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From a purple digit to left atrial appendage thrombus: the critical role of point-of-care transesophageal echocardiography in the emergency department: a case report

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Abstract

Digital ischemia may be the initial manifestation of Left Atrial Appendage (LAA) thrombosis, even in the absence of atrial fibrillation. We report the case of a 79-year-old woman presenting to the Emergency Department (ED) with transient right-hand hyposthenia, paresthesia, and cyanosis ("purple digit"). The patient had known mitral stenosis and regurgitation but no history of atrial fibrillation. Although Transthoracic Echocardiography (TTE) identified MS and atrial dilatation, it failed to visualize the embolic source, a focused bedside Transesophageal Echocardiography (TEE) performed by emergency physicians revealed a large thrombus within the LAA. This finding identified the cardioembolic source for both her digital and concomitant Chronic cerebral ischemic events finding in brain computed tomography. Anticoagulation was promptly started after TEE performed leading to clinical improvement. This case underscores that in patients with structural heart disease, early TEE in the ED is essential to detect occult embolic sources missed by TTE and to guide appropriate stroke-prevention therapy.

Key words: left atrial appendage thrombus; transesophageal echocardiography; transthoracic echocardiography; emergency department.

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Introduction

We present a clinical case of digital ischemia as the initial manifestation of Left Atrial Appendage (LAA) thrombosis in a patient with mitral valve stenosis and regurgitation, who was awaiting surgical valve replacement, with no prior or subsequent diagnosis of atrial fibrillation.

Case Report

A 79-year-old Caucasian woman presented to the Emergency Department (ED) with right-hand fingers hyposthenia and paresthesia which had begun about 3 hours before admission completely resolved upon presentation to the ED

Past medical history included systemic arterial hypertension, hypertensive heart disease, and known fibrocalcific degeneration of the mitral annulus with moderate-to-severe mitral stenosis-regurgitation. The patient was in therapy with beta-blockers, ACE inhibitors and diuretics. Upon admission, the patient showed cyanosis of the distal palmar region of the third digit of the right hand and complete improvement of the hyposthenia and paresthesia. No other deficits were detected on physical examination; specifically, the neurological examination of cranial and peripheral nerve was normal and the cardiopulmonary examination was normal except for a holosystolic murmur audible over all cardiac areas. During the initial evaluation in the ED, the differential diagnosis focused on ruling out local causes such as distal arterial thrombosis or rheumatologic conditions (*e.g.* Raynaud's phenomenon). Given her cardiac history, there was a strong suspicion of a cardioembolic event; therefore, continuous cardiac monitoring was initiated due to the high likelihood of atrial fibrillation.

Arterial blood gas analysis, ECG and blood test were within normal limits. X-ray of the right hand showed diffuse arthritic changes in the proximal and distal interphalangeal joints.

A vascular surgeon was consulted and documented a triphasic radial pulse on Doppler ultrasound, with preserved flow in proximal interdigital arteries.

Transthoracic Echocardiography (TTE) performed by the cardiologist consultant showed normal left ventricular global and segmental wall motion and confirmed the presence of moderate stenosis and severe regurgitation (valve area 1,21 cm^2 calculated with the value of PHT (182 msec), PASP 32 mmHg, mean gradient 7,6 mmHg) with left atrial dilation Left Atrial diastolic Volume (LADV) 217 ml, Left Atrial Systolic Volume (LASV) 121 ml, Left Atrial Volume Index 120 ml/m^2 (LAVI), Left Atrial Empty Fraction 44% (NV >45%).[1] Brain CT showed a right temporal lacunar lesion and suspected right cerebellar hemispheric ischemia reported both like a chronic lesion by a radiologist.

Considering non diagnostic evaluation and severity of mitral disease the physician decided to observe the patient in HDU (High Dependency Unit) to monitor cardiac rhythm and to extend clinical observation.

Given the non-diagnostic TTE and the high clinical suspicion of a cardioembolic source, a focused bedside Transesophageal Echocardiography (TEE) was performed by the emergency physi-

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cians in HDU. The exam revealed a large thrombus within the LAA (Figure 1) with flow velocities at the lower normal limit (Figure 2). Intravenous Unfractionated Heparin (UFH) was promptly initiated rather than low-molecular-weight heparin (LMWH) due to its short half-life and rapid reversibility. In the Emergency Department setting, this strategy was chosen to maintain therapeutic flexibility. UFH was selected to facilitate surgical synergy and streamline patient management within the interdisciplinary team. Further inpatient presurgery workup included coronary CT angiography showing normal coronary anatomy and CT angiography of the thoracoabdominal aorta, revealing diffuse atherosclerosis. The patient was transitioned to warfarin for long-term anticoagulation and discharged with a referral for cardiothoracic surgery consultation to evaluate for mitral valve repair. The patient was discharged after a 15-day hospital stay with anticoagulation therapy continued using warfarin. Clinically, there was an improvement in the paresthesia and local cyanosis. During hospitalization, she underwent all the necessary preoperative assessments, and approximately four months later she underwent successful transcatheter mitral valve implantation. At the time of consent acquisition, about two months after the procedure, the patient reported clinical improvement with no peripheral symptoms.

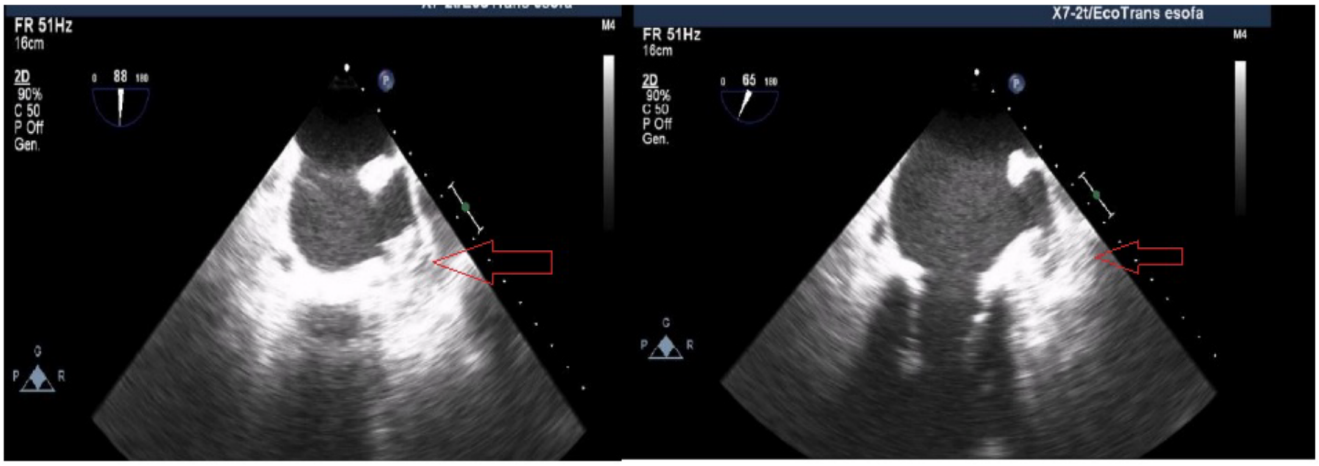


Figure 1. Left atrial appendage indicated by the arrow thrombosis on TEE view.

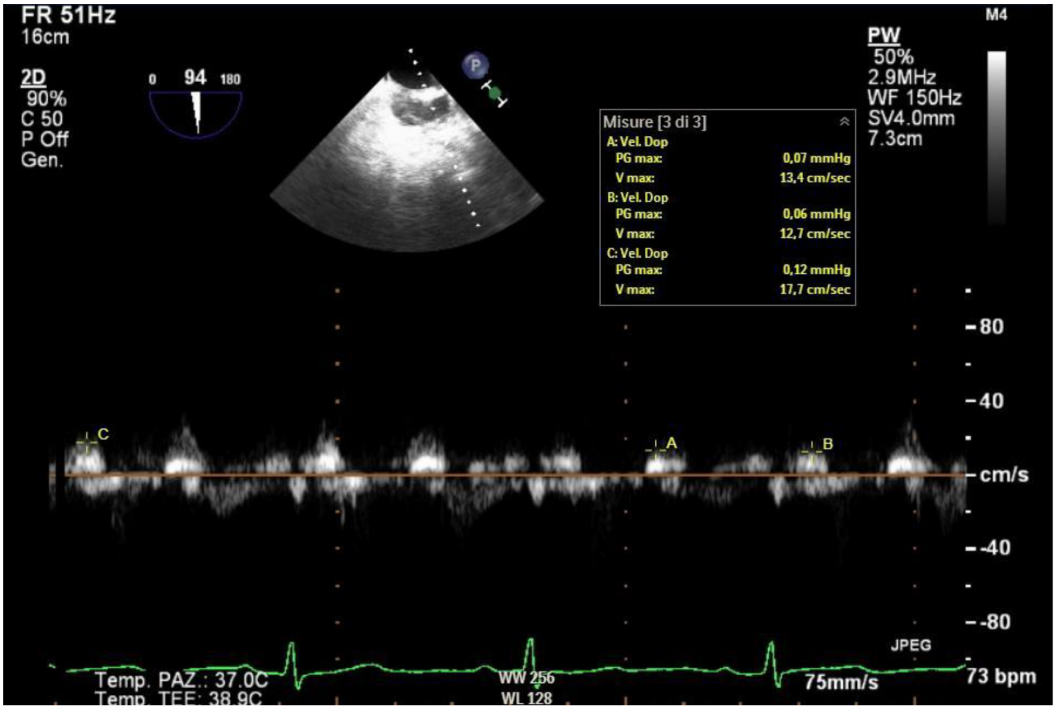


Figure 2. PW Doppler on Left Atrial appendage demonstrated markedly reduced emptying flow velocities (< 20 cm/s).

Discussion

In patients presenting with acute peripheral ischemia and structural heart disease, even in the absence of atrial fibrillation, it is essential to organize a rapid diagnostic pathway in the ED incorporating transesophageal echocardiography, to appropriately guide therapy and improve patient survival while avoiding complications.

This case highlights the importance of considering a cardiac embolic source in patients with acute distal ischemia even in the absence of a known history of embolic heart disease. In the ED, TEE allows for more accurate assessment of anatomical structures such as the LAA and valves compared to TTE.

TEE thus proves to be a key diagnostic tool in the evaluation of potential occult embolic sources including infective endocarditis. Given the rapid learning curve of TEE as a point-of-care technique, its low complication rate and fast execution it may be beneficial to incorporate TEE early in the diagnostic pathway in the ED.[2] We therefore suggest the use of TEE as a POCUS tool to answer binary clinical questions (simple "yes" or "no" answers). TEE could represent a crucial diagnostic instrument in the emergency department, especially in patients with suspected cardioembolic disease, infective endocarditis, or unexplained hemodynamic instability. Unlike TTE, TEE allows for a more detailed visualization of posterior cardiac structures, such as the left atrial appendage, heart valves, and thoracic aorta, significantly improving sensitivity in the detection of thrombi, vegetations, and embolic sources. Transesophageal Echocardiography (TEE) is currently the gold standard for LAA assessment due to its ability to visualize thrombi, spontaneous echocardiographic contrast ("smoke"), and reduced LAA emptying velocity, all indirect indicators of blood stasis and thrombotic risk.[3] Several TEE views allow for systematic LAA exploration, while the integration with pulsed- and color-Doppler enables the measurement of flow velocities. Values below 20–27 cm/s are associated with significantly increased thrombotic risk.

Notably, recent literature highlights the innovative use of TEE performed by emergency physicians as a focused bedside tool to support patients' assessment.[4]

In a patient with high suspicion for an embolic event, in the absence of atrial fibrillation and clinical or historical signs of peripheral vascular disease, clinical suspicion was redirected

toward a cardiac origin of the embolus. In this clinical scenario we performed a TEE to exclude a left atrial thrombus or other causes of arterial embolism (aortic atheroma, others aortic emboligenic disease). Although TTE is the first-line imaging modality for evaluating cardiac sources of embolism, its sensitivity for detecting LAA thrombi is limited. TEE, thanks to its acoustic window and higher spatial resolution, was crucial in identifying the embolic source in this patient. The finding of an LAA thrombus provided a plausible explanation for both the digital and cerebral ischemic events as diagnosed by imaging tests.

The absence of a known history of atrial fibrillation or flutter raises questions about the thrombus formation mechanism. The presence of "atrial cardiomyopathy" could be hypothesized in this patient, characterized by atrial myocardial fibrosis and LA dilation, which could cause thrombus formation and following embolic events. Fibrosis occurs due to collagen deposition, leading to structural and electrical remodeling of the atrium.

In a large community-based cohort, including only subjects without AF or prior stroke, Larsen *et al*. demonstrated that lower left atrium ejections fraction, as measured by TTE, was associated with future ischemic stroke, independently of detected AF, while LA dilation did not.[5] They concluded that reduced contractile function preceded dilation and was an earlier and more sensitive marker of atrial dysfunction.

The link between the LAA and cardiac embolism has been studied almost exclusively in patients with Atrial Fibrillation (AF), while the situation for those in sinus rhythm remains unclear. Whether this phenomenon precedes the onset of arrhythmia, develops concurrently with AF, or persists after cardioversion is still a matter of debate. Furthermore, it is uncertain whether LAA dysfunction arises solely in the presence of AF or if those with a pre-fibrillation status are also at risk, with relevant clinical consequences. In fact, the concept of atrial cardiomyopathy as a clinical entity requiring medical treatment in primary or secondary prevention, needs confirmation in prospective trials.[6]

Despite the presence of significant mitral regurgitation, which represents a protective factor, it is plausible that structural abnormalities of the LAA (the patient had a cauliflower-shaped LAA), mitral stenosis, and low flow velocities within the dilated atrium caused the formation of a thrombus in our patient's LAA.[7]

The prevalence of acute ischemic stroke and sinus rhythm on 12-lead ECG is 6.7% with demonstration of LAA thrombus although paroxysmal atrial fibrillation is identified as the most common underlying cause. The presence of Spontaneous Echo Contrast (SEC) emerged as a potential predictor of LAA thrombus in this patient population.[8]

However, the prevalence of LAA thrombus formation in patients without AF is unclear while the presence of SEC is a potential predictor of ischemic events in particular in patients with mitral valve stenosis.

This highlights that atrial fibrillation is the main but not the only risk factor for LAA thrombosis. Table 1 lists the most important risk factors for LAA thrombosis.

In the presence of clinical suspicion (e.g. acute ischemia of

Table 1. Most important risk factors for LAA thrombosis.

Risk factor	Description/notes
Mitral stenosis	Commonly associated with atrial enlargement and stasys
Aortic stenosis	May contributed to altered intracardiac hemodynamic
Spontaneus echocardiographic contrast (smoke effect)	Marker of blood stasis in the atrium
LAA flow velocity	< 20cm/s Indicated reduced emptying strongly associated with thrombi
Septic shock	Hypercoagulability and endothelial dysfunction
Malignancy	Prothrombotic state due to cancer-related factor
Cardiogenic shock	Severe stasis and reduced cardiac output
Endocarditis	Possible sourcec of thrombus formation or vegetation

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limb, mesenteric ischemia or radiological evidence of prior ischemic stroke) it may be useful to perform TEE to confirm or exclude a cardioembolic source particularly in the presence of the listed risk factors.

TEE should be performed early even in the ED to expedite diagnosis, reduce hospital stay and allow timely admission to an appropriate care setting.

The key message is that digital ischemia may be the first sign of left atrial appendage thrombus even without atrial fibrillation. In such patients with structural heart disease, early transesophageal echocardiography in the emergency department can promptly identify cardioembolic sources and guide life-saving treatment.

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