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CASE REPORT

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Acute myocardial infarction and left ventricular thrombus in a young male with thromboangiitis obliterans: a case report and literature review

Junyan Zhang[1†], Kai Li[1†], Li Rao[1], Yong He[1], Zhongxiu Chen[1*] and Xin Wei[1*]

Abstract

Thromboangiitis obliterans (TAO) is an inflammatory vascular disease that primarily affects the small- and medium-sized arteries of the extremities. Coronary artery involvement in TAO is rarely reported, and we present the first case of TAO involving the coronary arteries with concomitant left ventricular thrombus. In addition, our case report includes a literature review on coronary artery involvement in TAO and draws comparisons with systemic vasculitis with coronary artery involvement. The case report and literature review aims to offer physicians valuable insights for diagnosis and treatment of TAO with coronary arteries involvement, while also advocating for increased research efforts to explore therapeutic strategies for this rare condition, including both pharmacological treatment and interventional therapy.

Keywords Acute myocardial infarction, Thromboangiitis obliterans, Coronary artery, Young male smoker, Literature review

Introduction

Thromboangiitis obliterans (TAO), also known as Buerger's disease, is a segmental, non-atherosclerotic inflammatory condition that predominantly affects the small and medium-sized arteries, veins, and nerves of the limbs [1]. Reports of TAO involving the coronary arteries are exceedingly rare, and there is currently no consensus on the optimal revascularization strategies or specific antithrombotic regimens for these patients [2]. Herein, we present a case of a patient with established TAO who developed acute anterior myocardial infarction accompanied by a left ventricular thrombus. This case report also provides a comprehensive review of the literature on coronary involvement in TAO and compares it with systemic vasculitis, aiming to summarize current knowledge in this field. We call upon cardiovascular specialists to focus research efforts on this unique patient population, addressing both diagnostic and therapeutic approaches, including interventional and pharmacological strategies.

Case report

A 28-year-old male came into our emergency room with chief complaints of acute chest pain and sweating. The electrocardiogram showed ST segment elevation in V1-V4 and frequent ventricular bigeminy (Fig. 1). Serological test disclosed a significant increase in myoglobin

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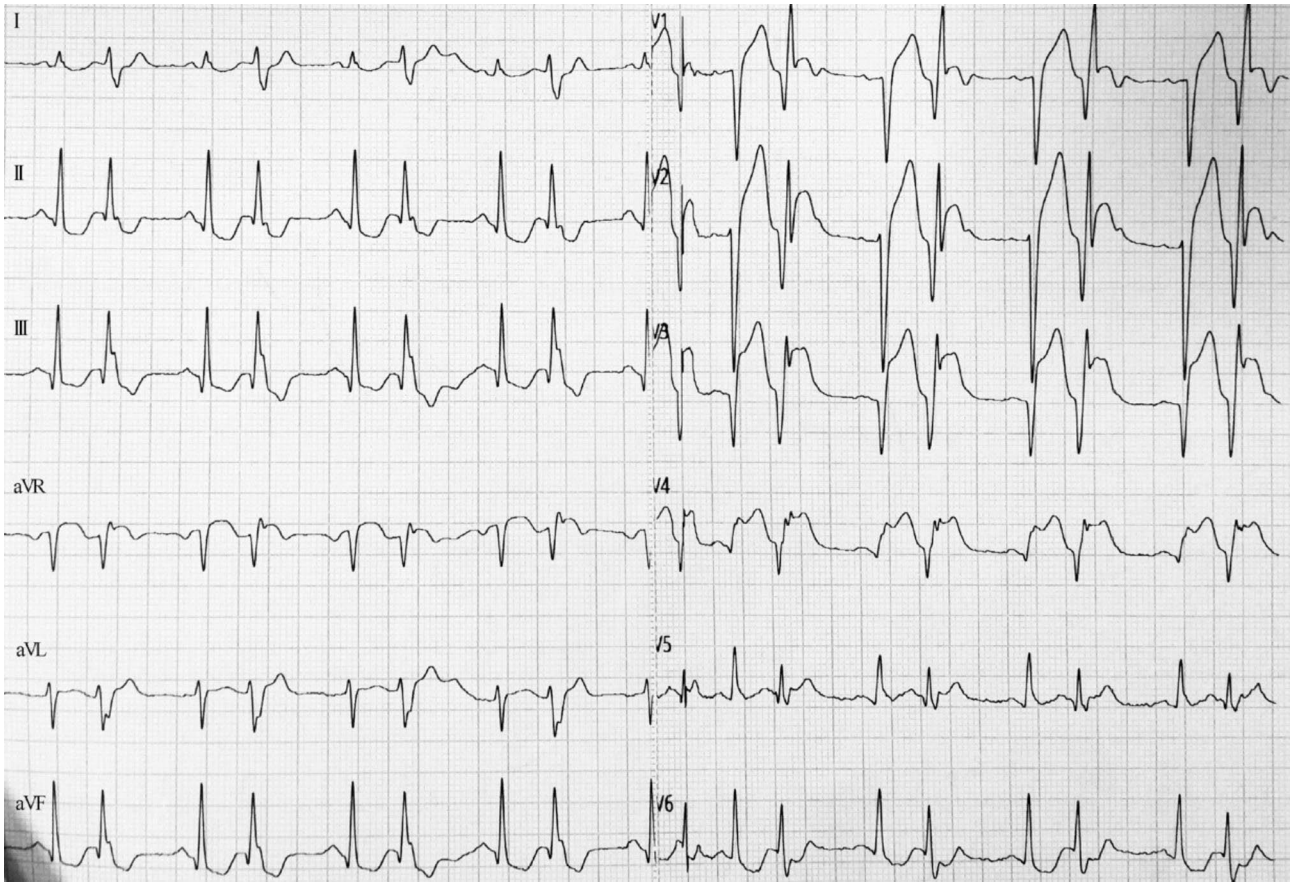

Fig. 1 Electrocardiogram. ST segment elevation in V1-V4 and frequent ventricular bigeminy

(196.20 ng/ml) and cardiac troponin T (>10000.0 ng/ml). He was diagnosed with acute anterior myocardial infarction and hospitalized in cardiac care unit. Emergent coronary angiography was performed and demonstrated the total occlusion of middle and distal segments of right coronary artery with the formation of multiple corkscrew-collaterals and reverse perfusion of left anterior descending coronary artery, while no stenosis was observed in left coronary artery (Fig. 2, Supplementary Material). Although there was no identified involvement of the left coronary artery by the coronary angiography, we considered that a transient thrombus could have been there.

Further medical history was taken. This young patient has been smoking one package of cigarettes per day for more than 10 years. He has no family history or other risk factors for coronary atherosclerotic heart disease. No evidences were found for hyperlipemia or diabetes mellitus. He has been diagnosed with TAO 5 years ago in our hospital, with the clinical manifestations of Raynaud's phenomenon of right hand and dry gangrene of right ring finger (Fig. 3). He has been taking aspirin, Sarpogrelate hydrochloride, Beraprost sodium and cilostazol after discharge. However, he didn't follow the instruction of smoking cessation. Ulceration of the distal portion of his left foot was found 2 years ago, and it was basically healed after carful nursing. Physical examination showed the absence of right ring finger and ischemia over the distal portion of left foot.

The computed tomography angiography further confirmed the severe segmental stenoses of the left popliteal artery, left peroneal artery, left anterior tibial artery and left posterior tibial artery with smooth vessels in non-involved region (Fig. 4). Ultrasonic cardiogram (UCG) revealed an abnormal motion of segmental left ventricular wall and the left ventricular ejection fraction was 41%. On the 27th day of admission, a left ventricular apical thrombus was noted by UCG, cardiovascular magnetic resonance imaging confirmed the myocardial lesion and thrombus (Fig. 5). Embolism-related indicators were tested. The results of anti-nuclear antibody, anti-phospholipid antibody, anti-neutrophil cytoplasmic antibody, anti-SCL-70 antibody, lupus anti-coagulant complex and tumor markers were all negative by laboratory studies. There were no mutations in coagulation factor II, coagulation factor V or JAK2 V617F. Furthermore, protein C and protein S were at the normal level. He received aspirin, warfarin, benazepril hydrochloride, β-blocker

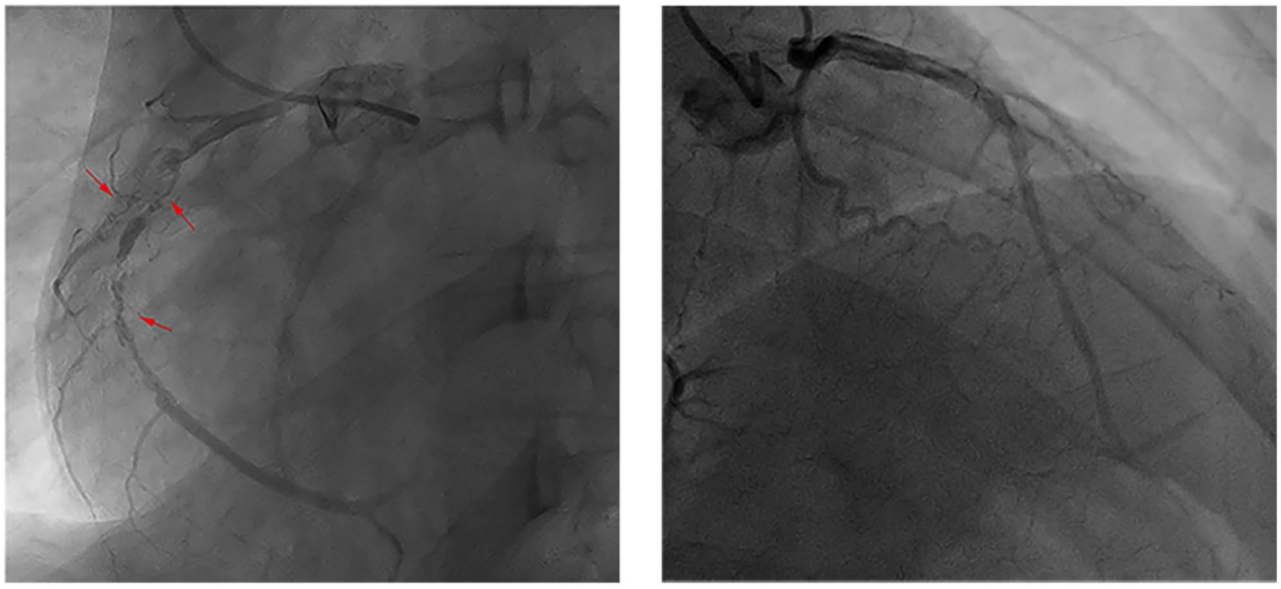

Fig. 2 Coronary angiography. Total occlusion of middle and distal segments of right coronary artery with the formation of multiple lateral branches (arrow, left). Angiography image of left coronary artery (right)

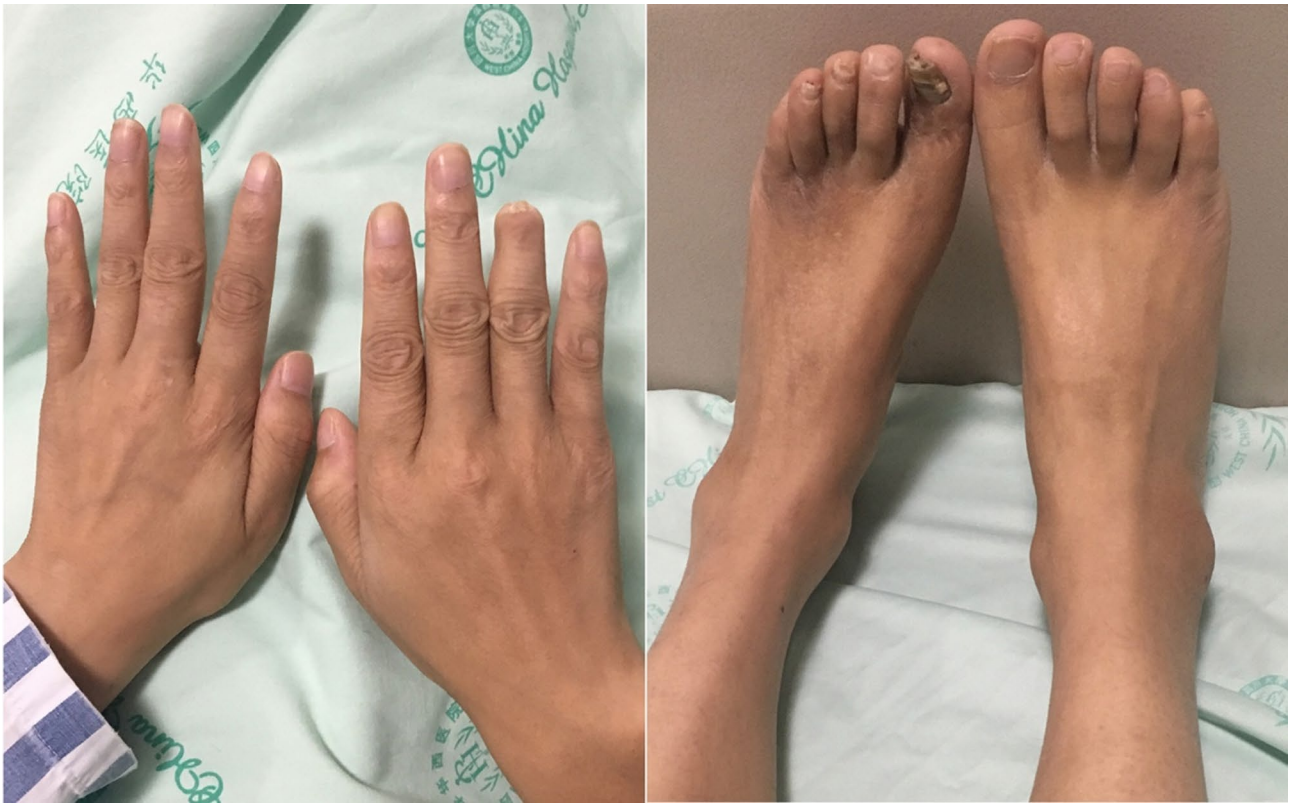

Fig. 3 The clinical manifestations of the patient's digits

and Sarpogrelate hydrochloride and was discharged after symptom remission. Three years after discharge, a telephone follow-up was conducted, during which the patient reported a satisfactory recovery with no recurrence of chest pain or other symptoms. Additionally, significant improvement was observed in the distal limb lesions. Due to the complete resolution of symptoms, the patient expressed a reluctance to undergo repeat coronary angiography.

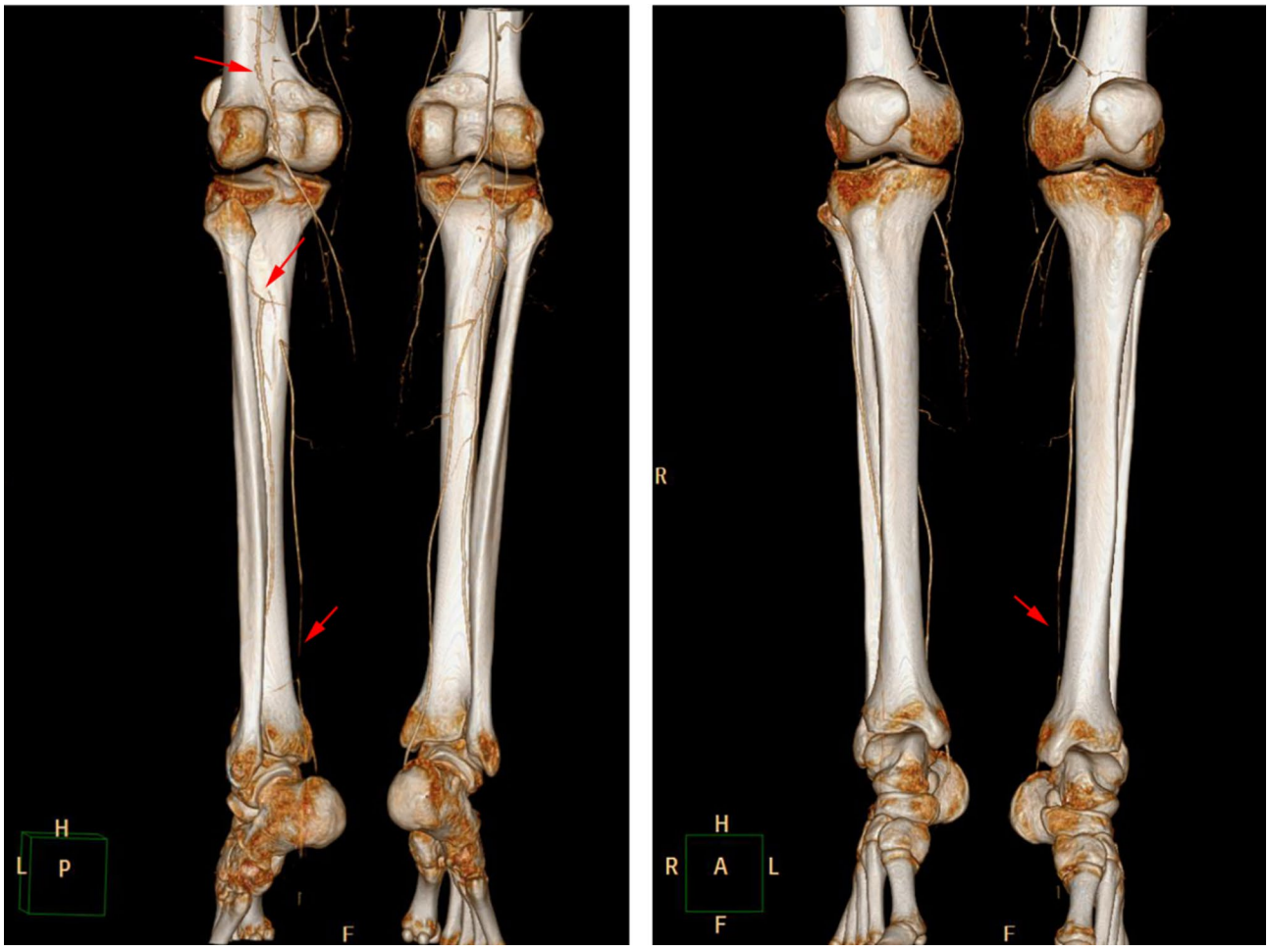

Fig. 4 Computed tomography angiography of lower limbs. Severe stenoses of left popliteal artery and left anterior tibial artery, and severe segmental stenoses in left peroneal artery and left posterior tibial artery (arrow)

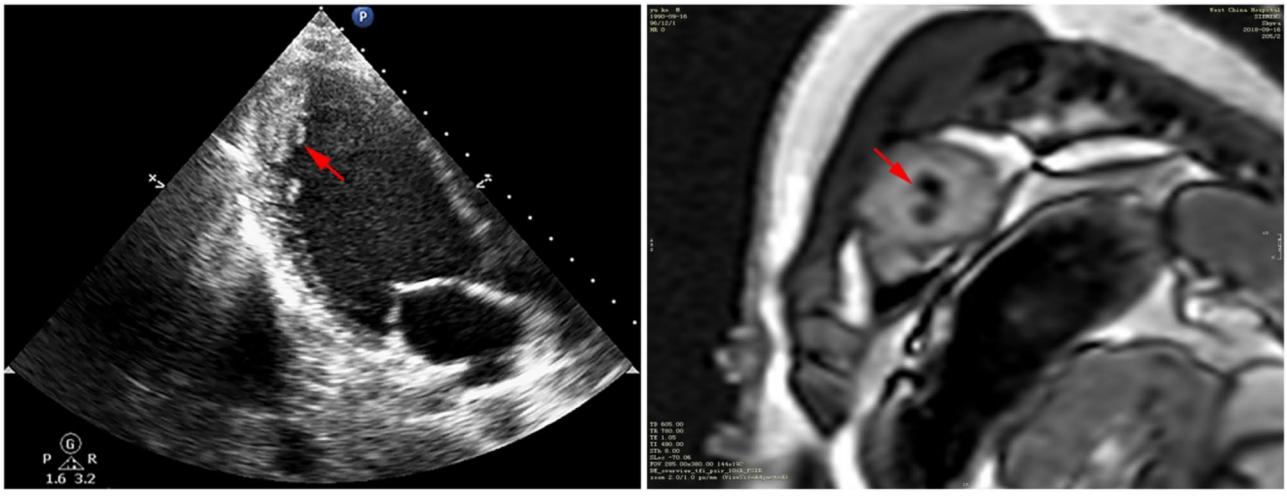

Fig. 5 Thrombus in left ventricular apex. A left ventricular apical thrombus demonstrated by ultrasonic cardiogram (arrow, left) and cardiovascular magnetic resonance (arrow, right)

Discussion

TAO is a segmental non-arteriosclerotic vasculitis disease of unknown origin that typically affects the small and medium-sized vessels of upper and lower extremities [3]. It is uncommon for TAO to involve large vessels, although there have been rare cases reported involving coronary arteries in patients with Buerger's disease (Table 1) [4–14]. Current published cases show no clear pattern of coronary artery involvement in TAO, with all three major vessels potentially affected and presenting primarily as acute myocardial infarction, particularly STEMI, but also as CTO lesions. It is worth noting that TAO patients may present with acute myocardial infarction and seek care from the cardiology department, which may possibly lead to misdiagnosis [2]. Therefore, it is crucial for cardiologists to be aware of this possibility. In this case report, we first present a unique case of a young TAO patient who developed acute myocardial infarction with left ventricular thrombus and aims to increase awareness and understanding of such presentations.

Despite being recognized for over a century, the precise pathological mechanism of TAO remains unclear [15]. Nevertheless, thrombosis and inflammation are widely acknowledged as pivotal components in the disease's pathological process [1, 16, 17]. Multiple studies have demonstrated the presence of inflammatory thrombi in the affected vessels of patients during the acute phase of TAO [18]. These thrombi are typically occlusive, accompanied by infiltration of inflammatory cells such as neutrophils and multinucleated giant cells [1]. This pattern of thrombosis distinguishes TAO from other systemic vasculitides that involve coronary arteries [19, 20]. The distinguishing features between TAO and systemic vasculitides involving the coronary arteries are summarized in Table 2. Systemic vasculitis is characterized by the presence of inflammatory leukocytes in vessel walls, leading to reactive damage to mural structures. In contrast, TAO is distinguished by intraluminal thrombosis without involvement of the vessel wall. Therefore, a thorough patient history and comprehensive auxiliary examinations are crucial for accurately determining the underlying cause of myocardial infarction.

Thrombus formation in TAO is primarily driven by endothelial dysfunction, chronic inflammation, immune responses, and oxidative stress [21]. Persistent inflammation damages endothelial cells, exposing collagen and tissue factor, which promote platelet adhesion and activation [22]. Elevated pro-inflammatory cytokines, such as interleukins and TNF-α, exacerbate endothelial injury and recruit leukocytes, while neutrophils release extracellular traps (NETs) that stabilize thrombi [23]. Immune complexes and autoantibodies further inhibit natural anticoagulants, leading to a hypercoagulable state. Oxidative stress, often induced by smoking, generates reactive oxygen species that impair endothelial function and activate the coagulation cascade [24]. Genetic predispositions may also increase thrombosis susceptibility [25]. This complex interplay fosters an environment conducive to thrombus development in TAO, suggesting potential therapeutic targets, including anti-inflammatory agents, antioxidants, and anticoagulants, to mitigate thrombogenesis and enhance patient outcomes.

Indeed, there is a lack of consensus regarding the optimal revascularization strategy for patients with coronary artery involvement in TAO and systemic vasculitis. In the case of STEMI patients, primary PCI is recommended to enhance patient outcomes [26]. On the other hand, it is essential for individuals with different types of coronary artery issues to focus on treating the primary disease because regression of coronary lesions may happen at the same time as alleviating the primary condition. For TAO patients, smoking cessation stands as the foremost treatment strategy, while vasodilators like prostaglandin analogs and phosphodiesterase inhibitors may alleviate symptoms [3]. Similarly, for the majority of systemic vasculitis patients, a treatment regimen involving glucocorticoids and immunosuppressants can ameliorate their condition [27]. Consequently, the coronary artery involvement in many patients may ameliorate with primary disease treatment. Hence, the necessity of immediate intervention therapy during the acute phase remains a topic of debate. Moreover, for patients necessitating delayed revascularization, the decision between PCI and CABG remains uncertain [28, 29]. In this case, after comprehensive discussion among the team, we ultimately opted for a conservative treatment strategy combining smoking cessation with medication, rather than proceeding with PCI or CABG. The favorable outcomes observed during follow-up further underscore the critical importance of smoking cessation as a therapeutic strategy.

Research indicates that patients with systemic vasculitis exhibit a heightened likelihood of stent restenosis post-PCI and graft vessel stenosis or occlusion following CABG [29–31]. This increased rate of restenosis is attributed to the inflammatory state of the arteries. Some studies even propose that individuals with coronary artery vasculitis should steer clear of coronary revascularization during active inflammation, as the latter elevates the risk of MACE by approximately tenfold [32]. Wang et al. also posit that if revascularization becomes imperative during active inflammation, CABG surpasses PCI, with their study revealing a notably higher incidence of MACE events in the PCI cohort compared to the CABG cohort among patients with Takayasu's arteritis [29]. In a study encompassing 806 patients with Takayasu's arteritis, Huang et al. observed that while cardiac mortality rates were akin in both CABG and PCI groups, the restenosis

Table 1 Summary of the main reported cases of Buerger's disease with coronary involvement

Author	Age	Sex	Smoke	CAD presentation forms	Vessel	CAG finding	Treatment	Diagnosis	Prognosis	Reference
Rey et al.	30	Male	Yes	STEMI	LAD	Thrombotic subocclusion	PCI	Cocaine-associated thromboangiitis obliterans	The cardiac rehabilitation was uneventful.	[5]
Tekin et al.	45	Male	Yes	STEMI	LAD	Coronary dissection	CABG	Buerger's disease	The patient was discharged five days post-surgery.	[6]
Tamura et al.	44	Male	Yes	Q wave in ECG	RCA	Corkscrew appearance	/	Buerger's disease	Not mentioned.	[7]
Hsu et al.	32	Male	Yes	STEMI	LAD	Thrombus	Thrombus aspiration and stent implantation	Buerger's disease	Discharged 5 days after coronary intervention.	[8]
Kim et al.	29	Male	Yes	STEMI	LAD	Partial segmental occlusion	Conservative managment	Buerger's disease	The patient was discharged on the 17th day with no recurrence of chest pain.	[9]
Abe et al.	25	Male	Not mentioned	CCS	LAD	CTO		Buerger's disease	Not mentioned.	[10]
Donatelli et al.	39	Female	No	Unstable angina	LAD + RCA	Severe left main disease and critical stenoses of the right coronary artery	CABG	Buerger's disease	At 8 months' follow-up the patient was doing well and was free of recurrent angina.	[11]
OHNO et al.	32	Male	Yes	STEMI	LAD + RCA	Thrombus	Urokinase	Buerger's disease	Not mentioned.	[12]
Becit et al.	36	Male	Yes	AMI	LAD + RCA	CTO	CABG	Buerger's disease	Seven months post-operation, he was readmitted with chest pain, and angiography showed occlusion of the aorta-LAD bypass graft.	[13]

Table 1 (continued)

Author	Age	Sex	Smoke	CAD presentation forms	Vessel	CAG finding	Treatment	Diagnosis	Prognosis	Reference
Akyuz et al.	43	Male	Yes	STEMI	LAD + RCA	Total occlusion	Thrombus aspiration and PTCA	Buerger's disease	Discharged with NYHA II functional capacity, fully recovered from hemiplegia, partially from hemianopsia.	[14]
Hong et al.	61	Male	Yes	Myocardial perfusion scan was performed that demonstrated reversible ischemia	D1 + LCX + RCA	Multiple, sequential, intraluminal filling defects	Conservative managment	Buerger's disease	Two weeks later, the patient underwent left carotid endarterectomy without complications.	[4]

Abbreviation: STEMI: st-elevation myocardial infarction; LAD: left anterior descending artery; PCI: percutaneous coronary intervention; CAD: coronary artery disease; CAG: coronary angiography; RCA: right coronary artery; CABG: coronary artery bypass grafting; ECG: electrocardiogram; CTO: chronic total occlusion; CCS: chronic coronary syndrome; AMI: acute myocardial infarction; PTCA: percutaneous transluminal coronary angioplasty; D1: first diagonal artery; LCX: left circumflex artery

Table 2 Comparison of characteristics between TAO and systemic vasculitis

	TAO	Systemic vasculitis							IgG4-related disease
		Large vessel vasculitis		Medium vessel vasculitis		Small vessel vasculitis		Various vessel vasculitis	
		Takayasu's arteritis	Giant cell arteritis	Kawasaki Disease	Polyarteritis nodosa	GPA	EGPA	Bechet's Disease	
Coronary artery involvement (%)	< 1%	10–30%	< 5%	20–30%	< 20%	< 20%	< 20%	< 0.5%	< 10%
Main angiography features	stenosis	Diffuse, stenosis, occlusion	stenosis	aneurysm, thrombosis	stenosis, occlusion, aneurysm	stenosis, occlusion	stenosis, occlusion	aneurysm, stenosis	stenosis
Histology feature	Thrombus including neutrophils and giant cells occludes the vessel lumen but spares the wall.	Transmural mononuclear cell infiltrate with fragmentation of internal elastic lamina, and granulomatous arteritis	Lymphoplasmacytic infiltrate, epithelioid macrophages, giant cells, elastic fragmentation	Lymphohistiocytic inflammatory infiltrate leading to destruction of the internal elastic lamina and necrosis	Panarteritis with fibrinoid necrosis and polymorphous inflammatory infiltrate	Necrotizing granulomatous inflammation without eosinophils	Necrotizing granulomatous inflammation with eosinophils	Endothelial damage and wall disruption cause red blood cell leakage, fibrinoid necrosis with thrombosis, and neutrophil-dominated inflammation in vessels and surrounding tissue.	Lymphoplasmacytic infiltrate, IgG4+ plasma cells, storiform fibrosis

Abbreviations: TAO, thromboangiitis obliterans; IgG4, immunoglobulin G4; GPA, granulomatosis with polyangiitis; EGPA, eosinophilic granulomatosis with polyangiitis

rate was markedly higher in the PCI cohort than in the CABG cohort [28]. Nonetheless, these conclusions predominantly stem from Takayasu's arteritis with a heightened likelihood of coronary artery involvement, rather than TAO. However, TAO can affect internal mammary arteries, which may be problematic for patients requiring coronary artery bypass grafting surgery [33].

Given the substantial pathophysiological variances between TAO and Takayasu's arteritis, these findings may not be universally applicable to TAO. The vascular wall inflammation in TAO is not as pronounced as in Takayasu's arteritis, suggesting that PCI might present a viable option for these patients.

In the present case report, the patient exhibited both acute myocardial infarction and left ventricular thrombus. While it is challenging to definitively ascertain whether the left ventricular thrombus originated from the TAO-related pathological process or from left ventricular dysfunction post-AMI, patients with TAO have a higher thrombotic burden compared to those with systemic vasculitis. Although the optimal antithrombotic regimen for such patients remains undetermined, it is likely that a more aggressive antithrombotic strategy may be required compared to that for systemic vasculitis.

Conclusion

TAO, an inflammatory vascular disease typically affecting the extremities' small- and medium-sized arteries, is rarely associated with coronary artery involvement. Our case report presents the first case of TAO with concurrent coronary artery involvement and left ventricular thrombus. We further reviewed the literature on coronary artery involvement in TAO and comparing it with systemic vasculitis affecting the coronary arteries. This case report and literature review aims to provide clinicians with critical insights into diagnosing and treating TAO with coronary artery involvement, while also championing for intensified research into therapeutic strategies for this uncommon condition, encompassing pharmacological and interventional approaches.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13019-025-03383-0.

Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

Supplementary Material 5

Supplementary Material 6

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Not applicable.

Author contributions
Junyan Zhang and Kai Li contributed equally to this work as co-first authors. They were involved in the conception and design of the study, data collection, and drafting the manuscript. Li Rao and Yong He assisted in provided critical revisions of the manuscript for important intellectual content. Xin Wei and Yong He were responsible for the clinical management of the patient and contributed to the data collection and manuscript preparation. Xin Wei and Zhongxiu Chen served as co-corresponding authors. They supervised the overall project, provided guidance throughout the study, and contributed to the manuscript's final revisions.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
None of the patient's personal identifcation was mentioned in the manuscript. The patient's consent was approved by Ethical committee of West China Hospital, Sichuan University.

Consent for publication
An informed written consent was obtained from the patient.

Competing interests
The authors declare no competing interests.

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