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Post-Myocardial Ventricular Septal Defect, Risk Factors and Epidemiological Aspects

Nabil Laktib[1*], Zakaria Lahlafi[1], Aymane Ouriaghli[2], Oussama Ait Kejjat[1], Fouad Nya[3], Mahdi Bamous[3], Noureddine Atmani[3], Anis Seghrouchni[3], Zouhair Lakhal[1], Younes Moutakillah[3], Aatif Benyass[4]

[1]Intensive Care Unit of the Cardiology Center of Mohammed V Military Teaching Hospital, Rabat, Morocco
[2]Emergency Department of Mohammed V Military Teaching Hospital, Rabat, Morocco
[3]Cardiac Surgery Department of the Cardiology Center of Mohammed V Military Teaching Hospital, Rabat, Morocco
[4]Cardiology Center of Mohammed V Military Teaching Hospital, Rabat, Morocco

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***Corresponding author:** Nabil Laktib
Intensive Care Unit of the Cardiology Center of Mohammed V Military Teaching Hospital, Rabat, Morocco

Abstract **Original Research Article**

Introduction and aims*:** Post-myocardial infarction ventricular septal defect is a rare dreadful complication of myocardial infarction. It usually occurs between the 3rd and 7th day of the infarction. Several risk factors for its occurrence have been identified. The aim of our work is to discuss these different risk factors through clinical cases with a literature review. ***Material and methods*:** This is a retrospective study conducted from 2015 to 2021 including 18 consecutive patients with post-myocardial ventricular septal defect presented in either cardiac intensive care unit or cardiac surgery department of Mohammed V Military Teaching Hospital Rabat. All patients had at least a clinical examination, an electrocardiogram, and an echocardiogram showing ventricular septal defect. Surgery was performed at cardiac surgery department. ***Results: Mean age was 65.2± 26.4 years. There were more men than women in our study (17 males/1 female). Diabetes (55.6%) and smoking (55.6%) were the two predominant cardiovascular risk factors. The average body mass index was 23.7 ± 2.98 kg/m². Anteroseptal was the most observed infarct location (38.9%.) 16 patients presented after 12 hours of pain onset. 2 of them underwent percutaneous intervention and 1 underwent coronary artery bypass graft intervention whereas 15 didn't undergo any revascularisation strategy. The culprit artery was left anterior descending artery in 13 patients. The death rate was 55.6%, 50% before surgery and 50% after surgery. ***Conclusion***: Delayed or absence of coronary reperfusion remains the main risk factor for post-myocardial infarction ventricular septal defect occurrence which explains its frequency decline since reperfusion strategies development.

Keywords: Myocardial infarction; Ventricular septal defect; Risk factor.

INTRODUCTION

Post-myocardial infarction ventricular septal defect (PMIVSD) is a rare and potentially lethal mechanical complication of myocardial infarction. It usually occurs between the 3rd and 7th day of the infarction [1]. The prevalence of PMIVSD was around 1 to 2% of all myocardial infarctions before the era of coronary reperfusion [1, 2]. It has been reduced to less than 0.2% thanks to early reperfusion strategies [1, 2]. PMIVSD is an extreme emergency. Its management must involve cardiologist, cardiovascular surgeon and resuscitator. The main complication of PMIVSD remains cardiogenic shock with hemodynamic instability which can rapidly cause death despite surgical or interventional treatment. Several risk factors for the occurrence of PMIVSD have been identified such as advanced age, hypertension, female gender, lower body mass index (BMI) [1, 3]. The aim of our work is to discuss these different risk factors through 18 clinical cases with a literature review.

MATERIAL AND METHODS

This is a retrospective study from 2015 to 2021 including 18 consecutive patients presented in either cardiac intensive care unit or cardiac surgery department of Mohammed V Military Teaching Hospital Rabat. Patients with PMIVSD were included. Patients with congenital or iatrogenic VSD and patients with incomplete medical records were excluded. All patients had at least a clinical examination, an electrocardiogram, and an echocardiogram showing

ventricular septal defect. Surgery was performed at cardiac surgery department.

Demographic data (age / sex, and common cardiovascular risk factors such as diabetes, hypertension, smoking, high serum low density lipoprotein cholesterol, obesity and coronary artery disease (CAD) family history), body mass index (BMI), ST-Elevation Myocardial Infarction (STEMI) location, culprit, Killip class, reperfusion strategy, ventricular septal defect location and death rate were recorded. Medical contact time limit was set to before and after 12 hours.

RESULTS

Table I: Patients characteristics (1)

	Values (n= 18)
Demographic data, CVRF	
Age (year-old)	65.2 ± 26.4
Female	1 (5.6 %)
Male	17 (94.4%)
Diabetes	10 (55.6%)
Hypertension	4 (22.2%)
Smoking	10 (55.6%)
Dyslipidaemia	4 (22.2%)
CAD family history	1 (5.6%)
Obesity	3 (16.7%)
BMI (kg/m²)	23.7 ± 2.98
STEMI location:	
Anteroseptal	7 (38.9%)
Extensive anterior	3 (16.7%)
Inferior	5 (27.8%)
Septal	3 (16.7%)
Medical contact time	
Less than 12 hours	3 (16.7%)
More than 12 hours	15 (83.3%)
Killip class and cardiogenic shock:	
Class I	1 (5.6%)
Class II	1 (5.6%)
Class III	9 (50%)
Cardiogenic shock	7 (38.9%)

Table II: Patients characteristics (2)

	Values (n= 18)
Reperfusion strategy	
Primary percutaneous coronary intervention	2 (11.2 %)
Thrombolysis	1 (5.6 %)
Coronary artery bypass graft	3 (16.7%)
No reperfusion	12 (66.5%)
Culprit artery	
Left anterior descending artery	13 (72.2%)
Right coronary artery	2 (11.1%)
Circumflex	3 (16.7%)
VSD location	
Apical	9 (50%)
Posterior	5 (27.8%)
Anterior	3 (16.7%)
Inferoseptal	1 (5.6%)
Death rate	
Before surgery	10 (55.6%)
After surgery	5 (50%)

DISCUSSION

PMIVSD is a defect in the interventricular septum secondary to transmural myocardial infarction. It is a rare and dreadful complication with a very high mortality rate (97% at one month follow-up despite coronary reperfusion) [1]. It should be suspected when a systolic murmur and signs of heart failure appear. The diagnosis is confirmed by transthoracic echocardiography or ideally, transoesophageal echocardiography. The ventricular septal defect, its location and size, the restrictive nature of the flow and the direction of the shunt should be assessed. The rupture is anterior in 69% of cases and posterior in 31% [4].

The patients in our study were elderly subjects. Mean age was 65.2 ± 26.4-year-old (minimum 51-year-old and maximum 81-year-old) (Table I). According to the SHOCK trial [5], age is a predictive factor for the occurrence of PMIVSD. The mean age of patients in this trial was 72 ± 10 years. The hypothesis suggested is myocardial structures ageing as well as the efficiency decrease of myocardial protective mechanisms that both lead to septal rupture occurrence [6]. The exact mechanisms of the heart self-protective capacity loss and myocardial senescence are not well known [7].

Patients gender differs between studies. In our study, most patients were males (Table I). In series before reperfusion era [8, 9], there was a male predominance, as in our study. In more recent studies, such as the SHOCK trial [5] and Ledakowicz-Polak *et al*., [6], a clear predominance of female patients was observed. The hypothesis put forward is that female heart is more susceptible to collagen fiber rupture due to its structure fragility [6].

In the Ledakowicz-Polak *et al*., study [6], left ventricular hypertrophy due to hypertension is a PMIVSD risk factor. In fact, septal hypertrophy, predisposes to PMIVSD occurrence due to myocardium structure alteration following uncontrolled hypertension [1, 3]. Furthermore, patients with low blood pressure are more likely to develop PMIVSD. As diastolic pressure is responsible for maintaining coronary flow, its reduction could lead to greater extension of necrosis and thus to septal rupture occurrence [10, 11].

A low BMI may predispose to the development of PMIVSD [6]. In several studies, most patients had a BMI <25 kg/m² compared with patients without PMIVSD. The exact mechanism is not clearly understood [6, 12]. Mean BMI in our study was <25kg/m² (Table I), which is consistent with the literature data.

Anterior, inferior and apical STEMI are often encountered in PMIVSD and the culprit artery is usually occluded with 1 or even 0 TIMI flow score according to the SHOCK trial [5]. PMIVSD occurrence probability increases in case of no performed revascularization and delayed myocardial infarction diagnosis [6]. In our study, myocardial infarction diagnosis was established within 12 hours in only 3 patients (Table I). Medical contact for most patients had been made after 12 hours since chest pain onset. Also, inaugural infarction and lack of collateral vessels leads to necrosis transmural extension and to PMIVSD occurrence [5]. The most involved artery in our study was the left anterior descending artery (LAD) (Table II). Its association with PMIVSD is due to a larger necrosis area as the LAD supplies a larger territory [6]. In the other hand, smoking is thought to be a protective factor which is not consistent with our study results. The causal relationship has not been established yet [1].

CONCLUSION

Delayed coronary reperfusion remains the main risk factor for PMIVSD occurrence which explains its frequency decline since reperfusion strategies development.

Compliance with ethical standards.
Conflicts of interest: The authors report no conflicts of interest.

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