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CASE BASED REVIEW

Takayasu arteritis associated with autoimmune/inflammatory syndrome induced by adjuvants: a case-based review

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Abstract

Takayasu's arteritis (TA) is a chronic granulomatous vasculitis that predominantly affects the aorta and its major branches. Despite advancements in the understanding of the pathogenic pathways of vascular inflammation, the etiology and predisposing factors of TA remain to be fully understood. In susceptible individuals, exposure to adjuvants may trigger, unlock or unmask an autoimmune disorder, presenting as non-specific constitutional symptoms or a fully developed autoimmune syndrome such as vasculitis. Here, we hypothesize that TA could be triggered by siliconosis, a subtype of the autoimmune/inflammatory syndrome induced by adjuvants (ASIA). ASIA, also known as Shoenfeld syndrome, encompasses a wide range of autoimmune and immune-mediated diseases resulting from dysregulation of the immune response after exposure to agents with adjuvant activity. This case report describes the development of large artery vasculitis, TA, in an individual one year following the placement of silicone breast implants. The patient initially presented with non-specific symptoms, and multiple imaging methods were employed, including ultrasound diagnostics, CT angiography, and 18-fluorodeoxyglucose positron emission tomography/CT. These techniques revealed vasculitic alterations in the carotid arteries and thoracic aorta. Initial treatment with glucocorticosteroids proved ineffective, prompting the addition of steroid-sparing immunosuppressive agents. Due to the distinct clinical symptoms, disease progression, implant-associated fibrosis, and resistance to therapy, the potential involvement of implants in the development of large-vessel vasculitis was considered, and a potential association with ASIA was postulated. Although there is limited evidence to support a direct link between adjuvants and the pathogenesis of TA, similarities in cellular immunity between the two conditions exist. The diagnosis of this complex and potentially debilitating condition requires a comprehensive clinical examination, laboratory evaluation, and instrumental assessment. This will aid in identifying potential contributing factors and ensuring successful treatment.

Keywords Autoimmune/inflammatory syndrome induced by adjuvants · Autoimmunity · Breast implants · Immunologic adjuvants · Silicone gels · Takayasu arteritis · Large vessel vasculitis · Case report

Introduction

Takayasu's arteritis (TA) is a chronic granulomatous vasculitis primarily affecting the aorta and its major branches, as well as the coronary and pulmonary arteries to a lesser extent. The underlying etiology of this disease is currently unknown, and it is most frequently diagnosed in young women [1]. A portion (10–20%) of patients with TA present as clinically asymptomatic, which can result in a delay in diagnosis and potential for irreversible vascular damage [2, 3]. Conversely, the remaining majority of patients experience systemic symptoms such as fatigue, weight loss, low-grade fever, and joint and muscle pain, as well as vascular signs and symptoms such as claudication, reduced or absent pulse, hypertension, carotidynia,

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dizziness, visual disturbances, and stroke. The manifestation of these symptoms may vary based on the location and severity of the affected arteries [2, 4].

The diagnosis of TA can be challenging and may take several years to confirm due to the diverse range of symptoms and a lack of standardized diagnostic criteria. The diagnostic process typically involves evaluating clinical signs and symptoms, laboratory results, particularly markers of inflammation, and instrumental methods. Despite recent updates to the classification criteria for TA [5], the 1990 criteria, which include five clinical and one imaging criterion [1, 6], continue to be widely used in both clinical practice and studies.

Adjuvant-associated autoimmune/autoinflammatory syndrome (ASIA), first reported by Shoenfeld et al. [7], is a syndrome involving conditions characterized by dysregulation of the immune response following exposure to agents with adjuvant properties (silicone, vaccines, infections, aluminum) [7–9]. Adjuvants are defined as any substance that accelerates, prolongs, or improves antigen-specific immune responses [8]. ASIA or Shoenfeld syndrome is characterized by general clinical manifestations such as myalgias, arthralgias, myositis, neurological manifestations, frailty, and chronic fatigue [10]. Major and minor criteria have been proposed that may aid in the diagnosis of patients with ASIA syndrome [7]. To date, several conditions associated with this syndrome have been described: Gulf War syndrome, postvaccination syndromes, macrophagic myofasciitis syndrome (MMF), and siliconosis [7–9].

Siliconosis is caused by exposure to silicone or silicone-based implants, which are commonly used adjuvants in medical devices and cosmetic procedures. As a particular case of ASIA, siliconosis was shown to have the potential to impact the immune response, leading to heightened production and activation of B and T cells [7, 10, 11]. In addition, the syndrome could be accompanied by a specific local immune reaction to silicone itself, silicone degradation products, or silicone particles bound to autologous proteins activating Th1/Th17 pathway [7, 12]. Similarly, Th1/Th17 cells are implicated in the pathogenesis of TA [13]. According to Deng et al. [14], Th1 immunity is associated with chronic, while Th17 immunity—is with acute vascular injury. Although both conditions share common driving mechanisms, a cause-and-effect relationship between ASIA syndrome and Takayasu disease has not yet been hypothesized or demonstrated.

Here, we aim to describe a clinical case of a patient with TA developed after the placement of silicone breast implants and to thoroughly investigate the potential etiological and pathogenetic mechanisms between ASIA and TA through a narrative review.

Case presentation

A 39-year-old woman presented with non-specific complaints such as pain in peripheral joints and neck area, diffuse myalgias, fatigue, tingling sensation in the fingers, headache, and fever up to 37.6 °C in the afternoon and evening hours. The onset of her complaints dated back to September 2019, one year after the patient underwent breast augmentation surgery. Cohesive silicone gel implants were placed in both breasts in August 2018, mainly for aesthetic reasons.

On the occasion of the above-mentioned complaints, combined with periodic palpitations and vertigo, the patient had outpatient consultations with an endocrinologist, cardiologist, and neurologist, and relevant thyroid, neurologic, or cardiac conditions were ruled out.

Due to the progression of her illness, the patient was admitted to the rheumatology department of the local university hospital. During the initial examination, the patient was afebrile, with similar blood pressure readings in both arms and only palpable pain in the cervical region of the spine. The conducted laboratory tests showed elevated acute-phase proteins and erythrocyte sedimentation rate, normocytic normochromic anemia, and significant bacteriuria. Given the available dysuric complaints subsequently reported by the patient and the isolated pathogenic *Escherichia coli* from microbiological cultures of urine, a diagnosis of urinary tract infection was made. Hence, treatment with antibiotics was carried out in line with the antibiogram results.

Consequently, several imaging examinations were also carried out—radiographs of the chest and cervical vertebrae, MRI of the brain, and CT of the abdomen and small pelvis with no detectable pathological changes. Consultations were held with a neurologist, gastroenterologist, obstetrician-gynecologist, and urologist, and no further infectious focus was found. Interestingly, elevated values of IgM and IgG antibodies against Borrelia burgdorferi were found using enzyme-linked immunosorbent assays (ELISA) for Lyme disease, without any patient history or objective signs of a tick bite. Since samples for immunoblot were collected over 30 days after symptom onset, only anti-Borrelia burgdorferi IgG immunoblot was performed (following APHL guidelines [15]) and found negative; therefore, a diagnosis of Lyme disease was ruled out by an infectious disease specialist.

For the first time, the diagnosis of systemic vasculitis was suspected when ultrasound diagnostics revealed thickened carotid arteries, resulting in segmental stenosis and "changes with the appearance of vasculitis". The finding was confirmed in the course of another hospital stay when a CT angiography of the chest and abdomen was

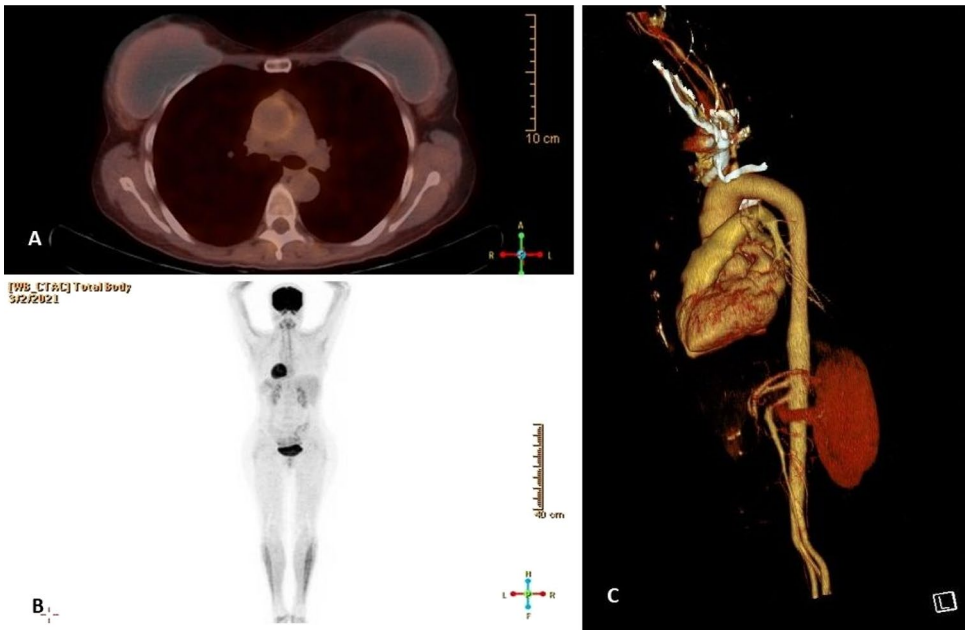

Fig. 1 Images of 18-FDG PET/CT and three-dimensional computed tomography showing: **A** an 18-FDG PET/CT showing an increased metabolic activity in the wall of the ascending aorta and aortic arch. Increased glucose metabolism is also detected around the implants and paraspinal muscles suggesting pericapsular inflammation and paraspinal myositis. **B** An 18-FDG PET/CT showing heightened glucose metabolism in lower legs and upper extremity proximal muscles. **C** A three-dimensional computed tomography (3D CT) reconstruction image of the descending aorta stenosis

performed. Then, evidence was found for the thickening of the wall of the thoracic aorta with a thickness of up to 7 mm, and the changes were visible up to the level of the cardia. There is also thickening in the wall of the three branches leaving the aortic arch with a thickness of up to 4 mm. Hence, taking into account the data from the instrumental studies, the elevated acute phase reactants and persistent constitutional symptoms, the diagnosis of Takayasu IIb aorto-arteritis was made and treatment with prednisolone equivalent 1 mg/kg body weight were started during the current hospitalization.

After discharge, the patient continued the treatment with glucocorticoids (GCs) at a dose of 1 mg/kg prednisolone equivalent slowly tapered over 6 months. Simultaneously, azathioprine 100 mg daily was also added as an initial steroid-sparing immunosuppressant. For the period from April 2020 to January 2021, the patient remained under the supervision of an outpatient rheumatologist. Upon attempting to decrease the glucocorticosteroid doses below the equivalent of 15 mg of prednisolone, the patient reported a resurgence of symptoms, including arthralgia, myalgia, headaches, low-grade fever, and episodes of supraventricular tachycardia of high frequency. This prompted re-admission to the hospital in February 2021 to carefully observe the progression of the disease and optimize treatment accordingly.

The carotid artery ultrasound performed at that time revealed diffuse thickening of the wall of the two common carotid arteries up to 2.0 mm with a norm of 0.9 mm. Subsequently, an 18-fluorodeoxyglucose (18-FDG) positron emission tomography/computed tomography (PET/CT) scan was performed in order to accurately assess the inflammatory alterations in the wall of the carotid arteries, ascending aorta, and aortic arch and to determine the progression and dissemination of the disease in question. The conspicuous enhancement of background activity in the vicinity of the implanted breast prostheses is noteworthy, which may be indicative of pericapsular fibrosis and related inflammatory changes. Furthermore, 18-FDG PET/CT revealed elevated glucose metabolism in the proximal muscle groups of the upper extremities, lower legs, and along the vertebral column, which could be linked to the concurrent presence of asymptomatic myositis (Fig. 1).

Progression of the disease with the involvement of new vessels necessitates a change in the immunosuppressants and escalation of GCs. Azathioprine was switched to methotrexate (MTX), given subcutaneously in a dose of 20 mg/week. Nevertheless, the disease remained active during the routine ambulatory checks after the 3rd month of MTX initiation. Due to the unsatisfactory effect of the ongoing therapy and maintenance of acute phase reactants, in June 2021, leflunomide 10 mg daily was added to the MTX therapy as a steroid-sparing therapy. Due to side reactions from the gastrointestinal tract in August 2021, MTX was discontinued and replaced with azathioprine 100 mg daily (approximately 2 mg/kg) in combination with leflunomide 10 mg. The therapy had a certain steroid-sparing effect and allowed the dose of glucocorticoids to be reduced to 8 mg methylprednisolone (10 mg prednisolone equivalent).

During another follow-up in April 2022, the patient arbitrarily discontinued leflunomide therapy, which led to a worsening of clinical complaints and laboratory parameters. CT angiography was performed again—without dynamics in the wall thickness of the ascending and descending thoracic aorta. A thickened wall is found in the proximal segment of

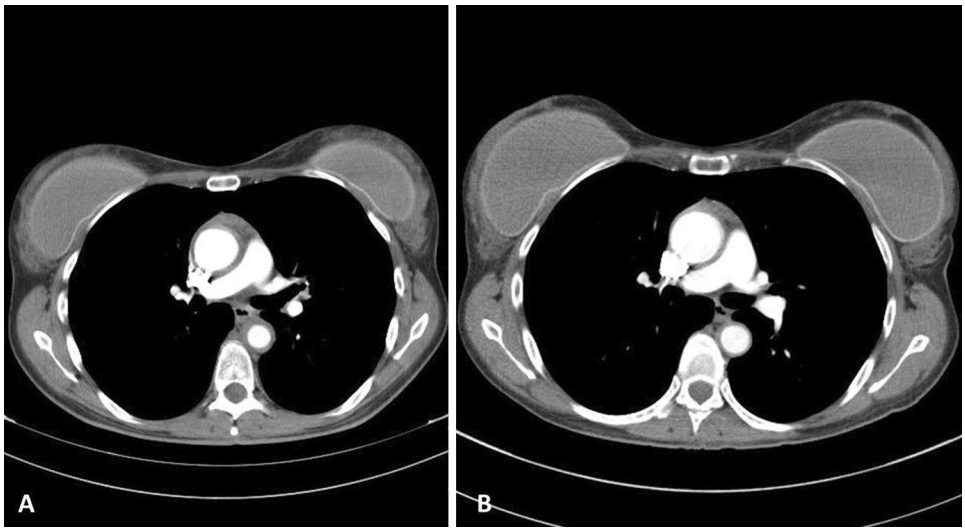

Fig. 2 Differences in the angiographic findings of the thoracic aorta between 2020 (**A**) and 2022 (**B**) show a significant regression in the thickness of the aortic wall and an increase in the vascular lumen

Table 1 Timeline of laboratory results depending on the applied therapy

Parameter	Initial hospitalization	GCs alone	AZA + GCs	MTX + GCs	MTX + LEF + GCs	AZA + LEF + GCs
Period of treatment	02.2020	02.2020–03.2020	04.2020–01.2021	02.2021–05.2021	06.2021–08.2021	09.2022–ongoing
Leukocytes (10^9/l)	6.90	7.45	8.50	6.90	6.07	9.95
Lymphocytes (%)	26.6	25.20	26.50	28.40	18.80	34.9
Platelets (10^9/l)	314	321	312	327	327	302
Hemoglobin (g/l)	105	113	104	109	105	106
CRP (mg/l)	84.41	116.35	11.65	31.34	7.95	3.9
ESR (mm/h)	116	104	47	45	17	23

AZA azathioprine, *CRP* c-reactive protein, *ESR* estimated sedimentation rate, *GCS* glucocorticoids, *LEF* leflunomide, *MTX* methotrexate

the right common carotid artery up to 2 mm, the area of the left common carotid artery up to 2.8 mm, and the area of the proximal segment of the left subclavian artery up to 2.2 mm, preceding up to 4 mm (Fig. 2). Nevertheless, regression of the finding in the left subclavian was observed.

Because of insufficient adherence to therapy, new stenoses of the carotid arteries were found. It was decided to restore the patient's basic therapy with leflunomide 20 mg every other day in combination with azathioprine 50 mg b.i.d. Timetable of the basic laboratory parameters according to the treatment is presented in Table 1.

Given the history of silicone breast implants and the chronological relationship to the disease, the tenuous course of the disease, the relative refractoriness to immunosuppressant treatment, the presence of fibrosis/inflammation around the implants, the varied clinical manifestations, including PET/CT evidence of myositis, the pathogenetic role of cohesive silicone implants in the genesis of large vessel vasculitis was discussed and a diagnosis of TA associated with ASIA was finally set by a multidisciplinary medical team. Given this, the patient was advised to have her breast implants removed as a potential trigger and cause of the disease, but at this stage she refused.

Search methodology and results

The recommendations of Gasparyan et al. for writing a narrative review [16] were adapted for writing a case-based biomedical review with a systematic approach. In an attempt to capture all relevant information, we searched the PubMed (MedLine) and Scopus databases from their inception to the 20th and 23rd November 2022, respectively, as part of our comprehensive literature review. The keywords "Takayasu" AND "ASIA syndrome", "autoimmune syndrome induced by adjuvant", "Shoenfeld's syndrome", "human adjuvant disease", "siliconosis", "silicone implant incompatibility syndrome", "silicone breast implant", "silicone adverse effects" OR "silicone related symptom complex" were entered in the boolean search console to identify 101 and 167 articles, respectively. After excluding irrelevant and duplicate articles and all reviews, original data were selected

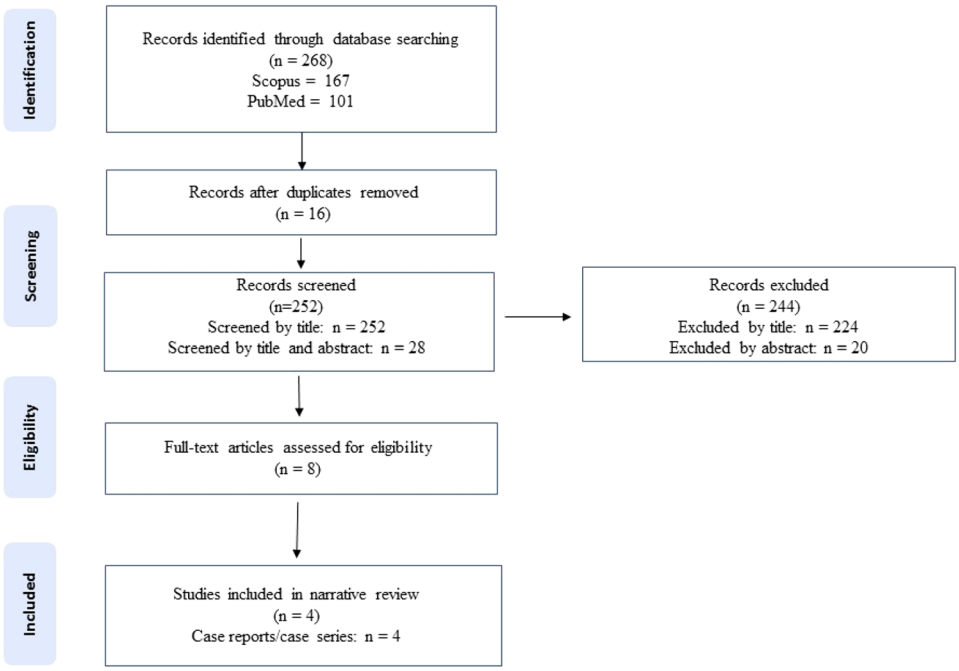

Fig. 3 Flow diagram showing the number of identified, screened, eligible and included studies

and included in the following paragraph. Figure 3 summarizes the search process. Research articles and reviews were included in the discussion section that relied upon data from references of retrieved articles, diverse database searches, and authors' professional expertise, experience, and opinions to transcribe this narrative review [16].

As a result of our literature review, no articles directly linking TA and ASIA syndrome were found. Nevertheless, we identified four articles that revealed a potential association between ASIA syndrome and other rheumatic diseases and were included as a result of our investigation of the literature. Such are the cases of patients who, in addition to TA, combine several autoimmune diseases such as ulcerative colitis, autoimmune thyroiditis, Sjögren's syndrome, and sarcoidosis [17–19]. A particular case of ASIA associated with large-vessel granulomatous vasculitis can also be considered in a case of giant-cell vasculitis that occurred after the injection of a COVID-19 vaccine [20].

Discussion

To the best of our knowledge, this is the first case of Takayasu arteritis associated with adjuvants that could be classified as an ASIA syndrome fulfilling the proposed classification criteria for both diseases.

The underlying mechanisms behind ASIA syndrome are predicated on the hypothesis that exposure to adjuvants triggers a catastrophic cascade of biological and immunological events, which may culminate in the manifestation of autoimmune disorders in susceptible individuals [12, 21]. Similar to other autoimmune and inflammatory conditions, classification criteria for ASIA syndrome have been proposed and can be divided into major and minor criteria [22]. In the case of our patient, prior to disease onset, exposure to a foreign stimulus (silicone) occurred, which eventually trigger or led to the development of an autoimmune disease. Additionally, the patient presented with typical clinical symptoms, including myositis, as evidenced by an 18-FDG PET/CT scan.

According to the current ACR recommendations, non-invasive imaging methods such as computed tomography angiography, magnetic resonance angiography or positron emission computed tomography with fluorine-18 fluorodeoxyglucose activity are preferred choices to visualize large vessel vasculitis. Those imaging modalities provide information about vessel wall inflammation, whereas catheter-based angiography primarily provides information about the vessel lumen [23]. PET/CT is useful for detecting active inflammation not only in patients with active TA before treatment but also in relapsed patients receiving immunosuppressive agents [24]. According to Meller et al., 18-FDG PET/CT imaging identifies more vascular areas involved in the inflammatory process than MRI [25]. In the case described above, 18-FDG PET/CT enabled us to evaluate the metabolic activity of the radiopharmaceutical in structures remote from the target vessels. The data on inflammatory activity around the patient's breast implants is probably the result of a foreign body-type reaction, which, according to several hypotheses, may trigger an autoinflammatory response in TA [26]. Furthermore, 18-FDG PET/CT enables us to identify myositis-like alterations in glucose utilization

suggesting the possible autoimmune phenomena outside the spectrum of the TA.

Silicone breast implants have been used in clinical practice since 1960, for aesthetic or reconstructive purposes. Some of the most frequently described complications are local pain, burning, redness, fibrous capsule formation, capsular contracture, implant rupture, and gel bleeding [27]. Silicone breast implants and associated pericapsular fibrosis activate a specific local immune response with Th1/Th17 accumulation in the capsule/body [12]. Importantly, Th1/Th17 dysregulation plays an important role in maintaining both systemic and vascular inflammation in TA. Cross-reactivity between foreign and self-pathogens has been already suggested to be involved in the pathogenesis of both TA [28] and ASIA syndrome, by stimulating autoreactive T or B cells. T cells and macrophages themselves have a key role in vasculitis, forming a vicious circle of leading to granulomatous inflammation in the arteries [13].

Current recommendations for the pharmacological management of TA include GCs alone [29] or in combination with a steroid-sparing immunosuppressive agent such as azathioprine, methotrexate, mycophenolate mofetil, or leflunomide [30]. In our case, we have initiated a combination of GCs and a single immunosuppressor as first-line treatment. Due to persistent disease activity and the inability to reduce the dose of GCs, we have used a combination of two conventional immunosuppressants similar to the practical approach to TA management presented by Keser et al. [31]. The approach required close monitoring to avoid adverse events. Despite the appropriate course of immunosuppressive therapy and combination, the disease remains relatively refractory to treatment and necessitates medium doses of GCs. At this stage, we may explain the fact with an "incorporated" adjuvant that triggers and maintain the vicious circle of autoimmunity/autoinflammation.

A characteristic feature of the ASIA syndrome includes major improvement after the elimination of the inciting agent [22]. Thus, we advised the patient to remove the cohesive silicone gel implants but she refused at the time of writing this article. This action deprives us of the possibility to test our hypothesis of a direct etiological/pathogenetic relationship between breast implants and immune-mediated phenomena, including the development of vasculitis. Moreover, the histopathological examination would provide additional information valuable for resolving differential diagnoses.

Conclusions

TA is a rare granulomatous vasculitis with an unknown etiology that could be triggered by exposure to environmental factors in individuals with a genetic predisposition. Both TA low prevalence and clinical heterogeneity present a major barrier to unveiling the exact driving mechanisms of the disease. Here, we present the first case that suggests a possible link of immune-mediated phenomena, following exposure to silicone, i.e. cohesive breast implants, that result in large vessel vasculitis.

Although there is no conclusive evidence that adjuvants could play a direct role in TA pathogenesis, a striking similarity of cell immunity in both conditions is present. Epiphenomenal cross-reactivity mediated by the adaptive immune system might initiate and amplificate inflammatory response in TA. In-depth clinical examination, laboratory, and instrumental evaluation is the key to diagnosis, identification of potential exacerbating factors, and successful treatment of patients with TA.

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Author contributions DS was responsible for composing, including conducting a literature review, editing, and revising the manuscript throughout all stages of production. TG played a role in the clinical management of the patient and contributed to writing, editing, and revising the manuscript. TS participated in the clinical management of the patient, manuscript editing, and revision at all stages of production. All co-authors approved final approval of the manuscript and take full responsibility for the integrity and accuracy of all aspects of the work.

Data Availability Upon request to the corresponding author, non-confidential data can be shared on a reasonable basis.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent A written informed consent was signed by the patient in the case concerning the publication of this case report. A digital copy of this consent is available upon reasonable request.

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