

Purple finger syndrome: an exceptionally rare complication of infective endocarditis

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A 68-year-old man with a history of gastric cancer resection presented with weight loss, dyspnea, and recurrent fever lasting several months. He had also suffered from 2 episodes of ischemic stroke: 7 months and 1 month before admission. Physical examination revealed mild right-handed hemiparesis and loud pansystolic heart murmur at the apex, radiating to the axilla. Vital signs were normal. Laboratory tests showed mild anemia and slightly elevated levels of inflammatory markers. Transthoracic and transesophageal echocardiography (TEE) revealed severe mitral regurgitation and moderate-sized vegetations attached to the mitral valve leaflets (FIGURE 1A–1C). Infective endocarditis was diagnosed and empirical antibiotic therapy was initiated. The diagnosis was confirmed by positive blood cultures (*Streptococcus sanguinis*). Further diagnostic workup did not reveal cancer relapse. On the third day of hospitalization, sequential neurologic symptoms occurred (dysarthria and left-sided hemiparesis). Three weeks later, the patient complained of pain in his left hand. We observed painful purple edema of almost the whole palmar area (FIGURE 1D). Purple finger syndrome (PFS) was diagnosed and

analgesic treatment was administered. The signs and symptoms in the hand resolved within several days. The antibiotic therapy was followed by mitral valve replacement. During the 3-year follow-up, the patient remained in good condition.

PFS, analogous to blue toe syndrome (BTS), can be defined as sudden-onset, nontraumatic painful skin discoloration, which is usually edematous. Discoloration may vary from purple to blue. A distal pulse is often palpable. Differential diagnosis includes Janeway lesions, which are painless and irregular macules, and Osler nodes, which are painful but, unlike PFS, they are nodular lesions. Janeway lesions are located chiefly on the palms and soles, but Osler nodes are usually found on the pads of the fingers or toes. The mechanism of BTS and PFS is similar but etiology has heterogeneous reasons. One of them is a thromboembolic event. There are a few possible sources of embolic material: atherosclerotic plaques of large-sized arteries¹ and intracardiac masses (eg, left atrial appendage thrombus, vegetations, mural thrombi, and cardiac tumors).² Other uncommon causes are vasoconstrictive, myeloproliferative, infectious, or connective tissue disorders.² It is

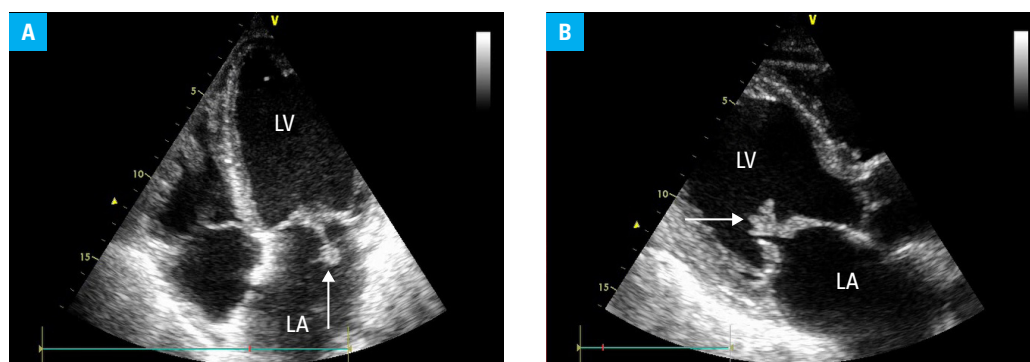


FIGURE 1 Mobile bacterial vegetations attached to the mitral valve leaflets (arrows); transthoracic echocardiography, 4-chamber view (A) and parasternal long-axis view (B). The largest size of the vegetation was 8 × 12 mm. Abbreviations: see on the next page

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Received: July 3, 2018.
Revision accepted: July 20, 2018.
Published online: July 27, 2018.
Conflict of interest: none declared.
Pol Arch Intern Med. 2018;
128 (9): 552-553
doi:10.20452/pamw.43 10
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highly likely that the etiology of PFS in our case

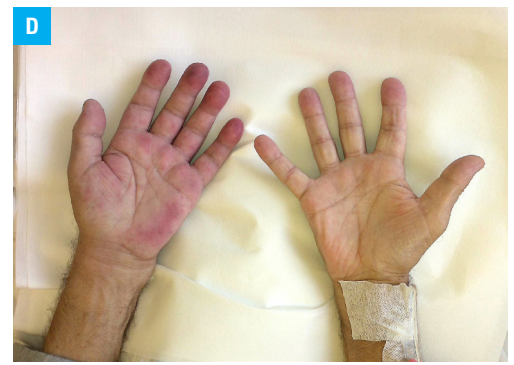
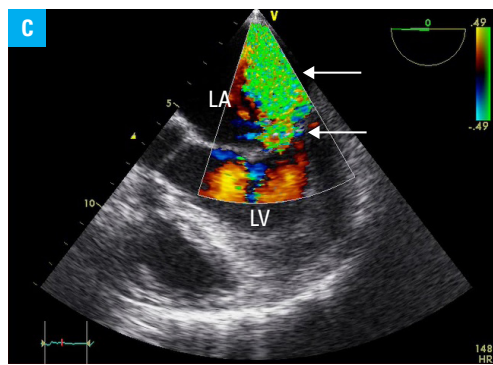


FIGURE 1 **C** – severe mitral regurgitation (arrows) due to infectious damage of the valve (transesophageal echocardiography, mid-esophageal view). The valve was finally replaced by biological prosthesis. **D** – painful purple edema of the palmar area of the left hand, effectively treated with oral tramadol within a few days
Abbreviations: LA, left atrium; LV, left ventricle

was an embolization from dislodged fragments of cardiac vegetations. The patient had no history of atrial tachyarrhythmias. Moreover, repeated TEE examinations excluded appendage clots or other intracardiac masses (except mitral vegetations). Paradoxical embolism was unlikely because atrial septum defect or persistent foramen ovale was not observed. TEE also revealed a normal-sized ascending aorta and an aortic arch without atheromatous plaques. Carotid ultrasound was normal. It is known that intravascular procedures may precipitate plaque disruption.³ However, coronary angiography required for cardiac surgery was performed a week after PFS onset. Angiography of the left upper extremity showed no stenoses or ectasia. In our patient, PFS was preceded by 3 cerebrovascular ischemic episodes. We concluded that PFS and ischemic events shared the same etiology. PFS may accompany a life-threatening condition, hence physicians should be alert to the need for a comprehensive diagnostic workup.^{4,5} According to available literature, this is the first described case of PFS caused by bacterial endocarditis.

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