

## Right Ventricular Infarction Associated with Anteroseptal Myocardial Infarction: A Clinicopathologic Study of Nine Cases

Naeem K. Tahirkheli, MD,\* William D. Edwards, MD,<sup>†</sup> Rick A. Nishimura, MD,\*  
and David R. Holmes, Jr., MD\*

*\*Division of Cardiovascular Diseases and Internal Medicine and the <sup>†</sup>Division of Anatomic Pathology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota, USA*



The association of right ventricular (RV) infarction with inferoseptal myocardial infarction is well established. However, a question remains about the occurrence of RV infarction in association with anteroseptal myocardial infarction. To determine the frequency and clinical correlates of this entity, we studied autopsied hearts from patients with isolated anteroseptal left ventricular (LV) infarcts. Among 3,249 autopsy specimens, 88 cases were identified. From each, sections were taken from the RV anterior, lateral, and inferior regions at basal, middle, and apical levels. All 1,584 slides were reviewed by blinded assessment. RV and LV infarcts were compared to confirm similarity in age. Patient records and cardiac investigations were reviewed for evidence of RV involvement. Of the 88 hearts with anteroseptal LV infarcts, 9 (10%) had coexistent RV infarction (6, old; 3, new). For these 9, the RV infarction involved 11% to 33% of the RV area, and the left anterior descending coronary artery was the infarct-related artery in each. All 3 patients who had an echocardiographic examination within 4 weeks of anteroseptal LV infarction had RV dysfunction. One patient, studied 15 years after infarction, had a normal right ventricle by echocardiography. In 3 patients with acute myocardial infarction, right heart catheterization during the acute phase revealed increased right-sided diastolic pressures out of proportion to left-sided diastolic pressures (right atrial pressure to pulmonary capillary wedge pressure, 60% to 95%). In conclusion, 10% of patients with an isolated anteroseptal LV infarct had evidence of RV free wall infarction. The RV infarction was associated with identifiable hemodynamic and echocardiographic features. *Cardiovasc Pathol* 2000;9:175–179  
© 2000 by Elsevier Science Inc.

The occurrence of right ventricular (RV) infarction as an extension of an inferoseptal left ventricular myocardial infarction (MI) is well recognized from both pathologic and clinical standpoints (1). However, RV infarction associated with an anteroseptal left ventricular MI is not a well-recog-

nized clinical entity, and controversy about the association has arisen from autopsy studies (2–8). The current investigation was undertaken to examine this possible association through a systematic histopathologic approach in patients with an isolated anteroseptal MI.

### Methods

#### Study Group

From 1988 through 1992, 3,249 autopsies were done at Mayo Clinic Rochester. From these, 88 patients were identified with an isolated anteroseptal MI. Patients with inferior MI, significant RV hypertrophy (RV wall > 0.8 cm), congenital heart disease, or previous coronary artery bypass grafting were excluded. Additionally, patients with circumferential MIs at midventricular or basal levels or with multifocal MIs

Manuscript received March 8, 2000; accepted March 31, 2000.

Present address for Dr. Tahirkheli: Southwest Cardiology Associates, 1111 44th Street SW, Oklahoma City, OK 73109, USA.

Presented in abstract form at American Heart Association Annual Scientific Sessions, 1996; and abstract subsequently published as follows: Tahirkheli NK, Edwards WD, Nishimura RA. Right ventricular infarction associated with anterior myocardial infarction without inferior myocardial infarction (abstract). *Circulation* 1996;96(Suppl 8):I73.

Address for correspondence: William D. Edwards, MD, Mayo Clinic, 200 First Street SW, Rochester, MN 55905.

were excluded because of the possible relation to global myocardial ischemia, such as hypotension or hypoxemia. However, because the left anterior descending coronary artery often wraps around the apex, anteroseptal infarcts with circumferential involvement at the apical level were included in the study.

### Pathology Data

The 88 hearts obtained from our institutional tissue registry had previously been dissected by the “bread slice” (short-axis) method. The major epicardial coronary arteries had been step-sectioned at 3-mm intervals. For each heart, the following additional studies were also performed.

After gross evaluation, sections were taken from anterior, lateral, and inferior regions of the right ventricles at basal, middle, and apical levels (9 blocks per heart). Hematoxylin-eosin and Masson trichrome stains were performed on each block, to produce 1,584 slides from the right ventricles. All were subjected to blinded assessment by the authors (N.K.T. and W.D.E) for presence, extent, and age of the infarction. Infarcts were designated as transmural, subendocardial, or mixed (>50% of wall thickness, <50%, or a combination, respectively). The age of each infarct was determined microscopically by previously published criteria (9).

For the 9 hearts with RV infarctions, the left ventricular slides were reviewed in a blinded manner to assess agreement in the ages of RV and left ventricular infarctions. The gross and microscopic findings were used for semiquantitative determination of the percentage of the left ventricle involved by infarction.

The size of the RV infarct was determined microscopically as follows: 5 of 6 segments at basal and middle levels were assigned 12% each, the anterobasal segment was assigned 13%, and the 3 apical segments were assigned 9%

each of the total RV myocardial mass. The percentage of each region involved by infarction was estimated to be 25% for subendocardial infarcts, 50% for mixed types, and 75% for transmural types.

### Clinical Data

Detailed histories of the patients who had RV infarction were reviewed. All cardiac investigations were available and reviewed, including electrocardiograms, echocardiograms, and cardiac catheterization findings. This study was approved by the Institutional Review Board.

## Results

### Pathology Findings

Twelve (14%) of the 88 hearts had evidence of RV infarction as an extension of an anteroseptal MI. Two of the infarcts were excluded because they were associated with a clinical history of non-Q-wave MI before the index event and did not match the age of the transmural infarct on the left side. Another RV infarction was excluded because of evidence of widespread tumor embolization that could have caused hitherto unrecognized RV infarction independent of the anteroseptal MI. The remaining 9 RV infarcts were thought to be definitely associated with the anteroseptal MI. The age of the RV infarction concurred with that of the left ventricular infarction in each.

All 9 left-sided infarcts were transmural, whereas 7 of the right-sided infarcts were mixed, 1 was subendocardial, and 1 was transmural. No mural thrombi were found in the right ventricle of any heart. Five of the infarcts were old, and the ages of the other 4 varied (Table 1). A mean of 32% (range, 19% to 46%) of left ventricular myocardial mass

**Table 1.** Autopsy Findings in Patients with Right Ventricular Infarction Associated with Anterior Myocardial Infarction

Patient	Left Ventricle				Right Ventricle				Coronary Arteries, % Stenosis		
	Location of MI	MI Size, % of LV	Type of MI	Age of MI	Location of MI	MI Size, % of LV	Type of MI	Age of MI	LAD	LCX	RCA
1	Anteroseptal, apical	46	TM	Old	Anteroapical, inferoapical	34	TM	Old	100	75	100
2	Anterolateral	37	TM	Old	Anterior	14	Mixed	Old	75	75	75
3	Anteroseptal	33	TM	Old	Anteroapical, inferoapical	30	Mixed	Old	75	<50	75
4	Anterolateral	31	TM	<24 h	Anterior	12	SE	<24 h	75 <sup>a</sup>	<50	<50
5	Anteroseptal	19	TM	3–4 wk	Anterior	15	Mixed	3–4 wk	75	75	50
6	Anterolateral	36	TM	4 wk	Anterior	25	Mixed	4 wk	<25	<25	<25
7	Anterior	23	TM	Old	Anterolateral	33	Mixed	Old	75	<50	50
8	Anterolateral	33	TM	7–9 days	Anterior	11	Mixed	7–9 days	100	<25	<25
9	Anterolateral	32	TM	Old	Anteroapical, inferoapical	24	Mixed	Old	75	75	<25

Abbreviations: LAD = left anterior descending artery; LCX = left circumflex artery; LV = left ventricle; MI = myocardial infarction; RCA = right coronary artery; SE = subendocardial; TM = transmural.

<sup>a</sup>With acute coronary thrombus.

was involved in the infarction, whereas a mean of 22% (range, 11% to 34%) of RV free wall mass was infarcted.

The left anterior descending coronary artery (LAD) was the infarct-related artery in each case. Three hearts had a large wraparound LAD causing both anterior apical and inferior apical RV infarction (Fig. 1). The other 6 had anterior or anterolateral RV wall infarction, presumably from impaired blood flow in right-sided diagonal branches of the LAD. The ventricular septum was involved by infarction in all 9 hearts.

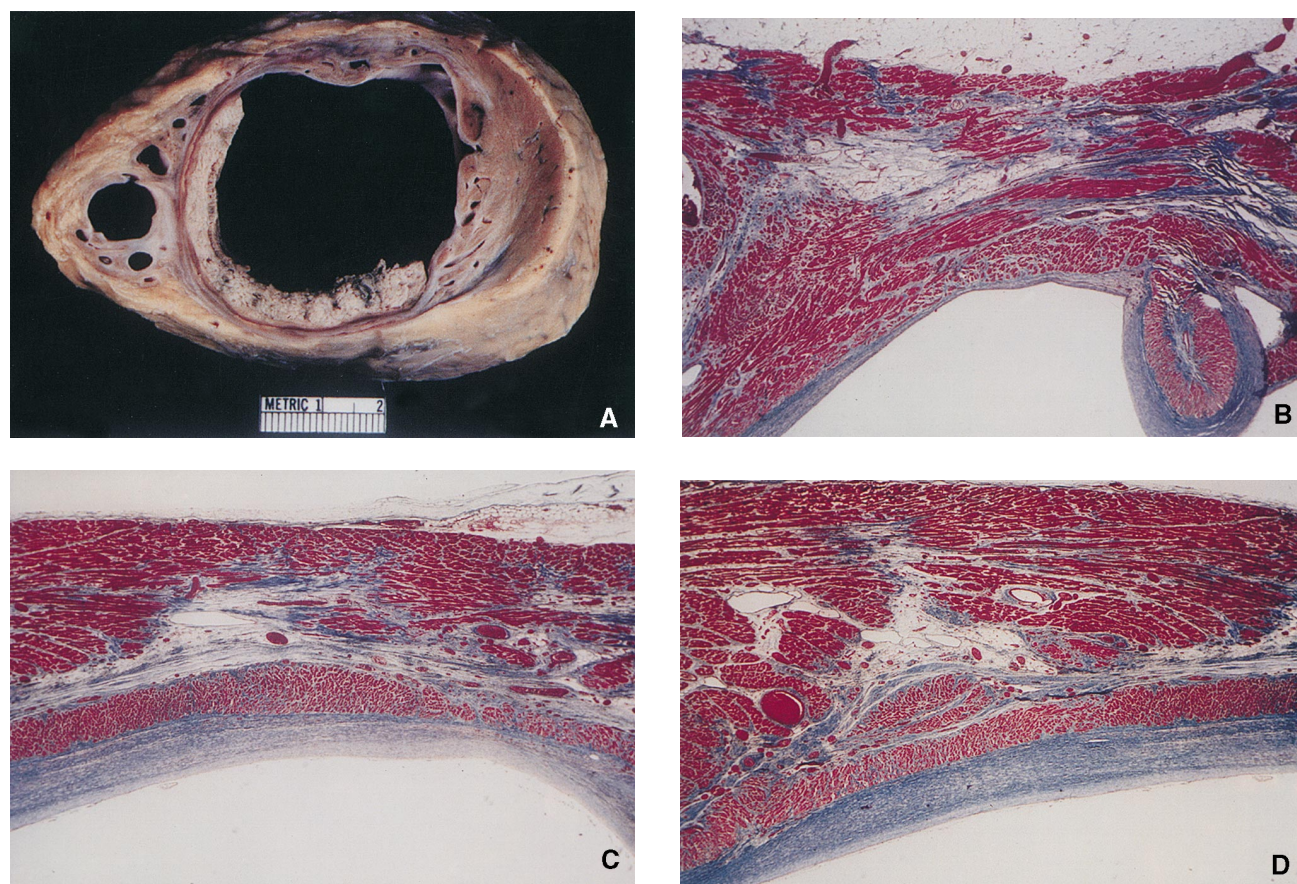
### Clinical Features

Five patients were male and 4 female (Table 2). Patient 6, age 22, with alcohol-related dilated cardiomyopathy, had a recent MI but did not have high-grade occlusive coronary artery disease. The other 8 patients, 51 to 86 years old, had coronary atherosclerosis.

None of the patients had had right precordial lead electrocardiography performed. The left-sided electrocardiograms obtained during and after acute MI were reviewed. We could not find any consistent pattern to suggest RV involvement.

Three patients had echocardiography within 72 hours of the MI. All had distinguishable RV dysfunction. Patient 5 had a dilated left ventricle with an estimated ejection fraction of 25%. The right ventricular cavity size was normal, but there was akinesis of the middle and distal anterolateral and anteroseptal walls of the RV. An echocardiogram obtained 2 months earlier did not show this regional wall motion abnormality in the right ventricle. Two others (patients 6 and 8) had RV dilatation in addition to a regional wall motion abnormality. However, in patient 3, who had an echocardiographic examination several years after the acute MI, the right ventricle was normal in size and function. In no patient had RV infarction been recognized clinically.

Four patients had right heart catheterization. All had increased right-sided and left-sided pressures. Patients 5, 6, and 8 had central monitoring within 72 hours of acute MI. The ratio of right atrial pressure to pulmonary artery wedge pressure was  $\geq 60\%$  in each patient (range, 60% to 95%). The ratio for the fourth patient (patient 7) was 53%, but this patient had an anteroseptal infarction 4 years earlier than the catheterization. All 9 patients had cardiac deaths. Patient 4



**Figure 1.** Patient 3. Gross and histopathologic sections showing an old transmural anteroseptal infarction with inferoseptal and right ventricular extensions at the apical level due to apical wraparound of the obstructed left anterior descending coronary artery. (A) Short-axis view of gross specimen demonstrates left ventricular remodeling (wall thinning and chamber dilation) with shallow mural thrombus. The right ventricle is involved by ill-defined gray-white fibrosis. (B-D) Low-power photomicrographs of slides stained with Masson trichrome better illustrate the extent of old right ventricular infarction along the anterior (B), lateral (C), and inferior (D) walls.

**Table 2.** Clinical Features of Patients with Right Ventricular Infarction Associated with Anteroseptal Myocardial Infarction

Patient	Age (yr)	Sex	ECG	Reason for Death	MI–Death Interval	Echo	RAP (mm Hg)	PAWP (mm Hg)
1	51	M	ST ↑ V1–V6	SCD	8 yr	—	—	—
2	84	F	LBBB	CHF	16 yr	—	—	—
3	66	M	ST ↑ V1–V4	SCD	19 yr	Normal RV	—	—
4	86	F	—	SCD <sup>a</sup>	<24 hr	—	—	—
5	77	M	LBBB	MI	23 days	RV dysfunction	15	25
6	22	M	ST ↑ V1–V6	SCD	31 days	RV dysfunction	21	28
7	82	M	RBBB, ST ↑ V1–V4	SCD	4 yr <sup>b</sup>	—	15	28
8	62	F	ST ↑ V1–V4	CHF	8 days	RV dysfunction	18	19
9	74	F	—	SCD	1.7 yr	—	—	—

Abbreviations: CHF = congestive heart failure; ECG = electrocardiogram; Echo = echocardiogram; LBBB = left bundle branch block; MI = myocardial infarction; PAWP = pulmonary artery wedge pressure; RAP = right atrial pressure; RBBB = right bundle branch block; RV = right ventricle or ventricular; SCD = sudden cardiac death; ST = ST interval. Arrow symbol (↑) signifies “increased.”

<sup>a</sup>Due to ruptured infarct.

<sup>b</sup>Probable interval.

died at home following rupture of the infarct; the proximal LAD showed coronary thrombosis.

## Discussion

RV infarction has been well recognized as a complication of inferoseptal infarction of the left ventricle (2,10). The right coronary artery supplies not only the inferoseptal wall of the left ventricle in a right-dominant circulation but also most of the RV free wall. Involvement of the right ventricle has been described in up to 50% of inferior MIs (2,11,12), and the typical associated clinical findings of increased right-sided diastolic pressures without pulmonary congestion (2,13,14) are noted in 15% to 20%. However, the association of RV involvement with an anteroseptal MI is not a well-recognized clinical entity, and controversy exists in the literature (2,7,8).

In 1938, Feil et al. (7) first reported a 22% incidence of RV infarction with anteroseptal MI at autopsy, and other reports of this association followed (3–6,15,16). Two large autopsy series (2,8) did not find RV extension among 97 and 35 patients, respectively, who had isolated anteroseptal MI. Cabin et al. (15) reported an incidence of 13% but performed histologic studies only if gross evaluation suggested RV involvement.

In the current investigation, we showed that on the basis of detailed microscopic evaluations of the RV myocardium, the incidence of RV involvement is 10% in patients with fatal isolated anteroseptal MI. A mean of 22% of the RV myocardial mass was infarcted. All three patients who underwent imaging and hemodynamic studies at the time of infarction demonstrated echocardiographic criteria of RV enlargement and catheterization evidence of increased

right-sided diastolic pressures out of proportion to left-sided diastolic pressures.

## Mechanism of Association

The blood supply to the anterior RV free wall is provided by the conus branch of the right coronary artery and the right ventricular branches of the LAD. James (17) reported that in 24% of human hearts, the right-sided branches of the LAD supply >30% of the RV free wall. This anatomical variation potentially could predispose the RV wall to involvement in patients with anteroseptal MI secondary to proximal or middle LAD occlusion. The inferior wall of the right ventricle is supplied by the acute marginal branches of the right coronary artery and partly by the posterior descending artery. However, in 22% of humans, the LAD wraps around the apex and ascends fairly high (>5 cm) into the posterior interventricular groove, thus supplying part of the inferior RV wall (17). The posterior descending artery in these instances is usually diminutive. An LAD-related anteroseptal MI in such patients could result in inferior infarction of both ventricles at the apical level.

Another potential mechanism is a high-grade right coronary artery stenosis (or chronic occlusion) that results in development of significant collaterals from the LAD. Finally, a frequent occurrence after coronary artery grafting is bypassing of the LAD by a left internal mammary artery in addition to vein grafts for the other arteries, including the right coronary artery. The natural history is that the left internal mammary artery graft to the LAD remains patent much longer than the vein grafts (18). In turn, a large portion of the right ventricle is supplied by the LAD via collaterals. In such a case, a subsequent anteroseptal MI could result in concomitant RV infarction.

### Effect of Exclusions

To avoid confounding variables, we studied a very select patient population. All inferoseptal infarctions at basal and mid-ventricular levels were excluded. Similarly, patients with RV hypertrophy, who may have a higher risk of infarction, and those with previous coronary artery bypass grafting were excluded. The incidence of RV infarction in association with anteroseptal MI may be different in an unselected MI population.

### References

1. Lew AS, Isner JM. Right ventricular infarction. *Cardiovasc Clin* 1987;17:203–217.
2. Isner JM, Roberts WC. Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease. Frequency, location, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. *Am J Cardiol* 1978;42:885–894.
3. Bean WB. Infarction of heart: Clinical course and morphological findings. *Ann Intern Med* 1938;12:71–94.
4. Myers GB, Klein HA, Hiratzka T. Correlation of electrocardiographic and pathologic findings in infarction of interventricular septum and right ventricle. *Am Heart J* 1949;37:720–770.
5. Sprague HB, Orgain ES. Electrocardiographic study of cases of coronary occlusion proved at autopsy at Massachusetts General Hospital, 1914–1934. *N Engl J Med* 1935;212:903–910.
6. Wartman WB, Hellerstein HK. Incidence of heart disease in 2,000 consecutive autopsies. *Ann Intern Med* 1948;28:41–65.
7. Feil H, Cushing EH, Hardesty JT. Accuracy in diagnosis and localization of myocardial infarction. *Am Heart J* 1938;15:721–738.
8. Ratliff NB, Hackel DB. Combined right and left ventricular infarction: Pathogenesis and clinicopathologic correlations. *Am J Cardiol* 1980;45:217–221.
9. Edwards WD. Pathology of myocardial infarction and reperfusion. In: Gersh BJ, Rahimtoola SH, eds. *Acute Myocardial Infarction (Current Topics in Cardiology)*. 2nd ed. New York, NY, USA: Chapman & Hall, 1997;16–50.
10. Cohn JN, Guiha NH, Broder MI, Limas CJ. Right ventricular infarction. Clinical and hemodynamic features. *Am J Cardiol* 1974;33:209–214.
11. Zehender M, Kasper W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. *N Engl J Med* 1993;328:981–988.
12. Erhardt LR, Sjogren A, Wahlberg I. Single right-sided precordial lead in the diagnosis of right ventricular involvement in inferior myocardial infarction. *Am Heart J* 1976;91:571–576.
13. Cintron GB, Hernandez E, Linares E, Aranda JM. Bedside recognition, incidence and clinical course of right ventricular infarction. *Am J Cardiol* 1981;47:224–227.
14. Lopez-Sendon J, Coma-Canella I, Gamallo C. Sensitivity and specificity of hemodynamic criteria in the diagnosis of acute right ventricular infarction. *Circulation* 1981;64:515–525.
15. Cabin HS, Clubb KS, Wackers FJ, Zaret BL. Right ventricular myocardial infarction with anterior wall left ventricular infarction: An autopsy study. *Am Heart J* 1987;113:16–23.
16. Andersen HR, Falk E, Nielsen D. Right ventricular infarction: frequency, size and topography in coronary heart disease: A prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am Coll Cardiol* 1987;10:1223–1232.
17. James TN. The arteries of the free ventricular walls in man. *Anat Rec* 1960;136:371–384.
18. Cameron A, Davis KB, Green G, Schaff HV. Coronary bypass surgery with internal-thoracic-artery grafts—Effects on survival over a 15-year period. *N Engl J Med* 1996;334:216–219.