Postinfarction Ventricular Septal Defect

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Incidence

- Postinfarction ventricular septal defects complicate approximately 1 to 2% of cases of AMIs and account for about 5% of early deaths after MI.
- The average time from infarction to rupture has been reported to be between 2 and 4 days, but it may be as short as a few hours or as long as 2 weeks.
- occur in men more often than women (3:2).
- The average age of patients with this complication is 62.5 years.
- The vast majority of patients who experience ventricular septal rupture do so after their initial infarction.
- The overall incidence of postinfarction ventricular septal rupture may have decreased slightly during the past decade as a result of aggressive pharmacologic treatment of ischemia and thrombolytic and interventional therapy in patients with evolving MI.

Incidence

- Angiographic evaluation of patients with postinfarction ventricular rupture indicates that septal rupture is usually associated with complete occlusion rather than severe stenosis of a coronary artery , as well as less developed septal collaterals than do other patients with CAD , The lack of collateral flow noted acutely may be secondary to anatomical configuration, edema, or associated arterial disease.
- Postinfarction ventricular septal defects are most commonly located in the **anteroapical septum** as the result of a full-thickness anterior infarction (in approximately **60%** of cases).
- These anterior septal ruptures are caused by anteroseptal MI after occlusion of the left anterior descending (LAD) artery.
- In about **40**% of patients, the rupture occurs in the **posterior septum** after an inferoseptal infarction, which is usually owing to occlusion of a dominant right coronary artery, or less frequently, a dominant circumflex artery .

Pathogenesis

- The infarct associated with septal rupture is **transmural** and generally quite extensive.
- **There are two types of rupture**:
- simple, consisting of a direct through-and-through defect usually located anteriorly.
- and complex, consisting of a serpiginous dissection tract remote from the primary septal defect, which is usually located inferiorly.
- Multiple defects, which may develop within several days of each other, occur in 5 to 11% of cases and are probably caused by infarct extension.
- Of the small number of patients who survive the early period of ventricular septal rupture, 35 to 68% go on to develop ventricular aneurysms through the process of ventricular remodeling.
- Postinfarction septal rupture, especially in the posterior septum, may be accompanied by mitral valve regurgitation resulting from papillary muscle infarction or dysfunction.
- In approximately one-third of cases of septal rupture, there is a degree of mitral insufficiency, usually functional in nature, secondary to left ventricular (LV) dysfunction with mitral annular dilation, which usually resolves with repair of the defect.

Pathogenesis

- The most important determinant of early outcome after postinfarction ventricular septal rupture is the development of heart failure (left, right, or both).
- The degree to which heart failure develops depends on the size of the ventricular infarction and the magnitude of the left to-right shunt.
- **Left ventricular dysfunction** resulting from extensive necrosis of the left ventricle is the primary determinant of CHF and cardiogenic shock in patients with anterior septal rupture.
- **Right ventricular dysfunction** secondary to extensive infarction of the right ventricle is the principal determinant of heart failure and cardiogenic shock in patients with posterior septal rupture.
- With the opening of a VSD, the heart is challenged by an increase in pulmonary blood flow, and a decrease in systemic blood flow, as a portion of each stroke volume is diverted to the pulmonary circuit.
- As a consequence of the sudden increase in hemodynamic load imposed on a heart already compromised by acute infarction, and possibly by a ventricular aneurysm, mitral valve dysfunction, or a combination of these problems, a severe low cardiac output state results.

Diagnosis

- The typical presentation of a ventricular septal rupture is that of a patient who has suffered an AMI, and who after convalescing for a few days develops a new systolic murmur, recurrent chest pain, and an abrupt deterioration in hemodynamics.
- The development of a loud systolic murmur, usually within the first week after an AMI, is the most consistent physical finding of postinfarction ventricular septal rupture (present in greater than 90% of patients).
- The murmur is usually harsh, pansystolic, and best heard at the left lower sternal border.
- The electrocardiographic (**ECG**) findings in patients with acute septal rupture relate to the changes associated with antecedent anterior, inferior, posterior, or septal infarction.
- Up to one-third of patients develop some degree of atrioventricular conduction block (usually transient) that may precede rupture.
- The chest radiograph usually shows increased pulmonary vascularity consistent with pulmonary venous hypertension.

Diagnosis

- It is important to realize that the sudden appearance of a systolic murmur and hemodynamic deterioration after infarction may also result from acute mitral regurgitation caused by a ruptured papillary muscle.
- systolic murmur associated with a septal rupture is more prominent at the left sternal border, whereas the murmur resulting from a ruptured papillary muscle is best heard at the **apex**.
- Second, the murmur associated with septal perforation is **loud** and associated with a **thrill** .
- Advances in transthoracic and transesophageal echocardiography, especially color flow Doppler mapping, have revolutionized the diagnosis of both the presence and site of septal rupture.
- Echocardiography can detect the defect, localize its site and size, determine right and left ventricular function, assess pulmonary artery and right ventricular pressures, and exclude coexisting mitral regurgitation or free wall rupture .

Management

- Use of the intra-aortic balloon pump (**IABP**), whenever technically feasible, frequently results in transient reversal of the hemodynamic deterioration.
- Persistence of **CHF** or marginal stabilization with rising blood urea nitrogen and borderline urine output necessitate aggressive therapy and prompt operation.
- patients in **cardiogenic shock** represent a true surgical emergency requiring immediate intervention, which may include surgery.
- Few patients who are completely stable, with no clinical deterioration, and who require no hemodynamic support, can undergo operative repair when convenient during that hospitalization.
- The large group of patients who are in an intermediate position between those with shock and those in stable condition should have intervention early (usually within 12 to 24 hours) after appropriate preoperative evaluation.

Preoperative Management

- Directed toward stabilization of the hemodynamic condition so that peripheral organ perfusion .
- 50 to 60% present with severe CHF and a low cardiac output state requiring intensive therapy.
- **The goals of preoperative management are to**:
- (1) reduce the systemic vascular resistance, and thus the left-to-right shunt.
- (2) maintain cardiac output and arterial pressure to ensure peripheral organ perfusion.
- (3) maintain or improve coronary artery blood flow.
- This is best accomplished by the IABP.
- Although counterpulsation produces an overall improvement in the patient's condition, a complete correction of the hemodynamic picture cannot be obtained.
- Pharmacologic therapy with inotropic agents and diuretics should be instituted promptly.

Operative Techniques

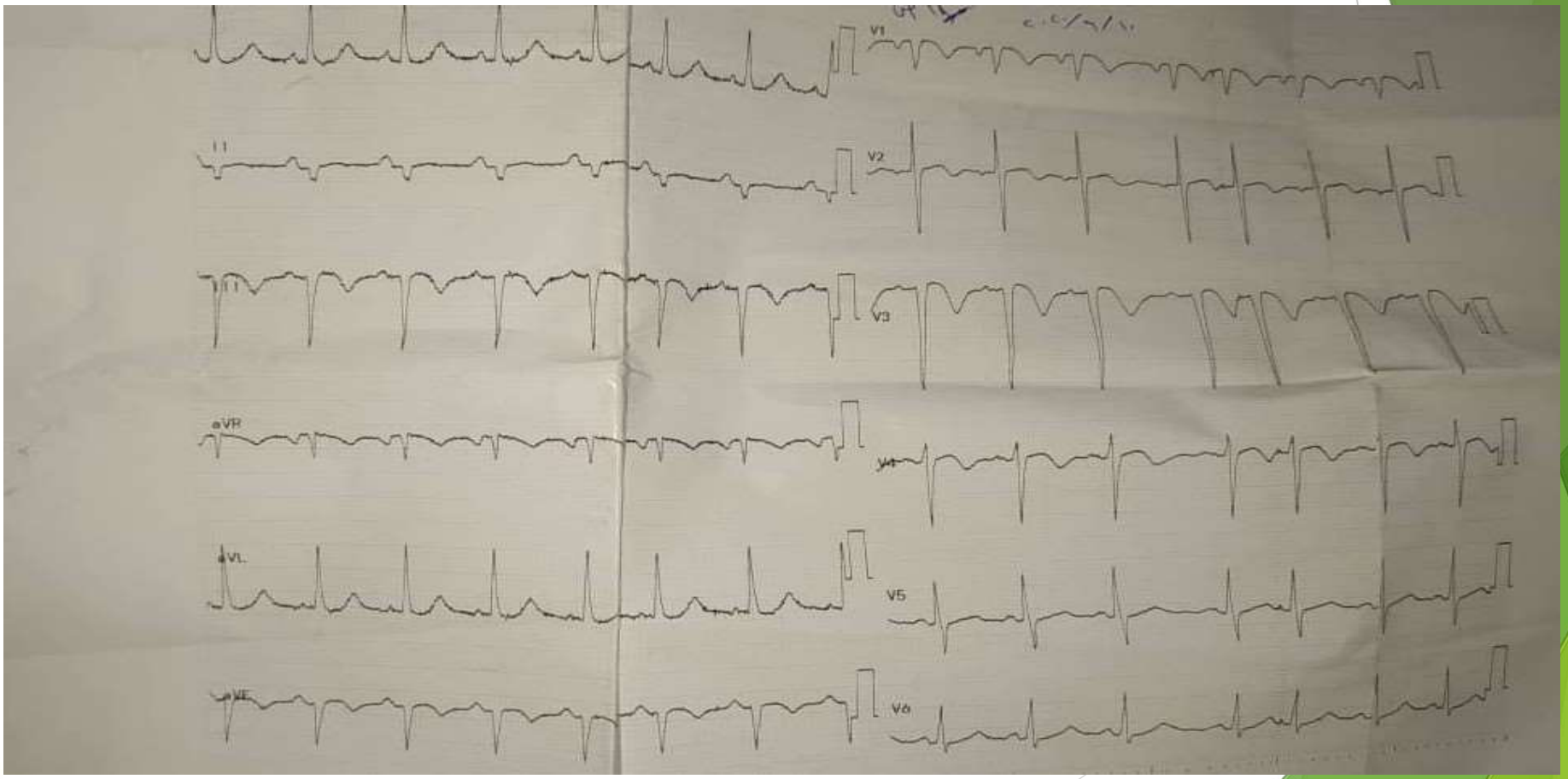
- **Transinfarct approach to ventricular septal defect**
- 1. Thorough trimming of the left ventricular margins of the infarct back to viable muscle to prevent delayed rupture of the closure .
- 2. Conservative trimming of the right ventricular muscle as required for complete visualization of the margins of the defect .
- 3. Inspection of the left ventricular papillary muscles and concomitant replacement of the mitral valve only if there is frank papillary muscular rupture.
- 4. Closure of the septal defect without tension, which in most instances will require the use of prosthetic material.
- 5. Closure of the infarctectomy without tension with generous use of prosthetic material as indicated, and epicardial placement of the patch to the free wall to avoid strain on the friable endocardial tissue.
- 6. Buttressing of the suture lines with pledgets or strips of Teflon felt or similar material to prevent sutures from cutting through friable muscle

CLINICAL CASE

History

- The patient Mohammad M , 64 years of age , was taken to the mouassat hospital on May 20 – 2020 after developing crushing retrosternal chest pain and was diagnosed with an acute MI .
- He was admitted into the ICU for 2 days , where then checked himself out on his behalf .
- After a couple of days he developed dyspenea that was worsening , and he went to a private hospital on May 30 ,2020 and underwent coronary catheterization and an angiogram . And was told that he needed emergent cardiac surgery .
- The patient is a heavy smoker and had T2DM.
- He also underwent a previous coronary catheterization with stent placement in 2014 to LAD and the following year in 2015 to LAD .

ECG



T - wave inversion (v1 to v4). Q development inferior

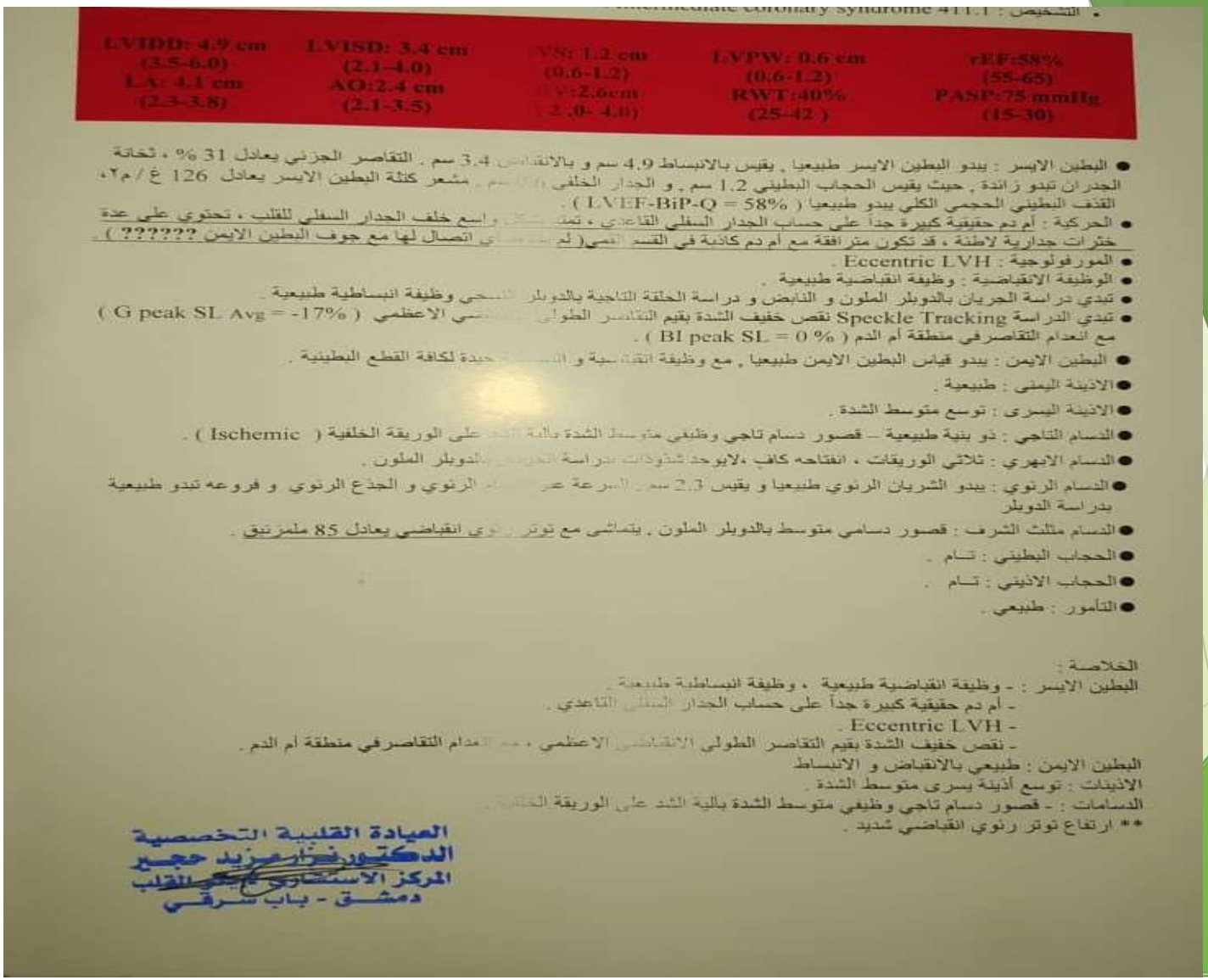
FT. CORONAY : L.M

L A D

C X

GHT CORONARY

FT VENTRICULOGRAM



- التشخيص : Intermediate coronary syndrome 411.1

LVIDD: 4.9 cm (3.5-6.0)	LVISD: 3.4 cm (2.1-4.0)	VS: 1.2 cm (0.6-1.2)	LVPW: 0.6 cm (0.6-1.2)	rEF:58% (55-65)
LA: 4.1 cm (2.3-3.8)	AO:2.4 cm (2.1-3.5)	:2.6cm (2.0- 4.0)	RWT:40% (25-42)	PASP:75 mmHg (15-30)

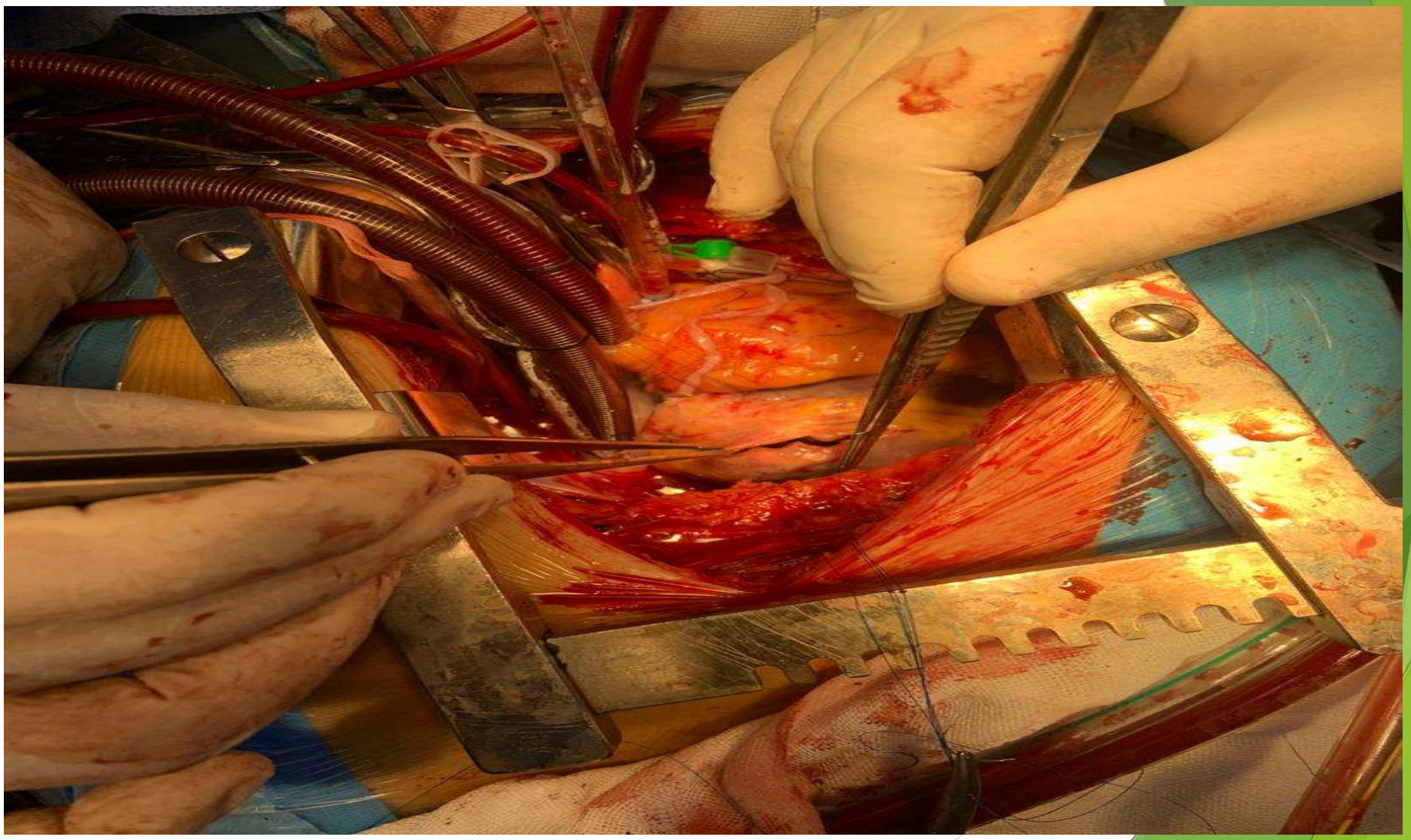
- البطين الايسر : يبدو البطين الايسر طبيعيا , يقيس بالانبساط 4.9 سم و بالانقباض 3.4 سم . التقاصر الجزئي يعادل 31 % ، ثخانة الجدران تبدو زائدة , حيث يقيس الحجاب البطيني 1.2 سم , و الجدار الخلفي سم , مشعر كتلة البطين الايسر يعادل 126 غ / م٢، القذف البطيني الحجمي الكلي يبدو طبيعيا (LVEF-BiP-Q = 58%) .
- الحركية : ثم دم حقيقية كبيرة جداً على حساب الجدار السفلي القاعدي ، تمد واسع خلف الجدار السفلي للقلب ، تحتوي على عدة جيوب جدارية لاطئة ، قد تكون مترافقة مع ثم دم كاذبة في القسم ي اتصال لها مع جوف البطين الايمن ؟؟؟؟؟) .
- المورفولوجية : Eccentric LVH .
- الوظيفة الانقباضية : وظيفة انقباضية طبيعية .
- تبدي دراسة الجريان بالدوبلر الملون و النابض و دراسة الحلقة التاجية بالدوبلر النسجي وظيفة انبساطية طبيعية .
- تبدي الدراسة Speckle Tracking نقص خفيف الشدة بقيم التقاصر الطولي سي الاعظمي (G peak SL Avg = -17%) مع انعدام التقاصر في منطقة أم الدم (BI peak SL = 0 %) .
- البطين الايمن : يبدو قياس البطين الايمن طبيعيا , مع وظيفة انقباضية و جيدة لكافة القطع البطينية .
- الاذينة اليمنى : طبيعية .
- الاذينة اليسرى : توسع متوسط الشدة .
- الدسام التاجي : ذو بنية طبيعية – قصور دسام تاجي وظيفي متوسط الشدة بالية على الوريقة الخلفية (Ischemic) .
- الدسام الابهري : ثلاثي الوريقات ، انفتاحه كافٍ ،لايوجد تشوهات بدراسة الدوبلر الملون .
- الدسام الرئوي : يبدو الشريان الرئوي طبيعيا و يقيس 2.3 سم , السرعة عبر الرئوي و الجذع الرئوي و فروعه تبدو طبيعية بدراسة الدوبلر
- الدسام مثلث الشرف : قصور دسامي متوسط بالدوبلر الملون , يتماشى مع توتر رئوي انقباضي يعادل 85 ملمزئبق .
- الحجاب البطيني : تام .
- الحجاب الاذيني : تام .
- التأمور : طبيعي .

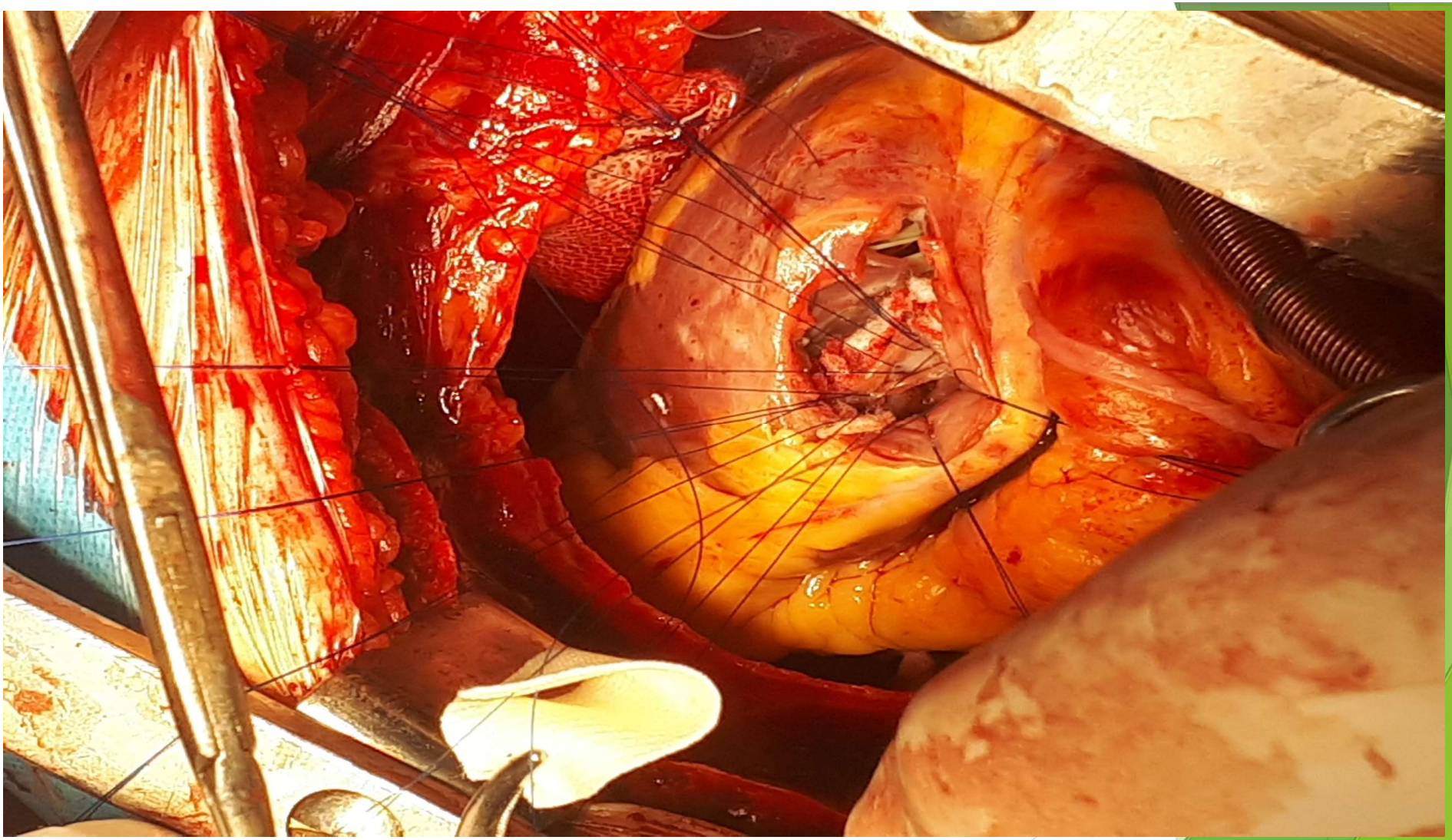
الخلاصة :
البطين الايسر : - وظيفة انقباضية طبيعية ، وظيفة انبساطية طبيعية .
- أم دم حقيقية كبيرة جداً على حساب الجدار السفلي القاعدي .
- Eccentric LVH .
- نقص خفيف الشدة بقيم التقاصر الطولي الانقباضي الاعظمي ، مع انعدام التقاصر في منطقة أم الدم .
البطين الايمن : طبيعي بالانقباض و الانبساط
الاذينات : توسع أذينة يسرى متوسط الشدة .
الدسامات : - قصور دسام تاجي وظيفي متوسط الشدة بالية الشد على الوريقة الخلفية .
** ارتفاع توتر رئوي انقباضي شديد .

العيادة القلبية التخصصية
الدكتور فراس يزيد حجير
المركز الاستشاري لأمراض القلب
دمشق - باب شرقي

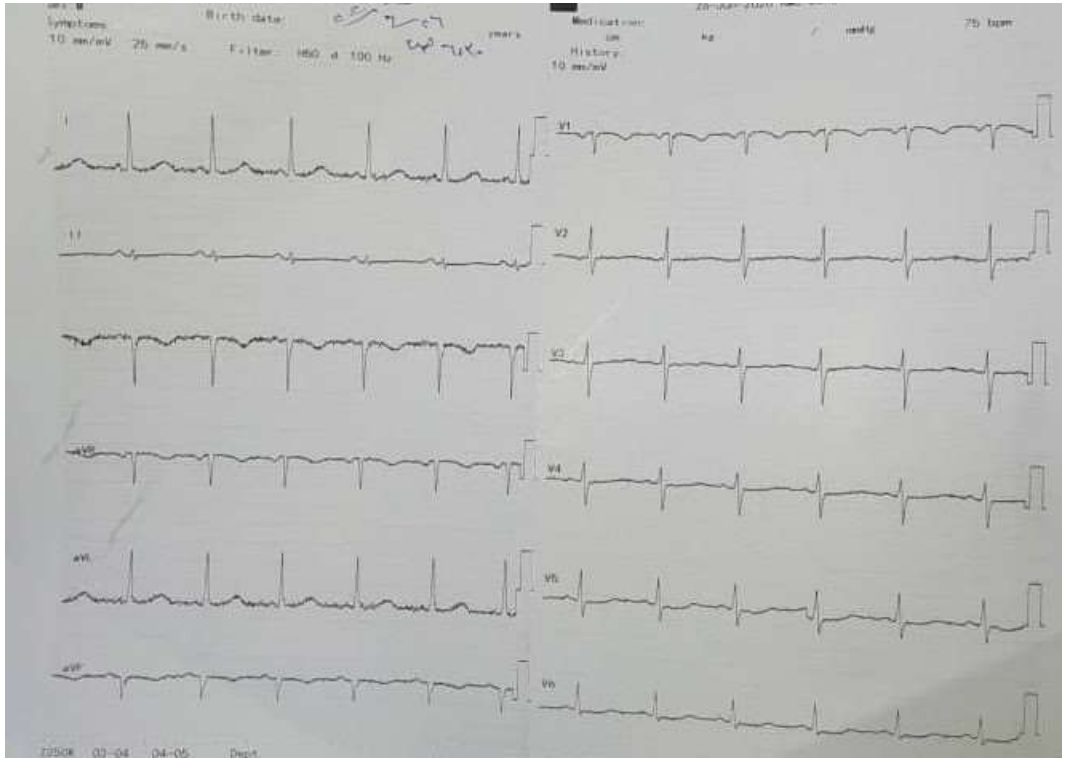
Operation : 23-6-2020.

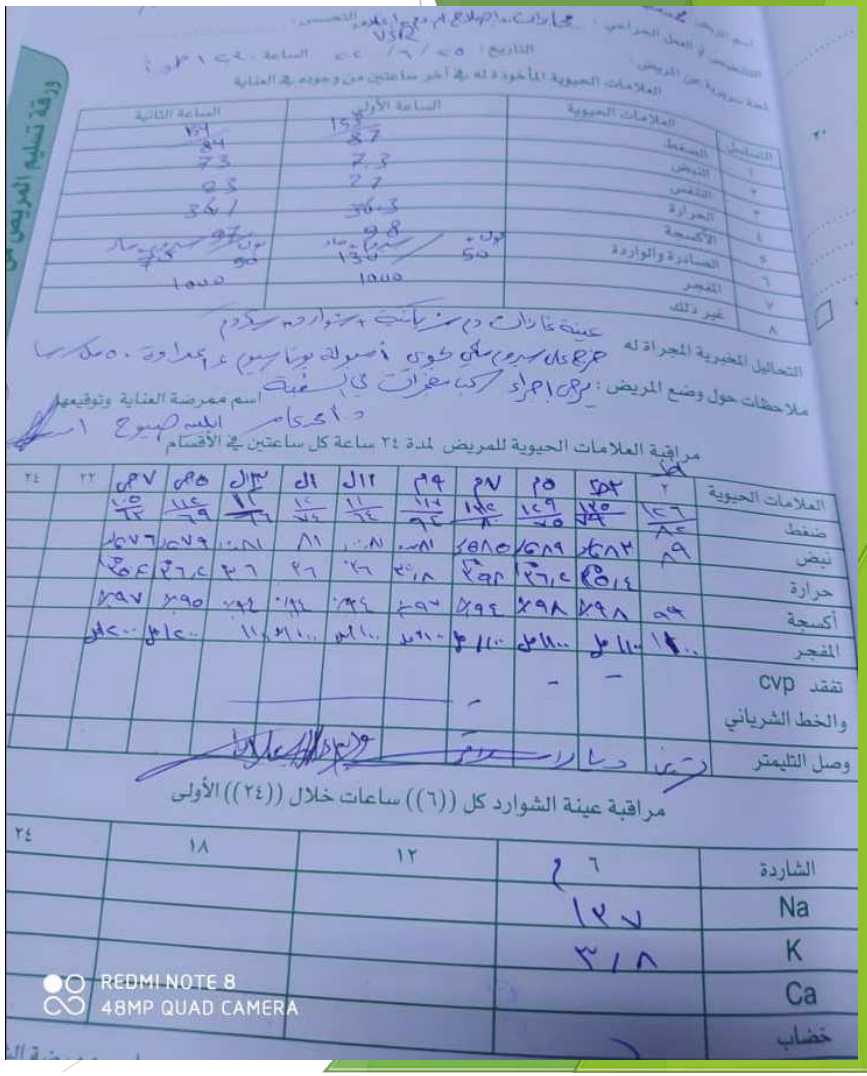
- Median sternotomy , heparin UFH , Bi-caval canullation .
- Antegrade blood cardioplasia .
- Distal anastomosis :
- Svg to LAD (PART 3) .
- Svg to Rca.
- **Transinfarct aneurism(2*2c.m) basal wall approach to ventricular septal defect incision in the inferior wall to L.V .**
- Inspection of the VSR (posterior ventricular septum with patent papillary muscles measure about 1.5 *1.5 c.m) and close with Dacron patch with Teflon pledgets without tension.
- Close the wall of left ventricle with Teflon strips (plication) .
- Proximal anastomosis to ascending aorta .
- De airing maneuver .
- Weanning of CPB… with introps
- Protamine and close the chest.
- **Surgen : Dr.Najdat Naseif (head of cadiac surgery department)**





IN the room




ورقة تسليم المريض
الساعة الأولى
الساعة الثانية
العلامات الحيوية
مراقبة العلامات الحيوية للمريض لمدة ٢٤ ساعة كل ساعتين في الأقسام
مراقبة عينة الشوارد كل ((٦)) ساعات خلال ((٢٤)) الأولى
الشاردة
Na
K
Ca
خضاب
REDMI NOTE 8
48MP QUAD CAMERA


THANK YOU
for listening!