Late stent thrombosis after wasp sting

Yabanarısı sokması sonrası geç stent trombozu

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Summary– Myocardial infarction (MI) following a bee sting is a highly unusual reaction. A 65-year-old man allergic to honeybee venom was admitted to the emergency department suffering from a wasp sting with urticaria. The patient had a history of bare metal stent (BMS) 9 months previously with regular drug use. He experienced chest pain after the sting and electrocardiography revealed ST-segment elevation in the chest leads. Subsequently, the patient developed ventricular tachycardia disrupting hemodynamics. Sinus rhythm was obtained by cardioversion. Coronary angiogram revealed total stent thrombosis (ST) in the midportion of the left anterior descending coronary artery. Primary coronary intervention was successfully performed. Presence of shared pathways in allergic reaction and MI pathogenesis may be responsible for *de novo* or ST. To our knowledge, this is the first case of total occlusive late ST in BMS following a wasp sting.

Özet– Arı sokması sonrası miyokart infarktüsü (Mİ) oldukça nadir görülür. Balarısı zehirine karşı alerjisi olduğu bilinen 65 yaşında erkek hasta acil servise yabanarısı sokması sonrası oluşan döküntüler ile başvurdu. Hastaya 9 ay önce çıplak metal stent (BMS) takıldığı ve hastanın antitrombotik ilaçlarını düzenli kullandığı öğrenildi. Hasta takibi sırasında göğüs ağrısı tanımladı. Elektrokardiyografisinde göğüs derivasyonlarında ST-segment yükselmesi gözlendi. Ardından hastada hemodinamiyi bozan ventriküler taşikardi oluştu ve kardiyoversiyon sonrası sinüs ritmi sağlandı. Koroner anjiyografide sol ön inen arter orta segmentinde tam tıkanma yapan stent trombozu (ST) gözlendi. Hedef lezyona başarılı bir şekilde girişim yapıldı. Alerjik reaksiyon ve trombojenik olay gelişmesinde Mİ patogenezlerin benzer olması nedeniyle, alerjik reaksiyonun stent trombozu gelişmesini tetiklediğini düşündük. Bizim bilgimiz dahilinde, bu hasta yabanarısı sokması sonrası gelişen ilk geç BMS ST olgusudur.

The clinical response following wasp sting may be a local reaction (e.g. urticaria), systemic reaction (e.g. anaphylaxis), or an unusual reaction (e.g. myocardial infarction (MI)).[1] To our knowledge, this is the first case involving total occlusive late stent thrombosis (ST) of a bare metal stent (BMS) following a wasp sting.

Abbreviations:

BMS	*Bare metal stent*
LAD	*Left anterior descending*
MI	*Myocardial infarction*
ST	*Stent thrombosis*

CASE REPORT

A 65-year-old man allergic to honeybee venom was admitted to the emergency department after suffering a wasp sting to the face. The patient had no complaint on admission except itchy rashes over the face and chest typical of those seen in urticaria. The patient had a history of hypertension and hypercholesterolemia, and a BMS had been implanted 9 months previously. He had been put on medical therapy consisting of acetylsalicyclic acid, clopidogrel, statin, and angiotensin converting enzyme inhibitor. On admission, his blood pressure was 140/80 mmHg, and heart rate was 85 beats/min with normal sinus rhythm. Cardiac auscultation showed a 2/6 grade systolo-diastolic murmur and the rest of physical examination, including respiratory sounds, was unremarkable. The patient was given an intra-

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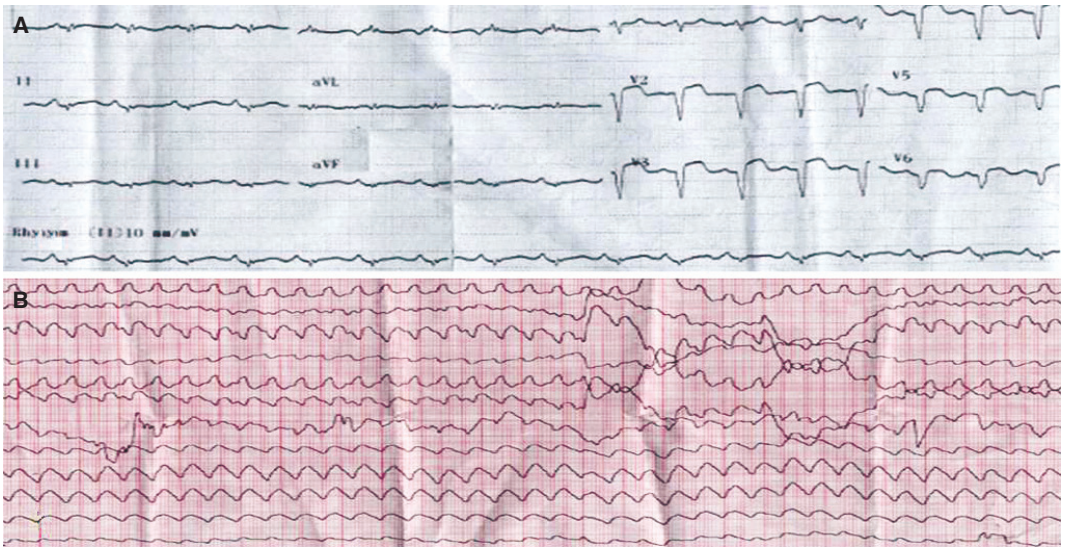

Figure 1. **(A)** Electrocardiogram with ST segment elevation. **(B)** Electrocardiogram with ventricular tachycardia.

venous antihistaminic agent. Since he stated that he experienced chest pain 60 minutes after the sting, electrocardiography was administered, revealing ST-segment elevation in chest leads V2 and V5 (Fig. 1A). Subsequently, the patient developed ventricular tachycardia disrupting the hemodynamics, and sinus rhythm was maintained by cardioversion (Fig. 1B). Coronary angiogram revealed total ST in the midportion of the left anterior descending (LAD) artery. Primary coronary intervention was performed to identify the culprit lesion and TIMI 3 flow was obtained (Fig. 2A, B). Transthoracic echocardiography revealed an ejection fraction of 45%. On the seventh day, he was discharged with medical therapy.

DISCUSSION

To our knownledge, this is the first case of total occlusive late ST of a BMS following a wasp sting. Greif et al. reported acute subtotal ST in a sirolimus-eluting stent after wasp sting.[2]

The pathogenesis of myocardial ischemia developing after an allergic reaction is complicated. The content of hymenopterous venom or the mediators

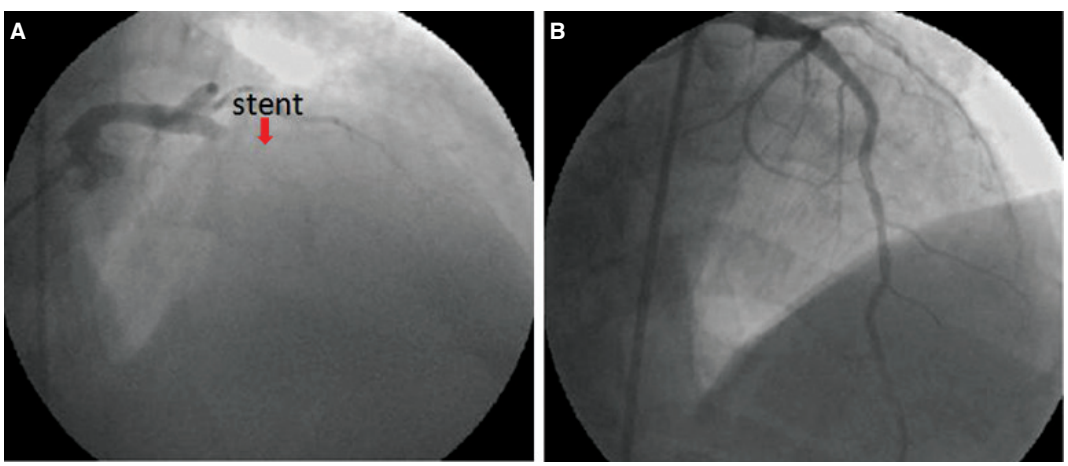

Figure 2. **(A)** Coronary angiogram of left coronary artery in anterior-posterior cranial projection showed stent thrombosis. **(B)** After successfully primary percutaneous coronary intervention TIMI 3 flow was obtained.

released due to those substances (e.g. histamine, serotonin, phospholipase A1, mellitin) have been shown to trigger vasospasm and thrombosis. Allergic reactions are governed by mast cells. The number of mast cells present in the margins of coronary plaques has been shown to be much higher, both during MI and normal homeostasis, in atopic individuals than in nonatopic individuals. In the event of contact with allergen, mast cells secrete sustantial amounts of proteolytic enzymes such as histamine, chymases, and tryptases. These mediators increase the oxygen requirement of the heart by reducing myocardial perfusion pressure or by inotropic and chronotropic effects.[3] In addition, previous studies have shown that wasp venoms contains higher amounts of phospolipase A1 in comparison to honeybee venoms. Yang et al.[4] have demonstrated phospholipase A1 induced platelet aggregation.These substances, and pro-inflammatory cytokines such as TNF-α, are known to degrade the collagen cover of the lipid core of plaques, resulting in plaque rupture.[5]

This is the first reported case of late ST of a BMS following a wasp sting. In this case, we believe that the wasp sting responsible for ST as wasp venoms can trigger thrombosis, and the patient had a history of regular drug use with very little interval between the sting and ST development. In this patient, a stung from a honeybee caused a local allergic reaction only, whereas a sting from a wasp resulted in ST with allergic reaction. This condition may be related to the increased phospolipase A1 content of wasp venoms in comparison to honeybee venoms as phospholipase A1 is known to play an important role in thrombosis.

In conclusion, the presence of shared pathways in allergic reaction and MI pathogenesis may be responsible for *de novo* or ST MI resulting from the triggering of a thrombogenic process following an allergic reaction.

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