Dear Editor,

Buerger's disease was first entered into the literature as endarteritis obliterans and later it was identified as obliterating thromboangiitis by Buerger. Buerger's disease is an inflammatory occlusive disease primarily affects the small and medium-sized muscular arteries of the extremities. In the diagnostic criteria of Buerger's disease; smoking, infrapopliteal arterial occlusive lesion, upper extremity vascular involvement or phlebitis migrans, the absence of other risk factors for cardiovascular perspective and a young age can be considered. In this case, we present a patient with a rare seen cardiac involvement in Buerger's disease.

26 years old male patient was first admitted to the hospital with moderate left lower extremity claudication. Buerger diagnosed patient's medical treatment started with pentoxifylline, bencyclane fumarate and acetylsalicylic acid and began to be followed. During his follow-ups the patient was admitted to the emergency service with chest pain and diagnosed with myocardial infarction (MI) 2 times in 1 year. His first angiography showed mid 70% lesion in the LAD artery and subtotal occlusion in the second diagonal branch (Figure 1).

Drug induced stent mounted in the mid region of LAD artery. The patient was discharged with klopidrogel and acetylsalicylic acid treatment. Patient quitted smoking after hospitalization. The patient was incompatible with his follow-ups and 5 months after his first MI the patient was again admitted to the hospital with chest pain and severe left lower extremity claudication. After the first examinations the patient was diagnosed with acute ST-elevation MI and coronary angiography and peripheral angiography was scheduled.

Coronary angiography showed in-stent thrombosis and in the peripheral angiography there was no significant lesions in the abdominal aorta, bilateral iliac, femoral and popliteal arteries but contrast transition to infrapopliteal vascular structures was going through collaterals (Figure 2A,2B). Percutaneous coronary balloon angioplasty was applied to in-stent thrombosis and thrombolytic therapy was applied to the patient. The patient was discharged after the succeed hospital period. Again the patient was incompatible with his follow-ups and 6 months after his last hospitalization he was admitted to the hospital. There were necrotic wounds on the left foot third finger and open sores on the left hand second and third finger (Figure 3).

The upper extremity arterial Doppler was reported as bilateral monophasic flows in the left radial and ulnar arteries. The lower extremity arterial Doppler showed monophasic flow in the left infrapopliteal vascular structures. The patient was scheduled for

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Figure 1. Third finger necrosis

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Geliş tarihi/Received: 13.10.2015 Kabul tarihi/Accepted: 23.11.2015
Contrast enhanced CT. Contrast enhanced CT of the patient showed there was no suitable lesion for surgical intervention. Orthopedics department were included in recommendations. Cilostazol was added to medical treatment and was discharged from the hospital. Burger's disease often occurs between 25-40 years of age in male smokers. Burger's pathological features of the disease have been demonstrated for the first time in 1879. The difference from the atherosclerotic disease was first described by Burger. The most important etiological factor is shown to be smoking.

Figure 2. A. Coronary arterial angiographical image; B: Peripheral angiographical images, normal superficial femoral artery and popliteal artery images; C: Distal peripheral arterial image, corkscrew collateral image.

Genetic factors, hypercoagulability, vascular endothelial structure and immunological mechanisms are also held responsible for the symptoms. Burger's disease symptoms is considered as resting pain in the legs, ischemic ulceration especially in the distal extremities, Raynaud's phenomenon, mobile thrombophlebitis attacks, cold sensitivity. In the diagnostic criteria of Burger's disease; smoking, infrapopliteal arterial occlusive lesion, upper extremity vascular involvement or phlebitis migrans, the absence of other risk factors like diabetes mellitus, hyperlipidemia or hypercoagulability for cardiovascular perspective and a young age can be considered.

Quitting smoking is the major treatment for Burger's disease. Amlodipine or nifedipine as medical support is available for vasospasm. Additionally, antiplatelet agent such as acetylsalicylic acid and other agents as pentoxifylline and cilostazol can be used. Prostaglandin analogues are widely used in non-surgical patients with advance disease. Also sympathectomy or permanent sympathetic blockade treatments are among the alternatives for preventing amputation. Systemic involvement of Burger's disease does not seem very common. Burger patients may have similar fundus findings as hypertensive retinopathy. A study conducted by Arslan and his colleagues showed this retinopathy cannot be associated with hypertension. Mavioğlu and colleagues issued a case of patient with similar symptoms in 2013.

Detailed cardiac examination is important in terms of early diagnosis and treatment changes in Burger's disease. Although rare, Burger's disease may show systemic involvement. Therefore, patients diagnosed with Burger's Diseases must be recognized promptly and detailed systemic examination must be done, for systemic involvement in order to treat the patient in time and to prevent further cardiovascular and systemic diseases.
REFERENCES