patients (27%) had inflammatory heart disease. The other patients showed normal histology (eight patients, 36%) or increased fibrosis (seven patients, 32%), which was accompanied by pathologic lipomatosis in only three patients (14%). In the present study we often found significant fibrolipomatosis (66%), leading to the diagnosis of arrhythmogenic right ventricular dysplasia. This high incidence of right ventricular dysplasia may relate to the more extensive procedure used, which obviously may increase the likelihood of finding clinically significant microscopic abnormalities. It may be objected that the high incidence may have been the result of less strict pathoanatomic criteria, compared with those used by other investigators.[3] In addition, lipomatosis may be considered to be a normal observation in endomyocardial right ventricular biopsies. However, in accordance with the report by Mehta et al.,[11] we considered the presence of adipose tissue significant only when it was accompanied by an increase in fibrous tissue.

Identification of a specific cause of idiopathic ventricular fibrillation has consequences for diagnosis and prognosis. Patients with a reversible cause, such as inflammatory heart disease, may especially have a favorable prognosis.[6] On the other hand, the prognosis may be rather variable in patients with right ventricular dysplasia or other cardiomyopathies. This explains the differences in prognosis of survivors of sudden death who have no cardiac disease in studies that did not use endomyocardial biopsies. The clinical value and feasibility of extensive right ventricular endomyocardial biopsy procedures in idiopathic ventricular fibrillation remain to be established in a larger cohort of patients. An extensive biopsy procedure could implicate a higher complication risk than a standard procedure, especially in the case of diseased right ventricles. During the biopsy procedures, care was taken to prevent deep transmural biopsies. The absence of complications in the present report may relate to the fact that biopsies were taken from patients in the early stages of the disease whose ventricular tissue was still relatively well preserved.

REFERENCES

1. Marcus FI, Fontaine GH, Guiraudon G, Frank R, Laurenceau JL, Malergue C, Grosgogeat Y. Right ventricular dysplasia: a report of 24 adult cases. Circulation 1982;65:384-98.
2. Wellens HJJ, Lemery R, Smeets JL, Brugada P, Gorgels AP, Cheriex EC, De Zwaan C. Sudden arrhythmic death without overt heart disease. Circulation 1992;85(suppl I):I-92-7.
3. Strain JE, Grose RM, Factor SM, Fisher JD. Results of endomyocardial biopsy in patients with spontaneous ventricular tachycardia but without apparent structural heart disease. Circulation 1983;68:1171-81.
4. Hosenpud JD, McAnulty JH, Niles NR. Unexpected myocardial disease in patients with life-threatening arrhythmias. Br Heart J 1986;56:55-61.
5. Iesaka Y, Hiroe M, Aonuma K, Nitta J, Nogami A, Tokunaga T, Amemiya H, Fujiwara H, Sekiguchi M. Usefulness of electrophysiologic study and endomyocardial biopsy in differentiating arrhythmogenic right ventricular dysplasia from idiopathic right ventricular tachycardia. Heart Vessels 1990; 5(suppl 5):65-9.
6. Vignola PA, Aonuma K, Swaye PS, Rozanski JJ, Blankstein RL, Benson J, Gosselin AJ, Lister JW. Lymphocytic myocarditis presenting as unexplained ventricular arrhythmias: diagnosis with endomyocardial biopsy and response to immunosuppression. J Am Coll Cardiol 1984;4:812-9.
7. Sugrue DD, Holmes DR, Gersh BJ, Edwards WD, McLaran CJ, Wood DL, Osborn MJ, Hammill SC. Cardiac histologic findings in patients with life-threatening ventricular arrhythmias of unknown origin. J Am Coll Cardiol 1984;4:952-7.
8. Topaz O, Perin E, Cox M, Mallon SM, Castellanos A, Meyerburg RJ. Young adult survivors of sudden cardiac arrest: analysis of invasive evaluation of 22 subjects. AM HEART J 1989;118:281-7.
9. Martini B, Nava A, Thiene G, Buja FG, Canciani B, Scognamiglio R, Daliento L, Dalla Volta S. Ventricular fibrillation without apparent heart disease: description of six cases. AM HEART J 1989;118:1203-9.
10. Buja G, Meneghello MP, Bellotto F. Isolated episode of exercise-related ventricular fibrillation in a healthy athlete. Int J Cardiol 1989;24:121-3.
11. Mehta D, Odawara H, Ward DE, McKenna WJ, Davies MJ, Camm AJ. Echocardiographic and histologic evaluation of the right ventricle in ventricular tachycardias of left bundle branch block morphology without overt cardiac abnormality. Am J Cardiol 1989;63:939-44.

Thrombolytic therapy and intravenous heparin in acute myocardial infarction do not affect the incidence of left ventricular mural thrombus formation

David W. Grambow, MD, Vallerie V. Valentini, MD, and William F. Armstrong, MD *Ann Arbor, Mich.*

Left ventricular mural thrombus is a frequent complication of acute myocardial infarction. Its occurrence has been reported after 15% to 20% of all myocardial infarctions and after 30% to 50% of anterior Q-wave infarctions.[1] Embolic events in the first 4 months after myocardial infarction have been reported to occur in up to 30% of patients with left ventricular mural thrombus in the absence of anticoagulation.[2] Two-dimensional echocardiography is a sensitive and specific, noninvasive method of detecting left ventricular mural thrombus.[3] Controversy exists as to whether the administration of thrombolytic therapy or the early use of intravenous heparin in acute myocardial infarction reduces the incidence of left ventricular mural thrombus formation.[4-10] The purpose of this study was to determine whether the early use of aspirin and intravenous heparin, with or without thrombolytic therapy, alters the incidence

From the Division of Cardiology, Department of Medicine, University of Michigan.

Reprint requests: William F. Armstrong, MD, Division of Cardiology, UH 9D Rm. 9800, University of Michigan, 1500 E. Medical Center Dr., Ann Arbor, MI 48109-0119.

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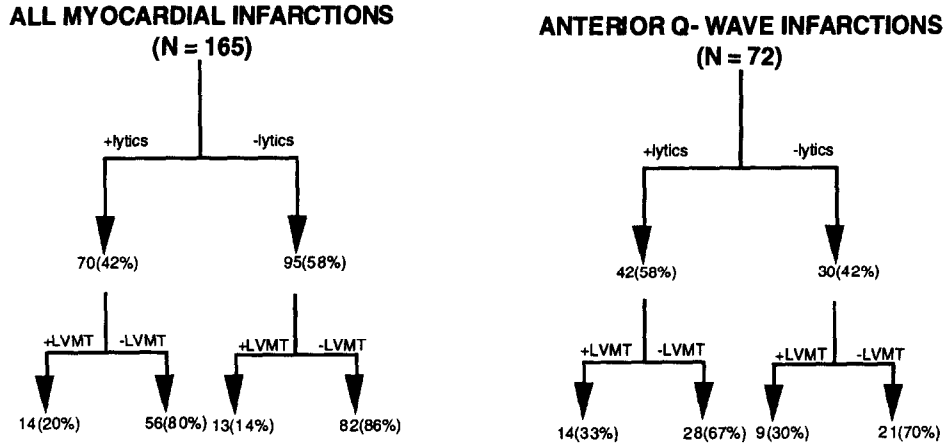

Fig. 1. Effects of thrombolytic therapy on left ventricular mural thrombus formation in acute myocardial infarction. *Lytics*, Thrombolytic therapy.

Table I. Patient demographics (n = 165)

Demographics	
Age (yr)	60.6 ± 13
Male gender	64%
Diabetes	23%
Hypertension	52%
Smoking	55%
Previous myocardial infarction	18%
History of congestive heart failure	6.1%
Previous coronary bypass grafting	11%

Table II. Location of myocardial infarction and percentage of left ventricular mural thrombus

Infarct location	*Left ventricular mural thrombus (%)*
Anterior Q-wave infarction (n = 72)	32
Inferior Q-wave infarction (n = 43)	4.7
Non-Q-wave infarction (n = 50)	4.0

of left ventricular mural thrombus in patients with acute myocardial infarction.

We reviewed the medical records of patients who were first seen between January 1990 and June 1991 at the University of Michigan Medical Center with chest pain and electrocardiographic and enzymatic evidence of acute myocardial infarction. The characteristics of the study population are shown in Table I. Attention was paid to the use of aspirin, intravenous heparin, and thrombolytic therapy within 12 hours of presentation. Patients given aspirin received a daily oral dose of 324 mg. Patients receiving intravenous heparin were given a bolus of 5000 units and started on a dose of 1000 U/hr. The dose of heparin was adjusted to maintain the activated partial thromboplastin time at 1.5 to 2 times control. Heparin was continued for 5.3 ± 3 days. Patients receiving thrombolytic therapy received 100 mg of tissue plasminogen activator intravenously over a period of 3 hours or 1.5 million U of streptokinase over a period of 1 hour. Each patient was subsequently managed in the coronary care unit in a standard manner. All patients underwent two-dimensional echocardiography with phased-array scanners during hospitalization 6.7 ± 4.2 days after infarction. Echocardiograms were analyzed by two observers who were blinded to medical therapy. Left ventricular mural thrombus was diagnosed by means of the criteria of Asinger et al.,[1] in which an echodense mass originating from the endocardial surface in an area of regional asynergy is noted in two orthogonal views at various depth settings. Left ventricular ejection fraction was calculated by Simpson's rule from apical echocardiographic views or at cardiac catherization with a prolate llipse geometric model. Aspirin was given to 88% of patients, 83% received intravenous heparin, and 42% of patients received aspirin and heparin in addition to thrombolytic therapy. Thrombolytic therapy was administered to 70 (42%) patients. The time to lytic therapy was 3.7 ± 2.2 hours after onset of chest pain in patients with thrombus and 4.0 ± 2.9 hours in patients without thrombus.

The overall incidence of left ventricular mural thrombus was 16%, with the highest incidence (32%) in anterior Q-wave infarctions (Table II). All thrombi associated with anterior Q-wave infarctions and with non-Q-wave infarctions were found in the left ventricular apex and were associated with areas of asynergy. The two thrombi complicating inferior wall myocardial infarction were seen with posterior wall aneurysms. The frequency of aspirin and intravenous heparin use was similar in patients with and without left ventricular mural thrombus. Thrombolytic therapy did not reduce the incidence of left ventricular mural thrombus formation (Fig. 1). Patients with left ventricular mural thrombus developing after anterior Q-wave infarction had lower left ventricular ejection fractions (29% vs 43%, p = 0.0001), had a higher rate of in-hospital congestive heart failure or death (7% vs 26%, p = 0.0009),

Table III. Characteristics of patients with and without left ventricular mural thrombus complicating acute myocardial infarction

	*Peak creatine kinase (IU/L)**	*Left ventricular ejection fraction (%)†*	*In-hospital CHF or death‡*
Left ventricular mural thrombus present (n = 27)	2740 ± 1909	29 ± 11	10 (37%)
Left ventricular mural thrombus absent (n = 139)	2035 ± 1994	43 ± 11	16 (11%)

CHF Congestive heart failure.
*p = 0.09.
†p = 0.0001.
‡p = 0.0009.

and tended to have higher postinfarction peak creatine kinase levels (2740 IU/L vs 2035 IU/L, p = 0.09) (Table III). Left ventricular mural thrombus was associated with akinesis or dyskinesis of the anteroapical wall in 89% of the cases. There was no difference in ejection fraction in patients with anterior Q-wave infarctions with (36.9%) or without (33%) thrombolytic therapy. No in-hospital embolic events were noted in any study patient.

Left ventricular mural thrombus formation after acute myocardial infarction is favored by stasis of blood and endocardial injury, both of which increase with greater extent of myocardial damage. Lysis or inhibition of thrombus formation on necrotic endocardial surfaces has been one proposed mechanism by which anticoagulants such as heparin or thrombolytic agents may reduce the incidence of left ventricular mural thrombus formation in acute myocardial infarction.[4,6] We did not observe a reduction in the incidence of left ventricular mural thrombus formation in patients receiving intravenous heparin, but the absence of embolic complications in our study population may be a result of the aggressive anticoagulation regimen used. Alternatively, reduction of infarct size with preservation of regional and overall left ventricular function has been suggested to be the primary mechanism by which thrombolytic therapy may reduce the incidence of left ventricular mural thrombus formation.[5] The lack of reduction in incidence of left ventricular mural thrombus formation in patients receiving thrombolytic therapy noted in this study may be explained by the similarity in postinfarction wall motion and overall left ventricular function when compared with non-lytic-treated patients. The results of this study suggest that the incidence of left ventricular mural thrombus formation is not reduced by the use of aspirin and intravenous heparin, alone or in combination with thrombolytic therapy, when compared with historical controls when anteroapical wall motion and overall left ventricular function are not preserved; however, in-hospital embolic complications are rare.

REFERENCES

1. Asinger RW, Mikell FL, Elsperger J, Hodges M. Incidence of left ventricular thrombosis after acute transmural myocardial infarction. N Engl J Med 1981;305:297-303.
2. Weinreich DJ, Burke JF, Ferrel JP. Left ventricular mural thrombi complicating acute myocardial infarction. Ann Intern Med 1984;100:789-94.
3. Stratton JR, Lighty GW, Pearlman AS, Ritchie JL. Detection of left ventricular thrombus by two dimensional echocardiography: sensitivity, specificity, and causes of uncertainty. Circulation 1982;66:156-66.
4. Eigler N, Maurer G, Shah P. Effect of early systemic thrombolytic therapy on left ventricular mural thrombus formation in acute anterior myocardial infarction. Am J Cardiol 1984; 54:261-3.
5. Held AC, Gore JM, Paraskos J, Ball SP, Corraco LM, Alpert JS. Impact of thrombolytic therapy on left ventricular mural thrombi in acute myocardial infarction. Am J Cardiol 1988; 62:310-1.
6. Nordrehaug JE, Johannessen KA, Von Der Lippe G. Usefulness of high-dose anticoagulants in preventing left ventricular thrombus in acute myocardial infarction. Am J Cardiol 1985; 55:1491-3.
7. Davis M, Ireland MA. Effect of early anticoagulation on the frequency of left ventricular thrombi after anterior wall acute myocardial infarction. Am J Cardiol 1986;57:1244-7.
8. Gueret P, Dubourg O, Ferrier A, Farcot J, Rigaud M, Bourdarias JP. Effects of full dose heparin anticoagulation on the development of left ventricular thrombosis in acute transmural myocardial infarction. J Am Coll Cardiol 1986;8:419-25.
9. Vecchio C, Chiarella F, Lupi G, Bellott P, Domenicucci S. Left ventricular thrombus in anterior acute myocardial infarction after thrombolysis: a GISSI-2 connected study. Circulation 1991;84:512-9.
10. Lupi G, Domenicucci S, Chiarella F, Bellott P, Vecchio C. Influence of thrombolytic treatment followed by full dose anticoagulation on the frequency of left ventricular thrombi in acute myocardial infarction. Am J Cardiol 1989;64:588-90.

Recurrent syncope in a patient with prominent J wave

Preben Bjerregaard, MD, DMSc, Ihor Gussak, MD, PhD, Susan L. Kotar, Joan E. Gessler, and Denise Janosik, MD *St. Louis, Mo.*

The patient is a 47-year-old African-American woman who was seen with her first syncopal episode at the age of 20 years. This was followed by multiple episodes of syncope, which were often accompanied by seizure activity. For a period of time she was treated with phenytoin without any obvious effect. She lived in a protected environment with

From the Division of Cardiology, St. Louis University Health Sciences Center.

Reprint requests: Preben Bjerregaard, MD, DMSc, St. Louis University Health Sciences Center, Division of Cardiology, 3635 Vista Ave. at Grand Blvd., St. Louis, MO 63110-0250.

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