

CASE REPORT

Interdisciplinary Approach in a Complex Case of STEMI

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ABSTRACT

We reported the case of a young man with ST-Segment Elevation Myocardial Infarction (STEMI), with ventricular fibrillation on debut and cardiogenic shock, who needed a complex interdisciplinary approach for a favourable long term outcome.

A 43-year-old man was admitted with inferior STEMI and cardiogenic shock. First coronary angiography revealed total chronic occlusion of left anterior descending artery (LAD) and tight stenosis with thrombus on right coronary artery (RCA). Thrombus aspiration and stent implantation on RCA was performed with good results. LAD couldn't be opened. Intraaortic balloon pump was implanted. Forty-eight hours later, we try again to open LAD, without success. After a lot of complications, all solved with difficulty, patient was discharged cachectic and with progressive exertion on mild exercise. Two months later an implantable cardioverter-defibrillator (ICD) was decided for persistent ventricular tachycardia and after one year he was referred to a cardiac surgery centre abroad for aneurismectomy with left ventricle (LV) reconstruction and mitral valve repair. The patient is currently asymptomatic with a normal social and professional life.

In conclusion, high performance cardiac surgery, after a complete interventional treatment, can improve quality of life and long-term outcome to a patient with severe cardiovascular disease.

Team work between clinical cardiologists, interventional cardiologists, electrophysiologists, intensivists and cardiac surgeons is the key to success.

Keywords: primary PCI, ICD in secondary prevention, aneurismectomy

CASE REPORT

A 43-year-old man, active smoker, with no significant medical history, presented to the Emergency Department of the Emergency University Hospital Bucharest, after being successfully resuscitated at his office, following cardiac arrest by ventricular fibrillation (VF). At presentation in the Emergency Department he

was on respiratory and vasopressor support (continuous infusion of adrenaline), with a blood pressure of 80/60 mmHg and heart rate of 120 bpm.

The electrocardiography (ECG) revealed sinus tachycardia, right bundle branch block (RBBB), Q waves in the anterior leads, ST segment elevation in the inferior leads and recurrent non-sustained ventricular tachycardia (VT) (Figure 1). The echocardiography identified an-

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terior and lateral wall akynesia, moderate inferior wall hypokinesia, an ejection fraction of the LV (LVEF) of 25% and moderate ischaemic mitral regurgitation.

Given to the clear diagnosis of cardiogenic shock secondary to inferior STEMI, the patient received loading dose of Aspirin and Clopidogrel (by naso-gastric tube) and was referred to the catheterization laboratory for emergency angiography. The coronary angiography revealed a chronic total occlusion of the left anterior descending artery (LAD), normal left circumflex artery (LCX) (Figure 2A) and a right coronary artery (RCA) with fresh thrombus in the proximal segment, a critical stenosis in the mid-segment and distal flow TIMI (Thrombolysis In Myocardial Infarction) 1-2 (Figure 2B).

The intraaortic balloon pump (IABP) was placed prior to any interventional therapeutic, vasopressor support was switched from adrenaline to dobutamine, and continuous i.v. lidocaine was maintained. The RCA lesions were quickly resolved by thrombus aspiration and stent placement in the mid-segment (3.5 x 19 mm), with a good final result (Figure 2C). Since the patient was presented with cardiogenic shock and was hemodynamically and electrically unstable, percutaneous coronary intervention (PCI) of the LAD was attempted, but with a mediocre result after multiple balloon inflations in the proximal and mid segments (Figure 2D). The patient was admitted in the Intensive Care Unit (ICU).

After 72h the IABP was removed, and PCI of the LAD was reattempted, but with the same mediocre result. The prolonged ICU admission (25 days) was due to difficult ventilation weaning, an associated severe respiratory infection, massive bilateral pleurisy, and iatrogenic pneumothorax. After the transfer to the Cardiology Ward, the patient remained in hospital for another 40 days.

Treatment on discharge comprised of dual antiplatelet therapy, oral anticoagulation for at least 3 months due to severely reduced LVEF, apical aneurysm of the LV and spontaneous contrast, antiarrhythmic (amiodarone), beta-blocker, angiotensin-converting-enzyme inhibitor (ACE inhibitor), loop diuretic, mineralocorticoid receptor antagonist, nitrate, statin and inotrope (digoxin).

At 2 months follow-up, the patient was electrically stable, without angina, but with exertion on mild exercise. After 4 months, he pre-

sented with symptomatic frequent ventricular premature beats, and the amiodarone dose was increased to 400 mg/day, and at 6 months he was transferred to our centre from his regional hospital with severe acute pulmonary oedema, secondary to sustained monomorphic VT. After hemodynamic stabilisation, an implantable cardioverter-defibrillator (ICD) was decided for the secondary prevention of VT.

At 1 year, the patient presented progressive exertion on mild exercise (<25 m walking distance), was cachectic (50 kg compared to 92 kg on initial presentation) and echocardiography

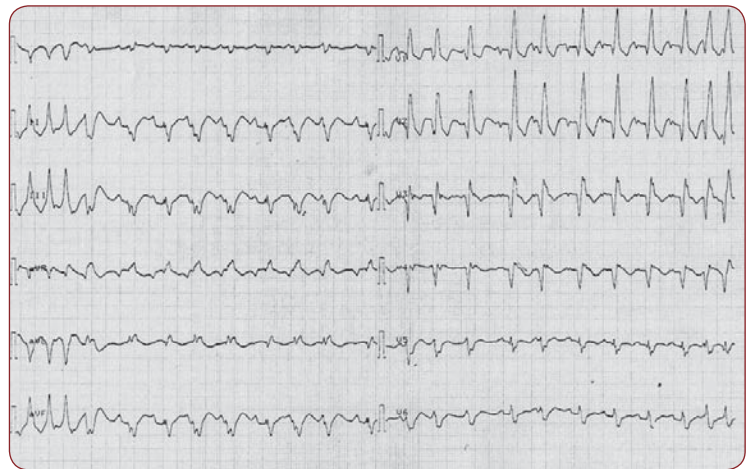


FIGURE 1. ECG at admission showing sinus tachycardia, RBBB, Q waves in anterior leads, ST elevation in inferior leads and periods of non-sustained VT.

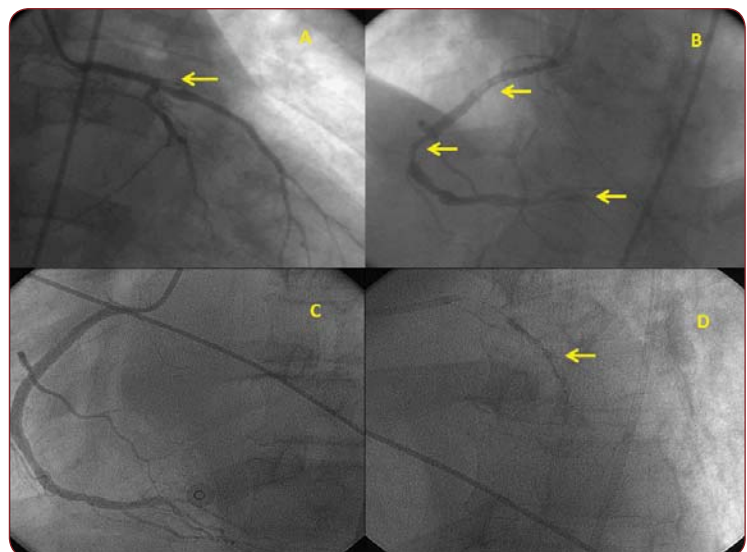


FIGURE 2. Admission coronary angiogram. **A.** Left coronary artery – chronic total occlusion of LAD, normal LCX. **B.** Right coronary artery – thrombus in proximal segment, critical lesion in the mid segment, TIMI 2 distal flow. **C.** Right coronary artery – result after thrombus aspiration and stenting of mid-segment; Distal flow TIMI 3. **D.** LAD – mediocre result after multiple balloon inflations in the proximal and mid segments.

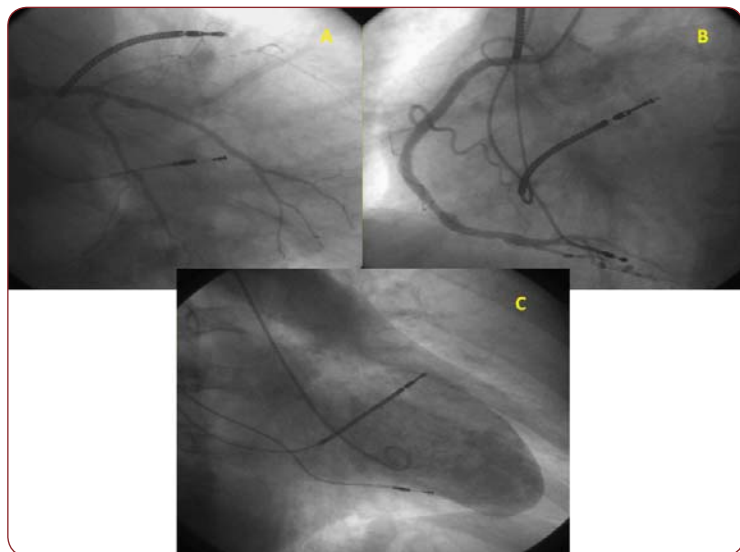


FIGURE 3. Echocardiography at 1 year follow-up. **A.** Apical 2 chamber view – dilated LV with apical aneurysm occupying 40% of LV volume. **B.** Apical 4 chamber view – severe mitral regurgitation (ischaemic and annulus dilation).

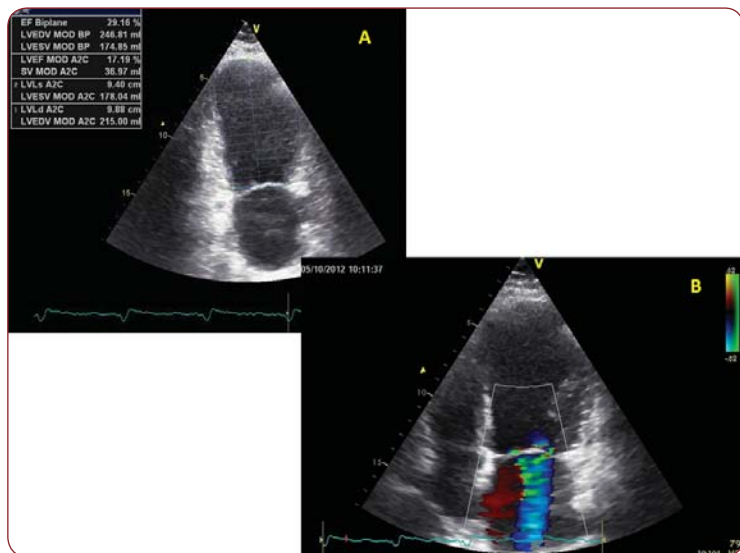


FIGURE 4. Preoperative coronary angiogram. **A.** Left coronary artery – chronic total occlusion of proximal LAD with weak collateralization, normal LCX. **B.** Right coronary artery – no in-stent proliferation, no “de novo” lesions. **C.** Left ventriculography – large apical aneurysm with akynesia of the anterior LV wall.

revealed a LVEF of 30%, a large apical LV aneurysm with ~40% of LV volume (Figure 3A) and severe secondary mitral regurgitation (ischaemic and mitral annular dilation) (Figure 3B). The patient was referred to a cardiac surgery centre abroad for aneurismectomy with LV reconstruction and mitral valve repair. Coronary angiography was performed prior to surgery, with a stationary status of the coronary arteries and large apical LV aneurysm (Figure 4A, 4B, 4C).

Surgery was performed at the Klinik für Herz und Gefäßchirurgie Robert Bosch Krankenhaus Stuttgart, with successful aneurismectomy and LV reconstruction. Initially, mitral valve repair was attempted, but due to residual moderate mitral regurgitation on intraoperative TOE, the final decision was in favour of mitral valve replacement with a metallic valve (Figure 5A, 5B). Of note, the patient could not be intubated oro-tracheally because of a tracheal stricture secondary to the prolonged intubation during the first hospital presentation, and required ventilation through tracheostomy.

After surgery, the patient developed mild bilateral pleurisy, and pericarditis without hemodynamic importance, which resolved uneventfully. The tracheal stricture was treated by laser therapy during the same admission. Postoperative echocardiography showed a mildly dilated LV (Figure 5C), with a normally functioning metallic mitral valve (Figure 5D).

Six months after surgery, the patient was asymptomatic, electrically stable, gained weight (75 kg) and was physically, socially and professionally active. □

DISCUSSION

Cardiogenic shock carries a very high in-hospital mortality rate (~50%), and treatment of its underlying cause, when possible, should be a top priority (1-2). In our case, emergency myocardial revascularization was life-saving, the operators trying to solve as many lesions as possible, besides the infarct related artery (indication class IIa) (3). Unfortunately, due to the characteristics of the proximal LAD lesion (probably old Q waves in the anterior leads), the final result after multiple balloon inflation was mediocre. Nevertheless, supportive therapy by an experienced intensive care staff was crucial for the patient's short term outcome.

It is well known that large myocardial infarctions, with severely depressed LVEF and extensive myocardial scarring are associated with long-term risk of malignant ventricular arrhythmias, this patient presenting with sustained VT and acute pulmonary oedema at 7 months after the myocardial infarction. ICD therapy is recommended in these cases for secondary prevention with a class I indication (4-8).

Despite full medical treatment, myocardial revascularization and secondary prevention of sudden cardiac death, cardiac remodelling is,

at times, an active and aggressive process, which plays an important role in patient quality of life and long-term outcome. In this case, apical aneurysm development, LV dilation and subsequent mitral regurgitation aggravation led to decreased functional capacity, low quality of life and cachexia. In such cases, despite the high operative risk, high performance cardiac surgery, when indicated correctly, can successfully complete the interventional treatment, and play a role in improving quality of life and long-term outcome. Despite LV aneurismectomy and reconstruction not being routinely recommended, the restoration of a more physiological LV volume and shape were extremely beneficial in this patient. Associated surgical myocardial revascularisation would have been ideal, but was not feasible in this case due to the LAD lesion characteristics (9).

Close follow-up and full compliance to medical therapy are essential in the management of such complex patients. Any change in functional capacity, new symptoms or associated comorbidities must be thoroughly investigated as they can have a large impact on long-term outcome and clinical decision making (10-11). ■

CONCLUSION

We report the case of a 43-year-old man, that presented with inferior STEMI and cardiogenic shock, who developed late sustained VT, and despite life-saving management by primary PCI, IABP, advanced life support and ICD implantation, a favourable long term outcome was not guaranteed.

High performance cardiac surgery, when indicated correctly, can successfully complete the interventional treatment, thus improving quality of life and long-term outcome.

Team work between clinical cardiologists, interventional cardiologists, electrophysiolo-

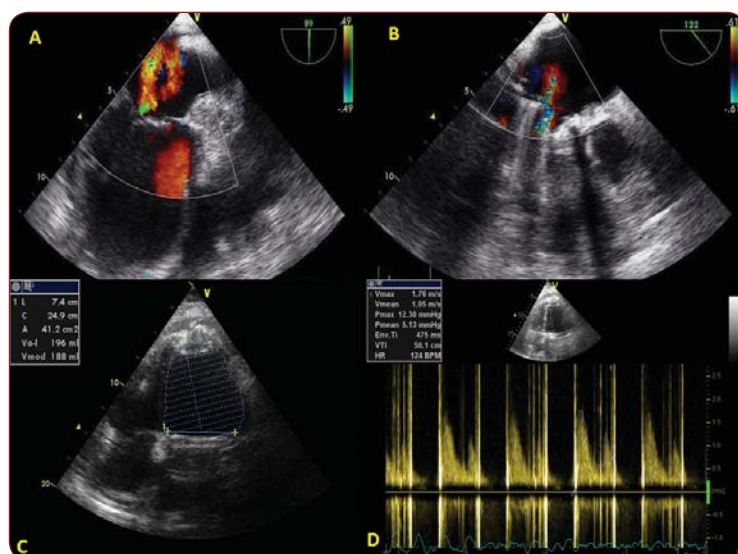


FIGURE 5. Intraoperative TOE and post-surgery TTE. **A.** Moderate residual mitral regurgitation after mitral valve repair attempt. **B.** Mild mitral regurgitation (normal) after metallic valve implantation. **C.** Apical 4 chamber view – mildly dilation of LV, LVEF 40%. **D.** CW Doppler in apical 4 chamber view, mitral valve level – normal functioning of metallic valve.

gists, intensivists and cardiac surgeons is the key to success.

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