**Pathological Extent of Interventricular Septal Infarction
in Patients with Acute Anteroseptal Myocardial Infarction
with and without Right Bundle Branch Block**

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SUMMARY

The purpose of the present study is to elucidate the difference in extent of myocardial infarction (MI) between those cases with and those without right bundle branch block (RBBB) occurring during the course of acute MI. We examined postmortem hearts from 20 patients with acute anteroseptal MI; 10 with (group A) and 10 without RBBB (group B). The extent of MI was studied pathologically in the interventricular septum (IVS) and reconstructed. The longitudinal extent of MI did not distinctly differ between groups A and B. In 5 hearts of group B, the anterior limit of the MI extended as high, or as close to the cardiac base, as in group A hearts. Transmural MI was relatively common and seen more frequently in group A than in group B. However, the MI did not always extend evenly to the left and right ventricular sides in the IVS. Left-sided predominance in extent of the MI was more frequently observed in group B than in group A. Right-sided predominance in the extent of septal MI was relatively rare and was seen in 5 cases at the portion where the right bundle branch came down. Four of these were group A patients. Thus, involvement of the right bundle branch might partly depend on the unevenness of the mural extent of the MI as well as the height of extension of the MI in the IVS. (Jpn Heart J 34: 121-129, 1993.)

Key Words:

Acute anteroseptal myocardial infarction Right bundle branch block
Interventricular septum

RIGHT bundle branch block (RBBB) newly acquired during the acute phase of myocardial infarction (MI) has a grave prognostic significance and is considered to be closely related to the extent of the MI.¹⁾⁻⁴⁾ In clinical practice, however, extensive MI of the left ventricle evidenced by electrocardiogram (ECG) or by echo- or angio-cardiograms is not always complicated by RBBB in

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Received for publication June 16, 1992.

Accepted September 9, 1992.

its acute phase. On the other hand, the pathological extent of MI has not been defined precisely in patients with conduction disturbances complicating acute MI, although histological changes of the atrioventricular (AV) conduction system have been described.⁵⁾⁻⁹⁾

The aim of the present study is to clarify the difference of pathological extent of MI between cases with and those without RBBB in the setting of acute anteroseptal MI.

METHODS

We studied the pathological extent of MI in 10 hearts from patients who developed RBBB during the course of acute anteroseptal MI (group A) and compared it to the extent of MI in 10 hearts from patients who died from acute anteroseptal MI uncomplicated by RBBB (group B). Patients who died within a day of the onset of MI were excluded so that the pathological analysis of MI would be reliable. Acute anteroseptal MI was diagnosed clinically and confirmed pathologically in both groups.¹⁰⁾ ECG criteria for RBBB were a QRS width longer than 0.12 sec with a QR configuration in V₁ and a slurred S wave in V₆.^{11),12)} RBBB was persistent until death in all the patients except one (case 8). Another patient (case 1) showed transient complete AV block in addition to RBBB. Table I summarizes the patient characteristics. None of the subjects had received acute coronary intervention such as thrombolytic therapy or angioplasty.

The hearts were examined after fixation in 10% buffered formalin. Anatomic patterns of distribution of the coronary arteries were defined according to Schlesinger's classification.¹³⁾ The extent of stenotic lesions of the epicardial coronary arteries was grossly estimated by examination of cross-sections at 3-mm intervals. Histologic sections were studied when 75% or more stenosis and/or coronary thrombi were suspected on gross observation. Atherosclerotic stenosis was graded as 1: 0-24%, 2: 25-49%, 3: 50-74% and 4: 75-100% based on the degree of luminal obliteration within the luminal circle bordered by the internal elastic membrane. Grade 4 stenosis due to either atherosclerosis or thrombosis and an obstructive thrombus was regarded as significant.

The extent of MI was studied throughout the whole IVS. Pathologically, we defined MI as coagulation necrosis of myocardial cells. The upper (basal) portion of the IVS was studied with coronal cutting for the purpose of simultaneous histological study of the AV conduction system,¹⁴⁾ and the lower (apical) portion was cut transversely at intervals of 5 mm. Histologic sections were prepared and stained with hematoxylin-eosin and with immuno-histologic staining of myoglobin if necessary. The extent of MI was studied on these macroscopic

Table I. Summary of the Subjects

Case	Age	Sex	Time from MI to Death (days)	Time from MI to RBBB	Cause of Death	Heart Weight (g)
group A						
1	63	M	8	3d	VT.Vf	570
2	75	M	3	<14h	Shock	420
3	64	M	4	2h	Shock	330
4	81	M	6	< 1h	Shock	350
5	71	F	2	<14h	Shock	410
6	82	M	16	< 5d	Shock	380
7	84	M	2	4h	Shock	550
8*	75	M	5	8h	VT.Vf	500
9	64	F	5	2d	Shock	550
10	57	M	3	2d	CHF	350
	71.6±9.3		5.4±4.2			441±93 (mean±SD)
group B						
11	77	M	8		VT.Vf	410
12	82	F	12		VT.Vf	400
13	68	M	11		CHF	410
14	75	M	2		Shock	580
15	81	M	2		CHF	520
16	65	M	5		VT.Vf	400
17	47	M	2		Shock	620
18	72	F	21		Shock	355
19	55	M	14		VT.Vf	600
20	86	M	3		Shock	440
	70.8±12.4		8.0±6.1			474±97 (mean±SD)

Abbreviations : MI = myocardial infarction; RBBB = right bundle branch block; M = male; F = female; d = days; h = hour(s); VT.Vf = ventricular tachycardia and/or fibrillation; Shock = cardiogenic shock; CHF = congestive heart failure.

* A case with transient RBBB.

Fig. 1. The three extending patterns of MI on the coronal sections of the IVS (immuno-histologic staining of myoglobin, ×5). Infarcted myocardium is not stained because of loss of myoglobin. The edges of infarction in the subendocardium of the left and right ventricular sides are indicated by small arrows. The right ventricular cavity is to the right. In type L or R, MI was more extensive in the left or right ventricular side than in the other side of the IVS, respectively. In type T, the extent of MI was exactly transmural.

cut surfaces and histologic sections grossly and with a magnifying glass ($\times 5$), and then reconstructed. On the coronal sections of the IVS, we classified extending patterns of MI into 3 types as shown in Fig. 1. In type T, the extent of MI was exactly transmural, since the difference in the distance from the crest of the IVS to the edge of the MI between the left and right ventricular sides was within 5 mm. In type L or R, the MI extended predominantly to the left or right ventricular side of the IVS, respectively. The incidence of these 3 types was analyzed.

RESULTS

Pathological findings of the coronary arteries

As shown in Table II, all 20 patients had significant lesions of the left anterior descending artery (LAD), which were proximal to the first septal branch in 9 group A and 5 group B hearts. Fresh LAD thrombosis was observed in 9

Table II. Pathological Findings of the Major Coronary Arteries

Case	Anatomic Pattern of the Coronary Arteries ⁽³⁾	Stenotic Lesions and Thrombosis of the Coronary Arteries			
		LMT	LAD	LCX (PD)	RCA (PD)
group A					
1	R-dominant	2	'2#	1	3 (1)
2	balanced	1	'2#	1	1 (1)
3	balanced	1	3	2	2 (1)
4	balanced	1	'4#	3	4 (3)
5	balanced	1	'3#	2	2 (4)
6	balanced	1	'4#	3	4 (4)
7	balanced	3	'4#	4	4 (2)
8*	balanced	4	'4###	4	4 (1)
9	balanced	4	'4###	3	4 (2)
10	balanced	2	'4	3	3# (1)
group B					
11	balanced	4	'4#	4	3 (4)
12	balanced	2	4#	2	2 (4)
13	balanced	4	'4	4	4 (3)
14	L-dominant	2	3#	2 (1)	4
15	balanced	1	'4#	4	4 (3)
16	balanced	2	'4#	2	2 (1)
17	balanced	1	'4	4	2 (4)
18	R-dominant	1	4	4	1 (4)
19	balanced	2	4###	3	1 (1)
20	L-dominant	1	4#	3 (4)	1

Abbreviations: LMT=left main trunk; LAD=left anterior descending artery; LCX=left circumflex artery; RCA=right coronary artery; PD=posterior descending artery; R- or L-dominant=the right or left coronary artery-dominant distribution.

* A case with transient RBBB, † proximal to the first septal branch, # or ## with a recent obstructive or non-obstructive thrombus, respectively.

Grading of atherosclerotic stenosis of the coronary arteries: 1: <25%, 2: 25-49%, 3: 50-74%, 4: >75% of luminal narrowing.

group A and 7 group B hearts. At the site of the LAD thrombosis, atherosclerotic stenosis was insignificant ($<75\%$) in 4 group A and 1 group B cases. The number of cases with one, two and three vessel disease was 3, 5 and 2 in group A; and 2, 3 and 5 in group B, respectively.

The pathological extent of MI in the IVS

Figure 2 shows the longitudinal extent of the MI which is reconstructed on a schematic diagram of the right ventricular surface of the IVS. In hearts of group A, all MIs reached the basal third of the IVS. In group B, however, the extent of septal MI differed case by case. Five group B patients (cases 11, 12, 13, 14, 16) had septal MIs which were as extensive as those of group A hearts.

Unevenness of the extent of MI in the IVS

Figure 3 shows the incidence of these 3 types at three different points; anterior edge of the IVS (anterior IVS), 20 mm posterior to the anterior IVS (mid-IVS) and 40 mm posterior to the anterior IVS (posterior IVS). Mid-IVS corresponded closely with the portion where the right bundle branch descended toward the moderator band. Type T was relatively common and observed more frequently in group A than in group B; 7 of 9 cases at the anterior IVS, 5 of 8 cases at the mid-IVS and 5 of 11 cases with type T at the posterior IVS were group A patients. Type L was seen more frequently in group B than in group A hearts. Type R was relatively rare and more frequent in group A than in group

Fig. 2. Pathological extent of the leading edge of MI in the IVS. Pathological extent of MI is reconstructed in a schematic diagram of the right ventricular surface of the IVS. Dotted area indicates the extent of MI, and the more darkly dotted area represents variations of extent of the leading edge of MI in each group. Numerals represent the case numbers.

Fig. 3. The incidences of types L, T and R are illustrated using histograms. The incidence was examined at three different points; anterior IVS (Ant. IVS = anterior edge of the IVS), mid-IVS (Mid-IVS = 20 mm posterior to Ant. IVS) and posterior IVS (Post. IVS = 40 mm posterior to Ant. IVS). Mid-IVS corresponded closely with the portion where the right bundle branch descended toward the moderator band (indicated by an arrow in the right upper inset).

B; 1 of 2 cases at the anterior IVS, 4 of 5 cases at the mid-IVS and 2 of 2 cases with type R at the posterior IVS were group A patients.

All of the group A patients showed type R or T at mid-IVS except for case 10. In this case, the MI reached the distal part of the right bundle branch (approximately up to 20 mm from the top of the IVS) which resulted in significant necrosis of the right bundle branch. Case 10 was the only patient who had a recent thrombus of the right coronary artery in addition to critical stenosis of the left anterior descending artery.

DISCUSSION

The present study revealed that half of the patients without RBBB showed high extension of MI in the IVS similar to those observed in group A. Extensive MI was not necessarily consistent with occurrence of RBBB, although high extension of the "transmural" MI (type T) seemed to be one of the determinants for development of RBBB. On the other hand, we had previously reported that the right bundle branch showed significant necrosis (MI) in all RBBB cases except for one case with transient RBBB and no necrosis in non-RBBB cases in the same subjects as in the present study.¹⁴⁾ Therefore, development of RBBB correlated excellently with necrosis of the right bundle branch, but not necessarily with the extent of MI. This discrepancy might be partly explained by our second finding that the MI was not always transmural, and that the MI sometimes extended unevenly to the left and right ventricular sides of the IVS. One of

the most impressive findings was that the MI could be more extensive in the right ventricular side than in the left ventricular side of the IVS.

Some pathological studies have indicated that the MI extended high in the IVS surrounding the AV conduction system in cases with conduction disturbances.^{6),9)} However, the precise extent of the MI has not been described. The difference in the extent of MI between cases with and those without conduction disturbances has not been clarified. Furthermore, to our knowledge, unevenness of mural extent of MI in the IVS has never been discussed.

The main blood supply to the IVS is derived from the anterior and posterior descending arteries.¹⁵⁾⁻¹⁷⁾ The septal branches from the LAD perfuse the anterior two thirds of the IVS, whereas the posterior septal branches and the AV nodal artery supply the posterior third of the IVS.¹⁵⁾⁻¹⁷⁾ The course of these septal branches closely follows the right ventricular cavity.^{15),17)} This is analogous to the epicardial arteries running in the free wall of the left ventricle where the extent of MI is predominantly in the subendocardium rather than in the subepicardium.¹⁸⁾ Therefore, in the IVS, the average MI is thought to be more extensive in the left ventricular than in the right ventricular side. Contrary to this expectation, some cases showed right-sided predominance, which might lead to involvement of the right bundle branch since such right-sided predominance was more frequently observed in group A than in group B hearts.

Some possible explanations for this observation can be proposed. One is that branches derived from the proximal coronary arteries may influence the arterial blood supply to the IVS. Rodrigues et al¹⁹⁾ found "the descending septal artery", from the proximal right coronary artery or directly from the aorta in 31 of 341 patients without coronary heart disease, and they described some of them spreading to replace the arborization of the anterior septal branches. Morphological studies^{15),17)} revealed septal branches coming off from the left main branch and uncommonly from the left circumflex artery. A second possible mechanism is collateral circulation which covers the area of the critically obstructed LAD. A third possibility is that arborization of the septal branches from the LAD may spread more extensively in the right ventricular side than in the left ventricular side of the IVS.

A few studies are available regarding the coronary arteries in patients with conduction disturbances.²⁰⁾⁻²²⁾ However, they have never discussed the coronary lesion qualitatively, i.e. whether it is a thrombus or an organic stenosis, since they were based on the findings of postmortem angiography²⁰⁾ or cineangiography.^{21),22)} In the present study, insignificant atherosclerotic stenosis coexisting with LAD thrombosis was more frequent in group A than in group B, as was the LAD thrombosis itself, although statistical significance was not obtained. In general^{23),24)} coronary occlusions are more likely to cause extensive transmural MI

when occurring suddenly, i.e. acute thrombosis, in vessels that are not critically obstructed and have no adequate and functional distal collaterals. From the viewpoint of coronary lesions, thrombosis associated with non-critical stenosis of the LAD might be responsible for the occurrence of RBBB.

In conclusion, development of RBBB might largely depend on high extension of the MI in the IVS and partly on the unevenness of the mural extent of the MI. Such fluctuation of the mural extent of the MI may in part explain the clinical inconsistency that extensive MI is not always complicated by RBBB.

ACKNOWLEDGMENTS

We thank Miss M. Kobayashi and N. Hirata for their technical assistance.

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