An unusual thrombosis of an ostial left internal mammary artery graft causing acute coronary syndrome five years after coronary bypass surgery

Koroner arter baypas operasyonundan 5 yıl sonra gelişen akut koroner sendrom nedeni sol internal mammarian arterin sıra dışı ostale trombozu

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Summary—For long-term patency, a left internal mammary artery (LIMA) graft is considered the gold standard for left anterior descending artery (LAD) revascularization. Subsequent occlusion of a LIMA graft may be related to atherosclerosis, narrowing at anastomotic locations, fixed torsions within the graft, straight tubular stenosis, dissection, or vasospasm. Late thrombotic occlusion of a LIMA, however, is rare, and the cause is not known, though case reports can be found in the literature. The present case is a description of the successful revascularization via percutaneous intervention of an ostial LIMA thrombotic occlusion occurring 5 years after bypass surgery in a 71-year old patient diagnosed with acute coronary syndrome.

The left internal mammary artery (LIMA) is the first conduit of choice for grafting the left anterior descending artery (LAD). LIMA grafts usually have good short- and long-term patency rates.[1,2] Acute thrombotic occlusion may be associated either with inadequate anti-thrombotic treatment in the early postoperative period or with an inappropriate surgical technique. Late LIMA thrombotic occlusion can develop in a distal anastomotic location or within a distal native artery. Proximal or ostial LIMA stenosis is extremely rare and the precise pathophysiology is unclear.[2,3] Presently described is a case of acute late thrombotic occlusion of the ostial LIMA occurring 5 years after bypass surgery and causing myocardial infarction that was successfully treated with percutaneous intervention (PCI).

CASE REPORT

A 71-year-old patient presented at the emergency department with a typical complaint of intermittent chest pain that had begun 1 day earlier. The physical examination did not reveal any abnormality. The patient gave a history of coronary artery bypass surgery (CABG) 5 years prior involving saphenous vein grafts from the aorta to the right coronary artery, obtuse marginal 1 and 2 sequential anastomosis, and a LIMA graft to the LAD. He was taking ramipril and metoprolol regularly, and acetylsalicylic acid irregularly. There was no history of hyperlipidemia, and
his low-density lipoprotein level was 110 mg/dL. An electrocardiogram showed a sinus rhythm with a diffuse ST-segment depression. The biochemical marker results included a cardiac troponin I level of 50 ng/mL (normal range: 0.0–0.01 ng/mL) and a creatine kinase-MB level of 90.37 ng/mL (normal range: 0–25 ng/mL). Echocardiography revealed an ejection fraction of 35% (with modified Simpson’s method) and hypokinesia of the anterior wall and apex. Coronary and graft angiograms were performed via a left radial arterial approach with the diagnosis of non-ST elevation myocardial infarction (non-STEMI). The saphenous vein grafts were found to be occluded. The right coronary artery contained diffuse stenoses, occlusion was observed in the LAD artery after the first diagonal artery, and 70% stenosis was detected in the obtuse marginal artery. It was also noted that thrombotic occlusion had caused 95% stenosis in the ostial LIMA (Fig. 1a, Video 1*). The patient’s hemodynamic state was stable. A glycoprotein IIb/IIIa blocker tirofiban infusion was administered at a dose of 0.15 μg/kg/minute for 24 hours. The patient was also treated with ticagrelor, enoxaparin, acetylsalicylic acid, ramipril, metoprolol, and atorvastatin. After 3 days, a control angiography was performed using a femoral arterial approach. The thrombus was reduced, but atherosclerotic stenosis was present in the LIMA ostium (Fig. 1b). Subclavian arterial stenosis was also found beyond the LIMA ostium, but medical follow-up was planned for the subclavian stenosis due to the non-critical and asymptomatic status. PCI was planned for the ostial LIMA lesion. A LIMA 6-F guiding catheter was used and a floppy wire was passed through the lesion. A 2.25x18-mm Xience Pro stent (Abbott Vascular, Inc., Santa Clara, CA, USA) was implanted (up to 18 atm directly) (Video 2*). The final LIMA angiogram revealed no residual stenosis, dissection, or thrombus embolization in the distal circulation (Fig. 1c). The patient was discharged after 48 hours without any complications.

**DISCUSSION**

Case reports presenting late thrombotic occlusion of a LIMA graft have been reported in the literature; however, the cause is still unclear. In situ thrombosis, atherosclerotic plaque rupture, localized spasm, embolism, or catheter-induced graft occlusion may cause a late LIMA occlusion.[1,2] The results of a thrombosis panel evaluation, as well as assessment of factor V Leiden and the prothrombin gene mutation were within normal limits in terms of predisposition to thrombosis in our patient. In addition, a sinus rhythm was present on the electrocardiography examination for a cardioembolic event. A follow-up coronary angiography suggested that the thrombus was located in the LIMA ostium with the atherosclerotic plaque. Atherosclerotic stenosis was also observed in the subclavian artery as far as the LIMA ostium, so the

![Figure 1.](image.png)

Figure 1. (A) Angiographic imaging of the thrombotic ostial left internal mammary artery (LIMA) lesion via the left radial arterial approach; (B) Angiographic imaging of the ostial LIMA thrombosis and atherosclerotic lesion after a tirofiban infusion with a right femoral arterial approach; (C) Angiographic imaging of the LIMA ostium after stent implantation.
atherosclerotic process may have progressed from the subclavian artery to the LIMA ostium. However, the atherosclerotic lesion couldn’t be demonstrated, since the intravascular ultrasound catheter could cause disruption of the coronary flow and lead to a distal embolism. There was no evidence indicating an embolic event or localized spasm. Yıldız et al.[4] and Akyüz et al.[5] have reported cases of late mid-region LIMA thrombotic lesions that were successfully treated using stent implantation. Yong et al.[1] reported a case of a patient presenting with a LIMA ostial thrombosis who had a fatal outcome. However, our case presented with non-STEMI, he was hemodynamically stable, and the ostial LIMA thrombotic lesion was treated successfully with PCI. A review of the current literature revealed that a definitive cause of a late LIMA thrombotic occlusion is not known: it might have a different pathophysiology or be a result of the progression of atherosclerosis, but can be successfully treated with PCI.[1,4,5]

Conclusion

A late ostial LIMA thrombotic occlusion is very rare and a conclusive cause remains unknown. We presented an unusual case of a patient who had a history of CABG surgery 5 years earlier who then presented with non-STEMI. The ostial LIMA thrombotic lesion was successfully treated with PCI. As indicated in the literature, PCI with potent antiaggregant therapy may be applied as a successful treatment method for LIMA thrombotic occlusion.

*Supplementary video file associated with this article can be found in the online version of the journal.

Peer-review: Externally peer-reviewed.

Conflict-of-interest: None.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.


REFERENCES

5. Akyüz Ş, Kemaloğlu Öz T, Özer N. Acute thrombosis of the left internal mammary artery graft 14 years after coronary bypass surgery. Anatol J Cardiol 2014;14:301–2. [CrossRef]

Keywords: Late thrombosis; left internal mammary artery; percutaneous intervention.

Anahtar sözcükler: Geç tromboz; sol internal mammarian arter; perkütan girişim.