International Research Journal of Pharmacy and Medical Sciences
ISSN (Online): 2581-3277

Takayasu's Arteritis Complicated with Myocardial Infarction in an Elderly Patient: A Rare Case Report

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Abstract*—Takayasu's arteritis (TA) is a condition similar to other forms of vasculitis, such as giant cell arteritis, which predominantly occurs in older adults. TA may present as a pulseless disease. This article describes a clinical case of TA in an 80-year-old woman who has been under observation since 2020. The patient came to the clinic with the following complaints: chest pain behind the sternum radiating to the left shoulder, dry cough, shortness of breath, and mild ataxia. Echocardiography was performed, which showed moderate dilatation of the left ventricle, calcification of the aortic annulus and mitral valve semilunar cusps, and moderate hypertrophy of the left ventricle. Additional studies were prescribed. Dopplerography showed left ventricular diastolic dysfunction, aortic and mitral valve regurgitation, and cor pulmonale with pulmonary hypertension. On the X-ray, the bronchovascular pattern of the lungs was enhanced, the right sinus was not illuminated, and the left was free. On the ECG, depression of the T and ST segments was pronounced without acute damage. As a result of ultrasonographic and angiological examinations, a diagnosis of TA was made, complicated by a myocardial infarction. Appropriate treatment with methotrexate was performed, accompanied by corticosteroids, namely prednisolone. In order to prevent complications, monitoring has been carried out for four years. At this stage, the disease is not progressing. No additional complications have occurred. This clinical case is interesting both in terms of the course of the disease and its management.*

I. Introduction

The first case of Takayasu's arteritis (TA) was described in 1908 by Japanese ophthalmologist Mikito Takayasu, who identified an unusual coronal-like arrangement of blood vessels in the retina, which he termed the corona sign. Japanese physicians Onishi and Kagoshima also reported similar retinal symptoms in individuals who had absent pulses in the wrists [1].

TA, also called pulseless disease or aortic arch syndrome, is a nonspecific aortic arteritis and a form of nonpulsatile granulomatous vasculitis with massive intimal fibrosis and vascular stenosis. It occurs more frequently in young to middle-aged women of Asian descent, although it can occur at any age [2-5]. It predominantly affects the aorta and its branches, as well as the pulmonary arteries. Women are affected approximately eight to nine times more often than men. Individuals with this disease develop symptomatic manifestations between the ages of 15 and 30. The incidence of TA varies across regions [6].

According to the literature, atherosclerosis is a more common cause of vascular occlusion than TA. TA is similar to other vasculitides, including giant cell arteritis, which usually occurs in older people [4]. Due to obstruction of the major branches of the aorta, including the left common carotid artery, brachiocephalic artery, and left subclavian artery, TA may present as a pulseless upper limb symptom. Pulses are weak or absent in the brachial, forearm, and carpal arteries [7,8].

In the early stages of the disease, some people develop an inflammatory phase, which manifests as a systemic illness: general weakness, fatigue, fever, night sweats, weight loss, joint pain, and tachycardia, which may be associated with carotid sinus hyperreactivity or subclavian artery compression syndrome [2]. Anemia and a sharp increase in ESR and C-reactive protein are common in the anamnesis. After the inflammatory phase, a pulseless phase develops. It is characterized by vascular insufficiency and narrowing of the intima of blood vessels, which is most often manifested by renal artery stenosis. These processes obviously cause hypertension. However, due to occlusion, cerebral blood vessel perfusion does not occur, and neurological manifestations are common, such as dizziness, confusion, seizures, and, in severe cases, hemorrhagic stroke [4,5].

It is worth noting that due to renal artery stenosis, high blood pressure (BP) causes hypoperfusion of the juxtaglomerular apparatus, which leads to excessive secretion of renin, increased aldosterone in the blood, water and salt retention, and increased BP [6,7,9].

Neurological symptoms of the disease vary depending on the degree of damage. The degree is determined by the area or percentage of luminal occlusion, so the range of neurological manifestations can vary from dizziness to generalized seizures. One of the rare but important features of TA is the development of visual field defects with subsequent blindness or retinal hemorrhage. In the late stages, the weakness of the arterial walls can lead to local aneurysms, increasing the risk of their rupture and bleeding, requiring monitoring. Coronary artery aneurysm is a very rare complication of this condition. Although the cause of TA is unknown, it is characterized by segmental focal granulomatous changes in the aorta and its main branches, which in turn leads to arterial stenosis, thrombosis, or aneurysm. Chronic vasculitis causes vascular fibrosis, sometimes leading to massive intimal fibrosis. Diagnosis is made by magnetic resonance angiography

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(MRA), computed tomography angiography (CTA), or digital arterial angiography (DSA) [8].

TA is genetically associated with HLA-B*52. A 2013 study identified multiple additional susceptibility loci in the chromosome, increasing the number of Takayasu genetic loci. Approximately 200,000 genetic variants were genotyped in two ethnically diverse TA cohorts from Turkey and North America using a genotyping platform (Immunochip). Additional genetic variants and classical HLA alleles were identified and analyzed. The study identified and confirmed two independent susceptibility loci in the HLA region (HLA-B/MICA and HLA-DQB1/HLA-DRB1) [10].

A recent GWAS identified genetic susceptibility loci for TA, including IL6 and an intergenic locus on chromosome 21q22. The genetic susceptibility locus RPS9/LILRB3 lies within the leukocyte receptor complex gene cluster on chromosome 19q13.4. Disease risk variants at this locus correlate with reduced expression of multiple genes, including the inhibitory leukocyte immunoglobulin-like receptor B3 gene [8]

Treatment of TA involves high-dose corticosteroids such as prednisone (1 mg/kg/day) with gradual tapering. Immunosuppressive agents like methotrexate, azathioprine, or mycophenolate are often added to reduce steroid dependence. In refractory cases, biologic therapies such as tocilizumab or infliximab may be used. Patients with critical vascular complications may require revascularization procedures like angioplasty or bypass surgery. Regular monitoring with imaging and inflammatory markers is essential to assess disease activity and treatment response [11,12].

II. Case Presentation

An 80-year-old woman presented to the Heart Clinic in 2020 with the following complaints: left shoulder pain, dry cough, shortness of breath, a feeling of choking, easy fatigue, confusion, and general weakness. Physical examination revealed general pallor and tremors in the extremities and lower jaw. Palpation revealed no pulse on the radial artery. BP (T/A) was 210/110 mm Hg. Respiratory rate (RR) was 29 breaths per minute. PO_2 was 89%. Percussion revealed clear lung sounds, with wet wheezing and crepitus audible below and behind the right shoulder along the axillary line. Cardiac auscultation revealed muffled heart sounds.

Based on initial investigations, a myocardial infarction (MI) was suspected. However, vascular diseases were ruled out following further assessment.

The final diagnosis was confirmed after a thorough evaluation, which included a synthetic analysis of multiple diagnostic tools: Doppler imaging of the fundus (Figure 1), chest X-ray (Figure 2), computed tomography (CT) (Figure 3), echocardiography (see below), and coronary angiography (see below).

The combination of these findings allowed for the definitive diagnosis of TA, complicated with myocardial infarction (MI).

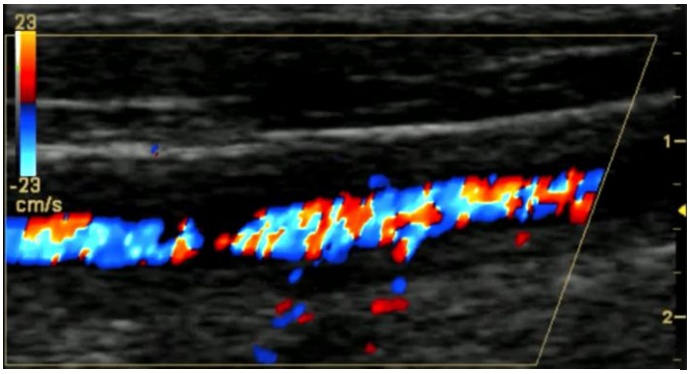

Figure 1: Pulse wave of blood flow in the central retinal artery (red) and vein (blue) measured by laser Doppler imaging in the fundus of a patient with TA.

Laser Doppler imaging with near-infrared digital holography can reveal characteristic blood flow waves in the central retinal artery and retinal vein in patients with vascular insufficiency. This can demonstrate a smooth systolic-diastolic pulse in the central retinal artery. This technique allows noninvasive functional microangiography with high-contrast measurements of endoluminal blood flow profiles in the posterior segment of the eye, with spatial resolution comparable to that of modern indocyanine green angiography.
Common carotid artery (CCA):
Right side: significant stenosis detected. Multiple calcified plaques are noted on both the left and right sides. The degree of stenosis is approximately 60%. The artery appears S-shaped. The peak systolic velocity is low.
Left side: multiple calcified plaques are noted on the left side. The degree of stenosis is approximately 50%. Peak systolic velocity is noted.
Vertebral arteries:
Right – diameter 3.8 mm. Stenosis > 50%.
Left – diameter 3.3 mm. Stenosis > 50%.

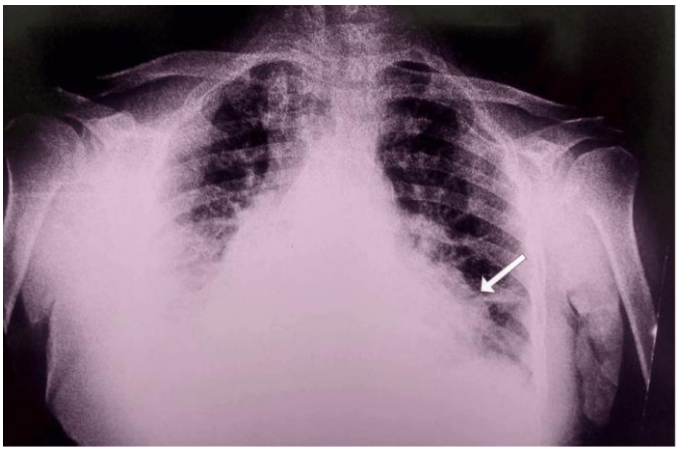

Figure 2: Chest X-ray

The bronchoalveolar image of the lung is enhanced, and the hila appear thickened. Against the backdrop of decreased transparency in the lower lung fields, a large, rounded, high-intensity shadow with uneven edges is observed on the left. The border of the heart is enlarged (indicated with an arrow).

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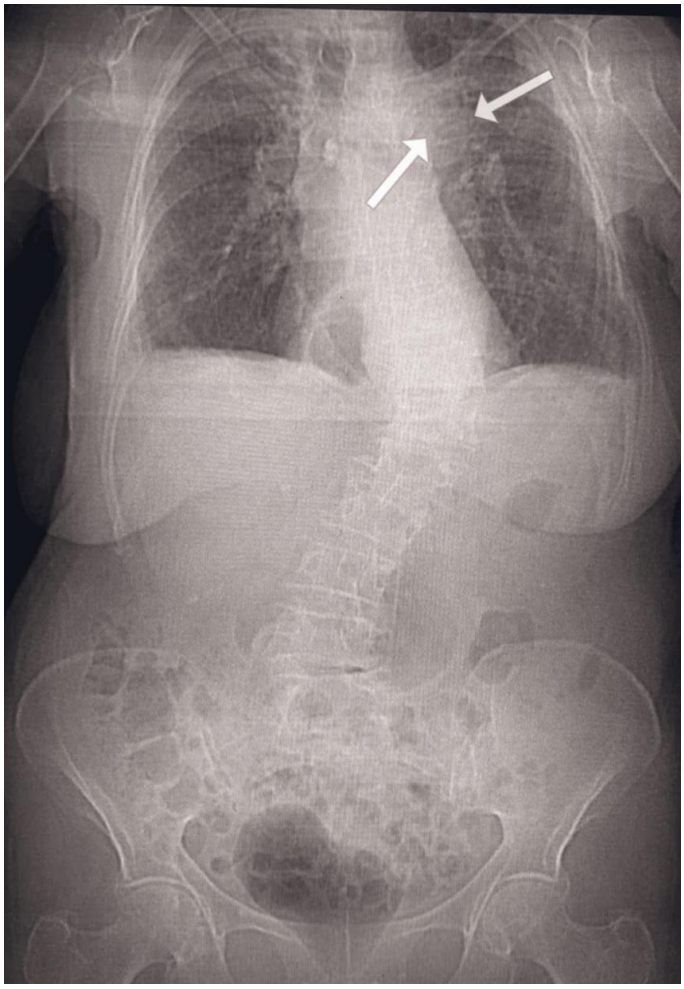

Figure 3: Computed Tomography Scan

Arch of Aorta: The aortic arch exhibits irregular thickening, with narrowing of the left subclavian artery and brachiocephalic trunk (indicated with arrows).
Pulmonary Vasculature: The pulmonary vasculature appears normal.

Echocardiography Report

The echocardiogram shows calcification of the mitral annulus and aortic annulus and valves. The left atrium, left ventricle, and right heart cavities are mildly dilated. Systolic function is reduced, likely due to hypokinesia of the lower basal segment. The interventricular septum is hypertrophic. Doppler imaging reveals biventricular diastolic dysfunction, classified as "worsened" relaxation type, with mitral II and tricuspid II dysfunction noted. A mild aortic regurgitation is present, with a maximal pressure gradient on the aorta of 30 mmHg. Additionally, a slight stenosis is observed with a mean pressure gradient (MPG) of 15 mmHg. The systolic pressure in the pulmonary artery is elevated, and the pulmonary artery pressure (PAPreac) is 34 mmHg (normal <25 mmHg).

Coronary Angiography Report

The left coronary artery trunk shows no significant changes and divides into two branches: the anterior descending and circumflex arteries. The anterior descending artery has an uneven wall but no significant stenosis. The diagonal branch also shows no stenosis. The circumflex artery is 30% stenosed proximally, and the first marginal branch is 50% stenosed at the ostium.

The right coronary artery shows 80% stenosis proximally and medially, with a right-type supply.

Blood pressure is recorded as TA Ao 140/80 mmHg.
The right coronary artery was intubated with a 6F JR-3.5 Mach 1 guiding catheter (Boston Scientific). Sol. Heparin 5000 U was administered intra-coronarily.
Two coronary stents were implanted medially and proximally in the right coronary artery:
REBEL (Boston Scientific) 3.0-16mm (14 atm)
REBEL (Boston Scientific) 3.0-16mm (16 atm)
Sol. Tsunami 350 was used, with a total volume of 200 ml.

Treatment and Follow-up

After confirming the diagnosis, it was decided to start treatment with methotrexate. Methotrexate treatment was initiated at 0.3 mg/kg once weekly and gradually increased to 25 mg/week. Prednisolone was also prescribed at 300 mg/day. Due to the side effects of long-term, high-dose prednisone use, the initial dose was gradually tapered to a maintenance dose over several weeks. Although treatment with mycophenolate and tocilizumab could have been alternative options, the patient's condition improved with the prescribed therapy.

TABLE 1: Dynamics of laboratory examination data by years

Date	**29.02.2020**	**18.05.2020**	**04.05.2021**	**26.01.2022**	**22.08.2022**	**06.02.2023**
ESR (mm/hr)	65 mm/hr	66 mm/hr	45 mm/hr	40 mm/hr	85 mm/hr	53 mm/hr
Fibrinogen concentration (mg/dL)	560 mg/dL	606 mg/dL	467 mg/dL	651 mg/dL	750 mg/dL	553 mg/dL
Creatinine (µmol/L)	99 µmol/L	117 µmol/L	115 µmol/L	108 µmol/L	113 µmol/L	87 µmol/L
Troponin (mg/L)	0.20-0.011 mg/L	0.01-0.010 mg/L	0.023-0.010 mg/L	0.01-0.01 mg/L	0.01-0.01 mg/L	0.1 mg/L
Prothrombin Index (%)	94%	93.3%	80%	83%	82%	82%
C-reactive protein (CRP) (mg/L)	174 mg/L	154 mg/L	159 mg/L	152 mg/L	149 mg/L	152 mg/L
Glucose (mmol/L)	Fasting - 7.4, Post-meal - 6.8	Fasting - 5.8, Post-meal - 5.7	Fasting - 6.7, Post-meal - 10.2	Fasting - 8.1, Post-meal - 7.6	Fasting - 7.9, Post-meal - 7.0	Fasting - 7.8, Post-meal - 7.2
Na+ (mmol/L)	128 mmol/L	130.4 mmol/L	134.7 mmol/L	131 mmol/L	137 mmol/L	136 mmol/L
K+ (mmol/L)	4.52 mmol/L	3.97 mmol/L	4.41 mmol/L	4.30 mmol/L	4.20 mmol/L	4.05 mmol/L

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Table shows the patient's laboratory results over multiple visits, including markers for inflammation (ESR, CRP, fibrinogen), kidney function (creatinine), cardiac injury (troponin), blood clotting (prothrombin index), glucose metabolism, and electrolyte levels (Na+ and K+). The laboratory results demonstrate mild fluctuations in inflammatory markers (ESR, CRP, fibrinogen) and stable renal function (creatinine) over time. There is mild variation in glucose levels, with some episodes of elevated fasting glucose, possibly indicating early-stage metabolic changes. Electrolyte levels (Na+ and K+) remained relatively stable. Overall, the patient's condition is being closely monitored with no significant adverse trends in the laboratory data.

TABLE 2: Dynamics of instrumental examination data by year

	29.02.2020	**18.02.2020**	**04.05.2021**	**26.01.2022**	**22.08.2022**	**06.02.2023**
Chest X-ray	Enhanced pulmonary bronchovascular pattern; Hardened hila; Non-homogeneous shadow (moderate intensity) in middle and lower right fields; Right sinuses not visible, left side clear; Enlarged left heart border, filled cardiac waist.	Enhanced pulmonary bronchovascular pattern; Right sinuses not visible, left side clear; Slightly enlarged heart borders (left & upper side); Filled cardiac waist.	Significantly enhanced pulmonary bronchovascular pattern; Hardened hila; Non-homogeneous shadow (moderate intensity) in lower right field; Enlarged left heart border.	Markedly enhanced pulmonary bronchovascular pattern; Severely hardened hila; High-intensity, non-homogeneous shadow in middle & lower right fields; Significantly enlarged left heart border.	Significantly enhanced pulmonary bronchovascular pattern; Hardened hila; All heart borders enlarged.	Markedly enhanced pulmonary bronchovascular pattern; Hardened areas in hila; All heart borders enlarged.
Chest Ultrasound	Moderate left atrial dilation; Aortic ring calcification; Valve fibrosis, mitral calcification; Significant septal enlargement; Moderate concentric hypertrophy of left ventricle.	Moderate left atrial enlargement; Aortic ring calcification; Valve fibrosis, mitral calcification; No significant septal enlargement; Concentric hypertrophy of left ventricle.	Left atrial dilation; Calcification of aortic & mitral rings; Significant septal enlargement; Moderate concentric hypertrophy of left ventricle.	Left atrial dilation; Aortic & mitral ring calcification; Significant septal enlargement; Massive concentric hypertrophy of left ventricle.	Left atrial dilation; Aortic ring calcification; Valve fibrosis, mitral calcification; Significant septal enlargement; Concentric hypertrophy of left ventricle remains.	Severe left atrial dilation; Aorto-mitral calcification; Significant septal enlargement; Concentric hypertrophy of left ventricle.
Doppler Ultrasound	Left ventricular diastolic dysfunction (impaired relaxation); Aortic regurgitation; Mitral regurgitation.	Partial left ventricular diastolic dysfunction; Aortic regurgitation.	Right & left ventricular diastolic dysfunction; Aorto-mitral dysfunction.	Diastolic dysfunction of both ventricles; Aorto-mitral dysfunction.	Diastolic dysfunction of both right & left ventricles; Severe aortic dysfunction.	Acute diastolic dysfunction of both ventricles; Aorto-mitral dysfunction.
Coronary Angiography & Angioplasty	Sinus rhythm; Leftward deviation of heart's electrical axis (-T); T-wave & ST-segment depression in lead I, aVL; No acute focal damage.	Sinus rhythm; T-wave & ST-segment depression in lead I, aVL; Minor focal damage.	Sinus rhythm; Leftward deviation of heart's electrical axis (-T); T-wave & ST-segment depression in lead I, aVL; Acute focal damage.	Non-sinus rhythm; Leftward deviation of heart's electrical axis (-T); Marked T-wave & ST-segment depression in leads I, II, aVL; Acute focal damage.	Sinus rhythm; Leftward deviation of heart's electrical axis (-T); T-wave & ST-segment depression in leads I, II, aVL; Acute focal damage.	Sinus rhythm; Leftward deviation of heart's electrical axis (-T); T-wave & ST-segment depression in lead I, aVL; Focal damage.

Table presents a summary of the patient's diagnostic imaging findings and cardiac function tests over a period of three years. The chest X-ray shows progressive enhancement of the pulmonary bronchovascular pattern, hardened hila, and enlarging heart borders. The chest ultrasound consistently shows left atrial dilation, calcification of the aortic and mitral rings, and left ventricular hypertrophy, with the severity increasing over time. Doppler ultrasound indicates diastolic dysfunction, with aortic and mitral regurgitation observed in various stages. Coronary angiography highlights focal damage, with changes in the heart's electrical axis and T-wave abnormalities, indicating ongoing ischemia. The table tracks the evolution of these findings, which reflect the patient's deteriorating cardiac condition.

III. Discussion

As mentioned before, TA is an uncommon disease, especially in elderly patients. In this case, the patient, an 80-year-old woman, presented with symptoms that were initially suggestive of MI. However, following further imaging, Takayasu arteritis was diagnosed, which is an atypical presentation at her age.

The patient's MI was likely a result of compromised coronary circulation due to the progressive stenosis of the aortic branches, which is a rare but serious complication of TA. Treatment with high-dose corticosteroids (prednisolone) and methotrexate was initiated, aimed at controlling inflammation and reducing vascular damage. While corticosteroids are essential for managing the disease, they come with long-term side effects, which is why methotrexate was used to reduce steroid dependency.

The patient's condition improved with this regimen, highlighting the importance of timely diagnosis and the effectiveness of immunosuppressive therapy. Regular monitoring is crucial to detect complications like aneurysms or further vascular occlusion, as they are common in TA.

Additionally, it is important to highlight that the woman had undergone a radical mastectomy, which can sometimes be complicated by a rapidly progressive tumor, such as

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lymphangiosarcoma-only around 150 cases have been reported worldwide.

IV. Conclusions

We believe the case described above holds significance both theoretically and practically. Noteworthy in this case is the absence of a pulse in the radial artery and the pronounced neurological symptoms present in the patient's history. The prognosis in elderly patients is often challenging, but with early diagnosis and appropriate treatment, positive outcomes can be expected.

This patient's disease has remained stable for five years, with no significant progression or complications. Although Takayasu arteritis is rare in older adults, this case demonstrates that with the right therapeutic approach, the disease can be managed successfully, even in elderly patients with complicated comorbidities. Regular follow-up and monitoring are essential to prevent further complications.

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