

Fatal derecruitment of occluded left anterior descending collaterals after left circumflex revascularization



Marouane Boukhris ^{a,b}, Salvatore D. Tomasello ^a, Alfredo R. Galassi ^{a,*}

^a Catheterization Laboratory and Cardiovascular Interventional Unit, Department of Clinical and Experimental Medicine, University of Catania, Catania

^b Faculty of Medicine of Tunis, University Tunis El Manar, Tunis

^a Italy

^b Tunisia

Coronary arteries are not definitely functionally terminal arteries, as previously thought; indeed, they are linked and interconnected by a rich network of collaterals. Chronic total occlusions (CTOs) represent a subset of frequent lesions encountered in everyday catheterization laboratory practice, generally associated with a developed system of collateral connections. These latter have the capacity to prevent myocardial necrosis and may even uphold metabolic supply to the ischemic territory to maintain its contractile capacity. Authors have reported a rapid and progressive reduction of collateral function and their decline after antegrade flow restoration, resulting in higher myocardial susceptibility to ischemia in the CTO territory. Here, we report the case of a fatal derecruitment of collaterals for a left anterior descending CTO not reopened, after left circumflex subocclusion revascularization.

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Introduction

In the presence of stenosis or occlusion, coronary collaterals are able to remodel and to expand in order to insure sufficient supply for the ischemic myocardial territory [1]. Thus, they have a relevant protective role in preserving ventricular function, preventing ventricular dilation, and reducing mortality [2].

The anatomic and functional characteristics of coronary collaterals have been well investigated, particularly in the setting of chronic total occlusions (CTOs), contributing to the development of the percutaneous treatment of such lesions [3–5]. After CTO recanalization and the restoration of an antegrade flow, Zimarino et al. [6] showed the rapid *derecruitment* of collateral circulation, hence increasing the myocardial susceptibility to ischemia.

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* Corresponding author at: Via Antonello da Messina 75, Acicastello, 95021 Catania, Italy.

E-mail address: argalassi@gmail.com (A.R. Galassi).



P.O. Box 2925 Riyadh – 11461KSA

Tel: +966 1 2520088 ext 40151

Fax: +966 1 2520718

Email: sha@sha.org.sa

URL: www.sha.org.sa



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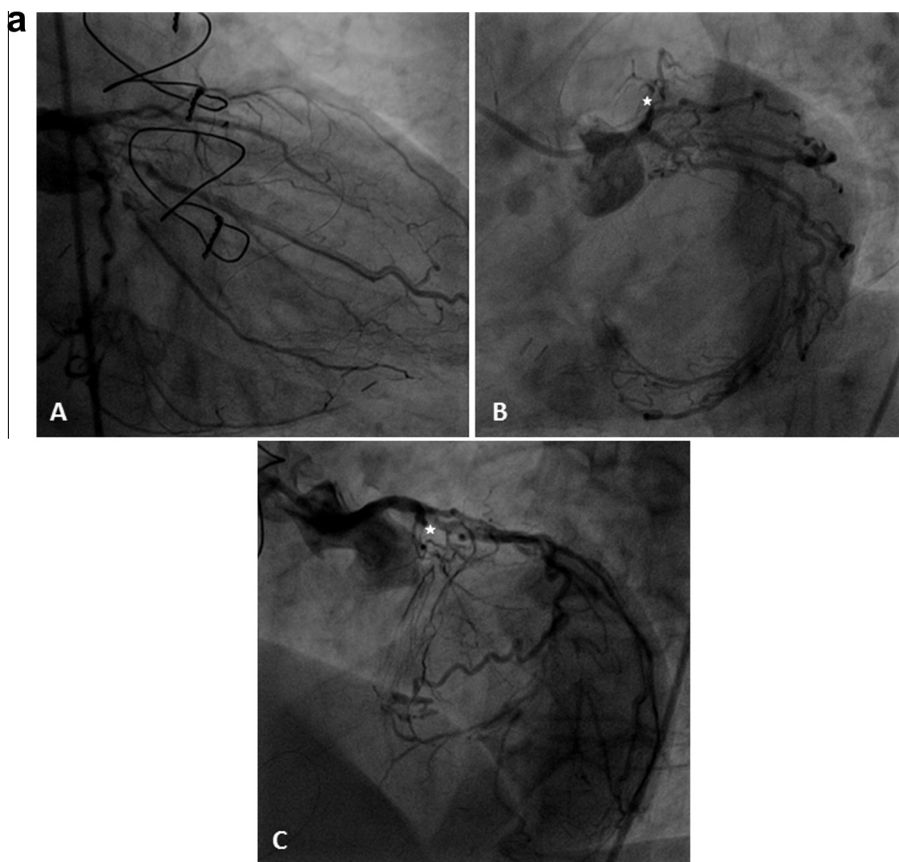


Figure 1. (A) (a) Right caudal view; (b) left caudal view; and (c) left cranial view. Left angiogram showing a proximal chronic total occlusion of LAD with septal and epicardial ipsilateral collaterals, and a co-dominant LCx with subocclusion of the ostium of OM1, OM2, and OM3. (B) Right angiogram showing a subocclusion. (a) Left view and (b) cranial view. Right angiogram showing a subocclusion of the second segment of a RCA giving septal and epicardial collaterals to LAD. (C) Control of coronary artery bypass graftings. (a) Occlusion of saphenous venous graft to RCA; (b) occlusion of left IMA to LAD; and (c) stenosis of the distal anastomosis of right IMA to OM2. IMA = internal mammary artery; LAD = left anterior descending; OM = obtus marginal; RCA = right coronary artery.

We report the case of a fatal derecruitment of an occluded left anterior descending (LAD) collaterals occurring after left circumflex (LCx) subocclusion revascularization.

Case report

A 60 year-old ex-smoker and dyslipidemic male was admitted for dyspnea class III and typical angina class III, according to New York Heart Association and the Canadian Cardiovascular Society classifications, respectively. Three months earlier, he underwent a coronary artery bypass graft intervention for a three-vessel coronary artery disease: left internal mammary artery (IMA) to LAD, right IMA to first obtus marginal (OM) and saphenous venous graft (SVG) to right coronary artery (RCA). No abnormalities were found on his chest X-ray. Transthoracic echocardiography (ECG) was performed, showing a mildly impaired left ejection fraction (50%) with

global hypokinesia. No Q wave was observed in 12-lead ECG. A coronary angiography was then indicated. The left angiogram showed a proximal CTO of LAD with septal and epicardial ipsilateral collaterals from a co-dominant LCx with subocclusion of OM1, OM2, and OM3 (Fig. 1A). The right coronary angiogram revealed a subocclusion of the second segment of a RCA giving a network of collaterals to LAD and LCx (Fig. 1B). The control of coronary artery bypass graftings revealed the occlusion of SVG to RCA, the occlusion of left IMA to LAD, and a critical stenosis at the distal anastomosis of right IMA to OM1 (Fig. 1C). The decision of the local heart team was to opt for percutaneous coronary intervention (PCI) following a two-step strategy: a first procedure with intra-aortic balloon pump support to revascularize RCA and LCx, while the second procedure would be scheduled 1 month later to attempt LAD CTO.

A double 7F femoral access was used. A PCI of the second segment of RCA with one

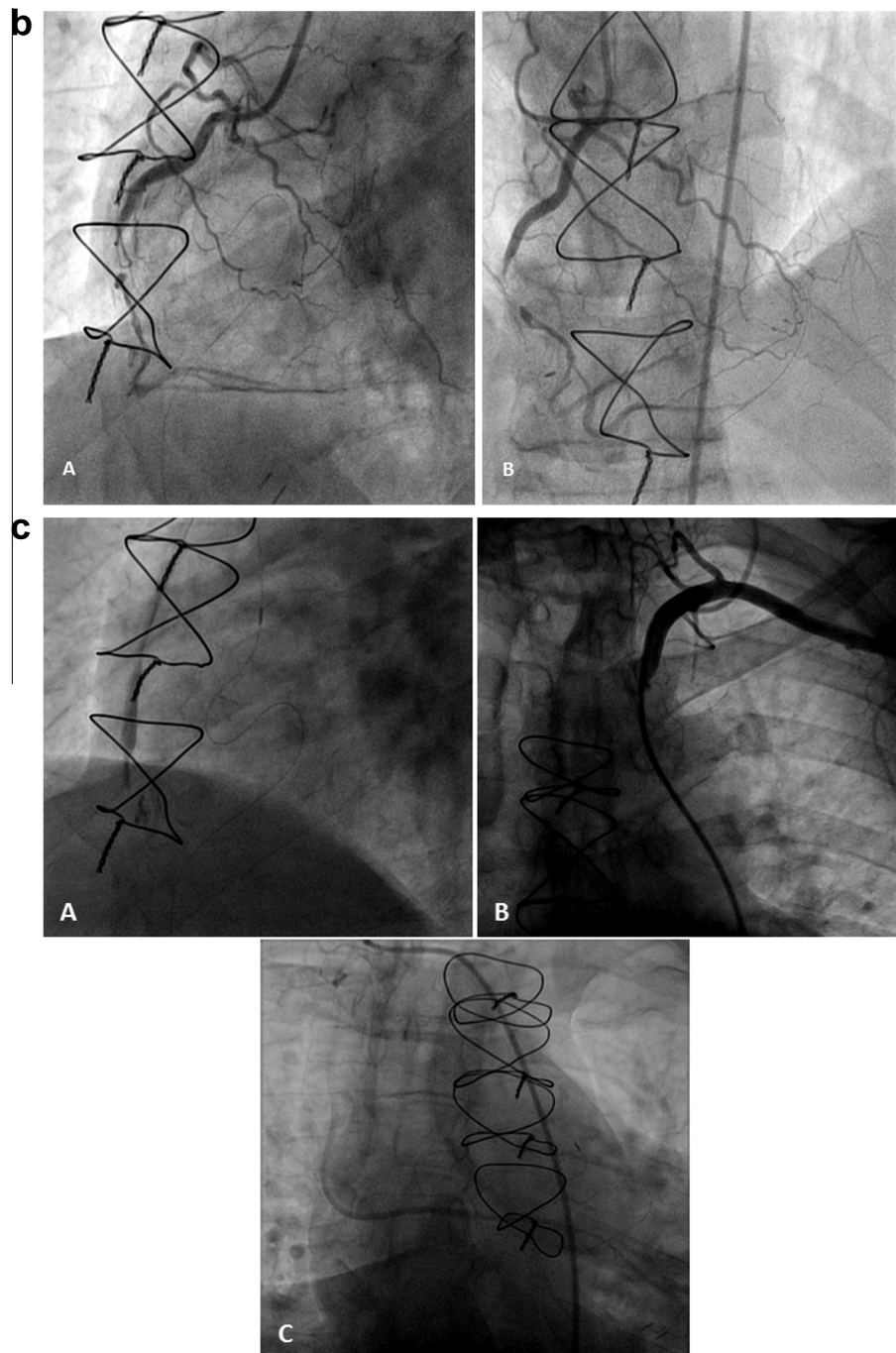


Figure 1. (continued)

drug-eluting stent (DES) was performed with good angiographic result (Fig. 2). Notably, prolonged right angiogram showed an improved flow in the network of collaterals with retrograde filling (Rentrop 3) of LAD via epicardial (CC2) and septal (CC1 and CC2) collaterals, in addition to collaterals to LCx (septal and epicardial).

Then, double-kiss crush technique was employed twice to treat the double bifurcations

OM3–OM2, and OM2–OM1, respectively, and a total of seven DESs were implanted in the LCx. The final angiographic result was satisfactory; although the distal LCx was occluded with a decreased ipsilateral filling of LAD, a final thrombolysis in myocardial infarction flow was achieved in the three OMs and the patient was asymptomatic. Importantly, the patient received intravenous unfractionated heparin (100 IU/kg)

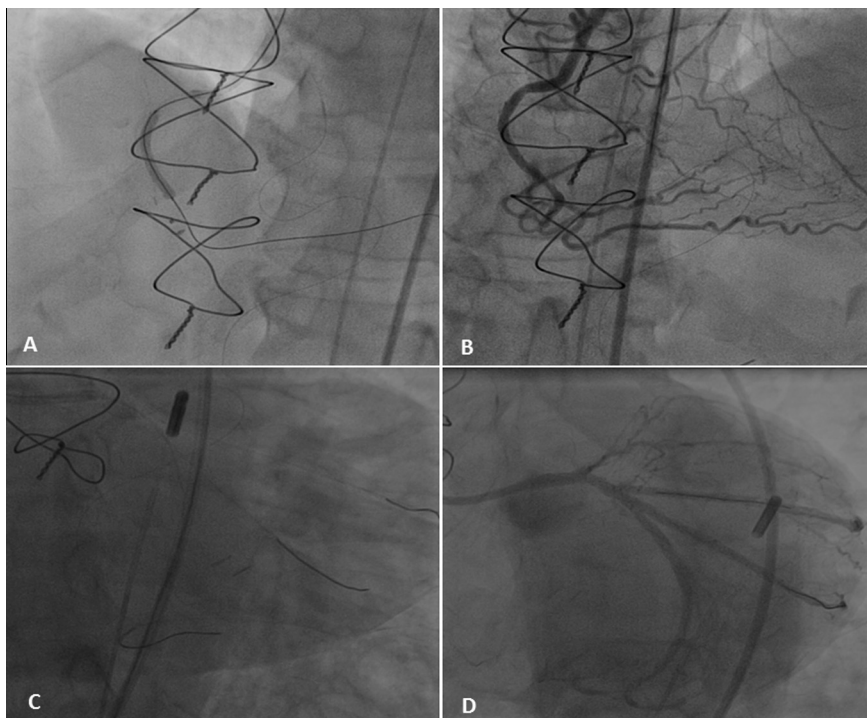


Figure 2. (A) PCI of RCA with one DES implantation; (B) right angiogram after RCA PCI, revealing collaterals (septal and epicardial) for LCx and LAD, with visualization of distal LAD. (C) Positioning of three Fielder FC (Asahi, Japan) in OM1, OM2, and OM3. (D) Left angiogram after DK crush technique in both OM3-OM2 and OM2-OM1 bifurcations, and a total of 7 DES implantation, showing a good angiographic result with collaterals communicating with RCA and septals for LAD. DES = drug eluting stent; DK = double kiss; LAD = left anterior descending; LCx = left circumflex; OM = obtus marginal; PCI = percutaneous coronary intervention; RCA = right coronary artery.

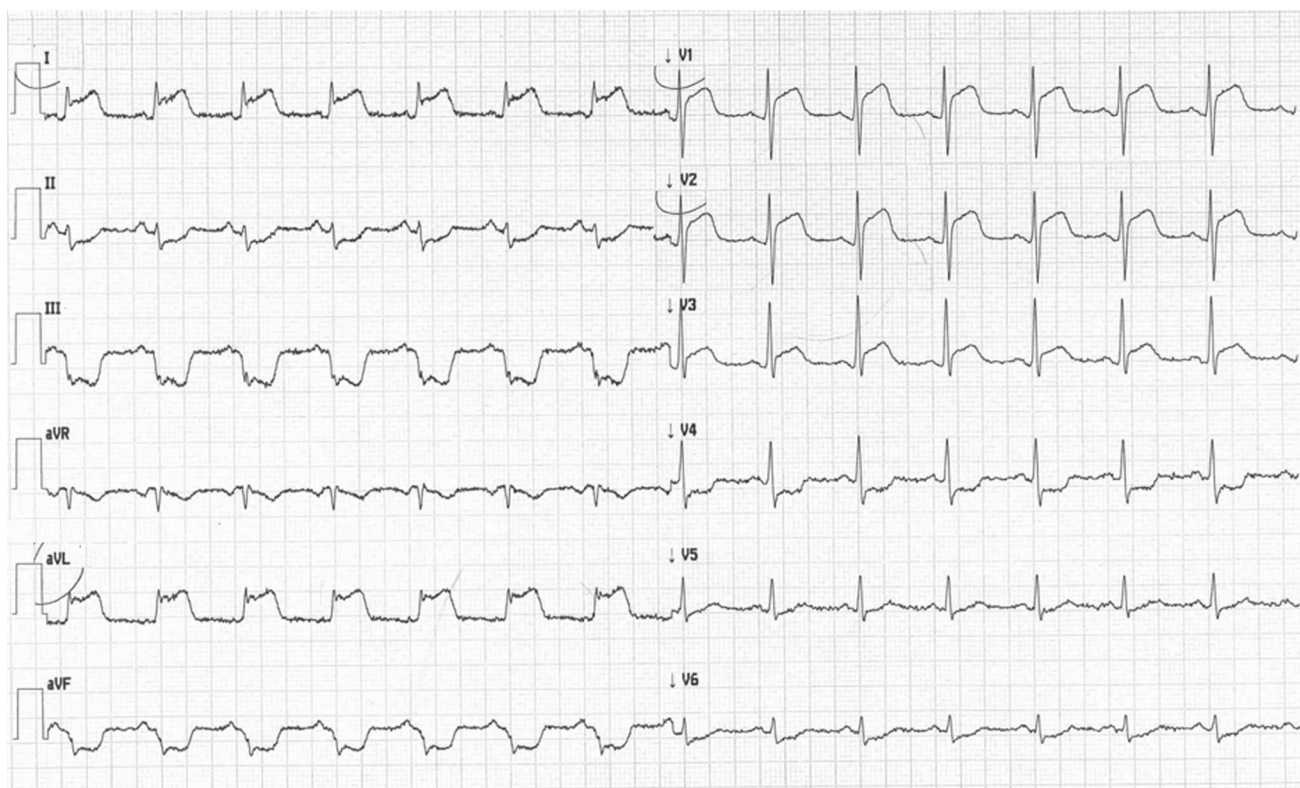


Figure 3. Twelve-lead electrocardiogram, 20 minutes after percutaneous coronary intervention procedure, concomitant with chest pain, showing ST elevation in V1-V3 and D1-aVL with ST depression in V4-V6 and inferior leads.

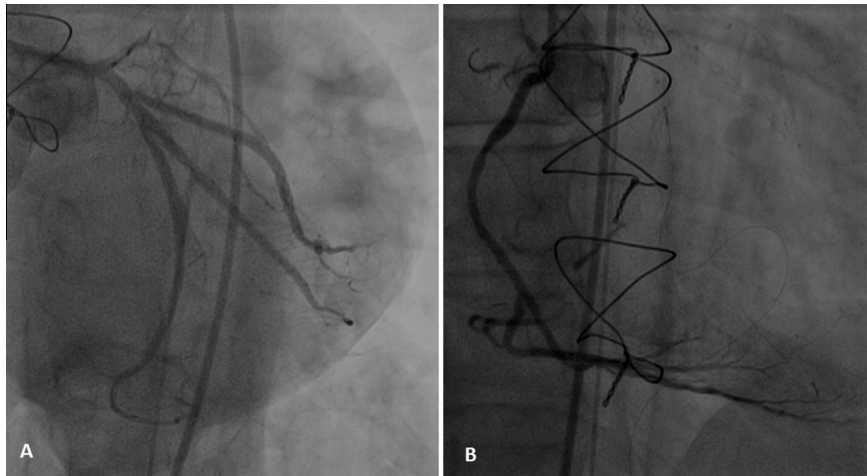


Figure 4. Control coronary angiography showing the absence of stent thrombosis in both (A) left circumflex and (B) right coronary artery with absence of visualization of left anterior descending.

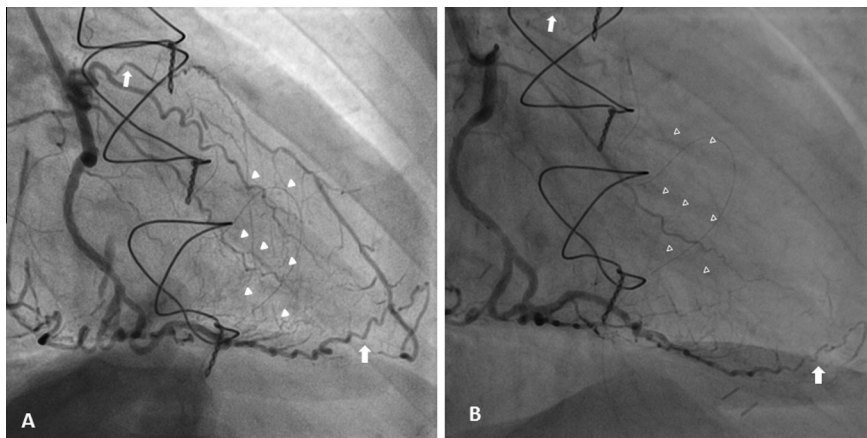


Figure 5. Comparison between right angiograms (right view) between (A) first coronary angiography, (B) after RCA PCI, and (C) at control after chest pain onset. (A) Underestimation of RCA collaterals before RCA PCI due to second segment stenosis resulting in coronary steal. (B) RCA angiogram PCI showing important collaterals (epicardial and septal) for LAD (with distal LAD visualization) and left circumflex. (C) Derecruitment of collaterals originating from RCA with absence of visualization of LAD. LAD = left anterior descending; PCI = percutaneous coronary intervention; RCA = right coronary artery.

to maintain an activated clotting time (ACT) > 300 seconds. The ACT was monitored every 30 minutes throughout the procedure to determine if an additional bolus of unfractionated heparin was necessary; at the end of the procedure, ACT was 335 seconds.

The immediate outcome was good and the patient was transferred to the intensive care unit. Twenty minutes later, he showed an acute constrictive chest pain, and a 12-lead ECG revealed an ST-elevation in V1-V3, D1, and aVL (Fig. 3). Immediate control coronary angiography was performed and did not show any stent thrombosis in both the right and left angiograms (Fig. 4); however, we observed the absence of retrograde filling

of LAD from RCA epicardial and septal collaterals, which showed an important decline as compared with different previous angiograms during the PCI procedure (Figs. 4B and 5).

The patient became hemodynamically unstable with hypotension requiring catecholamines administration, followed by cardiac arrest. After intubation and 45 minutes of unsuccessful resuscitation, the patient was declared dead.

Discussion

In the setting of a CTO, collaterals have the capacity to prevent myocardial necrosis and may even uphold metabolic supply to the territory

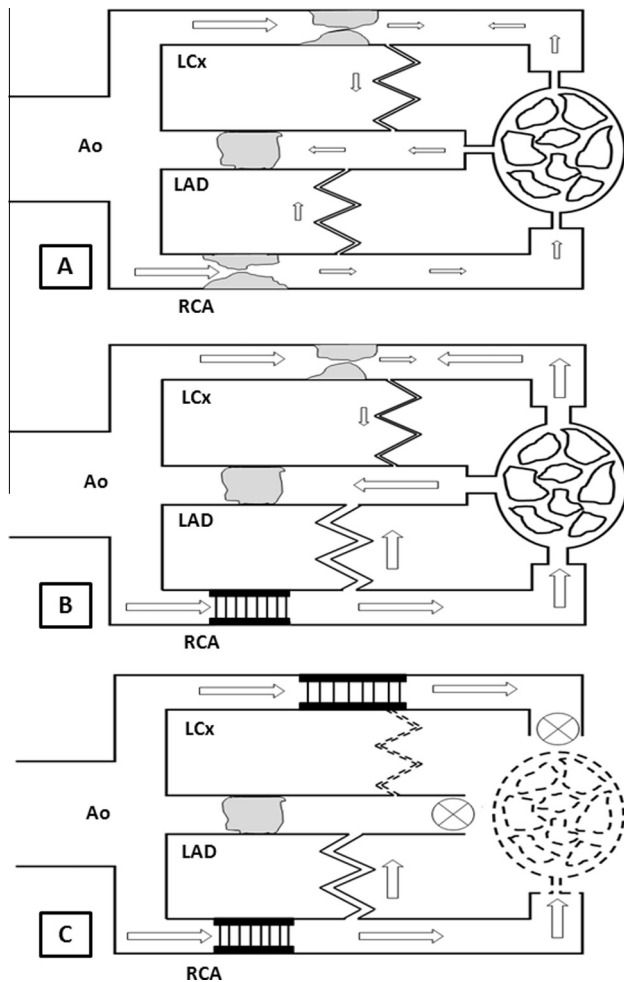


Figure 6. Schematic illustration of the de-recruitment phenomenon observed in our case. (A) Steal phenomenon due to RCA stenosis underestimating the flow in the collateral network to LAD and LCx. (B) After RCA stenting, increase in the collaterals flow to both LAD and Cx. (C) After LCx stenting, the antegrade flow was responsible for the de-recruitment of the network of collaterals for both LCx and occluded LAD, resulting in acute ischemia and infarction in the territory of this latter coronary artery. LAD = left anterior descending; LCx = left circumflex; RCA = right coronary artery.

subtending the occlusion or stenosis, in order to maintain its contractile capacity. After the restoration of antegrade flow with successful PCI of CTO, authors reported a rapid and progressive reduction of collateral function previously supplying the CTO territory [6–8]. The collateral regression is even more evident when the revascularized artery is persistently patent at a 5-month follow-up [9]. Moreover, Zimarino et al. [6] demonstrated a contextual reduction in the tolerance to ischemia, documented in 80% of cases by ECG modifications (ST elevation) and in 60% by angina. The clinical implication of these findings is that previously developed collaterals do not systematically exert a protective effect in case of late reocclusion

(e.g., stent thrombosis) of the target vessel, and their derecruitment increased the myocardial susceptibility to ischemia [10].

However, the ECG modifications and ischemia due to decline of collaterals were reported in the territory of the reopened artery [6]. The originality of our case consists of the fact that LCx revascularization leads not only to the decline of LCx collaterals, but also to the derecruitment of those of LAD. In fact, RCA provided an interconnected network of collaterals to both LCx and LAD, better observed after RCA PCI (Fig. 5). Once the three OMs revascularized, the improvement of their antegrade flow resulted in a derecruitment involving all the network of collaterals originating from RCA, including those supplying LAD territory. Indeed, the ST elevation occurred in the territory of LAD, which was already occluded and not revascularized, resulting in acute hemodynamic failure and cardiac arrest (Fig. 6).

For the first time, we reported such interdependence between the revascularization of a coronary artery and the occurrence of a fatal acute myocardial infarction due to a decline of the collaterals supplying another artery. Such a possible phenomenon might impact on the strategy of revascularization by giving priority to the vessel subtending the largest at risk myocardial territory.

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