

Paraneoplastic Arterial Thrombosis Masquerading as Buerger's Disease: A Case Report and Literature Review

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Abstract

Background: Arterial occlusion as the sentinel manifestation of an occult malignancy is an infrequent but critical clinical occurrence. This case report describes an atypical presentation of acute limb ischemia in a non-smoker, initially mimicking Buerger's disease (Thromboangiitis Obliterans).

Case Presentation: A 46-year-old male fisherman, a lifelong non-smoker, presented with Rutherford Class IIb acute limb Ischemia of the left foot. Physical examination revealed the "5Ps" (pain, pallor, pulselessness, paresthesia, and paralysis) with absent distal pulses. Although the patient's age and distal arterial involvement initially suggested Thromboangiitis Obliterans (Buerger's disease), the absence of tobacco exposure—a mandatory Shionoya diagnostic criterion—prompted a systemic search for malignancy.

Contrast-enhanced computed tomography (CECT) revealed a 4.2 cm pancreatic body tumor with multiple hepatic metastases. Biopsy confirmed Stage IV pancreatic adenocarcinoma. Despite anticoagulation with low-molecular-weight heparin, the prognosis remained poor due to the advanced stage of the malignancy.

Conclusion: This case underscores the necessity of malignancy screening in patients with unexplained arterial occlusion who lack traditional vascular risk factors. It advocates for the inclusion of paraneoplastic syndromes in the differential diagnosis of atypical distal vasculopathy.

Key words: Buerger's disease, arterial thrombosis, paraneoplastic syndrome pancreatic cancer.

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تجلط الشرايين المرتبط بالأورام كقتاع لمرض بورجر: تقرير حالة ومراجعة للأدبيات الطبية

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الملخص

المقدمة: يُعتبر الانسداد الشرياني كعلامة أولية على وجود ورم خبيث غير مُكتشف حدثاً سريريًا نادرًا ولكنه بالغ الأهمية. يصف تقرير الحالة هذا عرضًا غير تقليدي لنقص التروية الحاد في الأطراف لدى فرد غير مدخن، حيث تشابهت أعراضه في البداية مع مرض بورجر (التهاب الأوعية الخثاري المسد).

عرض الحالة: استعرضت العيادة حالة صياد يبلغ من العمر 46 عامًا، غير مدخن، يعاني من ألم حاد في القدم اليسرى مصحوب بشحوب وخدر. أكد الفحص السريري وجود انسداد شرياني حاد (وفقًا للأعراض الخمسة المعروفة 5Ps). وعلى الرغم من أن فنته العمرية وإصابة الأوعية الطرفية قد توحى بمرض بورجر، إلا أن غياب استهلاك التبغ - وهو ركن تشخيصي إلزامي للمرض - استدعى إجراء فحص شامل على مستوى الجسم. التشخيص والتدبير: أظهر التصوير المقطعي المحوسب مع التباين وجود كتلة في جسم البنكرياس بحجم 4.2 سم مع نقائل كبدية متعددة. أسفر التشخيص النهائي عن أنه سرطان البنكرياس الغدي من المرحلة الرابعة، والذي تجلّى عبر تجلط شرياني مرتبط بالورم أحد أشكال متلازمة تروس. على الرغم من بدء العلاج بمضادات التخثر الهيبارين منخفض الوزن الجزيئي، إلا أن التوقعات الطبية ظلت سيئة نظرًا للمرحلة المتقدمة للمرض.

الخلاصة: تؤكد هذه الحالة على ضرورة استقصاء الأورام الخبيثة لدى المرضى الذين يعانون من انسداد شرياني غير مبرر ويفتقرون لعوامل الخطر الوعائية التقليدية. كما تبرز أهمية إدراج المتلازمات المصاحبة للأورام ضمن التشخيص التفريقي لأمراض الأوعية الدموية الطرفية غير النمطية.

الكلمات المفتاحية: مرض بورجر، تجلط الشرايين،

Introduction and literature Review:

Acute arterial occlusion in young adults (under 50 years) typically directs the clinician toward a differential diagnosis of vasculitis, premature atherosclerosis, or Thromboangiitis Obliterans (Buerger's disease). Highly inflammatory, segmental, non-atherosclerotic occlusive lesions of the small and medium-sized arteries and veins in the extremities (1) characterize Buerger's disease. A fundamental requirement for this diagnosis, as outlined in the Shionoya criteria, is a significant history of tobacco use (2). When a patient presents with an identical vascular phenotype but lacks tobacco exposure, the clinician must pivot toward identifying secondary causes, most notably paraneoplastic hypercoagulable states.

The association between malignancy and thromboembolism, colloquially known as Trousseau's syndrome, was first documented in 1865 (3). The relative risk of both ATE and VTE are significantly higher in persons with cancer (4). While venous thromboembolism (VTE) is a well-established complication occurring in up to 20% of cancer patients, arterial thromboembolism events (ATE) are rarer, occurring in approximately 2–5% of patients with advanced solid tumors (5). Among cancer patients, ATE was associated with an increased risk of mortality (6), patients with cancer who develop ATE are at a 3-fold increased risk of mortality (7). The increased risk of arterial thromboembolic events began 5 months before cancer was officially diagnosed and peaked in the month prior (8).

Historically, pancreatic adenocarcinoma is rarely diagnosed in persons younger than 40 years of age, with a median age at diagnosis of 71 years (9). However, this epidemiological landscape is shifting. Recent large-scale studies have identified a troubling new trend: the incidence of pancreatic cancer is rising significantly among younger adults. An analysis of Surveillance, Epidemiology, and End Results (SEER) data from 2000 to 2021 found that among individuals aged 15 to 34 years, the annual percentage change (APC) for pancreatic adenocarcinoma was a striking 4.35% (10, 11). This rate of increase is substantially higher than that seen in older age groups and has been described as a "truly shocking statistic" by clinicians (12). The increase is particularly pronounced in young females, with one study reporting an APC of 9.37% in women aged 18–26 compared to 4.43% in men of the same age group (13). This data challenges the traditional view of pancreatic cancer as exclusively a disease of the elderly and mandates its consideration in younger patients with unexplained symptoms.

This rising incidence is paralleled by an increasing prevalence of modifiable risk factors in the same young adult population.

Conditions like new-onset type 2 diabetes (T2D) are a significant contributor, with studies showing that patients with new-onset T2D have a 3.8- to 5.2-fold higher risk of pancreatic cancer, a risk that escalates to 6- to 8-fold in those over 50 (14, 15).

Obesity, particularly central adiposity, contributes through chronic inflammation and insulin resistance, and excess body weight is now estimated to contribute to nearly 18% of pancreatic cancer cases in the United States (16, 17). Smoking remains a potent risk factor, responsible for approximately 14% of cases, and its role, along with alcohol consumption, is hypothesized to be a key driver of the increasing incidence in younger demographics (11, 12). The interplay of these factors creates a "perfect storm," where a younger patient with undiagnosed pancreatic cancer may present with a thrombotic event, mimicking a primary vascular disorder like Buerger's disease.

Pancreatic adenocarcinoma is uniquely aggressive in its pro-thrombotic potential, with an estimated incidence of thrombosis reaching 36% in some cohorts (18). The literature identifies three primary mechanisms for this:

1. Tissue Factor (TF) Expression: Pancreatic tumor cells release microvesicles (MVs) into the systemic circulation that are heavily enriched with TF, the primary initiator of the coagulation cascade (19).

2. Mucin-Selectin Interactions: Carcinoma-produced mucins can interact with P-selectin and L-selectin on platelets and endothelial cells, leading to the formation of platelet-rich microthrombi without primary vessel wall injury (20).

3. Direct Factor X Activation: The secretion of "cancer procoagulant," a cysteine protease, allows for the direct activation of Factor X, bypassing the traditional clotting pathway (21).

Grilz *et al.* suggests that ATEs in cancer patients carry a significantly higher mortality rate than VTEs, often indicating a terminal or rapidly progressive stage of the underlying malignancy (22). The incidence of cancer-associated thrombosis has increased over time and is associated with an increased risk of mortality for all malignancies (23). While rare, the presentation of pancreatic cancer as acute limb ischemia is a documented, albeit ominous, phenomenon. For instance, case reports have described patients with unusual distal arterial occlusions who were later found to have large pancreatic masses and hepatic metastases, with a rapidly deteriorating course (24, 25). This paraneoplastic presentation can also manifest as paraneoplastic acral vascular syndrome (PAVS), further broadening the vascular phenotype associated with occult malignancy (26).

Case report:

Detailed history and physical examination was taken from the patient after obtaining consent from him. A 46-year-old male, employed as a fisherman, presented to the emergency department with a 48-hour history of sudden-onset, severe pain in his left foot. He described the pain as "stabbing" and reported associated coldness and a "pins and needles" sensation (paresthesia) extending to the ankle. History: The patient was a lifelong non-smoker. His medical history was unremarkable for diabetes mellitus, hypertension, or hyperlipidemia. There was no family history of early-onset cardiovascular disease or thrombophilia.

Physical Examination: Upon presentation, the left foot exhibited significant pallor and poikilothermia (coldness) compared to the contralateral limb. Vascular examination revealed absent dorsalis pedis and posterior tibial pulses, while the popliteal and femoral pulses were palpable. The capillary refill time was delayed (>5 seconds). Sensory testing confirmed a reduction in light touch sensation over the distal phalanges, although motor function (dorsiflexion/plantar flexion) was initially preserved.

Diagnostic Workup:

- **Laboratory Testing:** Routine blood counts and metabolic panels were within normal limits. HbA1c and lipid profiles ruled out metabolic syndrome and premature atherosclerosis.

- **Hypercoagulability Screen:** Tests for Protein C, Protein S, Antithrombin III, and Factor V Leiden were negative. ANA and ANCA titers were within normal limits.

- **Imaging:** Doppler ultrasound of the lower extremities showed no flow in the anterior and posterior tibial arteries. CT Angiography (CTA) confirmed an abrupt "cutoff" of the distal tibial vessels with minimal collateral formation, a pattern often seen in Buerger's disease.

- **Echocardiography:** A transthoracic echocardiogram was negative for intracardiac thrombi, vegetation's, or atrial myxoma. The absence of a smoking history in a 46-year-old with "distal-type" occlusion was a significant clinical incongruity. Consequently, a Contrast-Enhanced Computed Tomography (CECT) of the chest, abdomen, and pelvis was performed to investigate for an occult visceral source. Final Diagnosis: The CECT of the abdomen revealed an ill-defined, 4.2cm hypo-attenuating mass in the body of the pancreas, involving the peripancreatic fat. Multiple "target-like" lesions were observed throughout the liver parenchyma, consistent with hepatic metastases. ACT-guided core needle biopsy of a liver lesion confirmed metastatic pancreatic adenocarcinoma.

- The final diagnosis by biopsy was Stage IV Pancreatic Adenocarcinoma presenting with paraneoplastic arterial thrombosis (Trousseau's syndrome variant).



Figure 1: Early signs of ischemia



Figure 2: Signs of ischemic: color change after one month of assessment

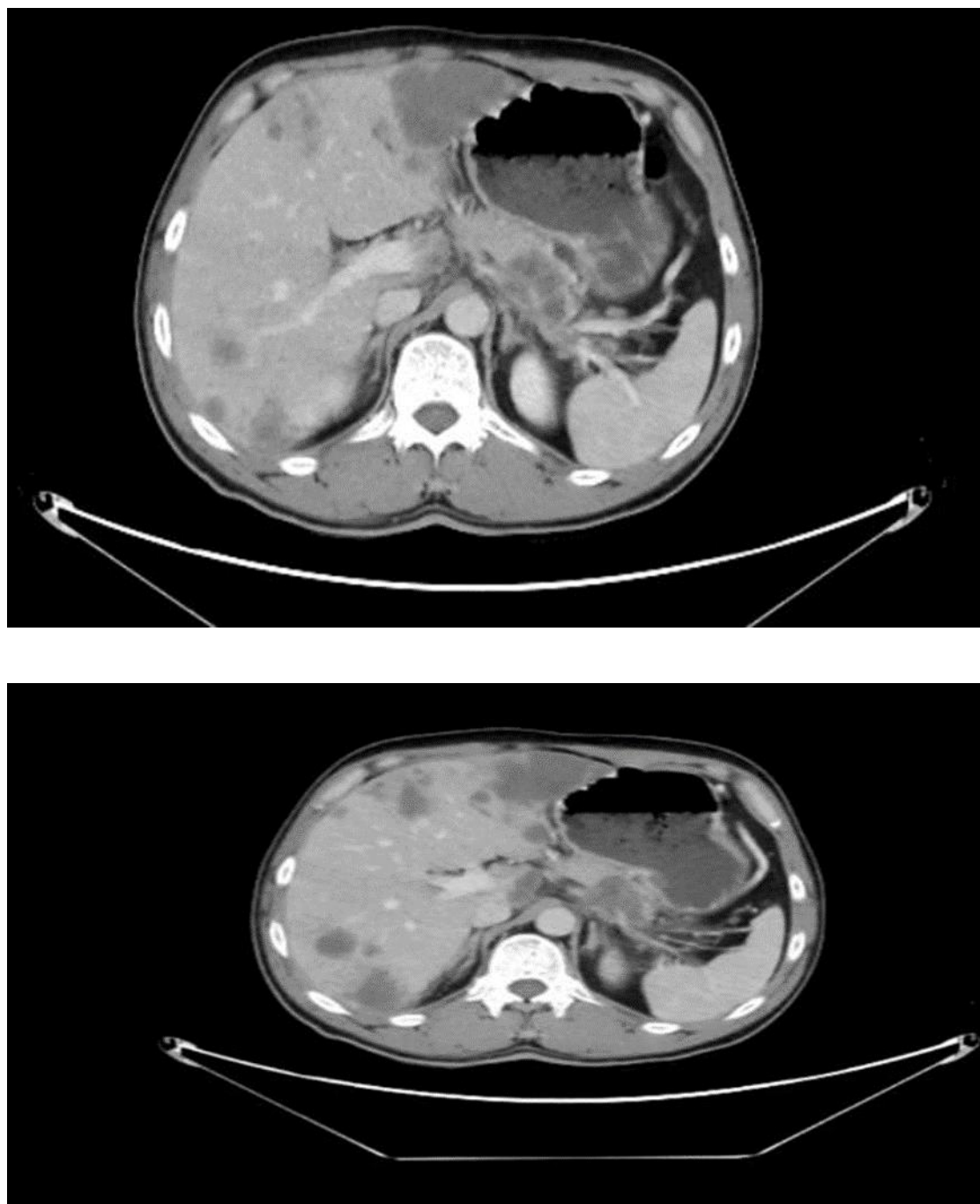


Figure 3&4: Pancreatic mass in the body of pancreases with hepatic metastasis as shown on the arterial phase of abdominal CT scan with contras.

Discussion:

This case underscores the profound diagnostic complexity inherent in acute limb ischemia when it occurs in the absence of traditional cardiovascular risk factors. While Thromboangiitis Obliterans (Buerger's disease) is frequently the initial suspicion for distal arterial occlusions in patients under 50 years of age, such a diagnosis demands very careful examination in a patient who does not smoke. The well-established rule by Olin remains foundational: a diagnosis of Buerger's disease must be actively questioned and is likely untenable in any patient without a current or historical exposure to tobacco (1). When this key social history is absent, the clinical paradigm must shift dramatically away from primary vasculitis towards a systematic search for secondary, systemic etiologies, with occult malignancy at the forefront of differential considerations.

The manifestation of pancreatic adenocarcinoma as a catastrophic arterial thrombotic event represents a severe paraneoplastic syndrome. Unlike the gradual, plaque-based pathophysiology of atherosclerosis, which favors arterial bifurcations and is characterized by vessel wall calcification and remodeling, paraneoplastic thrombosis can occur abruptly in otherwise angiographically normal vessel segments (5, 24). In this patient, the systemic release of procoagulant factors—primarily tissue factor (TF) and cancer-associated mucins—from the undiagnosed tumor created a potent hypercoagulable condition. This environment predisposes to spontaneous "*in-situ*" thrombosis of distal tibial and digital arteries, mimicking the clinical picture of a distal vasculitis (19, 20).

The prothrombotic state in pancreatic cancer is multifactorial, driven by direct tumor cell expression of TF, the shedding of TF-rich microparticles into the circulation, and mucin-mediated interactions with selections on platelets and endothelium, which promote microvascular thrombosis and platelet aggregation (21, 25).

This case also forces a re-examination of the typical demographic for pancreatic cancer. Historically considered a disease of the elderly, with a median age at diagnosis of 71 years (9), our 46-year-old patient aligns with a disturbing new epidemiological trend. Recent analyses of large datasets, such as the SEER database, have revealed a significant rise in pancreatic adenocarcinoma among younger adults. Studies from 2024 and 2025 report an annual percentage increase of over 4% in individuals under 35, with the rise being particularly steep in young women (10, 13).

This increase is not occurring in a vacuum but parallels a rising prevalence of key risk factors in the same population, including new-onset diabetes, obesity, and smoking (14, 15).

Clinicians must therefore be aware that the absence of "old age" can no longer be used to dismiss the possibility of pancreatic cancer in a young adult with unexplained arterial thrombosis. As one commentary noted, this data is a call to action for increased awareness and research into the causes driving this shift (12).

Management of cancer-associated arterial thrombosis (CAAT) is notoriously difficult and often disheartening. Vitamin K antagonists (e.g., warfarin) have historically proven ineffective, as the underlying procoagulant stimulus bypasses or overwhelms the traditional vitamin-dependent pathway (27). Low-molecular-weight heparin (LMWH) has remained the therapeutic cornerstone for cancer-associated thrombosis (CAT) for over two decades, not only for its more reliable anticoagulant effect but also due to proposed modest anti-tumor and anti-metastatic properties, potentially through inhibition of mucin-selectin interactions and other mechanisms (28, 29).

However, even with optimal anticoagulation, revascularization efforts (surgical or endovascular) in these patients are frequently unsuccessful. The reason is fundamental: the relentless systemic stimulus for thrombosis—the active malignancy—persists unabated, leading to prohibitively high rates of early re-occlusion and procedural complications (30). The prognostic implication of such an event is grave; the development of arterial thrombosis in a cancer patient is associated with a significantly increased risk of mortality, often indicating a terminal or rapidly progressive stage of the underlying disease (7, 22).

This case powerfully advocates for the adoption of a strict "red flag" protocol in vascular medicine. When a patient presents with a vasculitis-mimicking picture (young age, distal, segmental occlusions) but lacks the requisite exposure history (tobacco), an aggressive and expeditious investigation for an occult malignancy is not merely advisable—it is mandatory. The diagnostic workup must extend beyond routine limb imaging. Contemporary practice should incorporate comprehensive cross-sectional imaging of the chest, abdomen, and pelvis with contrast-enhanced CT. Given the particular association with pancreatic malignancy and the limitations of CT for small lesions, endoscopic ultrasound (EUS) has emerged as a superior modality for detecting occult pancreatic tumors and obtaining histologic confirmation (31, 32). Biomarkers such as a markedly elevated D-dimer disproportionate to the ischemic event and cancer antigen CA 19-9 can provide supportive clues, though they lack specificity alone (33).

The management challenges have evolved but not diminished. While direct oral anticoagulants (DOACs) have revolutionized treatment for cancer-associated venous thromboembolism, their role in arterial thrombosis remains off-label and inadequately studied, particularly in the context of gastrointestinal cancers with high bleeding risk.

Current international guidelines, including those from the International Initiative on Thrombosis and Cancer (ITAC) and ASCO, continue to recommend LMWH as first-line therapy for complex CAT, which includes arterial events (34, 35). Ultimately, the only strategy with the potential to alter the devastating thrombotic diathesis is rapid initiation of systemic anticancer therapy. For pancreatic adenocarcinoma, regimens such as FOLFIRINOX or gemcitabine/nab-paclitaxel constitute the most critical intervention. Given the exceedingly poor prognosis associated with CAAT, early integration of palliative care is essential to align medical interventions with realistic goals of care and to manage symptoms effectively (36).

Conclusion:

Acute limb ischemia in a young non-smoker requires doctors to make a crucial and urgent diagnostic choice. It should trigger a high-index of suspicion for a paraneoplastic syndrome, with pancreatic adenocarcinoma often being the cause. The diagnostic journey requires a proactive, multimodal approach to uncover the occult malignancy. Management requires a team of specialists. They must balance ineffective attempts to save the limb with systemic anticoagulation and the urgent need for cancer treatment, all while offering compassionate palliative support. This case reinforces that the vascular system can be the first warning sign of a hidden, aggressive malignancy paints its initial, life-threatening presentation.

Consent:

Written informed consent was obtained from the patient for publication and any accompanying images.

Ethical Approval:

Faculty of medicine and health science - Hadhramout university Ethics committee, provided ethical approval for this case. Written Informed consent was obtained from the patient for publication of this case and accompanying images. The patient and their family were informed clearly that no additional benefits will be given to them when agreement participate in this research. In addition, no harm will be caused to them in case of refusing participation.

Author Contributions:

All authors contributed to patient care and writing and revising the manuscript and figures. All authors contributed to the article and approved the submitted version.

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Declaration of competing interest:

The authors declare no conflict of interest regarding publication of this case report.

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