

# Cardiac arrest during percutaneous coronary intervention in a patient 'resistant' to clopidogrel – successful 50-minute mechanical chest compression

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## Abstract

We report a case of 72-year-old female patient with end-stage chronic kidney disease, undergoing percutaneous coronary intervention (PCI) that resulted in a cardiac arrest caused by a thrombus mediated flow limitation in the left coronary artery. With mechanical cardiopulmonary resuscitation (CPR) PCI of the left main artery was performed successfully during 50 min cardiac arrest. The patient was discharged from the hospital without compromising cardiac function and neurological deficits.

**Key words:** percutaneous coronary intervention, cardiac arrest, cardiopulmonary resuscitation, thrombolytic therapy.

## Introduction

Ischemic complications of percutaneous coronary intervention (PCI) are infrequent but prognostically important. They are strongly associated with subsequent adverse outcomes [1] but fatal PCI complications are rare and mostly due to left ventricular failure, neurological causes and arrhythmia [2]. Chronic kidney disease (CKD) is associated with a greater risk of coronary artery disease (CAD), which is the leading cause of mortality in this population [3]. This high risk group of patients very often requires revascularization [4]. On the other hand, CKD is associated with increased risk of in-hospital mortality as well as adverse ischemic, hemorrhagic, cardiac and cerebrovascular events during treatment with PCI, as compared to patients without CKD [5–7].

## Case report

A 71-year-old woman with end-stage chronic kidney disease (haemodialysis 3 times a week), with a history of non-ST-elevation myocardial infarction (NSTEMI) treated with PCI of the left main (LM)/left anterior descending (LAD)/circumflex (Cx) artery with two everolimus-eluting stents, with type 2 diabetes, long-standing history of hypertension, and moderate mitral regurgitation, was admitt-

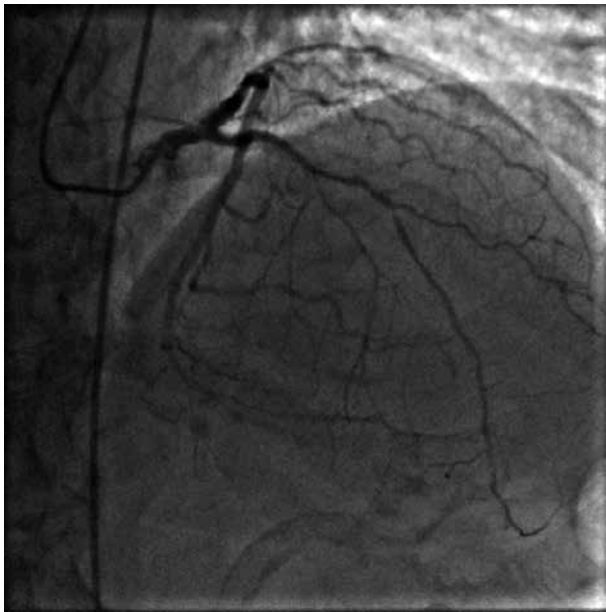
ed to the Cardiology Department with a diagnosis of NSTEMI. The echocardiographic examination performed on admission was comparable to the previous study and demonstrated extensive wall motion abnormalities with decreased global ejection fraction (EF = 35%). The coronary angiography revealed right coronary artery occlusion (as before) and properly functioning stents in the LM and the proximal segments of the LAD and CX. The culprit lesion was a critical narrowing in the medial LAD (Figure 1). As the patient was already receiving aspirin and clopidogrel due to previous myocardial infarction (MI) a decision to perform *ad hoc* PCI was taken. Despite initial balloon predilatation of the stenosis (Trek 2.0 mm × 8 mm, 10 atm, Abbott Vascular), having good support of the guiding catheter, the stent could not be advanced through the ostium of the LAD. During prolonged manipulations formation of thrombus in the LM, proximal LAD and CX segments (ACT time after heparin bolus 70 U/kg equal to 267 s) occurred (Figure 2). The patient developed cardiogenic shock and subsequently cardiac arrest in the mechanism of pulseless electrical activity (PEA). Immediate manual resuscitation was started and after the patient was intubated a Lund University Cardiac Arrest System (LUCAS) device was engaged to continue automatic chest compression. Despite the administration of intracoronary bolus followed

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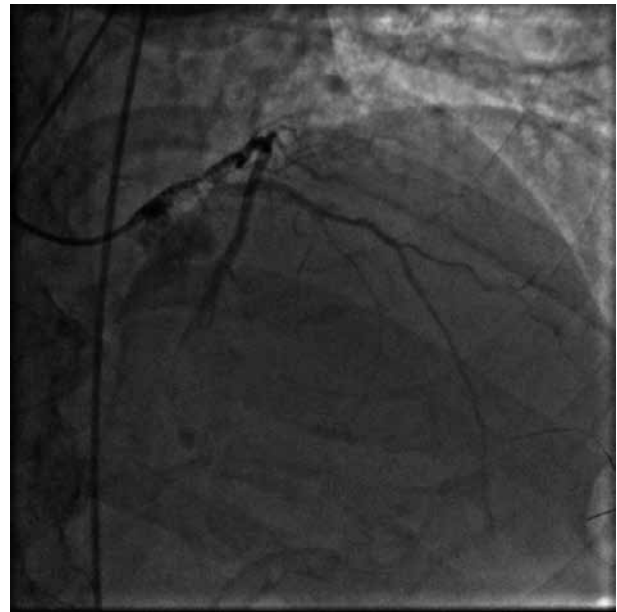
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**Fig. 1.** Angiography of the left coronary artery with properly functioning previously implanted stents



**Fig. 2.** Thrombus in the left main, proximal left anterior descending and circumflex arteries

by intravenous infusion of abciximab along with multiple thrombus aspirations with an Export catheter (Medtronic, USA) the coronary angiogram remained unchanged. Spontaneous circulation had not returned. Despite the potential risk of bleeding, 5 mg of intracoronary alteplase was administered. After a few minutes the thrombus began to dissolve but only slight improvement of the flow was observed. Because of the suspicion of coronary artery dissection, a 4.5 mm × 20 mm stent at 15 atm (Resolute, Medtronic) in the LM/CX was implanted and finally kissing balloon inflation was performed with two 3.0 mm × 20 mm balloons (Sprinter, Medtronic). After the PCI spontaneous return of circulation and TIMI-3 flow in the left coronary artery were observed (Figure 3). The whole PCI lasted over 50 min during which ongoing LUCAS support was continuously used. After the procedure the patient with blood pressure of 160/80 mm Hg and heart rate 110/min on adrenaline and noradrenaline infusion was transferred to the intensive cardiac unit. Two days later the patient was extubated. Because of the double stent layer in left main coronary artery the patient was subjected to genetic examination of the CYP2C19 gene and light transmission aggregometry (LTA) was performed to assess platelet activity. There was no polymorphism within the CYP2C19 gene but the aggregometry test revealed excessive platelet aggregation of 63% after stimulation with 5 µg of ADP. With this result we decided to change antiplatelet therapy to a more potent platelet inhibitor – ticagrelor. This therapy resulted in proper, 40%, platelet aggregation. The echocardiography examination performed before discharge showed a slight improvement in left ventricular systolic function. No neurological deficits were diagnosed. The patient was discharged from the hospital 10 days later.

## Discussion

The described case shows that a patient at high risk, even with an isolated, simple lesion, can develop very serious complications that should always be reckoned with. The management of cardiac arrest during coronary intervention presents a substantial challenge and effective cardiopulmonary resuscitation with chest compressions is the primary method of circulatory support.

There have been some observations in the past in which continuous mechanical chest compression was



**Fig. 3.** Restoration of TIMI3 flow in left coronary artery

used as a bridge to perform a successful PCI procedure during resuscitation efforts [8–10]. Another aspect of the case described is the use of intracoronary thrombolytic, which, despite the potential complications of bleeding, may be the only effective strategy to deal with a massive intracoronary thrombus [11–13]. Although neither method has been reflected yet in the corresponding guidelines, available publications and the case described above demonstrate the effectiveness of these methods in critical situations.

Finally, an optimal antiplatelet therapy is crucial for successful treatment of ischemic heart events. Chronic kidney disease is one of the reasons for inadequate platelet inhibition with clopidogrel [14, 15]. We believe that the cause of the serious complication was mechanical but we should remember that high platelet activity is one of the known causes of ischemic complications after PCI procedures [15]. Prasugrel and ticagrelor, new P2Y12 platelet inhibitors, achieve faster and greater platelet inhibition. In this special, CKD patient, balancing between adequate platelet response and potential bleeding complications, we decided to put our patient on ticagrelor. The drug, in contrast to prasugrel, was shown to reduce major adverse ischaemic events without extensive risk of major bleeding [16]. Fortunately we managed this complication and the patient is alive and not neurologically compromised.

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