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Case Report

Left Ventricular Aneurysm and Ventricular Septal Defect Following Myocardial Infarction: A Dangerous Cocktail

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ABSTRACT

An ischemic ventricular septal defect (VSD) is a lethal complication of myocardial infarction (MI), commonly from 24 hours to up to 5 days of presentation with AMI. Despite the improvement of surgical techniques, the mortality is still very high with poor prognosis. Left ventricular aneurysm (LVA) may also be a fatal mechanical complication of MI but rarely occurs in the posterior or inferior portion of the interventricular septum. Concomitant AMI mechanical complications in the same patient are less than infrequent with poor prognosis, particularly with late hospital arrival.

We present an unusual case of post-myocardial infarction ventricular septal rupture (PI-VSR) combined with left ventricular inferior/inferoseptal aneurysm that was managed surgically.

The aim of this article is to make clinician alerted in case of mechanical complication, especially when post-MI patients become hemodynamically unstable with refractory congestive heart failure.

KEYWORDS: Ventricular septal rupture, left ventricular aneurysm, inferior wall, myocardial infarction.

INTRODUCTION

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality among ischemic heart diseases worldwide.

Ventricular septal defect (VSD) is one of the most serious mechanical complications of acute myocardial infarction (AMI) leading to acute heart failure, cardiogenic shock (CS) and high mortality rate. It's an abnormal communication between the left and right ventricle through a defect in the septal wall of the heart. Many risk factors are described as female sex, advanced age and systemic hypertension. Surgical repair is the definitive treatment and percutaneous closure is an alternative in high-risk patients. Ventricular septal rupture (VSR) rarely occurs in the posterior or inferior portion of the inter-ventricular septum.

Coronary artery disease and MI cause left ventricular aneurysm. It is mainly due to severe transmural MI after a large coronary artery occlusion. LVA can cause many complications: Cardiac failure, ventricular arrhythmia, systemic embolism and ventricular rupture, all of which can be life-threatening. Only 9% of Left Ventricular Aneurysm (LVA) involves the inferior or posterior left ventricular wall. However, LVA and VSR as two mechanical complications of MI in the same patient are extremely rare.

In this article, we present an unusual case of post-myocardial infarction ventricular septal rupture (PI-VSR) combined with left ventricular aneurysm identified by transthoracic echocardiography (TTE).

CASE PRESENTATION

We herein report a case of a 64-year old male patient who presented to the emergency department with a medical history of diabetes and smoking, shortness of breath, paroxysmal nocturnal dyspnea, and orthopnea lasting several days with bilateral lower limb edema since 2 weeks.

Physical examination documented a blood pressure 130/80 mmHg, tachycardia 110 beats/min, respiratory rate 16 per minute, saturating at 92 % on room air, temperature 37.5 °C. Cardiovascular examination revealed a 4/6 harsh, pan-systolic murmur was appreciated on his left parasternal area and across the pericardium. The patient also had jugular vein distention. The abdominal examination found that the liver was not palpable and bowel sounds were within normal limits.

Electrocardiogram (EKG) shows normal sinus rhythm and QS morphology in leads II, III and AVF suggesting an inferior myocardial infarction (MI). Heart failure treatment was initiated and titrated (Furosemide).

His complete blood count (CBC) revealed a hyperleukocytosis with a white blood cell (WBC) count of 11.8 K/uL (4-10 K/uL), normal renal functions with creatinine level of 13 mg/L (reference range of 6-13 mg/L), measured glomerular filtration rate of 51 mL/min (reference range > 60 mL/min/1.73 m²). Other parameters are within normal limits.

Chest X-ray revealed bilateral reticular opacities with small bilateral pleural effusions. Polymerase chain reaction (PCR) for COVID was negative.

A TEE was performed documenting a hypertensive and ischemic cardiomyopathy, a wide neck aneurysm in the basal segment of the inferior and infero-septal wall of the left ventricle (35x28 mm short axis). There was also a muscular ventricular septal defect in the wall of this aneurysm with a left-to-right shunt (Figure 2-3). Although the aneurysmal wall was dyskinetic, the remaining left ventricular segments were normokinetic with a left ventricular ejection fraction (LVEF) of 48 % (aneurysm included) and a moderate pericardial effusion (Figure 1). A second TEE scanning revealed a double muscular ventricular septal defect in the wall of the aneurysm. The patient underwent coronary angiography revealing complete total occlusion of the distal right coronary artery (RCA) (Figure 4). He was claustrophobic and refuses MRI. He was afterwards referred to cardiac surgery for surgical closure 3 days after (Figure 5).

A postoperative transthoracic echocardiography evaluation (TTE) was performed 6 days after surgery. The VSD was successfully closed. No lethal arrhythmias developed during in-hospital and the patient was discharged from hospital in satisfactory condition (Figure 6).

DISCUSSION

Acute myocardial infarction can cause 3 mechanical complications: Ventricular septal rupture (VSR), papillary muscle rupture and severe mitral regurgitation (MR).

Left ventricular aneurysm (LVA), first reported by Fulton is the most common mechanical complication of ST-segment elevation myocardial infarction (STEMI) [1]. Aneurysms in the inferior wall are uncommon. The incidence of LVA is less than what Coronary Artery Surgery Study (CASS) reported (7.6%) [3].

Aneurysm formation increases the mortality of acute coronary syndrome up to six times beside the patients without aneurysms. Mortality usually occurs as sudden death (4). This is probably due to ventricular arrhythmias, which is one of the most frequent complications of ventricular aneurysms (5). True aneurysms generally do not rupture to pericardium. But they steal the kinetic energy of ventricle by ballooning in systole (6). They are always a potential source of thromboembolism (7)

Ventricular septal defect (VSD) is a rare mechanical complication of myocardial infarction (MI). It usually occurs days 2 to 9 following a trans-mural infarction secondary to complete occlusion of any of the coronary vessels with septal branches supplying the inter-ventricular septum in the absence of collaterals. Occlusion of the left anterior descending (LAD) artery is the most common cause. The incidence of this complication was less than 2 % of all acute myocardial infarction (AMI) without reperfusion [2].

Ventricular septal rupture (VSR) is an abnormal communication between the left and right ventricle through a defect in the septal wall of the heart. It can involve both anterior and inferior MI. The defect caused by anterior MI is apical and simple. VSR due to inferior MI can be more complex with more significant tissue destruction. There have been prior reports of concomitant LV inferior/inferoseptal aneurysm and Ventricular septal defect as parallel complications of an acute inferior ST elevation myocardial infarction from in-stent thrombosis of a drug-eluting stent to the saphenous venous graft-right coronary artery (SVG-RCA) in a patient after coronary artery bypass graft surgery [3]

Our patient developed a VSR following an old inferior myocardial infarction (MI). Haemodynamic deterioration and cardiogenic shock are common in such cases [4]. Some studies found that the in-hospital mortality with an inferior STEMI was increased when combined with VSR [5]. Rarely, however, patients may show only signs of congestive heart failure like our patient.

The risk factors for post-infarction ventricular rupture (PI-VSR) are described as: female sex, advanced age, systemic hypertension, late-hospital arrival. The mortality rate remains high despite the decrease of PI-VSR due to the current era of perfusion and optimal medical treatment [6]. The incidence tends to be 0.2 % nowadays [7]. The co-existence of left ventricle aneurysm (LVA) and VSD is even less common. They have been prior reports of concomitant LVA and VSD. For example, a successful closure of a post-myocardial infarction VSD and an inferior wall aneurysm was performed in the same session at 17 days after an inferior wall MI [8].

The gold standard in the diagnosis of VSR is clinical examination and more specifically cardiovascular auscultation during an acute myocardial infarction. The transthoracic echocardiography is essential as it shows information about the size and location of the rupture. It remains a non-invasive test and the most available tool for diagnosis of such complications in patients who develop heart failure after myocardial infarction.

Surgery is recommended as the first-choice treatment for LVA, and conservative therapy can be considered for appropriate patients [11].

True LV aneurysm can be managed both surgically and medically. Medical management focuses on reducing the risk of embolism and treating underlying congestive cardiac failure. The aim of surgical therapy is restoration of LV geometry, LV volume reduction, and the relief of ischemia by Coronary Artery Bypass Grafting (CABG) in the presence of concomitant coronary artery disease in viable myocardial territory [12]. Many studies demonstrated that concomitant

VSR closure and CABG increases life expectancy compared to VSR closure without CABG. Barker and al. reported survival rates in 30 days, 1 year, 2 years and 4 years in patients with VSR without concomitant CABG of 79.1 %, 58.8%, 49.1% and 32.2% while VSR with CABG reached to 96.2%, 91.6%, 88.8% and 82.8% [13].

In the current case, surgical closure for VSD, although challenging, remains the definitive treatment, with emergent closure for unstable patients. The success rate is 50-86 % and the 30-day mortality rate is 20-39 % if the closure is performed at 14 days or more after MI [9]. American Cardiology College/American Heart Association (ACC-AHA) 2013 practice guidelines have recommended an early surgical closure for unstable and stable patients whereas the 2017 European Society of Cardiology promotes delayed elective repair in patients who initially respond to aggressive conservative management [10].

CONCLUSION

VSR and LVA are uncommon complications of post-MI, particularly with late-hospital arrival. The management requires substantial critical care, imaging, intervention and efficient surgical techniques. Thus, the clinician should be alerted with post-MI patients who become hemodynamically unstable with refractory congestive heart failure. Magnetic resonance gives excellent three-dimensional images of aneurysm but it's unavailable in most of the emergent situations**.**Early diagnosis and surgical intervention are crucial to reduce mortality in this category of patient. However, perioperative mortality of aneurysmal repair is high especially if additional coronary by-pass surgery is needed.

Consent

Informed consent was obtained from the patient for the publication of this case report and its accompanying images.

Competing interests

The authors declare that they have no competing interest.

List of abbreviations:

AMI: Acute myocardial infarction
CS: Cardiogenic shock
LVA: Left ventricular aneurysm
MRI: Magnetic resonance imaging
TTE: Transthoracic echocardiography
VSR: Ventricular septal defect

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FIGURES

Figure 1: Apical two-chamber view of transthoracic echocardiogram showing large aneurysm localized at the basal region of the infero-septal and inferior wall.

Figure 2 – 3: Parasternal short axis (PSAX) view of transthoracic echocardiogram showing a huge left ventricular aneurysm (LVA) with a double ventricular septal defect (VSD) with tortuous path.

Figure 4: Right anterior oblique view (RAO) showing a complete total occlusion of the distal right coronary artery (RCA).

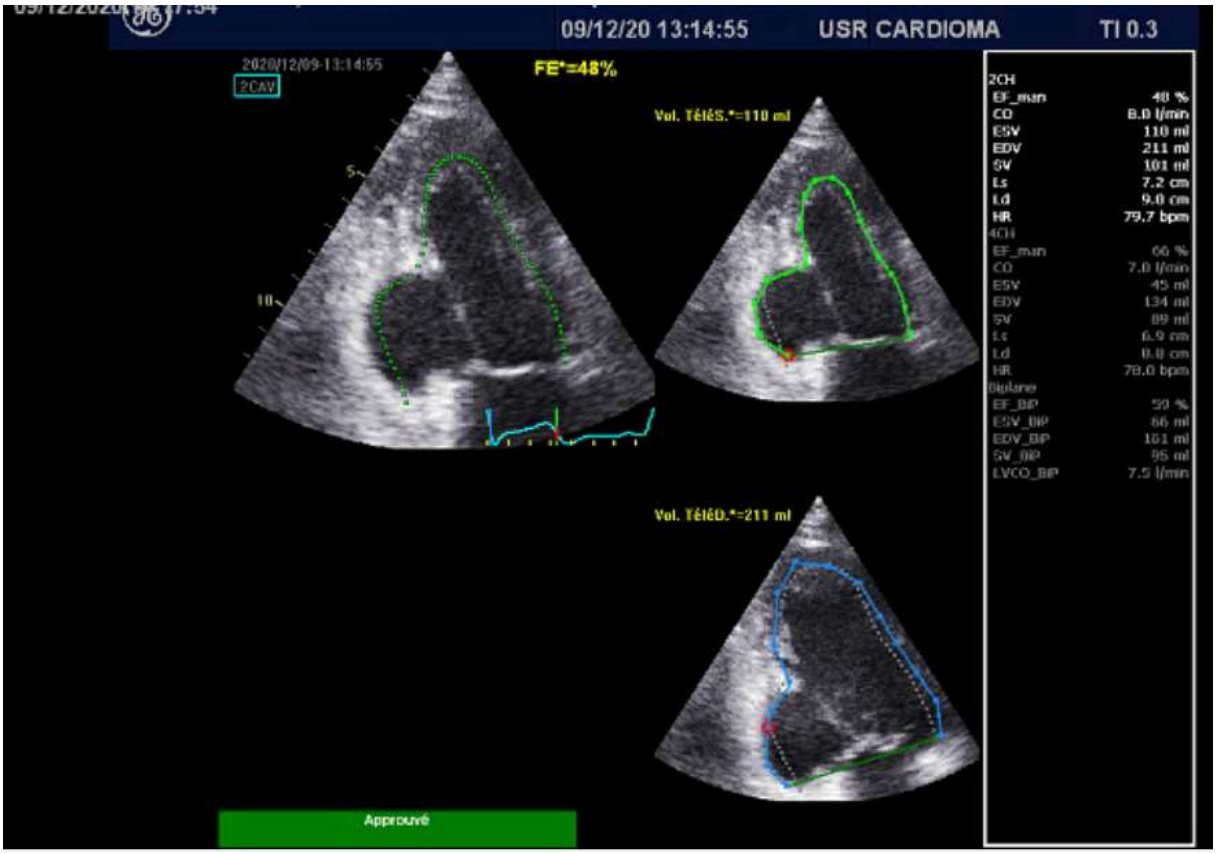
Figure 5: Per-operative view of the aneurysm

(A) After the establishment of cardiopulmonary bypass and identification of the wall perforation.

(B) The left ventricular wall repaired externally with a linear technique.

(C) Double patch (Dacron) was employed to close the septal defect internally and externally

Figure 6: Post-operative echocardiography images with the VSR successfully closed with residual aneurysm of inferioseptal wall.



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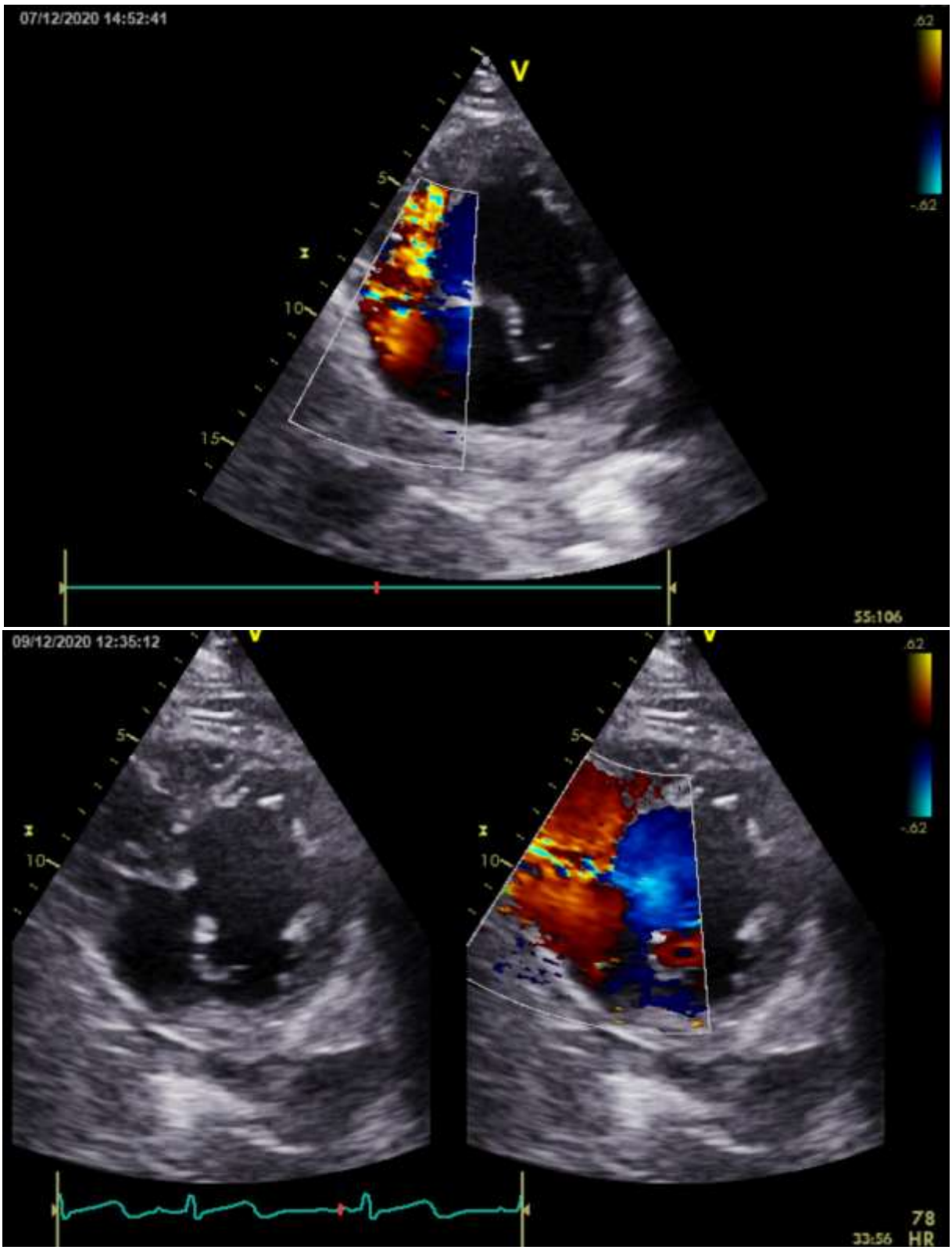


Figure 2-3: Echocardiography images Parasternal short axis (PSAX) view of transthoracic echocardiogram showing a huge left ventricular aneurysm (LVA) with a double ventricular septal defect (VSD) with tortuous path.

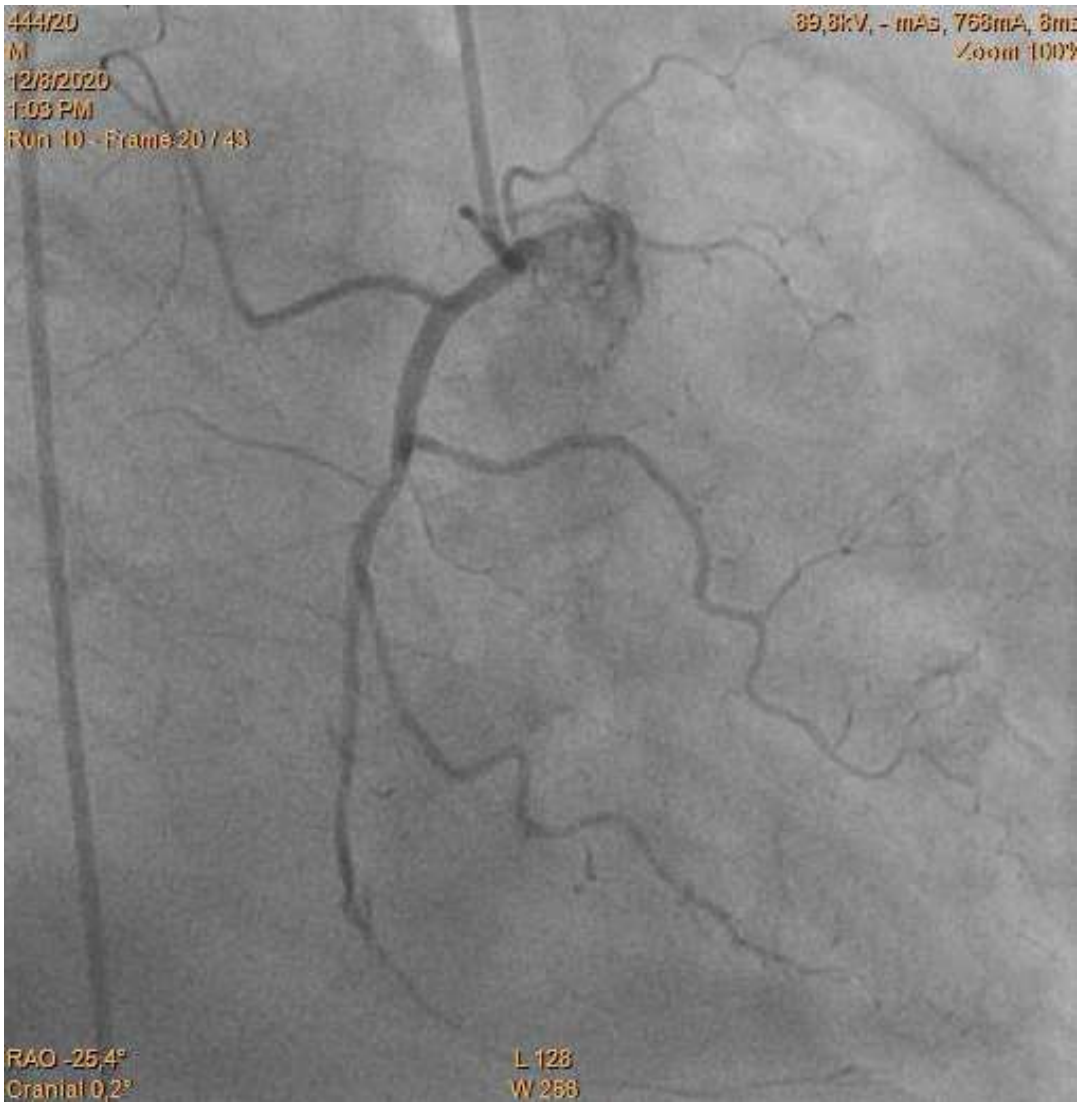
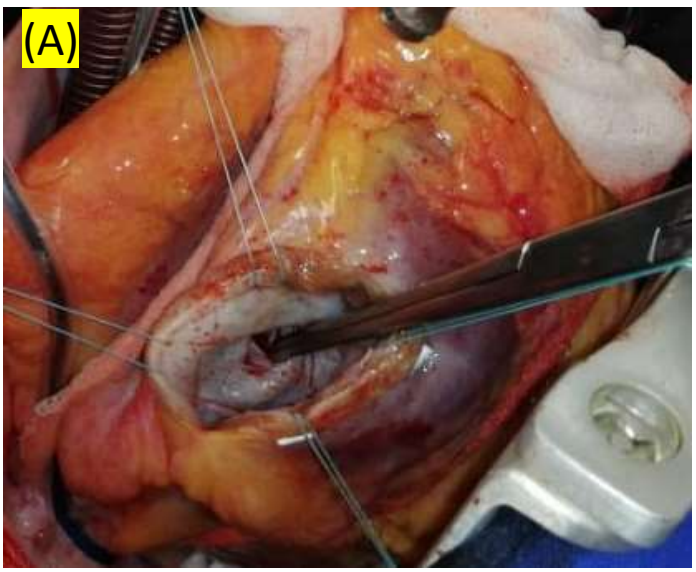
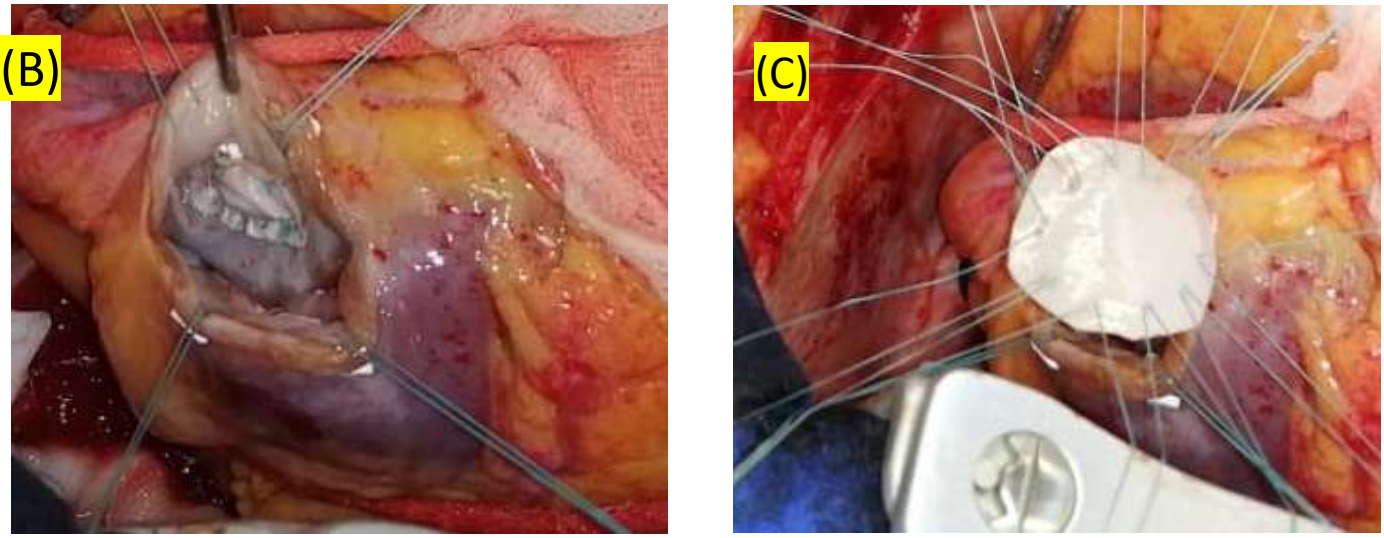


Figure 4: Coronary angiography: Right anterior oblique view (RAO) showing a complete total occlusion of the distal right coronary artery (RCA).





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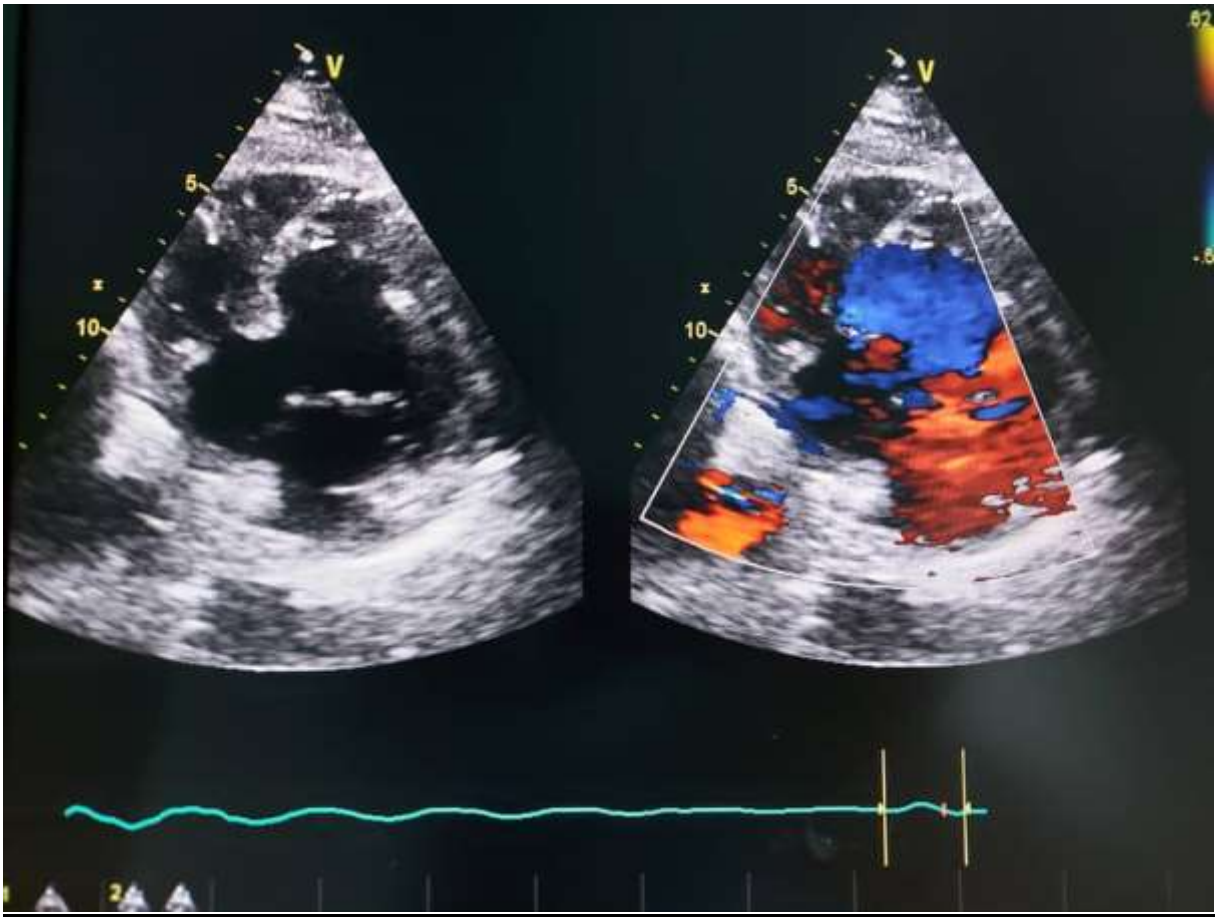

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