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Original Article

Effect of thrombolytic therapy on the patterns of post myocardial infarction ventricular septal rupture

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ABSTRACT

Objectives: Ventricular septal rupture (VSR) is a rare but feared complication after myocardial infarction (MI). The objective of this study was to investigate the effects of thrombolytic therapy on the patterns of VSR following MI.
Methods: 30 consecutive patients admitted to a single tertiary level cardiac hospital with a diagnosis of acute MI and developed VSR in the hospital were included. The effect on thrombolytic therapy on the formation of VSR and its clinical outcome was studied.
Results: Out of 30 patients, 15 patients received thrombolytic therapy.10 received early (<12 h) and 5 received late (>12 h). The median time to post MI VSR formation was significantly shorter in thrombolysis group compared to non thrombolysis group at 1 vs 3 days($p = 0.026$). The median time for VSR formation was shorter in early thrombolysis group compared to late thrombolysis group at 1 vs 3 days ($p = 0.022$). There was no difference between late and no thrombolytic therapy (3 vs 3 days, $p = 0.672$). There was no significant difference in the mortality between thrombolytic and no thrombolytic therapy ($p = 0.690$). Patients treated medically had a significant higher mortality compared to patients treated surgically ($p = 0.005$).
Conclusion: Thrombolytic therapy results in an earlier presentation of VSR after MI. This earlier presentation may be due to reduction in the number of patients developing late VSR after thrombolytic therapy, while the number of patients developing an early VSR remaining unaffected. Despite improvements in medical therapy and percutaneous and surgical techniques, mortality with this complication remains extremely high.

1. Introduction

Ventricular septal rupture (VSR) is a rare but often fatal complication of acute myocardial infarction (MI). In prethrombolytic era the reported incidence of post MI VSR was 1%–2%.[1,2] However introduction of reperfusion therapy like thrombolysis and primary percutaneous intervention (PCI) has significantly reduced its incidence with only 0.2% reported in the GUSTO I trial[3]. Several factors have been identified to be associated with an increased risk of developing a post MI VSR like female sex, hypertension, anterior MI, prior angina, and increasing age.[3,4] In the prethrombolytic era, outcomes after the development of VSR were extremely poor, with an in-hospital mortality of 45% in surgically treated patients and 90% in those managed medically.[1,2,5–7] Factors predicting increased mortality include cardiogenic shock, longer duration of bypass, previous infarct, right heart compromise, inferior myocardial infarction and size of infarct.[3,4,8–15]

In prethrombolytic era it was reported that VSR typically occurs within the first week of MI with a mean time from symptom onset of 3–5days.[1,8,9] In thrombolytic era studies have given conflicting evidence regarding the time of onset of VSR after MI. In GUSTO-1 trial the mean time of development of VSR after thrombolysis was 1 day.[3] However in the SHOCK trial there was no difference in the time of development of VSR between the thrombolytic and non thrombolytic groups.[16]

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The aim of the present study was to evaluate the difference in the time of onset of VSR after MI between thrombolytic and non-thrombolytic patients admitted to a single tertiary care cardiac hospital. Furthermore we sought to study the factors associated with the increased mortality in patients who develop VSR.

2. Methods

In this prospective study done between January 2012 to December 2012, 30 patients with diagnosis of post MI VSR were included. Enrolment criteria included patients presenting with chest pain lasting ≥ 20 min and satisfying the World Health Organization criteria of acute MI and developing VSR within the hospital. Exclusion criteria included patients who had developed VSR outside the hospital and referred for further management. Informed consent was taken from all patients. Ethical clearance was taken before conducting the study.

The time of onset of chest pain was considered as the time of onset of the acute myocardial infarction. Acute myocardial infarction was diagnosed using the standard 12 lead electrocardiogram and cardiac enzyme (World Health Organization criteria). The time from onset of myocardial infarction to thrombolysis was separated into early (<12 h) or late (>12 h); this was the time from initial onset of pain to the time when the patient received thrombolysis. The time of onset of the VSR was assumed to coincide with the abrupt onset of symptoms and new murmur as documented in the patient's clinical record. The diagnosis of VSR was confirmed with Trans thoracic echocardiography in all cases. Patients were divided into two groups- thrombolytic group and non-thrombolytic group. The thrombolytic group was further divided into patients receiving thrombolysis within 12 h and those receiving thrombolysis 12 h or later. Streptokinase was used as thrombolytic agent in all the patients. All patients were analyzed for risk factors, demographics, clinical profile, treatment and prognosis.

Table 1
Baseline clinical characteristics.

Variable	Total (n = 30)	Thrombolysis group (n = 15)	Non Thrombolysis group (n = 15)
Age in years (Mean ± SD)	63.4 ± 9.8	66.07 ± 6.1	60.73 ± 12.1
Sex (Male)	18 (60%)	10 (66.66%)	8 (53.3%)
Risk factors			
Diabetes	15(50%)	8(53.3%)	7(46.6%)
Hypertension	12(40%)	7(46.6%)	8(53.3%)
Dyslipidemia	5(33.33%)	3(20%)	2(13.33%)
Smoking	10(33.33%)	5(33.33%)	5(33.33%)
History of angina	4(13.33%)	1(6.6%)	3(20%)
Family history	4(13.33%)	3(20%)	1(6.6%)
Old MI	3(10%)	1(6.6%)	2(13.33%)
Presentation			
AWMI	18(60%)	8(53.33%)	10(66.66%)
IWMI	12(40%)	7(46.66%)	5(33.33%)
Ejection fraction (Mean ± SD)	44.07± 6.4	45.87 ± 7.1	42.27 ± 5.1
CK-MB (Mean ± SD)	121.5± 139.84	82.47 ± 76.4	160.53 ± 177.1
Treatment			
Heparin	27(90%)	13(86.66%)	14(93.33%)
Aspirin	30(100%)	15(100%)	15(100%)
Clopidogrel	29(96.66%)	15(100%)	14(93.33%)
Beta blocker	14 (46.66%)	8(53.33%)	6(40%)
ACE Inhibitor	15 (50%)	9(60%)	6(40%)
Diuretics	15(50%)	7(46.66%)	8(53.33%)
Inotropes	26(86.66%)	13(86.66%)	13(86.66%)
NTG	3(10%)	1(6.6%)	2(13.33%)
Examination			
Pulse >100	19(63.33%)	9(60%)	10(66.66%)
Systolic BP < 90	19(63.33%)	11(73.33%)	8(53.33%)
Raised JVP	22(73.33%)	10(66.66%)	12(80%)
Killip I	13(43.33%)	9(60%)	4(26.66%)
Killip II	10(33.33%)	3(20%)	7(46.66%)
Killip III	2(6.66%)	1(6.66%)	1(6.66%)
Killip IV	5 (16.66%)	2 (13.33%)	3 (20%)
Urea (mg/dl) [Mean ± SD]	58.40 ± 49.59	61.67 ± 52.79	55.13 ± 47.81
Creatinine(mg/dl) [Mean ± SD]	1.35 ± 0.72	1.24 ± 0.51	1.45 ± 0.89
IABP	19(63.33%)	11(73.33%)	8(53.33%)
Total occlusion of infarct artery	21(70%)	9(60%)	12 (80%)
30 days mortality	16 (53.33%)	8(53.33%)	8(53.33%)
1 year mortality	21(70%)	11(73.33%)	10(60%)

MI: Myocardial infarction; AWMI: Anterior wall myocardial infarction; IWMI: Inferior wall myocardial infarction; NTG: Nitroglycerin; JVP: Jugular venous pulse; IABP: Intra aortic balloon pump.

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