Pathomorphological Criteria for Cardiosclerosis and Angioarchitectonics of the Hypertrophic Myocardium in Hypertensive Heart

V. D. Rozenberg and L. M. Nepomnyashchikh

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We performed a pathomorphological study of 200 hearts with cicatricial changes from patients died from hypertensive disease. Most postinfarction scars in men were transmural and localized in the anterior and posterior wall of the left ventricle and in the interventricular septum. Non-transmural scars were revealed in the lateral wall (primarily in women). Pathognomonic changes in the architectonics of the heart included reduction of regional blood flow and segmentary, discontinuously extended, and diffusely extended atherosclerotic obstruction. Changes in the index of blood supply to the myocardium corresponded to pronounced decrease in vascularization of the hypertrophic left ventricle. A correlation was found between the index of blood supply to the myocardium and mean systolic and diastolic blood pressure. Therefore, the coronary bed did not satisfy the demands of hypertensive heart with cicatricial changes.

Key Words: *hypertensive disease; myocardial hypertrophy; postinfarction and microfocal cardiosclerosis; coronarography; morphometry*

Hypertensive heart (HH) is a functional and morphological complex of symptoms characterized by phasic development of functional, hemodynamic, and pathomorphological changes in various regions of the heart. This term was proposed after detailed studies of the mechanisms of hypertrophy of the left ventricle, which develops in response to the increase in blood pressure (BP) during hypertension [1,10]. Postinfarction and microfocal scar injuries in the myocardium (SIM) are of particular importance. These disturbances contribute to insufficiency of the hypertrophic myocardium. Much attention is given to factors of patho- and thanatogenesis [11,14].

Here studied the incidence and typical characteristics of SIM in HH and evaluated the relationship between cardiosclerosis and type of atherosclerotic obstruction in the major coronary arteries (CA) in men and women with hypertensive disease.

Department of General Pathology and Pathomorphology, Institute of Regional Pathology and Pathomorphology, Siberian Division of the Russian Academy of Medical Sciences, Novosibirsk. ***Address for correspondence:*** pathol@cyber.ma.nsc.ru. Nepomnyashchikh L. M.

MATERIALS AND METHODS

We examined 200 HH with SIM from 120 men and 80 women with hypertensive disease (mean age 54.2±0.6 years). Sixty hearts without pathomorphological signs of SIM were taken from individuals of comparable age after accidental death and served as the control. The severity of SIM in the functionally overloaded left ventricle was evaluated by the method recommended by the World Health Organization and involving macroscopic examination of transverse serial sections of the heart. The development of postinfarction myocardial scars or microfocal cardiosclerosis was confirmed by the appearance of connective tissue focuses with an area of more or less than 2 cm^2, respectively. Morphometric and histological examination, separate weighing of the ventricular myocardium, and volume-weight and planimetric cardiometry were performed to estimate functional capacities of HH [6]. Changes in the coronary bed of HH were determined by a modified method of polypositional postmortem coronarography [5]. Atherosclerotic obstructive dam-

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TABLE 1. Incidence and Depth of SIM in Patients with HH

Type of scar injury	Number of patients		Sex		Transmural		Non-transmural	
	abs.	%	men	women	abs.	%	abs.	%
Postinfarction scars	125	62.5	80	45	70	56	55	44
Microfocal sclerosis	75	37.5	40	35				
Total	200	100	120	80	70	56	55	44

ages to major CA in the cicatrized myocardium were classified by length (segmentary, discontinuously extended, and diffusely extended changes).

For light microscopy, myocardial samples were fixed in 10% neutral formalin. The staining procedure for paraffin sections included hematoxylin-eosin and van Gieson's picrofuchsin. Elastic fibers were stained with Weigert's picrofuchsin-fuchseline. Three-color staining procedure was used to visualize the muscle and connective tissue [4]. Volume density of the vascular bed (vascularization of the left ventricle wall) and connective tissue was determined by planimetry. The index of blood supply to the myocardium (IBSM) was determined after weighing of heart chambers [7]. Mean BP was calculated by the criteria recommended by the World Health Organization. The results were analyzed by methods of variational statistics (Student's *t* test).

RESULTS

The study of the incidence, localization, and depth of SIM in HH showed that myocardial scars were most abundant (62.5%, Table 1). This type of scar injury in men was observed more frequently than in women (by 1.8 times). The incidence of transmural scars was maximum (56%). Microfocal cardiosclerosis was revealed in 37.5% HH. It should be emphasized that the incidence of this disorder in men was 1.1 times higher than in women.

Postinfarction myocardial scars were predominantly localized in the posterior wall of the hypertrophic left ventricle (Table 2). The incidence of these transmural scars in men was 2 times higher than in women. The interventricular septum ranked second as a site of postinfarction SIM in the left ventricle of HH. It should be emphasized that 25.7% scars were transmural. Their incidence in men was 10-fold higher than women. The anterior wall of the left ventricle was the third most frequent site of injury. Transmural scars were mainly found in men.

Postinfarction scars were present in 13.6% samples from the lateral wall of the hypertrophic left ventricle. These non-transmural postinfarction scars were found mainly in women. Postinfarction scars were revealed only in 8% samples from the apex of the heart. Non-transmural scars prevailed in men (Table 2). The incidence of circular postinfarction scars was minimum and did not differ in men and women. Most damages were non-transmural.

These changes were pathognomonic and differed from those observed during other heart diseases [2]. Coronarography and pathomorphological study revealed a correlation between postinfarction scars in the myocardium of HH and type of obstructive and atherosclerotic damage to major CA. The incidence of segmentary obstruction was highest (39.2%). The pathological process involved individual (mainly proximal) segments of major CA. Diffusely extended obstruction with pronounced stenosis of various segments was the second most frequent type of damage (37.6%). Discontinuously extended obstructive atherosclerotic injury with damage to several segments of major CA

TABLE 2. Incidence, Localization, and Depth of Postinfarction Scars in the Left Ventricle of Patients with HH

Region of the left ventricle	Number of patients		Sex		Transmural		Non-transmural	
	abs.	%	men	women	abs.	%	abs.	%
Anterior wall	20	16.0	12	8	12	17.1	8	14.5
Posterior wall	48	38.4	32	16	28	40.0	20	36.4
Lateral wall	17	13.6	6	11	8	11.4	9	16.4
Interventricular septum	22	17.6	20	2	18	25.7	4	7.3
Apex	10	8.0	6	4	2	2.9	8	14.5
Circular localization	8	6.4	4	4	2	2.9	6	10.9
Total	125	100	80	45	70	100	55	100

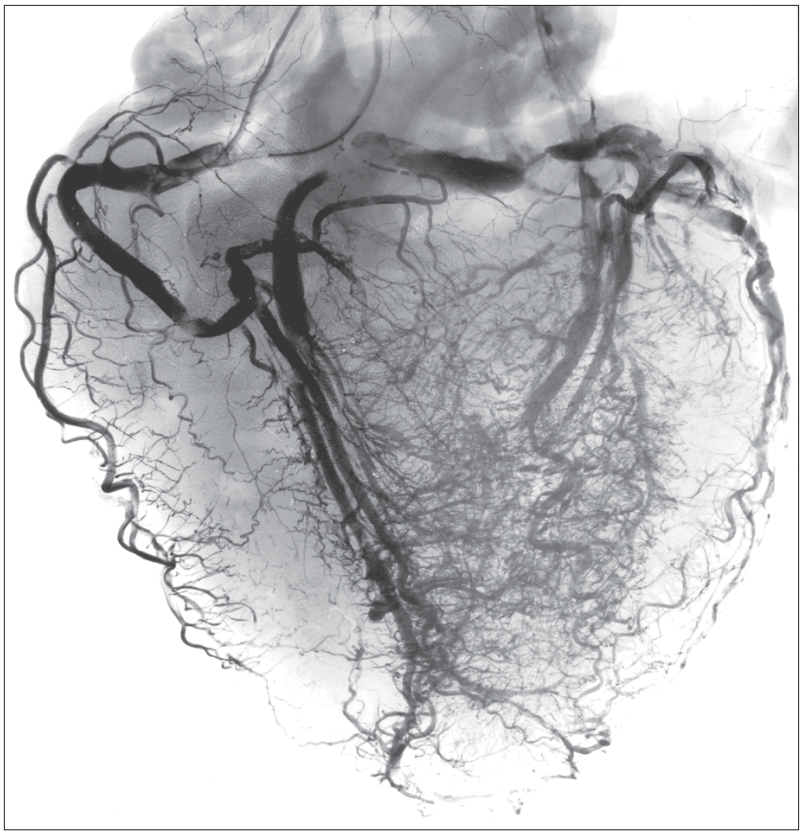

Fig. 1. Segmentary obstructive changes in the anterior interventricular branch of the left coronary artery and syndrome of complete collateral blood flow during postinfarction transmural cicatrization of the posterior wall in the left ventricle (patient V., 56 years). Postmortem cardioventriculography.

was rarely observed (23.2%). The incidence of discontinuously extended atherosclerotic obstruction was 4.2 times lower than that of segmentary and diffusely extended damage to major CA of HH.

We evaluated the relationship between changes in angioarchitectonics of HH with SIM and the type of atherosclerotic obstruction in the major CA. Pathognomonic reconstruction of the bed was accompanied by reduction of regional blood flow. Segmentary coronary obstruction was followed by pronounced recalibration. We observed coronarographic syndrome of complete collateral blood flow. The closed "coronary circuit" of new blood flow pathways encircled the microvascular collateral network, which was formed around major CA with severe stenotic damage (Fig. 1). Complete collateral blood flow enclosed alternating regions of terminal microvascular hypervascularization and "vascular exhaustion" in the "coronary circuit" (Fig. 2). Severe diffusely extended stenosis was followed by changes in the architectonics of HH and loss of magistral characteristics of major CA and primary branches (Fig. 3).

The degree of vascularization in the functionally overloaded left ventricle of HH differed in various topographic zones (Table 3). In both subgroups the degree of vascularization was maximum in the anterior and posterior wall of the left ventricle, but decreased in the septum and apex of the heart. Vascularization of the myocardium was minimum in the lateral wall of the left ventricle during postinfarction and microfocal cardiosclerosis.

Changes in IBSM corresponded to a pronounced decrease in vascularization of the hypertrophic left ventricle in HH (Table 3). The mean value of systolic and diastolic BP in both subgroups markedly differed from the control (Table 3).

A correlation was found between IBSM and mean systolic and diastolic BP. Therefore, the anatomical functional inadequacy of the coronary bed to demands of the hypertrophic and cicatrized myocardium is not compensated by propulsive function of BP. This is followed by a sharp decrease in contractility of the myocardium and changes in phasic activity of the ventricles (lengthening of the strain phase and shortening of the filling phase for the left ventricle) [3,9]. The loss of regional contractility in cicatricial zones of the myocardium is to a lesser extent compensated by collateral blood flow [6,7].

Polymorphic changes in the vascular bed of cicatrized HH depend on the area and localization of scar tissue. Recalibration of the coronary bed accompanies pronounced atherosclerotic obstruction, which reflects progressive cicatrization. Progressive stenosis of anastomosing branches and functional insufficiency of compensatory microvascular plexus promote these changes. The latter dysfunction determines pronoun-

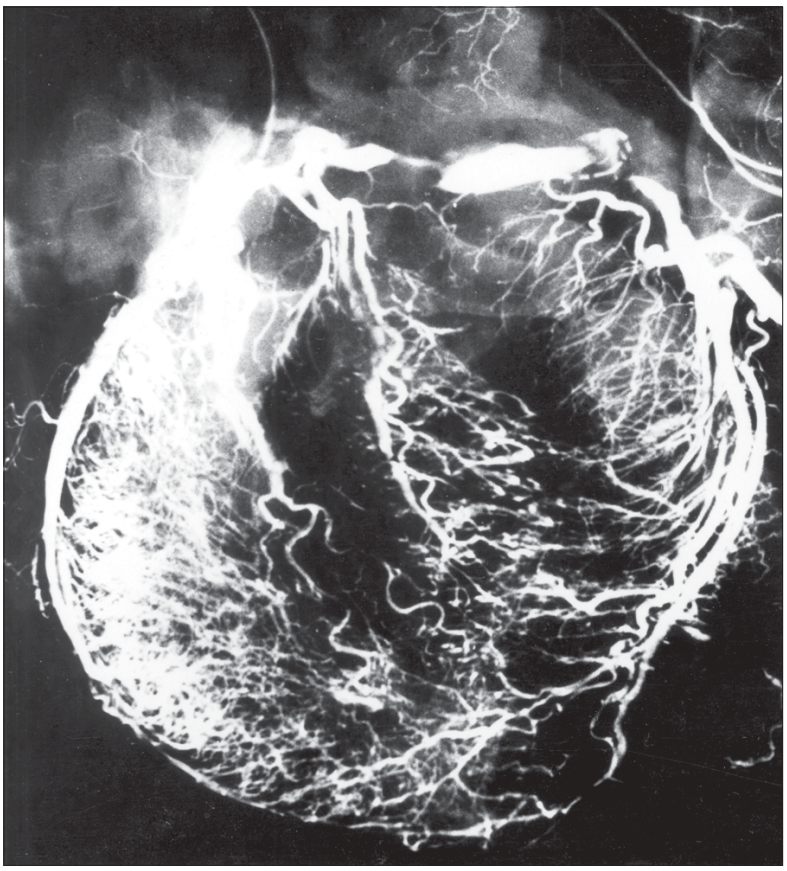

Fig. 2. Discontinuously extended obstructive changes in vessels of the left coronary bed, loss of magistral characteristics, and syndrome of complete collateral blood flow around alternating regions of microvascular hypervascularization and "vascular exhaustion" during postinfarction transmural cicatrization of the interventricular septum (patient M., 58 years). Coronarogram of the opened heart.

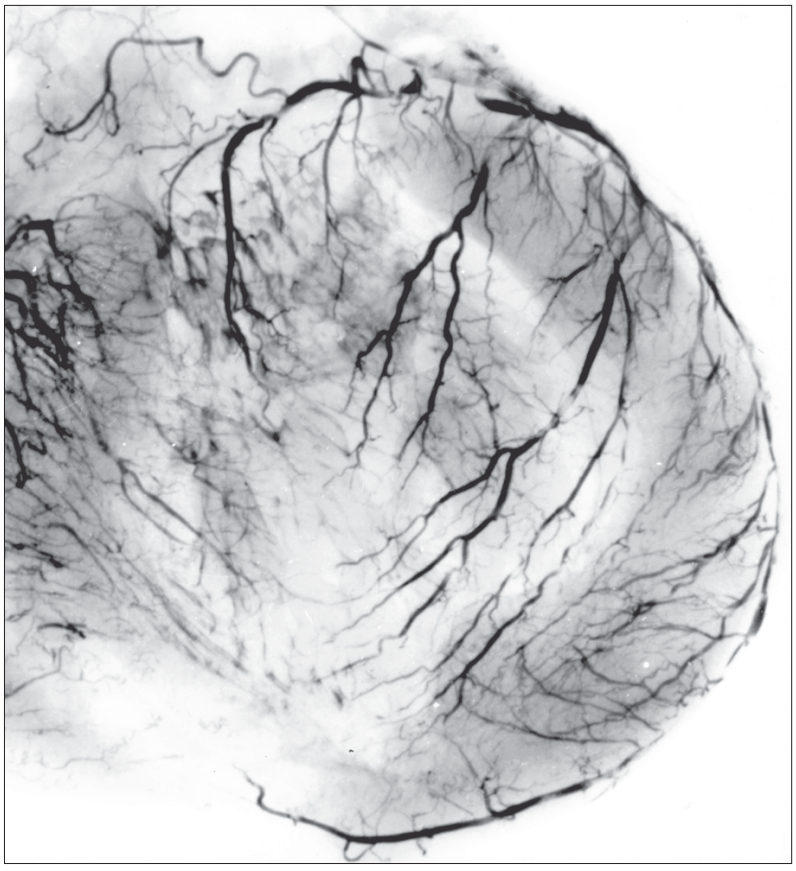

Fig. 3. Diffusely extended obstructive changes in vessels of the left coronary bed, loss of magistral characteristics, and presence of vertical coronary vessels characterized by straight-line branching and formation of stamps. Syndrome of "small-branching dead tree" during postinfarction transmural circular cicatrization (patient N., 60 years). Coronarogram of the opened heart.

TABLE 3. BP, Volume Density of Vessels (Degree of Vascularization) in the Left Ventricle, and IBSM during Postinfarction and Microfocal Cardiosclerosis in Patients with HH (*M*±*m*)

Type of scar injury	Number of patients	BP, mm Hg		Volume density of vessels, %				IBSM, g/mm^2
		systolic	diastolic	anterior wall	posterior wall	lateral wall	septum and apex	
Postinfarction scars	60	210.5±4.1*	109.1±1.8*	40.0±1.7	37.3±1.3*	30.4±3.2	36.1±1.7	23.8
Control	10	134.1±3.2	85.1±2.4	48.8±3.1	45.5±1.2	36.1±1.7	37.9±1.9	19.7
Microfocal sclerosis	60	194.5±2.2*	101.3±1.3*	42.2±0.6*	40.7±1.5*	31.3±1.5*	36.4±1.2	21.6
Control	10	126.1±2.2	80.2±1.4	51.1±1.7	50.3±1.7	38.7±1.3	38.3±1.2	18.9

Note. *$p<0.01$ compared to the control.

ced coronary reconstruction of the so-called "hibernated" and "thundered" myocardium in the area of scar injury and distant regions [8,12].

Severe ischemia of the left ventricle in HH alters contractility of the myocardium, which is followed by progression of hypoxia and abolishes the compensatory response. Pronounced coronary hypoxia usually accompanies the influence of extreme factors and overload of HH. These changes produce decompensation of the cicatrized hypertrophic heart, which contributes to dilation of cavities and formation of a "large flaccid heart" [13]. The presence of these symptoms in most dead patients attests to a particular course of hypertensive disease [11].

The relationship between SIM and changes in architectonics of HH illustrates the genesis of clinical dysfunction and clinicopathomorphological dissociation. The decrease in functional capacity of HH during various types of cardiosclerosis is associated with insufficiency of blood supply to the hypertrophic myocardium, rather then with the loss of muscle tissue. Reduced vascularization of HH and value of IBSM reflect blood flow velocity and its adequacy to the requirements of the myocardium. These changes determine polypathic pathogenesis of cardiac insufficiency during HH.

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