doi: 10.5505/abantmedj.2014.46320

Abant Medical Journal

Editöre Mektup / *Letter to the Editor*

*Volume* Cilt 3 *Issue* Sayı 3 *Year* Yıl 2014

# Intracoronary Thrombus with Normal Coronary Flow during Acute Anterior Myocardial Infarction: a case report

## *Akut Ön Duvar Miyokard İnfarktüsü Esnasında Normal Koroner Akımla Birlikte İntrakoroner Trombüs: vaka sunumu*

**Kemal Karaağaç[1], Ali Emul[2], Sezgin Albayrak[3], Fahriye Vatansever[1], Özlem Arıcan Özlük[1], Mustafa Yılmaz[1]**

[1]Yüksek İhtisas Eğitim Araştırma Hastanesi,kardiyoloji Bölümü, Bursa, Türkiye
[2]Şevket Yıılmaz Eğitim Araştırma Hastanesi,kardiyoloji Bölümü, Bursa, Türkiye
[3]Boztepe Devlet Hastanesi, Kardiyoloji Bölümü, Ordu, Türkiye

Acute myocardial infarction (MI) is a clinical syndrome characterized by acute occlusion of a coronary artery caused mainly by the development of a thrombus on the substrate of various types of atherosclerotic (1). We present a case of female patient with evolving anterior acute MI referred for primary percutan transluminal coronary angioplasty (PTCA) who were successfully managed tirofiban infusion therapy after angiographic documentation of an infarct related artery with a large thrombus, normal coronary flow and absence of detectable atherosclerosis in the rest of the coronary tree.

52 years-old women patients presented with newly onset of typical complaint of chest pain and exertional dyspnea to the emergency department. The patient had electrocardiographic evidence of AMI (Figure 1).

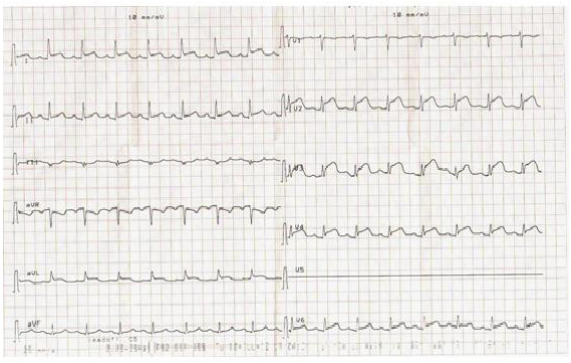

**Figure 1.** Initial 12-lead electrocardiogram performed in the emergency department. ST-elevation in Lead I, aVL and V2 through V6 was concordant with acute anterior MI

Electrocardiography showed 2 mm ST segment elevation in V1-V6 leads. A diagnosis of acute anterior MI was made and she was referred to the cardiac catheterization laboratory for primary coronary angioplasty. Medical treatment was started with 300 mg aspirin, 600 mg clopidogrel, intravenous nitrate infusion and unfractionated heparin infusion. Urgent coronary angiography showed large intracoronary thrombus and normal coronary flow in the infarct related artery (Figure 2).

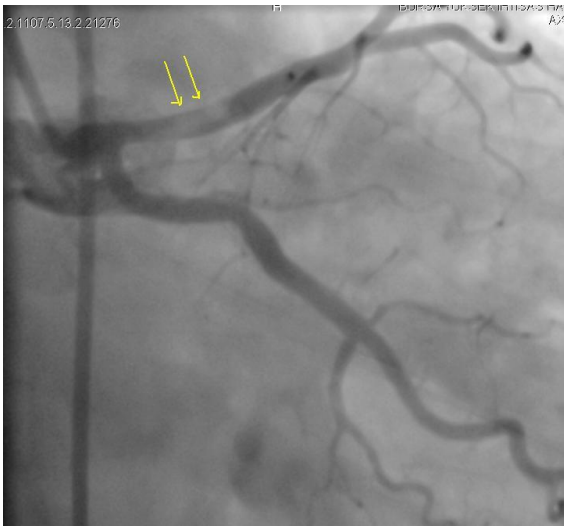

**Figure 2.** Right anterior oblique (RAO) caudal projection. Showing the intraluminal filling defects in the proximal segment of the left anterior descending artery (yellow arrow)

Due to the rejected stenting, percutaneous coronary intervention was not performed and aggressive antiplatelet/anticoagulant treatment was administered (acetylsalicylic acid, clopidogrel, tirofiban and heparin). GlycoproteinIIb/IIIa inhibitör tirofiban as intravenous infusion bolus (10µg/kg/min) was administered and followed by a continuous infusion (0.15/kg/min) administered for 48 hours. In a

**İletişim Bilgisi / Correspondence**
Uzm. Dr. Kemal Karaağaç, 152 Evler Mahallesi, Prof Tezok Caddesi, No: 2 Yıldırım/bursa Bursa - Türkiye
**E-mail:** drkaraagac2001@gmail.com

**Geliş tarihi / *Received*:** 15.04.2013 **Kabul tarihi / Accepted:** 26.06.2013 **Çıkar Çatışması / Conflict of Interest:** Yok / None

case early angiographic control (3 days after MI) showed disappearance of thrombus, no significant residual stenosis and normal flow (Figure 3).

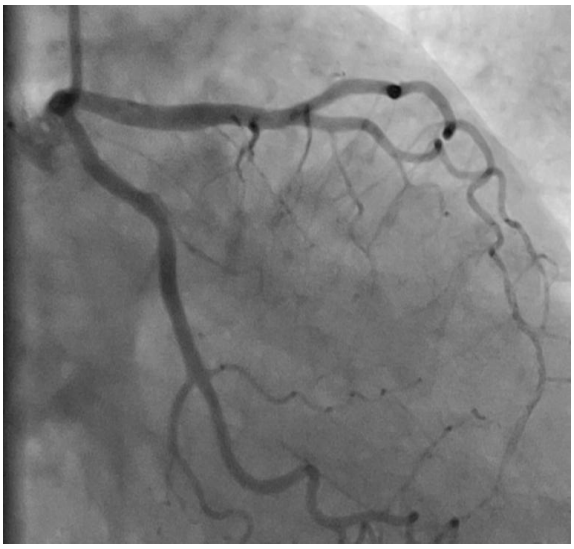

**Figure 3.** Right anterior oblique (RAO) caudal projection. Resolving the intraluminal filling defects after the tirofiban therapy.

Her coagulation factor levels were normal. After seven days and she was followed-up at the outpatient clinic uneventfully. Intracoronary thrombus is accepted as a difficult and mostly encountered problem in acute coronary syndromes. Treatment of intracoronary thrombus by PTCA is associated with complications. The most complications during PTCA are abrupt coronary vessel closure, distal embolization and no-reflow phenomenon (2). The prognosis of patients with MI developing due to intracoronary thrombus has usually adverse outcomes. Even though several anti-platelet drugs, such as thienopyridine and glycoprotein IIb/IIIa inhibitors, have been providing a better outcomes of this lesion (3).

In the present report we describe a relatively rare finding in the setting of urgent coronary angiography for evolving MI: the presence of an angiographic pattern indicative of large intraluminal thrombus (thrombus score 2 or 3) without critical impairment of the anterograde flow (TIMI 2 or 3) in a coronary tree free of detectable atherosclerosis. In this setting the optimal medical or interventional strategy is unknown. Indeed, because of the characteristics of thrombus rich target lesions (4), the use of angioplasty could have led to flow deterioration and impairment of adjacent side branches, as well as to an appreciable restenosis rate. Moreover, when urgent angiography shows good flow in the infarct related artery, a watchful conservative treatment can be adopted (5). On the other hand, because of the dynamic process of thrombus formation and dissolution during acute coronary syndromes (6), the choice of a conservative strategy can increase the risk of deterioration of coronary flow through the lesion in the following hours to days. This may occur especially in patients with a high degree of coronary stenosis even in the presence of TIMI 3 flow (7).

It is reasonable to speculate that the combination of drugs we used facilitated the disappearance of thrombus. In particular the use of glycoprotein IIb/IIIa inhibitors could have exerted a beneficial effect, in agreement with recent data showing that the administration of abciximab reduces the rate of angiographically detectable thrombus in acute MI (8).

Hereby, there is no certain algorithm available for the treatment of coronary artery trombosis. In such situations, as alternative therapy versus percutan coronary intervention, glycoprotein IIb/IIIa receptor antagonist may be an effective agents for trombus resolution.

## References

1. Burzotta F, Hamon M, Sabatier R, Prati F, Boccanelli A, Grollier G. Large intracoronary thrombi with good TIMI flow during acute myocardial infarction:four cases of successful aggressive medical management in patients without angiographically detectable coronary atherosclerosis. Heart. 2002;88(5):e6.
2. Heper G, Yetkin E. Dissolution of massive intracoronary thrombus by medical therapy: case report. Turkiye Klinikleri J Cardiovasc. Sci 2012;24(3):295-8.
3. Arora RR, Platko WP, Bhadwar K, Simpfendorfer C. Role of intracoronary thrombus in acute complications during percutaneous transluminal coronary angioplasty. Cathet Cardiovasc Diagn 1989;16(4):226-9.
4. Topol EJ, Yadav JS. Recognition of the importance of embolization in atherosclerotic vascular disease. Circulation 2000;101(5):570-80.
5. Steg PG, Himbert D, Benamer H, Karrillon G, Boccara A, Aubry P, Juliard JM. Conservative management of patients with acute myocardial infarction and spontaneous

acute patency of the infarct-related artery. Am Heart J 1997;134(2 pt 1):248–52.
6. Davies MJ, Thomas AC. Plaque fissuring: the cause of acute myocardial infarction, sudden ischemic death and crescendo angina. Br Heart J 1985;53(4):363–73
7. Grech ED, Sutton AG, Campbell PG, Ashton VJ, Price DJ, Hall JA, de Belder MA. Reappraising the role of immediate intervention following thrombolytic recanalization in acute myocardial infarction. Am J Cardiol 2000;86(4):400–5.
8. Gibson CM, de Lemos JA, Murphy SA, McCabe CH, Cannon CP, Antman EM, Braunwald E; TIMI Study Group. Combination therapy with abciximab reduces angiographically evident thrombus in acute myocardial infarction. A TIMI 14 substudy. Circulation 2001;103(21):2550–4.