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CASE REPORT
Diseases of the heart

A successful treatment of ischemic cardiomyopathy associated with left ventricular aneurysm and chronic ischaemic mitral regurgitation (RCD code: III-1B.9.o)

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Abstract

Complications of myocardial infarction such as left ventricular aneurysm, chronic ischemic mitral regurgitation and heart failure are associated with a poor prognosis. The management of complex defects is challenging and requires an individualized strategy. We describe a case of a 44-year-old male with multiple complications of myocardial infarction including heart failure with reduced ejection fraction, left ventricular aneurysm and mitral regurgitation, treated with mitral valve replacement and left ventricular reconstruction. We discuss pathophysiology and treatment of chronic ischemic mitral regurgitation and left ventricular aneurysm. JRCD 2017; 3 (4): 122–128

Key words: chronic ischemic mitral regurgitation, heart failure, ventricular remodeling, left ventricular aneurysm, mitral valve replacement

Background

Chronic ischemic mitral regurgitation (CIMR) is a disease of left ventricle (LV) and not of mitral valve (MV) itself. Severe ventricular remodeling induces changes in MV geometry and results in valve incompetence. CIMR is a frequent complication of coronary artery disease or myocardial infarction (MI) and is associated with adverse prognosis [1,2]. The main mechanism of CIMR is related with distortion of the spatial relationships between the MV and papillary muscles (PMs) secondary to ventricular remodeling. CIMR is characterized mechanically by valve leaflets that coapt apically within the LV, restricting leaflet closure in a pattern known as incomplete mitral leaflet closure. This process can then become self-perpetuating as CIMR leads to ventricular dilatation, which in turn leads to further PM displacement, annular dilatation, and progression of CIMR [1,3].

Important advances in the understanding of pathophysiology, evaluation, and prognosis have occurred during recent years and confirmed that CIMR has many specific features which differentiates it from organic regurgitations. In patients who have severe CIMR but no indication for coronary revascularization, generally because there is no myocardial viability, the indication for isolated mitral surgery is controversial. The evidence that the treatment of CIMR improves long-term survival still remains unclear, and further studies are needed to determine whether invasive treatment will improve survival and/or symptoms [4,5].

Case presentation

A 44-year-old man with worsening symptoms of heart failure was admitted to the Centre for Rare Cardiovascular Disease of the John Paul II Hospital in March 2016. He presented with resting dyspnea, chronic fatigue and severely reduced exercise tolerance [New York Heart Association (NYHA) class IV], and angina [Canadian Cardiovascular Society (CCS) class III] for one month.

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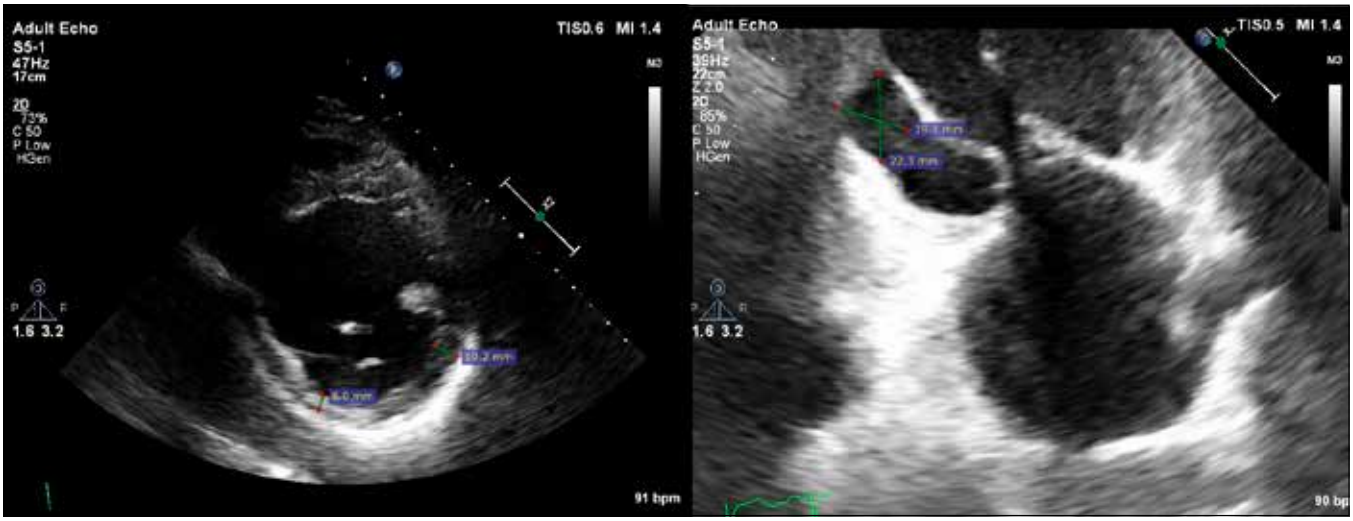

Figure 1. Transthoracic echocardiography – parasternal short axis view (left), apical two-chamber view (right). Post-infarct cardiac remodeling – a true aneurysm of the inferior wall (size 22x19 mm, arrow), thin and akinetic posterior wall, restriction of the posterior mitral leaflet. LA- left atrium; LV- left ventricle

About 5 months before the admission he had experienced anginal chest pain that lasted for a couple of hours but he did not visit emergency room.

The patient had typical cardiovascular risk factors, such as hypertension, hyperlipidemia, diabetes mellitus type 2, obesity and nicotine addiction. In 2004 he was diagnosed with pulmonary tuberculosis. He had no family history of cardiac diseases or sudden cardiac death. On admission to the hospital the patient had acute heart failure with signs of pulmonary congestion and ascites. His blood pressure was 100/60 mm Hg and his heart rate was 110 bpm. The oxygen saturation was 88% on room air and with oxygen therapy given by nasal cannulae at 2–4 L/min reached 95%. A loud systolic murmur was heard at the fifth intercostal space in the midclavicular line, radiating to the left chest border. The electrocardiogram revealed sinus tachycardia of 110 bpm and large Q waves in leads II, III, aVF. Laboratory test results showed elevated N-terminal pro brain natriuretic peptide (8316 pg/mL; normal values <125 pg/mL), troponin T (first assessment – 0.036 ng/mL, repeated 6 h later – 0.033 ng/mL; normal values <0.014 ng/mL) and D-dimer (1348 μg/L; normal values <500 μg/L) [6]. The cardiac markers results excluded acute myocardial infarction. Transthoracic echocardiography showed: enlargement of all heart chambers, severely impaired function of both ventricles, inferior and posterolateral LV wall aneurysm, with severe functional mitral regurgitation (MR) resulting from restricted leaflet motion and valvular annulus dilatation (type I and IIIb by Carpentier Classification), moderate tricuspid regurgitation, elevated LV filling pressures (Doppler E velocity to tissue Doppler E′ velocity ratio was 25, normal range <8), increased right ventricular systolic pressure (50 mm Hg, normal range <36 mm Hg), small amount of fluid in the pericardial space (up to 5 mm in front of the right ventricle) and moderate pleural effusion (Figures 1-3; Tables 1-3). Computed tomography (CT) of the chest excluded pulmonary embolism. Coronary angiography revealed non-significant lesions in the left anterior descending coronary artery (LAD) and a borderline stenosis in the right coronary artery (RCA) (Figure 4). Cardiac magnetic resonance imaging (MRI) revealed a true aneurysm of the inferior wall of LV (20 × 20 mm), akinetic inferior and posterior walls, apical segment of the lateral wall, septum and apex. Total scar volume was 76ml (Figure 5). LV ejection fraction (LVEF) was reduced (29%, normal range: 52–72%) as well as stroke volume (63 ml, normal range: 68-144 ml). Right ventricular end-diastolic volume and mass were within the limits, 221 ml (normal range: 118–250 ml) and 39 g (normal range: 25–57 g), respectively [7].

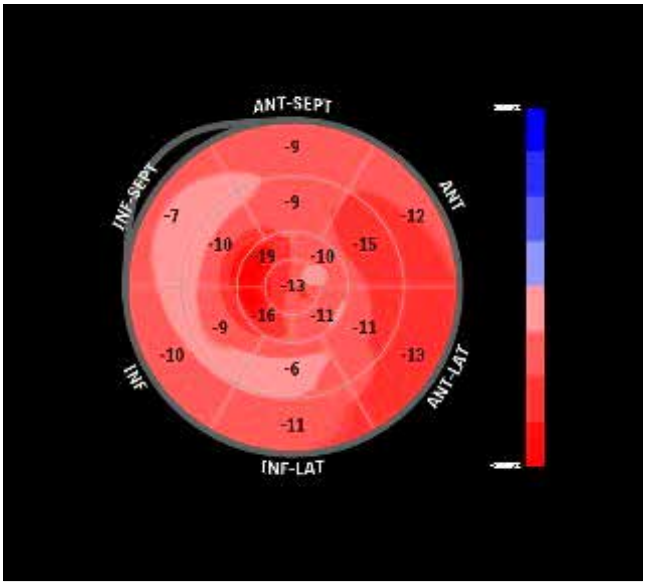

Figure 2. Speckle tracking echocardiography. Reduced left ventricular global longitudinal strain (GLS -11%). Severely impaired longitudinal function of *the* inferior and posterolateral wall

Patient management and follow-up

The patient's case was discussed during a multidisciplinary consultation meeting and he was scheduled for surgical repair of LV aneurysm despite known surgical risk factors for repair failure.

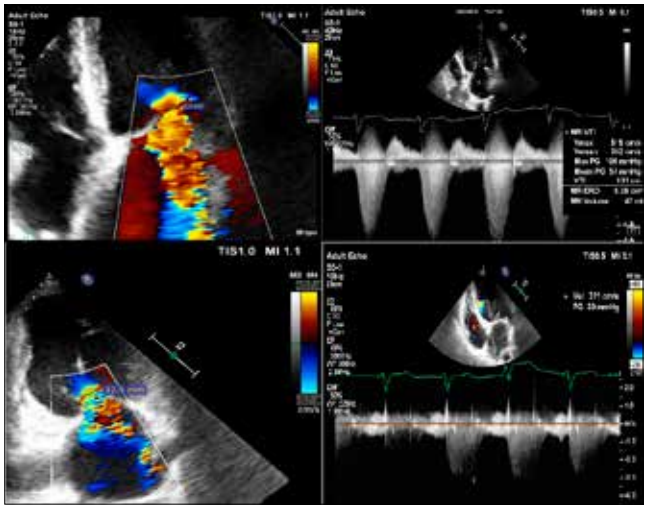

Figure 3. Transthoracic echocardiography. Semiquantitative and quantitative assessment of severity of mitral regurgitation. Vena contracta width 9–12 mm (apical views), effective regurgitant orifice area 0.38 cm^2, regurgitant volume 47 ml. Tricuspid regurgitation peak velocity 3.1 m/s

Table 1. Selected transthoracic echocardiography parameters

	Values observed	Normal values for male (2SD range) [34]
Left ventricular size and function		
end-diastolic diameter (mm)	72	42–58
end-systolic diameter (mm)	52	25–40
end-diastolic volume (ml)	260	62–150
end-systolic volume (ml)	198	21–61
end-diastolic volume index (ml/m^2 BSA)	116	34–74
end-systolic volume index (ml/m^2 BSA)	88	11–31
left ventricular ejection fraction (%)	24	52–72
Right ventricular size and function		
basal diameter (mm)	38	25–41
mid diameter (mm)	25	19–35
longitudinal diameter (mm)	81	59–83
parasternal long-axis diameter (mm)	38	20–30
outflow tract end-diastolic area (cm^2)	25	10–24
outflow tract end-diastolic area index (cm^2/ m^2 BSA)	11	5–12.6
tricuspid annular plane systolic excursion (mm)	9	17–31
pulsed Doppler S' wave (cm/sec)	5	9.5–18.7
Left atrial volume index (ml/ m^2 BSA)	73	16–34
Right atrial volume (ml/ m^2 BSA)	48	11–39

SD – standard deviation, BSA – body surface area

In June 2016 he underwent LV reconstruction (also known as the Dor procedure) and MV replacement with St. Jude Medical™ 27M prosthesis with posterior leaflet preservation. The postoperative course was uncomplicated. Several months after the operation he is well, feels no pain in the chest and walks 1 km without taking a rest. The implanted mechanical MV works well. A control transthoracic echocardiography showed a decreased LV dimension, an improved LVEF of 38%, normally functioning mechanical MV and mild tricuspid regurgitation.

Review of literature

Chronic ischemic mitral regurgitation

Mechanisms of CIMR are very complex. Proper function of the MV depends not only on the components of the valve apparatus (leaflets, chordae tendineae, PMs, and/or annulus) but also on the LV size and function. CIMR is most frequently caused by dysfunction of the LV and changes in the MV annulus. LV remodeling leads to the dilatation of the posterior portion of the MV annulus (type I by Carpentier Classification) and its flattening, losing its physiological saddle-shape conformation [4,8]. Regardless of the etiology, LV dilatation and alternations in its geometry result in PMs displacement from each other and from the annulus, which worsens coaptation of the leaflets. This mechanism is known as restricted leaflet motion (type IIIb by Carpentier Classification) [1,2,4].

Depending on the localization of a contractile dysfunction there are two types of restriction. Symmetric restriction is characterized by a predominant apical tethering of both leaflets – the regurgitant jet has usually a central origin and direction. This pattern occurs after global LV remodeling (e.g. in patients after antero-posterior MI). Asymmetric restriction is characterized by the predominant posterior tethering of the posterior leaflet – the regurgitant jet is thus eccentric (e.g. due to altered geometrical localization of the posterior PM, as in patients after postero-inferior MI) [5,9].

CIMR causes chronic volume overload of the LV, which augments its eccentric hypertrophy, further dilatation and flattening of the annulus and in the self-perpetuating mechanism leads to the progression of CIMR (CIMR begets CIMR) and worsening of cardiac failure [6,10]. Given the aforementioned mechanism, indications for surgical therapy exist in less severe CIMR than they are for patients with chronic primary MR [effective regurgitant orifice area (EROA) of 0.2 cm^2 vs. 0.4 cm^2, regurgitant volume of 20 ml vs. 40 ml]. An exercise-induced increase of ≥13 mm^2 of the EROA has been shown to be associated with a large increase in the relative risk of death and hospitalization for cardiac decompensation [11–13].

CIMR more often develops in patients with inferior MI (65%) than anterior MI (35%) and in more than 80% of patients with antero-inferior MI [4,14]. It is due to the different blood supply to the PMs. Anterior PM has dual blood supply from both the LAD and a diagonal or marginal coronary artery. On the other hand, posterior PM has a single blood supply in 95% of cases, most commonly from the posterior descending branch of the RCA. Moreover, posterior PM ruptures are 6–12 times more often than the anterior PM, which requires immediate diagnosis and urgent surgical intervention [2,12].

Table 2. Global and regional longitudinal left ventricular strain values

Regional longitudinal strain (%)	Values observed			Normal values for male (mean±SD) [35]		
	Basal	Mid	Apical	Basal	Mid	Apical
Anterior	-12	-15	-10	–20.7 (3.8)	–21.3 (3.3)	–22.5 (4.1)
Anteroseptal	-9	-9	-19	–17.8 (2.5)	–21.9 (2.8)	–23.5 (6.0)
Inferoseptal	-7	-10		–18.0 (3.2)	–20.4 (4.6)	
Inferior	-10	-11	-16	–21.5 (3.8)	–22.1 (3.3)	–23.5 (3.6)
Inferolateral	-11	-6	-11	–20.2 (4.0)	–19.8 (3.6)	–20.9 (3.7)
Anterolateral	-13	-11		–21.4 (3.8)	–20.1 (3.2)	
Global longitudinal strain (%)	-11			–20.7 (2.0)		

Treatment of chronic ischemic mitral regurgitation

Functional MR can be treated conservatively (pharmacologic therapy) or invasively (interventional and surgical options) [3]. Effective conservative therapy that reverses LV remodeling may reduce MR and continued efforts should be made to optimize treatment of these patients (we observed the aforementioned effect in our patient – LVEF increased by 5–10% in a month before surgery). Among patients with CIMR that are symptomatic despite optimal pharmacologic therapy, invasive treatment should be considered. Current guidelines do not suggest whether it should be MV repair or replacement [15,16].

In clinical practice MV repair is the surgical treatment of choice for MR. In centers that are experienced in MR therapy, MV repair makes up 90% of procedures [5,17]. However, there are situations in which the repair is technically impossible or so difficult that its long term effects remain unsure. The choice of a surgical procedure depends on the following factors: degree of morphological and functional valve dysfunction, presence and extent of calcifications and mitral annular diameter. Long-term predictive factors of MV repair failure are: long distance between PMs, severely restricted posterior mitral leaflet and considerable LV dilatation. In such cases MV replacement may be recommended instead of MV repair [11,18]. Successful MV repair in patients with secondary MR is less likely in the presence of factors listed in Table 3.

MV repair requires intraoperative echocardiography in order to assess possible post-procedural regurgitation (before a patient leaves the operating room). Presence of persistent significant MR requires revision of the repair or valve replacement. MV replacement surgery (for a mechanical or bioprosthetic valve) should be done with the aim of preserving the subvalvular apparatus because its excision has a negative impact on LV function [15,16].

Inoperable, high-risk, symptomatic patients may benefit from transcatheter MV repair. Currently, there are two procedures of transcatheter MV repair. The first one reduces mitral annular diameter with a ring implant inserted through the coronary sinus. The other one – percutaneous leaflet plication – consists in suturing the regurgitant orifice of the MV leaflets together through a transseptal puncture (the so called edge-to-edge repair – MitraClip) [19]. Percutaneous edge-to-edge repair for secondary MR is a low-risk option, but it is inferior to surgery in terms of MR reduction. It can improve symptoms, functional capacity and quality of life and may induce reverse LV remodeling [20]. When revascularization is not indicated and surgical risk is not low, a percutaneous edge-to-edge procedure remain an option in patients with severe secondary MR and LVEF >30% who are symptomatic in spite of optimal medical management and who have a suitable valve morphology as assessed by echocardiography [3]. A novel device, the Harpoon (expanded polytetrafluoroethylene artificial cord implantation device) is designed to provide a minimally invasive surgical solution for degenerative MR. Instead of opening the chest, surgery using the Harpoon device enters the heart through a mini-thoracotomy on the left side of the torso. Utilizing echocardiographic imaging, the surgeon brings the tip of the device to the mitral leaflets, where it stabilizes the flaps and anchors artificial cords that will do the work of the ailing natural cords. Early clinical evidence has shown reduction in MV regurgitation, improvement in symptoms, positive safety profile and no need for long-term oral anticoagulation. This approach has the potential to decrease invasiveness and surgical morbidity, but further follow-up is necessary to assess long-term efficacy [21].

Surgical correction of CIMR improves functional status of the patients but its impact on survival is questionable. MV repair results in better short-term outcomes than surgical replacement but long-term benefits are similar. The rate of recurrent of moderate or severe MR at 1 year follow-up is higher by 30% in patients undergoing the repair [15,16,22]. Therefore, long-term observation of patients undergoing invasive treatment of CIMR is necessary.

Left ventricular aneurysm

Loss of cardiac muscle cells e.g. due to MI overburdens the myocardium and leads to contractility disorders and, with time, to alternations in ventricular shape and size. Progressive chamber dilatation is a part of a self-perpetuating mechanism of excessive overload, increased stimulation and further myocardial damage, which impairs LV mechanical function. In contrast to the typical remodeling of the LV, in some cases its shape may change asym-

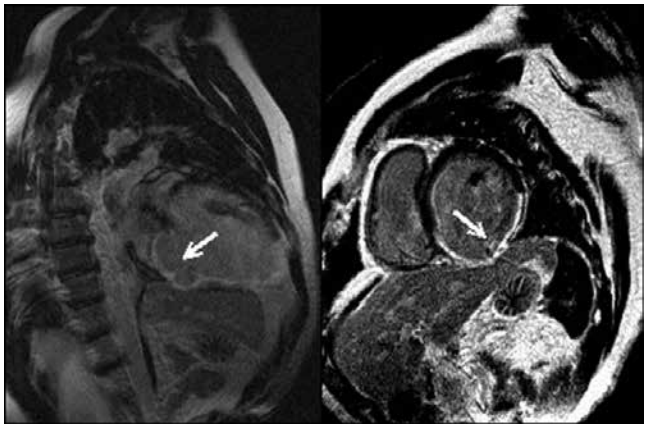

Figure 4. Magnetic resonance late enhancement imaging, two chamber and short axis views. Myocardial infarction of the inferior and posterolateral wall complicated with aneurysm (arrow)

metrically with the formation of an aneurysm of a wall. Extensive transmural MI causes ventricular aneurysm in every fifth patient (5–7 times more often in men) [23,24]. They are more common in younger patients because of a poor coronary collateral circulation and more extensive MIs in this group. LV aneurysms develop in 13% of patients under 40 whereas among patients over 60 in 5.5% of cases [25].

LV aneurysms may develop early, even during muscle necrosis (then it's of small size), within weeks or late, after months or years from the MI. Post-MI aneurysms are in 98% of cases true aneurysms (made up of a scarred ventricular wall) and only in 2% pseudoaneurysms, where cardiac rupture is contained by pericardium [23]. An LV aneurysms is most commonly the result of MI, usually involving the anterior wall (>80%) with the apex and the antero-lateral wall. They develop due to total occlusion of the LAD, rarely due to changes in other coronary arteries (as in our patient) [25].

Our patient had complications typical for previous MI, although significant coronary artery disease was not present. Acute coronary syndrome may develop from the erosion or rupture of obstructive or nonobstructive coronary atherosclerotic plaques. The latter condition is less common than obstructive coronary artery disease, with an estimated prevalence between 5% to 25% [26] but the rates of events largely varied across single studies. Nonobstructive coronary artery disease may encompass epicardial artery coronary vasospasm, spontaneous coronary artery dissection, takotsubo cardiomyopathy, cocaine abuse and acute myocarditis with acute coronary syndrome-like presentation [27].

More than a half of patients with post-MI LV aneurysms experience angina at rest and with exertion and signs of heart failure, 15–30% of patients have arrhythmias, including life-threatening ventricular arrhythmias, whereas thromboembolic complications are rare (2–5%) [23,28]. Echocardiography and ventriculography are the basic diagnostic techniques of post-MI LV aneurysms whereas MRI and CT are used in uncertain cases [3,29]. Sometimes, though, these modalities cannot distinguish between a true aneurysm and pseudoaneurysm. Many authors emphasize diagnostic difficulties in distinguishing between these two clinical entities, especially if invasive procedures are unavailable [24,29].

Table 3. Echocardiographic assessment of functional mitral regurgitation and probability of successful mitral valve repair

	Values observed	Severely abnormal [11,18]
Mitral regurgitation: semiquantitative and quantitative assessment		
effective regurgitant orifice area (cm^2)	0.38	≥0.20
regurgitant volume (ml)	47	≥30
regurgitant fraction (%)	0.75	≥0.50
vena contracta width (mm)	8	≥7 (>8 for biplane)
mitral E wave velocity (m/s)	1.5	>1.5
pulmonary vein flow – systolic flow reversal	+	+
right ventricular systolic pressure (mmHg)	50	≥ 36
Predictors of mitral valve repair		
Mitral valve deformation		
coaptation height (cm)	1.4	≥1
tenting area (cm^2)	4.3	>2.5–3.0
posterolateral angle (°)	54	>45
Local left ventricular remodelling		
interpapillary muscle distance (mm)	38	>20
posterior papillary muscle – fibrosa distance (mm)	51	>40
lateral wall motion abnormality	+	+
Global left ventricular remodelling		
end-diatolic diameter (mm)	72	>65
end-systolic diameter (ml)	52	>51
end-systolic volume (ml)	198	>140
systolic sphericity index	0.87	>0.7

Treatment of left ventricular aneurysm

Pseudoaneurysms are an absolute indication for surgical repair because untreated pseudoaneurysms have a 50 percent risk of rupture which results in death. True asymptomatic or mildly symptomatic aneurysms should undergo conservative treatment because an anticipated five-year survival of patients is up to 90% [30,31]. The treatment of choice for symptomatic true LV aneurysms is surgery – LV reconstructive surgery also known as LV plasty or Dor procedure. The procedure consists in excision of the scar and placing a patch in its place. It reconstructs ventricular shape, size and restores appropriate geometrical relationship between the ventricle and valve apparatus, improving secondary MR. If the postoperative LV chamber size is too small, pulmo-

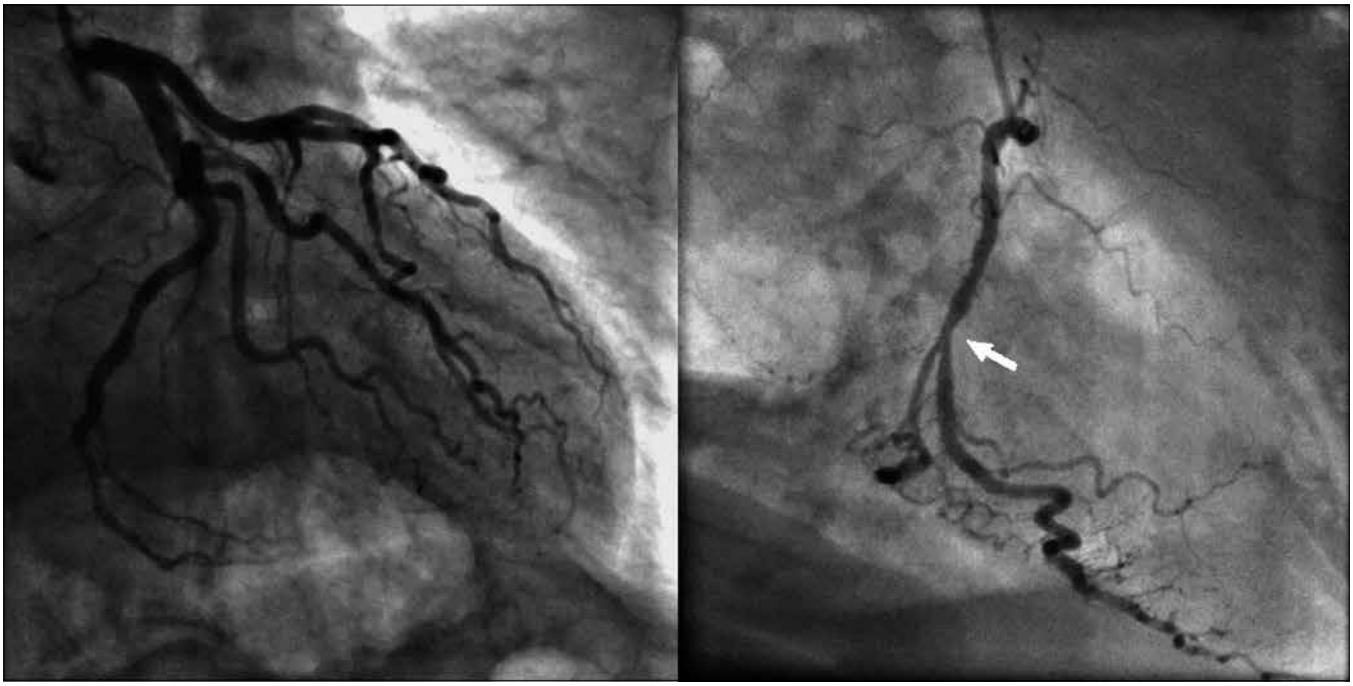

Figure 5. Coronary angiogram. Right anterior oblique view with caudal angulation, non-significant lesions in the left anterior descending artery (left). Right anterior oblique view, a borderline stenosis in the right coronary artery (arrow, right)

nary hypertension may develop, whereas if the ventricle is still too large, hemodynamic parameters may hardly improve. Unique and very sophisticated myocardial fiber architecture is also irrevocably lost [32,33].

The STICH trial (Surgical Treatment for Ischemic Heart Failure) questioned the benefits of LV plasty, that accompanied coronary artery bypass graft procedure, but the trial focused on restoring appropriate chamber volume and not its shape. The trial showed no benefits of LV plasty in terms of short and long term mortality and hospitalization for heart failure. Both trial groups experienced improvement in NYHA, CCS class, 6-minute walk distance and in reduction in symptoms [33].

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