

Recognition of acute right ventricular infarction by right Precordial Leads. ★

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SUMMARY:

Fifty consecutive patients admitted to NICVD Karachi were screened for presence of ECG criteria of right ventricular infarction. As right ventricular infarction is a fairly frequent occurrence (6 of 50 i.e. 12%) in our population, the recording of lead $V_{3-4}R$ should be an intrinsic part of ECG recording of all patients suspected of acute myocardial infarction.

The potentially serious and unique hemodynamic consequences of right ventricular infarction were first pointed out by Cohn et al in 1974(1). Its incidence once thought to be low has been reported to be as high as 24-45% (2,3,4,5,6) in patients with inferior wall myocardial infarction. The clinical recognition of right ventricular infarction now rests largely on a typical but not uniformly present clinical picture, backed by laboratory evidence of right ventricular dilation and dysfunction (7,8). Early recognition of right ventricular dysfunction is important because time of on-set of its often profound hemodynamic sequelae is unpredictable and these may be prevented by the administration of an appropriate intravenous volume load. If right ventricular infarction is neglected or treated as left heart failure with diuretics cardiogenic shock may supervene. Because routine right heart catheterisation in every patient presenting with acute infarction is impracticable, non invasive diagnosis by electrocardiographic methods to detect or raise the suspicion of important right ventricular infarction at an early stage would be helpful.

The purpose of this study is to detect the prevalence of right ventricular infarction in our population presenting with acute myocardial infarction in the light of previously validated ECG criteria of right ventricular infarction.

Material and Methods

Fifty consecutive patients with acute myocardial infarction who got admission at National Institute of Cardiovascular Diseases, from April 1 to July 31, 1986 form the basis of this study.

The diagnosis of myocardial infarction was based on typical history, serial electrocardiographic changes and elevated serum cardiac enzymes (Presence of all three). No patient had clinical evidence of prior myocardial infarction, pre-existing heart failure, cardiomyopathy, Valvular diseases, pulmonary diseases or associated precordial diseases. Patients with left bundle branch block were also excluded from this study.

Twelve leads standard ECG was recorded daily along with V_{3R} and V_{4R} . Position of V_{3R} - V_{4R} is exactly same as standard V_3 and V_4 on left side. Position of electrodes were marked by red marker on very First day so as to keep position of electrodes during subsequent ECG a constant one. The presence of pathological Q waves leads V_{4R} V_{3R} V_1 V_2 and V_3 and elevation of S-T Segment (measured in millivolts) in the same leads were analyzed in the each case. The S-T segment shift was considered to be significant if it is elevated 0.5mm or more above the isoelectric line.

All patients underwent careful clinical evaluations. Clinically right ventricular infarction was diagnosed if following features were present simultaneously, persisting hypotension systolic blood pressure < 90mm Hg, raised Jugular venous pressure clear lung field (clinically and radiologically) cold extremities, and oliguria < 300 ml urine per twentyfour hours.

Results

Out of total 50 patients 46 were men, 4 were women and their ages ranged from 40 to 65 years (mean 52.5). Table I gives break-up of patients based upon electrocardiographic site of myocardial infarction.

* NICVD Karachi.

TABLE I

Site		Number of patients (%)	
Transmural	inferior infarction=	24	(48)
Transmural	anterior infarction=	18	(36)
Transmural	antrio inferior infarction=	4	(8)
Transmural	posterior infarction=	2	(4)
Transmural	Inferior posterior wall infarction=	2	(4)

Six patients showed clinical features of right ventricular infarction, 4 patients were from inferior infarction group and 2 were from inferior posterior wall group. None was from anterior group. One patient showed S-T changes but no clinical evidence of right ventricular infarction. It may be transient ischemic changes which have reverted back spontaneously. Time course of S-T elevation was interesting S-T segment became normal with one day in 4 patients and remained elevated for more than one day in other 2 patients. 2 patients died in the hospital. One went into asystole and other died due to renal shut down following continuous hypertension. 5 patients from non right ventricular infarction group who died in the hospital. 4 were from anterior infarction group and one from anterior inferior group. 3 died due to pump failure and 2 due to electrical complications.

Table II gives analysis of right ventricular infarction group and non right ventricular infarction group.

TABLE II

	No of patients	Male (%)	Female (%)
Right ventricular infarction=	50	46 (92)	4 (8)
Non right ventricular infarction=	6	6 (12)	0
Hospital Mortality=	44	40 (80)	4 (8)
Mortality right ventricular=	7	6 (12)	1 (2)
Mortality in Non right Infarction group=	5	4 (8)	1 (2)
Infarction group=	2	2 (8)	1 (2)

Discussion

Several authors (9) have recognised of right ventricular dysfunction in the context of acute myocardial infarction but existence of right ventricular infarction as a clinical entity has been boosted recently by the refinements in non-invasive diagnostic techniques (electrocardiography, Nuclear Ventriculography, pyrophosphates scintigraphy and echocardiography) (10). In clinical setting there is no substitute for right heart catheterisation to identify deranged hemodynamic function produced by right ventricular infarction and to monitor treatment but use of this technique in all patients is impracticable and unnecessary. The full blown clinical picture of right ventricular infarction is now easily recognised. However the hemodynamic consequences of right ventricular infarction may appear unexpectedly after what appears at first to be uncomplicated inferior wall myocardial infarction and it is therefore evidently desirable to have an early simple and sensitive diagnostic clue to the presence of or possibility of right ventricular infarction before complications ensue. V3R-V4R is not the only diagnostic criteria that will decide whether are not right ventricular infarction is present but it is very useful early simple warnings signal before specific complications set in (11). But there are certain factors which can influence the degree of S-T elevation in V3R-V4R. Firstly S-T elevation will not be as prominent in V3R-V4R if it is not prominent in II, III and avF. Secondly S-T elevation in V4R is right word as well as anteriorly oriented vector thus left word S-T deviation such as that seen in V5, V6 because of extension of infarction to the lateral wall could cancel out S-T elevation in V4R. But even in the light of these factors, V3R-V4R should be recorded as early as possible particularly in the presence of inferior and posterior wall infarction. S-T elevation in V3R, V4R carries a reasonably high sensitivity, specificity and predictive value for right ventricular infarction (or at least ischemia). V3R-V4R in the presence of posterior and inferior wall myocardial infarction and in the absence of left bundle branch block or other causes of anteriorly oriented vector (12,13). Carries a sensitivity for right ventricular infarction from 80% to 90%, sepcificity of 68% to 70%, positive predictive value of 70% to 80% and negative predictive value of

85% to 100% (14,15). Clinical recognition of right ventricular infarction may be important in patients management. Right ventricular infarction should be considered in any patient with an acute inferior or posterior wall infarction along with evidence of systemic venous engorgement and hypotension.

Rapid infusion of fluid may reverse shock in these patients, of course, clinical presentation of right ventricular infarction can resemble cardiac tamponade. Constrictive pericarditis or pulmonary embolism. Right ventricular infarction should also be suspected in severe left ventricular dysfunction and Cardiogenic shock since volume overloading may enhance the hemodynamic evidence of right ventricular dysfunction as well as improve performance of left ventricle.

V3R-V4R should be recorded as early as possible in the presence of acute inferior and posterior wall infarction, rather it should be an intrinsic part of the early electrocardiographic recording of every patient, evaluation who is entering into hospital.

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