Pre-PCI angina post-PCI ischaemia: tales of ordinary madness

Mario Marzilli* and Doralisa Morrone

Cardiovascular Medicine, University of Pisa, Pisa, Italy

An 82-year-old male patient was admitted to the Emergency Room due to exertional dyspnoea and orthopnoea. Medical history revealed a long history of hypertension, diabetes mellitus type II, and the following sequence of events (Table 1A):

In year 2000, patient had effort angina. No medical treatment was prescribed, and the patient was immediately referred for percutaneous coronary intervention (PCI) on first obtuse marginal (OM1); the PCI procedure was complicated by occlusive dissection of the vessel. After this first procedure, the patient denied angina up to 2006, when he was referred for an exercise test. The patient was asymptomatic, but the test was diagnosed as positive for myocardial ischaemia based on the electrocardiogram (ECG). The patient was immediately referred for repeat coronary angiography that was followed by PCI on the left anterior descending (LAD).

In 2007, during a follow-up visit, a dipyridamole test was performed that elicited no angina, but was diagnosed as positive for myocardial ischaemia again. A coronary angiography was repeated, but no new obstructive lesion that could have been dilated.

From 2007 to 2015, several provocative tests were performed, all diagnosed as positive for myocardial ischaemia, with the patient asymptomatic.

In June 2015, an additional stress echocardiography positive for myocardial ischaemia without symptoms, was followed by repeat coronary angiography that showed stenosis of the right coronary artery (RCA) and OM1. PCI was again performed.

In July 2015, the patient suffered an acute coronary syndrome (ACS) due to intrastent thrombosis of the RCA, treated with repeat PCI.

In February 2017, the patient was admitted to the hospital for infective pneumonia; during admission, he suffered an episode of paroxysmal atrial fibrillation (AF). During this admission, a coronary angiography was repeated that showed no new coronary lesions. At discharge, ejection fraction (EF) was 43%. Beta-blocker and oral anticoagulant were introduced.

In March 2017, during a follow-up visit, EKG showed II degree atrio-ventricular (AV) block (Mobitz type 1).

In July 2017, patient underwent an exercise echocardiography test that resulted positive at low workload (50 W) with hypokinesis of the apical segments of all walls and of mid-interventricular septum (IVS) and infero-lateral walls. This observation was followed by increased beta-blocker dosage.

In August 2017, patient had haematuria from a urinary papilloma and was scheduled for surgery.

In September 2017, the patient was extremely bradycardic, therefore a biventricular Pacemaker (PM) was implanted, justified by the plan to ‘power up’ anti-ischaemic therapy and beta-blockers dosage. Patient was sent home on: bisoprolol 7.5 mg b.i.d., valsartan/hydrochlorothiazide 80/12.5 mg, dabigatran 150 mg b.i.d., ASA 100 mg, atorvastatin 20 mg, omeprazole 20 mg, metformin 500 mg trid, and nitroglycerin nitrates through the skin (NTG TTS) 10 mg h B-20.

On October 7, 2017, the patient was admitted in the hospital for dyspnoea at rest. Clinical examination showed: blood pressure (BP) 155/85 mmHg and sO2 95%. reduced breath sounds at basal lung fields, fine crackles on mid-basal fields. Regular heart rhythm, normal S1-2, holosystolic murmur at apex. Blood chemistry: troponin ultra sensitive (hsTn) 826 ng/mL; creatine Kinase MB (CK-MB) 1.9 ng/mL; WBC 9064 RBC 3.2×10⁶, Hb 9.9, Plt 313000, AST 10, Tot Proteins 7.1, Creat 1.56 mg/dL; Gluc 192 mg/dL; brain natriuretic peptide (BNP) 1593 pg/dL. EKG and X-ray at admission are showed in Figures 1 and 2. Echocardiographic examination showed left ventricular mild end-diastolic dilatation and wall thickness increase; severe reduction of global systolic function (EF 34%) with apical and inferior wall akinesia plus dyssynergic contraction of IVS; mild pulmonary hypertension (pulmonary artery pressures (PAPs) 45 mmHg) and mild mitral regurgitation. A diagnosis of acute decompensated heart failure was made and the following therapy was started: intravenous diuretics, BB and...
ASA were interrupted and trimetazidine was added; the PM was re-programmed, increasing AV delay to favour spontaneous rhythm. The patient became progressively asymptomatic; BP returned in the normal range (145/75 mmHg); 

$sO_2$ 97% increased (FiO2 21%). Clinical examination showed reduced breath sounds at basal fields, fine crackles on basal fields with regular heart rhythm; normal S1-2, holosystolic murmur at apex.

Blood chemistry showed decrease in troponin and BNP (hsTn 709 ng/mL; CK-MB 1.53 ng/mL; WBC 8013 RBC 3.33 × 10^6; Hb 10.1, Plt 302000, AST 10, Tot Proteins 7, Creat 1.37 mg/dL; Gluc 209 mg/dL; BNP 729 pg/dL). EKG and chest X-ray after treatment are showed in Figures 3 and 4. Echocardiographic examination before discharge showed: left ventricle (LV): mild end-diastolic volume and wall thickness increase; mild reduction in global systolic function (EF 46%) with akinosis of apex and inferior wall, mild pulmonary hypertension (PAPs 32 mmHg), and mild mitral regurgitation.

At a follow-up visit on October 16, the patient was well compensated, with normal BP values (135/70 mmHg) and saturation $sO_2$ 97%. Normal breath sounds at basal fields, no crackles. Regular heart rhythm; normal S1-2, holosystolic murmur at apex.

Blood chemistry showed hsTn 110 ng/mL; WBC 6066 RBC 3.41 × 10^6; Hb 10.3, Plt 242000, AST 15, Tot Proteins 6.4, Creat 1.51 mg/dL; Gluc 123 mg/dL; BNP 461 pg/dL.

As of today, the patient remains asymptomatic with the following therapy: valsartan/ hydrochlorothiazide 80/12.5 mg; dabigatran 150 mg b.i.d.; atorvastatin 20 mg; omeprazole 20 mg; metformin 500 mg trid; isosorbide dinitrate 20 mg trid, trimetazidine 20 mg trid; furosemide 25 mg.
Discussion

This is an impressive example of how far the so-called ‘real world’ of Cardiology can be from Guidelines recommendations.

Since year 2000, this patient underwent five invasive coronary angiography, four revascularization procedures, six stress echocardiograms, and a bicameral PM implantation. Most of these treatments were not recommended according to current guidelines.\textsuperscript{1,2} To mention some, the first PCI procedure was performed in a patient receiving no antianginal medication, repeat PCI procedures were performed in the absence of any symptom, beta-blockers were recommended for a presumed heart failure with an EF > 40%, a PM was implanted for a iatrogenic A–V block. And some of these treatments were associated with serious complications, including occlusive coronary dissection, late stent thrombosis, worsening LV function, and high degree A–V block.

The question is how exceptional is this case or how ‘normal’. In cardiology meetings, Clinical Cardiologists, Electrophysiologists, and Interventional Cardiologists are frequently encouraged to follow guidelines and to practice ‘evidence-based medicine’. Are these recommendations applied to daily practice? Beyond this extreme case, available data are discouraging. Almost half the patients undergoing elective PCI in the USA, are reported to be taking no antianginal medication and not to have documented myocardial ischaemia. PCI procedures are reported to cause complications\textsuperscript{3} (Table 1), which could not be considered as ‘minor’, especially in cases, in which the procedure is not relevant (Table 1B). So, when undertaking one, it should be certain, that the benefits prevail the risk appropriateness criteria and restrictions to reimbursement have been introduced in the USA that have cut down on the number useless procedures, but in most countries no such control exist and decisions are left to the discretion of the treating physician.
Some thought should be given to the ‘real world’ of Cardiology to prevent that seeing a Cardiologist becomes a risk factor.

Consent statement

The patient consent to report the case has been obtained.

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**Table 1** History summary/PCI adverse events

| Year | Event Description |
|------|-------------------|
| 2000 | PCI of OM1 (dissection of LAD) |
| 2006 | positive echostress → PCI of LAD |
| 2007 | positive echostress → PCI of LAD |
| 2007-2014 | 2 positive echostress |
| 2015 | positive echostress → PCI of RCA and OM1, stent thrombosis of RCA treated with PCI |
| 2017 | dyspnea during pneumonia diagnosed as heart failure (negative coronary angiography) → beta-blockers |
| 2017 | Ambulatory ECG: II degree Mobitz type 1 AV block |
| 2017 | Increased beta-blocker dosage |
| 2017 | Biventricular PM implanted to increase beta-blocker dosage |
| 2018 | Patient had exertional angina |

| Rate of adverse events in the approximately 500,000 patients who undergo PCI for symptomatic relief of stable angina in the USA and Europe each year: |
|--------------------|-----------------|-----------------|
| Death 0.65% | 3250 deaths |
| Myocardial infarction 15% | 78000 MI |
| Renal injury 13% | 65000 CKD |
| Stroke 0.2% | 1000 TCEs |
| Vascular complications 2-6% | 6000-30000 vascular complications |

Data from Dehmer GJ et al., *J Am Coll Cardiol* 2012;67:2017-31