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Thromboangiitis Obliterans Complicated by Myocardial Infarction in a Young Male Patient

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Abstract

Background: Thromboangiitis Obliterans (Buerger's disease) is a rare, non-atherosclerotic, segmental inflammatory vascular disease that predominantly affects young male smokers. Its cardiac involvement is extremely uncommon and may mimic atherosclerotic coronary artery disease. **Case Presentation:** We present the case of a 29-year-old male patient who was admitted with acute myocardial infarction. During cardiac rehabilitation, a more detailed medical history revealed previous episodes of thrombophlebitis, Raynaud's phenomenon, intermittent claudication, and a history of heavy smoking. Based on these findings, Thromboangiitis Obliterans was suspected and subsequently confirmed by further diagnostic evaluation. **Conclusion:** This case emphasizes the crucial importance of thorough medical history taking and comprehensive diagnostic evaluation, especially in young patients presenting with acute coronary events. By presenting this case, we aim to highlight that even subtle vascular or thrombotic symptoms may provide essential diagnostic clues and should not be overlooked.

Keywords

Thromboangiitis Obliterans, Intermittent Claudication, Myocardial Infarction

1. Introduction

The syndrome was first described by Leo Buerger in 1908 in his seminal work "*Thromboangiitis obliterans: A study of the vascular lesions leading to presenile spontaneous gangrene*" [1]. Although von Winiwarter already reported a similar condition in 1879, Buerger was the first to clearly define it as a distinct entity through detailed histological and clinical observations. In 1966, Schatz associated Raynaud's phenomenon with the disease.

Buerger's disease is an idiopathic condition most commonly observed in young (<45 years), heavy-smoking males, though the proportion of affected females has been increasing, likely reflecting changing smoking habits. Thus, the disease is no longer considered exclusive to young men. Disease onset typically occurs between 17 and 45 years of age, being rare in childhood or after age 50. Between 80% and 99% of patients are active smokers [2]. Intermittent claudication may occur but it is more characteristic of atherosclerotic obliterans. Doppler ultrasound can detect vascular abnormalities even in asymptomatic individuals. Raynaud's phenomenon and lower-limb thrombophlebitis are common accompanying features, with superficial venous inflammation observed in 40% - 60% of cases. The disease course is marked by acute exacerbations and prolonged remissions. Recurrent exacerbations may extend the process to cerebral, pulmonary, coronary, or other visceral arteries. Coronary involvement leading to myocardial infarction is extremely rare but does not exclude the diagnosis of Buerger's disease. [3]

Immunological factors may contribute to its development, although their precise nature remains unclear. A potential genetic predisposition has also been proposed (HLA-A9, HLA-B5, HLA-B54). [4]

Geographical variation is notable: the incidence is lowest in Western Europe and North America, while significantly higher rates are reported in Japan, Türkiye, India, Korea, and Israel [5]. Differences in regional smoking patterns and ethnic susceptibility may both play a role. [6]

Diagnosis is based on clinical presentation, medical history, and imaging findings, as no single laboratory test is diagnostic. Hemoglobin, hematocrit, white blood cell count, erythrocyte count, ESR, serum glucose, and lipid profiles are typically within normal limits. The presence of polycythemia or thrombocytosis practically rules out TAO. (**Table 1**)

Table 1. Differential diagnostic features of *Thromboangiitis Obliterans* (TAO) and *Atherosclerosis Obliterans* (ASO), summarizing key clinical, pathological, and angiographic distinctions between the two entities.

	TAO	ASO
Age at disease onset	20 - 40 years	>40 years
Sex ratio (male: female)	7:1 to 3:1	>50 years: 1:1
Histology	1. Intimal proliferation 2. Cellular thrombus formation 3. Marked recanalization 4. Perithrombotic transformation 5. Absence of necrosis 6. Lamina elastica interna intact	1. Plaque formation 2. Calcification
Localization	Small and medium-sized arteries	Large arteries
Type of vascular lesion	Localized, segmental	Generalized
Venous involvement	Frequent (40% - 60%)	None
Upper extremity involvement	16% - 74%	Rare (~10%)
Intermittent claudication	Instep claudication	Calf, thigh, or gluteal muscles

Continued

Clinical course	Sudden onset, episodes with remission	Slowly progressive
Coronary sclerosis	Rare	Common
Microcirculatory damage	Present already in early stage	Develops only in late stage
Raynaud's phenomenon	Common (~40%)	Rare
Gangrene	Frequent (40% - 70%)	Less common (10% - 20%)
Amputation	Common	Less frequent
Life expectancy	Similar to general population	About 10 years shorter than general population
Smoking	>90%	50% - 60%
Diabetes mellitus	Rare	Common
Hypertonia	Rare	Common
Dyslipidaemia	Rare	Common
Angiographic findings	Segmental occlusions alternating with intact segments; "corkscrew"-like collaterals	Stenosis, occlusion, and calcification of major arteries

Source: Landi, A.—"Belgyógyászati Angiológia". Medintel Publishing, 1999.

In light of these findings, the international literature currently uses the terms Winiwarter-Buerger disease or Thromboangiitis Obliterans (TAO) to describe the clinical syndrome observed in young smokers, characterized by arterial and venous inflammation, distal-type arterial occlusion, and the presence of Raynaud's phenomenon.

2. Case Report

A 29-year-old male patient was admitted for cardiac rehabilitation following an inferior STEMI. Urgent coronary angiography revealed a long, irregular, partially critical stenosis in the mid-to-distal RCA. The lesion did not exhibit the typical features of atherosclerosis. PCI was performed, deploying a Xience Alpine (DES) 4 mm × 28 mm stent using a direct stenting technique. The procedure was uneventful.

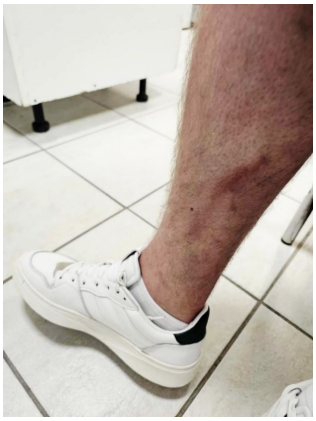

Figure 1. Ultrasound excluded deep vein thrombosis, but confirmed superficial thrombophlebitis with adjacent subcutaneous inflammation.

The patient was a heavy smoker (24 pack-years). In the year preceding the infarction, he had twice been treated for migratory lower-extremity phlebitis (as shown in Figure 1). Upon admission, he reported recurrent episodes of digital cyanosis (as shown in Figure 2) and cramping pain in the soles and calves after walking 300 - 400 meters. Family history of cardiovascular disease was negative.

Figure 2. Raynaud's phenomenon was observed during physical examination.

Laboratory evaluation revealed elevated hemoglobin (17.2 g/dL), attributed to secondary polyglobulia from heavy smoking. White blood cell count ($9.64 \times 10^3/\mu L$), platelet count ($236 \times 10^3/\mu L$), Lp(a) (0.06 g/L), and fibrinogen (3.2 g/L) were within normal limits. HbA1c was normal (5.5%). (Table 2)

Pre-intervention lipid values were elevated but normalized by the time of rehabilitation.

Table 2. Summary of the patient's laboratory parameters.

Test	Result	Reference Rage
Erythrocyte Sedimentation Rate (ESR)	2.0 mm/hr	2.00 - 10.00 mm/hr
White Blood Cell Count (WBC)	$9.64 \times 10^3/\mu l$	4.00 - 10.00 $\times 10^3/\mu l$
Red Blood Cell Count (RBC)	$5.03 \times 10^6/\mu l$	4.30 - 5.80 $\times 10^6/\mu l$
Hemoglobin (Hgb)	17.2 g/dl	13.00 - 18.00 g/dl
Hematocrit (Hct)	48.5%	40.00 - 50.00%
Mean Corpuscular Volume (MCV)	96.4 fL	80.00 - 95.00 fL
Mean Corpuscular Hemoglobin (MCH)	34.2 pg	26.00 - 34.00 pg
Mean Corpuscular Hemoglobin Concentration (MCHC)	35.5 g/dl	30.00 - 36.00 g/dl

Continued

Platelet Count (PLT)	236 $\times 10^3/\mu l$	150.00 - 400.00 $\times$ $110^3/\mu l$
Fibrinogen	3.2 g/l	2.1 - 3.5 g/l
Lipoprotein A (LPA)	0.06 g/l	<0.3 g/l
Total Cholesterol	2.62 mmol/l	3.9 - 5.2 mmol/l
HDL Cholesterol	0.76 mmol/l	0.90 - 2.07 mmol/l
LDL Cholesterol	1.42 mmol/l	0.00 – 3.40 mmol/l
Triglycerides	0.97 mmol/l	0.65 - 1.85 mmol/l
Blood Glucose	4.7 mmol/l	3.9 - 6.00 mmol/l
Hemoglobin A1C (HbA1c)	5.5%	—

The patient's medication regimen included: acetylsalicylic acid 100 mg once daily, prasugrel 10 mg once daily, atorvastatin 40 mg once daily, ezetimibe 10 mg once daily, pantoprazole 40 mg once daily, nebivolol 2.5 mg once daily, and ramipril 2.5 mg once daily.

Doppler ultrasound revealed segmental perfusion deficit in the left lower limb (ABI: 0.7), supporting peripheral arterial involvement. Echocardiography showed preserved left ventricular systolic function (EF: 76%), without regional wall-motion abnormalities or valvular disease. Carotid ultrasound showed normal vessel walls and patent major cervical arteries.

During treadmill testing, the patient demonstrated excellent physical capacity, but the test was terminated at 11.3 METs due to cramping pain in the soles and calves, further supporting peripheral arterial disease.

3. Discussion

Thromboangiitis Obliterans (Buerger's Disease) is a rare, non-atherosclerotic, inflammatory vascular disorder that primarily affects young, heavy-smoking males. Its pathogenesis is multifactorial, involving complex immunological, genetic, and environmental interactions. Although many aspects of its etiology remain unclear, clinical and epidemiological evidence consistently identifies smoking as the central causative factor, exerting a crucial influence on both disease onset and progression. Triggers induced by nicotine and other tobacco smoke components play a key role in the development of peripheral vascular abnormalities. Endothelial injury and fragmentation of capillaries can be demonstrated in the vasospastic episodes characteristic of Raynaud's phenomenon, which, in severe cases, may lead to digital ulceration or gangrene. This phenomenon is also observed in a wide range of autoimmune disorders, such as Systemic Lupus Erythematosus (SLE) and Mixed Connective Tissue Disease (MCTD) [7]. Another cardinal feature of the disease is the occurrence of migratory superficial thrombophlebitis, which may involve both the upper and lower extremities. The inflammatory process may also affect the arteries—most commonly in the lower limbs—although the presence of upper extremity arterial involvement, in conjunction with any two of the three cardinal

symptoms, is sufficient for establishing the diagnosis of TAO.

Most patients have a favorable prognosis if the triggering factor—tobacco use—is successfully eliminated. In such cases, symptoms may regress significantly, and complete remission may even occur, allowing life expectancy to approximate that of the general population, in contrast to Atherosclerosis Obliterans (ASO).

In the majority of cases reported in the literature, the diagnosis of TAO preceded the occurrence of acute myocardial infarction, with subsequent cardiac involvement representing a rare visceral manifestation of the disease [8]. Angiographic findings in these instances typically reveal segmental, non-atherosclerotic lesions, often—but not invariably—associated with "corkscrew"-like collateral vessel formation. Coronary involvement in TAO is exceptionally rare in clinical practice. [9]

In our case report, a 29-year-old man presented with myocardial infarction as the initial manifestation of the disease. Coronary angiography revealed lesions not characteristic of atherosclerotic disease, but rather segmental and irregular narrowing—findings consistent with a visceral manifestation of TAO. A detailed medical history confirmed previous episodes of migratory thrombophlebitis, Raynaud's phenomenon, intermittent claudication, and a significant smoking history, together supporting the diagnosis of Thromboangiitis Obliterans.

This case is unique in that the patient was first evaluated due to acute myocardial infarction, and only subsequently was the diagnosis of TAO established—contrary to most reported cases, where TAO had already been recognized prior to cardiac involvement. Following the myocardial infarction, the patient discontinued smoking, resulting in remarkable improvement over a four-month follow-up period: Raynaud's symptoms markedly subsided, and his walking distance improved to several kilometers without discomfort. However, after resuming smoking four months later, new episodes of superficial thrombophlebitis developed in the upper arm.

This observation underscores the pivotal role of complete smoking cessation as the cornerstone of Buerger's disease management. Abstinence from tobacco not only carries prognostic significance but also exerts a near-causal effect on disease pathogenesis, serving as the most essential and effective therapeutic intervention available.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix

Abbreviations:

TAO	Thromboangiitis Obliterans
ASO	Atherosclerosis Obliterans