an underlying rheumatic disease, who also have non-rheumatic diseases in 17%, and patients without underlying rheumatic disease who show a newly diagnosed rheumatic disease in 52% [25]. Other common conditions with a highly elevated ESR of up to 100 mm/h include hypersensitivity vasculitis, giant cell arteritis, de Quervain's thyroiditis, metastatic cancer, chronic infection, hyperfibrinogenemia, multiple myeloma, leukemia, nephrotic syndrome, or a combination of different causes such as hemolysis or severe anemia [26]. This case emphasizes the clinical relevance of ESR due to a discrepancy between CRP and ESR in some infectious and rheumatic diseases [25]. Yet ESR is less sensitive and less specific than CRP. Therefore, false-positive and false-negative results occur more frequently. ESR is still valuable in detecting low-grade bone infection and lupus erythematosus [27].

There is a wide range of possible differential diagnosis for optic neuritis. There are inherited (Leber's hereditary optic neuropathy), infectious (viral, rickettsial, bacterial, fungal, parasitic), inflammatory (MS, neuromyelitis optica spectrum disorders, systemic lupus erythematosus, granulomatosis with polyangiitis, sarcoidosis, Tolosa-Hunt), paraneoplastic (small cell lung cancer), vascular (arteritic, ischemic, retinal vasospasms, diabetic papillopathy), toxic (vitamin B12/folate or copper deficiency), drug-associated (isoniazid, linezolid), and compressive causes (thyroid eye disease, orbital myositis, tuberculoma, lymphoma) [9].

In this case, it was important to distinguish hypertensive retinopathy from optic neuritis correctly since

Table 1. Overview of reported cases in literature (1–3) and our case (4)

Case	First symptoms	Clinical findings	Laboratory	Imaging	Treatment	Outcome
1 [9]	TA at age 19: chest pain MS at age 34: blurred vision, diplopia, confusion, left hemiparesis and hemihypesthesia, episodic dystonia	Brisker reflexes on the left, left-sided weakness, intention tremor bilaterally, gait ataxia, left plantar response extensor	ESR 54 mm/h (TA), normal during MS relapse, oligoclonal IgG in CSF	Multiple severe aortic arch stenoses (TA), extensive white matter disease with enhancing lesion (MS)	1 year of prednisone	Chest discomfort improved with prednisone Relapses also improved with cortisone
2 [8]	MS at age 37: symptoms not stated TA at age 52: claudication in the left arm, fever, headache, joint pain	Severe stenosis in left axillary artery, increased aorta thickness	High CRP (240 mg/L), ESR (153 mm/h), mild thrombocytosis	CT: increased thickness in the thoracic and abdominal aorta, coeliac stenosis	IV methylprednisolone, oral prednisolone, methotrexate, azathioprine (discontinued)	Severe deterioration after re-treatment with IFN beta-1a, improvement after second prednisone treatment, and discontinuation of IFN beta-1a
3 [10]	TA at age 23: malaise, weight loss, fatigue, claudication in upper extremities, anemia, palpitations, dry cough MS at age 24: bilateral blurred vision	Low blood pressure, distal pulselessness, (radial and brachial), tachycardia	ESR 34 mm/h, CRP 25 mg/dL, 7 OCBs in CSF	CT: diffuse wall thickening and stenosis of aortic branch vessels, echocardiography showed an ejection fraction of about 5–10%, valve dysfunction, brain MRI showed multiple FLAIR high-signal lesions in the white matter of cerebral and cerebellar hemispheres, some with diffusion restriction and some with enhancement	Pulse of methylprednisolone 1 g for 3 days, then oral prednisolone and cyclophosphamide 750 mg IV, mycophenolate mofetil added, then infliximab, 2 months later another pulse of methylprednisolone 1 g for 5 days, then tocilizumab when watershed infarctions were seen	Died within 15 days after tocilizumab
4 (our case)	MS according to the patient at age 4 TA at age 57: hypertensive retinopathy, papillitis, retinal bleedings	Bilateral papillitis, vasculitis, focal bleedings	Thrombocytopenia, anemia, elevated ESR (TA)	Bilateral optic nerve enhancement, periventricular lesions typical for MS, vessel wall thickening of internal carotid and subclavian artery	Antihypertensives, discontinuation of IFN	Sonographic improvement of vessel wall thickness, improvement of retinopathy

MS, multiple sclerosis; TA, Takayasu arteritis; ESR, erythrocyte sedimentation rate; IFN, interferon; CSF, cerebrospinal fluid.

steroids increase blood pressure and might have aggravated the hypertensive retinopathy. Otherwise, steroids would have treated TA and the secondary hypertension in the long run.

The meager scope of reported cases of large vessel vasculitis in MS patients may be explained by underreporting. It remains unclear whether the incidence of TA is higher or lower than to be expected in patients with MS.

In two women, TA developed after MS under the treatment with IFN beta and both women improved after its discontinuation. So far, the only known pathophysiological contribution of IFNs in TA are elevated serum levels and higher proportions of IFN-producing CD8+ T cells were found for the IFN-gamma subtype in TA patients [28]. This remains hypothetical and warrants further research.

Statement of Ethics

Ethical approval is not required for this study in accordance with local guidelines. This retrospective review of patient data did not require ethical approval in accordance with local/national guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

The authors declare no potential conflicts of interest with respect to the research, authorship, and publication of this article.

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The authors received no funding.

Author Contributions

Andrea Wesser: methodology, original draft preparation, and review. Andreas Albert Braun: conceptualization, methodology, data collection, writing, original draft preparation, and data curation.

Data Availability Statement

The data of the case report are not publicly available due to information that could compromise the privacy of the patient, but the data are available from the corresponding author Andreas Albert Braun upon reasonable request.

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