Original Article

Journal of Cardiovascular Medicine and Surgery
Volume 3 Number 2, July - December 2017
DOI: http://dx.doi.org/10.21088/jcms.2454.7123.3217.16

Winking of an Infarct Related Artery Overlying Ventricular Septal Rupture Complicating Acute MI: An Angiographic "Winking Coronary" Sign

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Recived on 15.06.2017,
Accepted on 08.08.2017

Abstract

Background: Post myocardial infarction is a fatal condition needing immediate attention. *Objectives:* This study intends to report a unique angiographic finding – "Winking coronary" sign of infarct related artery (IRA) overlying the ventricular septal rupture (VSR) complicating acute myocardial infarction (AMI). *Materials and Methods:* Retrospective analysis of 15 patients (mean age - 62.43 ± 8.4 years) having VSR after AMI were included and coronary angiogram of all these patients were evaluated. Thirteen patients had anterior wall and 2 had inferior wall AMI. *Results:* Majority of the patients had LAD (n=13; 86.7%) and only 13.3% (n=2) had RCA as an involved IRA. None of the patient showed association of LCX as a culprit artery. Only 1 patient had completely occluded IRA, where as all other (n=14; 93.3%) patients showed winking coronary sign on coronary angiogram. In all 4 patients' who had undergone surgical closure of VSR, the anatomic site of the "wink" was well correlated with the defect at the time of surgery. *Conclusions:* "Winking coronary" sign of the IRA is sensitive and specific feature of underlying VSR complicating a case of AMI on coronary angiogram.

Keywords: Acute Myocardial Infarction; Ventricular Septal Rupture; Infarct Related Artery; Coronary Angiography.

Background

Ventricular septal rupture (VSR) is an uncommon but life threatening complication of acute myocardial infarction (AMI) with significantly high mortality. Though its management is quite challenging, quick diagnosis and definitive surgery remains the treatment of choices for this condition. Concomitant coronary artery bypass grafting (CABG) is known to improve the long term survival outcome in patients undergoing post MI VSR surgery.

The septal blood supply comes from the branches of left anterior descending coronary artery, the posterior descending branch of the right coronary artery, or the circumflex artery when it is dominant. The nature of infarction contributing to VSR is often transmural and extensive. In contrast to previous reports, where time for VSR development after infarction was found to be 3 – 5 days, recent studies claim it to be 1st day, indicating role played by the different pathophysiological mechanisms [1-3]. Popular and extensive usage of thrombolytic therapies may have contributed to this as it may also induce "myocardial haemorrhage" leading to reduction in time span needed for VSR manifestation. The quantum of left-to-right shunt is a critical factor involved in the hemodynamic instability complicating post MI VSR. This often results in right ventricular volume overload, increased pulmonary blood flow, and secondary volume overload of the left atrium and ventricle. Moreover, the extent of shunting largely depend on the size of the septal rupture, left and right ventricular function, the level of pulmonary vascular resistance and systemic vascular resistance and the ratio of the two.

The frequency of septal rupture is reported to be high with infarction of anterior wall MI (60%) as

compared to inferior wall MI (contributing in about 40% of post MI VSR) [2,4,5-7]. Though the multi-vessel coronary artery disease was found to be associated with septal rupture, the prevalence of single vessel disease (54%) was higher amongst the patients with VSR [2,4,8]. One of the key angiographic finding noted in post MI VSR incidences is the total occlusion of the infarct related artery (IRA) [1,5,10].

This could be due to associated arterial disease, anatomic anomalies, or myocardial oedema. Post MI, coronary collateral circulation flow to the affected area is escalated as a compensatory healing mechanism and is associated with improved outcome [9]. However this protective action is found to be compromised in patients with VSR supporting the hypothesis that collateral circulation reduces the risk of rupture of the cardiac free wall as well as septal rupture [4,10]. Previously two independent studies have shown, total occlusion of the infarct-related artery in 57% of patients with VSR, whereas 82% of the population had fairly compromised or absent collateral circulation to the infarct territory [5,10].

Objectives

Herewith we intend to report a unique angiographic finding – "Winking coronary" sign (partial occlusion) of IRA overlying the VSR complicating Acute MI.

Materials and Methods

Study Design and Patient Selection

A retrospective analysis was performed on 15 consecutive patients with VSR following AMI who were presented to 4 cardiac catheterisation labs in the city of Ahmedabad, India between August 2008 and February 2017. Coronary angiograms of all these patients were analysed.

Inclusion criteria's were any patient admitted for AMI who had undergone cardiac catheterization with echocardiographic evidence of VSR. Diagnosis of AMI was made based on clinical symptoms and elevation of serum troponin-T (>0.1mg/dL) with or without electrocardiogram (EKG) evidence of >2mm ST-segment elevation in the precordial leads or >1mm ST-segment elevation in the limb leads. All patients were analysed for their demographic profile, treatments including medical and surgical. All 14 coronary angiograms were analysed by three different interventional cardiologists for the presence of the "Winking coronary" sign and sign was considered present if confirmed by all 3 of them.

Cardiac Catheterization

Patients with diagnosis of AMI with VSR, were taken for early coronary angiography with or without placement of IABP. These 15 patients had prior echocardiographic confirmation of VSR.

Coronary artery disease (CAD) was defined based the degree of obstruction. No apparent CAD was defined as no stenosis greater than 20%. Non-obstructive CAD included at least 1 or more lesions with stenosis greater than 20% but less than 70%. Obstructive CAD was defined as any stenosis greater than 70% or left main stenosis greater than 50% with a distribution involving 1, 2 or 3 vessels. Only 3 patients underwent successful PCI with coronary stents.

Echocardiography

All patients underwent an echocardiogram for confirmation of VSR by transthoracic echocardiography. Diagnosis of VSR was defined as disruption in the ventricular septum with evidence of left-to-right shunt by color Doppler. The location of the VSR was identified via transthoracic echocardiogram (TTE). Ventricular septum rupture location was recorded as basal septum, mid septum, or distal septum.

Statistical Analysis

All statistical analysis was performed using SPSS v 20.0 (Chicago, IL, USA). Continuous variables were summarized as mean±standard deviation (SD) whereas categorical variables were expressed as percentage of the sample.

Results

A total of 15 subjects were diagnosed with VSR after MI between the years of August 2009 to February 2017 (Table 1). Mean age was 62.4 ± 8.4 years of which 12 patients (80%) were males and 3 were females (20%). Eight (53.3%) patients were hypertensive and 3 (20%) were diabetic and only 1 (6.7%) patient had a history of prior coronary artery disease (CAD). Almost half of the patients (n=8; 53.3%) having post AMI VSR had single vessel disease, followed by 33.3% (n=5) having DVD. Only 6.7% (n=1) had triple vessel disease or non-obstructive disease. Mean left ventricle ejection fraction (LVEF) at time of VSR diagnosis was 38.2±6.8% with the mid septum being the most common location for VSR in 60%.

Out of 15 patients, 13 (86.6%) had anterior wall myocardial infarction and 2 (13.3%) had inferior wall MI. Majority (n=13, 86.6%) of patients had Left Anterior Descending (LAD) as an involved IRA, whereas in 13.3% (n=2) patients Right Coronary Artery (RCA) was involved. None of the patient had LCX as the culprit artery in the study population. Three patients underwent PCI to open the culprit artery prior to VSR closure due to ongoing angina, 3 had VSR device closure after CABG and 4 underwent surgical VSR closure along with revascularization. Five patients succumbed before revascularization or VSR closure could be attempted. In the study population, only 1 patient showed completely occluded IRA where as "Winking coronary" sign (of infarct related artery) was found in remaining 14 (93.3%) patients having post AMI VSR. Out of 4 patient's that underwent surgical closure to VSR, the anatomic site of the "wink" correlated with the site of VSR defect as seen at the time of surgery (100%). This pattern of transient occlusion of IRA during systole is shown in figure 1 (LAD) and 2 (PDA), whereas the normal flow of the same artery during diastole is indicated in figure 3 and 4 respectively.

Table 1: Demographic, risk factor and angiographic profile of study population

Sr. No	Variable	Mean (SD)/N (%)
1	Age	62.4 (±13.8)
2	Male (%)	12 (80)
3	DM (%)	3 (20)
4	HTN (%)	8 (53.3)
5	**Preoperative coronary angiogram N (%)**	
	Nonobstructive	1 (6.7)
	Single vessel disease	8 (53.3)
	Double vessel disease	5 (33.3)
	Triple vessel disease	1 (6.7)
6	**Culprit vessel N (%)**	
	LAD	13 (86.7)
	RCA	2 (13.3)
	LCX	0 (0)
7	**Location of VSR N (%)**	
	Basal	2 (13.3)
	Mid Septal	9 (60)
	Apical	4 (26.7)

SD, standard deviation; DM, diabetes mellitus; HTN, hypertension; LAD, left anterior descending; RCA, right coronary artery; LCX, left circumflex artery, VSR, ventricular septal rupture

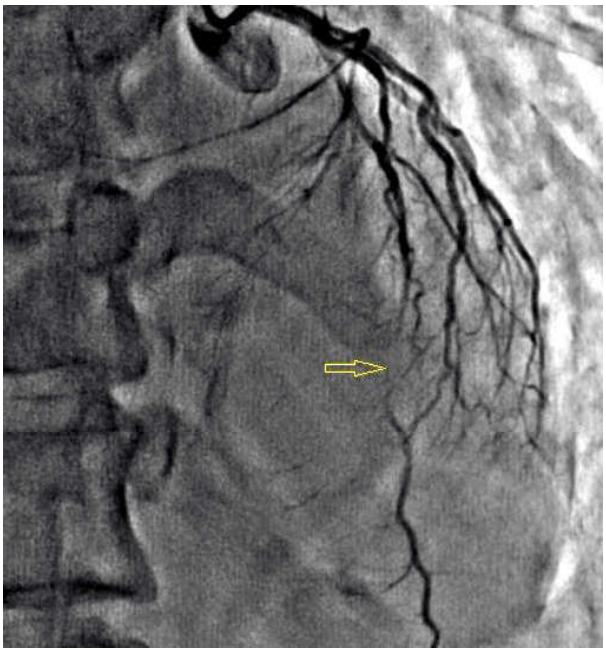

Fig. 1: Systolic blinking of LAD in VSR (arrow showing winking coronary sign)

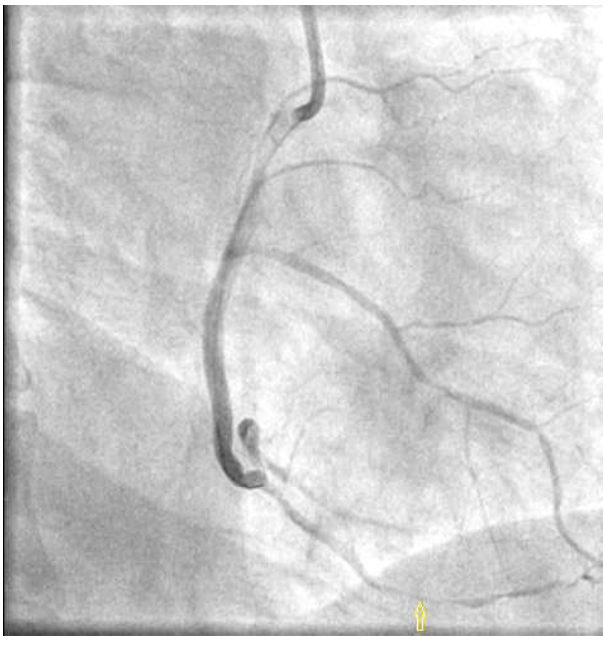

Fig. 2: Systolic blinking of PDA in VSR (arrow showing winking coronary sign)

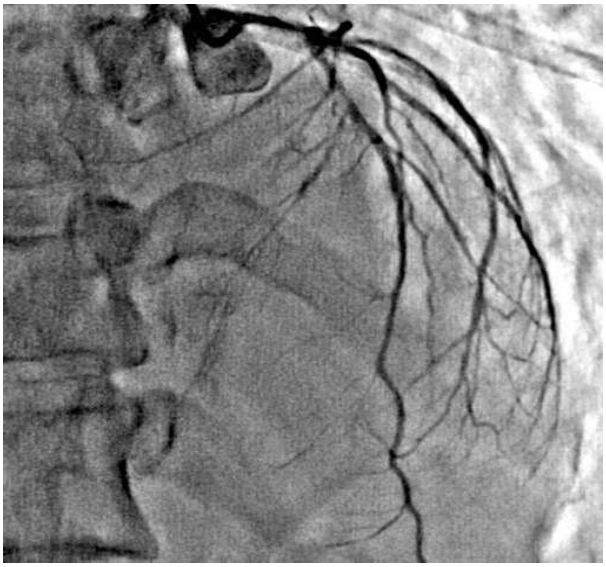

Fig. 3: Diastolic normal flow in LAD

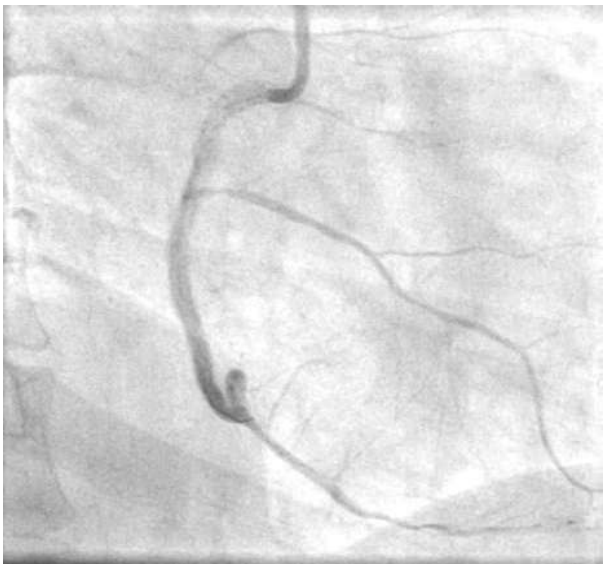

Fig. 4: Diastolic normal flow in PDA

Discussion

"Winking coronary" sign of VSR could be described as a partial occlusion of the infarct related culprit artery overlying the site of VSR during ventricular systole but gets filled in diastole. In this study, we document a clinical sign that was observed consistently as a distinctive pattern of IRA– "Winking coronary" in patients with post-infarction septal rupture overlying the anatomic site of the same.

"Winking coronary" sign is different from an anomaly known as "Myocardial bridging" that has been associated with clinical manifestation of various cardiac conditions such as unstable angina, myocardial infarction, arrhythmia, and sudden death [11-15. Myocardial bridging is a malformation characterised by a muscle bridge over a segment of coronary arteries. Systolic contractions of the muscle bridge could lead to narrowing of the artery, coronary compression, myocardial ischemia, myocardial infarction and sudden cardiac death. Unlike myocardial bridge (intramyocardial course) where the coronary artery gets compressed from either epicardial surface first (outside- in) or both epicardial and endocardial surface simultaneously (circumferentially), in winking coronary sign the involved culprit artery is compressed from the endocardial surface (inside- out) during systole.

Various Plausible Mechanisms of "Winking Coronary Sign" are Proposed

1. Venturi effect - The blood flow in this artery could be reduced during systole because of "Venturi effects" that could be explained by the hypothesis that the rapid shunt across the VSD leads to rapid drop in pressure across the defect causing overlying coronary to collapse leading to systolic compression of the artery overlying the rupture. The defect disturbs dynamics of local flow related gradients across ventricular septum, inducing drop in pressure and ultimately reducing the volume blood passing through IRA during systole
2. Mechanical theory–septum adjacent to rupture is fragile, dyskinetic and necrotic, which may mechanically compress the artery due to instability of the septum.
3. Hemodynamic theory- Mid systolic fall in velocity of coronary perfusion due to shunting across the VSR may lead to transient occlusion of the artery in systole while perfusion is maintained in diastole. Study of natural history of post infarction VSR has indicated compromised systemic perfusion due to massive left to right shunt along with infarct associated insult causing compromised pumping function of left ventricle. Consequence of post MI VSR involves deterioration of left ventricle functioning, decline in forward flow and activating compensatory vasoconstriction that may in turn cause partial occlusion of IRA. The results of GUSTO-trial showed a negative correlations between enrolment systolic (≤130 mm Hg) and diastolic (≤ 75 mm Hg) blood pressures with the incidence of VSD, that might be due to hemodynamic instability associated with extensive MI or right ventricular infarction [5].

Conclusion

- "Winking coronary" sign of the IRA is sensitive and specific sign of underlying VSR complicating a case of acute MI.
- It should be watched out for in patients undergoing coronary angiogram and may help salvage the operator blushes if he has missed VSR on other modalities of imaging or if the VSR has happened between the index echocardiography and angiogram.

Authors' Contribution

All the authors contributed significantly in the drafting and designing of the study and the first author provided the idea.

Funding/Support

Not applicable

Financial Disclosure

The authors don't have any financial conflict of interest to disclose.

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