MORPHOLOGY AND PATHOMORPHOLOGY

Pathomorphology and Pathogenic Role of Myocardial Bridges in Sudden Cardiac Death

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Pathognomonic incidence of myocardial bridges during obstructive hypertrophic cardiomyopathy, hypertension, and ischemic heart disease was established. Myocardial bridges were predominantly found in the median segments of major coronary arteries with prevailence of bridge-like obstructions in the anterior interventricular branch of the left coronary artery. Typical changes in cardiac angioarchitectonics indicating pronounced inadequacy of coronary blood flow were determined depending on the segmentary directionality of bridge obstruction. The data attest to pronounced pathogenetic role of myocardial bridges in sudden cardiac death.

Key Words: *sudden cardiac death; myocardial bridges; pathomorphology; coronarography*

According to WHO definition, sudden cardiac death (SCD) is a fatal outcome occurring unexpectedly 1-6 h after the onset of cardiac attack in patients with cardiovascular diseases, which were considered healthy or in a satisfactory state [3]. Possible reasons and mechanisms of SCD are ventricular fibrillation, electrical instability of the heart, changes in the heart conduction system and hypothalamus, and abnormalities in the myocardium and coronary arteries (CA). Increasing incidence of SCD in individuals with undamaged CA attests to an important role of myocardial bridges (MB) in triggering SCD [4,8].

MB are peculiar anatomical structures (muscle bundles, loops, and intersections), which cross or overleap the frontal aspects of CA. During systole, MB constrict the lumen of CA by more than 75% followed by complete (possible) recovery of the initial diameter during diastole [7]. MB crossing or overleaping CA induce myocardial ischemia, angina pectoris, myocardial infarction, and SCD. Despite considerable role of MB in the pathogenesis of SCD, the pathomorphology of MB received little attention.

Our aim was to examine the incidence, pathomorphological peculiarities, and pathogenetic importance of MB for SCD diagnostics in patients with different cardiac pathologies.

MATERIALS AND METHODS

The examined organs were the hearts of patients (n=540), who died of cardiac diseases of various genesis: ischemic heart disease (IHD, n=300), hypertension (n=120), hypertrophic cardiomyopathy (n=120, 60 cases of obstructive and 60 cases of non-obstructive forms of the disease). The control group comprised 60 age-matched persons, which casually died and had no MB and the above diseases.

The state of CA was examined by the modified method of postmortem contrast polypositional coronarography, which was used individually or in combination with WHO standard anatomical method [6]. Three main CA were examined in angiograms of specially opened and flattened hearts: the right, anterior interventricular, and circumflex branch of left CA. Each vessel was divided into four segments with equal inti-

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mal area preliminary calculated by WHO standard anatomic method [6]. The volume of vascularization for all subdivisions of the left ventricle was determined by angiometry [6]. The combined use of WHO standard method and volume-mass cardiometry made it possible to calculate the index of myocardial blood supply (the ratio of net muscular weight of the heart to the total lumenal area of CA). In parallel with examination of CA, MB were visualized by detailed macro- and micropreparation of the myocardium under stereomicroscope [7]. The length and width of MB were determined together with their ratio to the degree of atherosclerotic manifestations in each intersected segment of CA. In addition, the target histotopographic method was used. The data were processed statistically using Student's *t* test for paired samples.

RESULTS

Complex pathomorphological analysis of 540 hearts revealed MB in 220 cases (40.7%). In 150 cases, they provoked SCD (98 men and 52 women, the mean age 48.4±0.2 years). The greatest number of MB was scored in IHD patients (118 of 300 cases or 39.3%, Table 1). In this group, MB triggered SCD in 61% cases. In 40% cases, MB were observed in hypertension patients, where they caused SCD in 58.3% cases. In patients with obstructive and non-obstructive forms of hypertrophic cardiomyopathy, MB were detected in 60 and 30% cases, respectively. In patients with obstructive hypertrophic cardiomyopathy, they provoked SCD in 88.9% cases, while in patients with non-obstructive form they caused SCD in all examined cases. Acute coronary insufficiency and acute myocardial infarction rarely triggered SCD.

MB were the main cause of death in all types of cardiac pathology (Table 1). The data attest to pronounced pathognomonic potency of MB as the cause of SCD in patients with hypertrophic cardiomyopathy, especially in those with its obstructive form. Complex geometry of heart cavities caused by catenoidal shape of the interventricular septum and specific character of myocardial hypertrophy explain the "diving" course of CA characteristic of "bridge compression" [6,10]. In essential hypertension and coronary heart diseases, the individual segments of CA are located intramurally, which promoted bridge obstruction of CA [5,9].

Evaluation of the incidence of MB in three main CA, their length, width, and degree of atherosclerotic manifestations in the intersected segment revealed maximum vulnerability (102 cases) of the anterior interventricular branch of the left CA (Table 2). The length and width of MB in this CA were maximum (26.0±0.6 and 3.0±0.2 mm, respectively). In most cases (n=80), the atherosclerotic lesions were absent in the intersected segment of this CA. In the circumflex branch of the left CA, the incidence of MB was 2.8 times lower (n=36) than in the anterior interventricular branch, their length (22.8±0.8 mm) and width (2.4±0.4 mm) were also smaller. At the same time, in most cases (n=26) the atherosclerotic manifestations were absent in the intersected segments of this CA. The minimum number of MB (n=12) was found in the right CA, where their length (20.2±0.4 mm) and width (2.2±0.2 mm) were the smallest. In this vessel, most intersected segments had no atherosclerotic lesions.

When analyzing these data, one can say that most segments of CA with detected MB had no atherosclerotic lesions in the intersected segments. At the same time, a positive correlation was found between the incidence of MB and their length, width, and number of intact segments in CA. These data definitely substantiate reported findings [12-14] that MB "protect" the intersected segments of CA from atherosclerosis [7]. This hypothesis is also corroborated by clinical observations [1].

Analysis of the distribution of MB in segments of the main CA revealed predominant incidence of these structures in II and III segments (Table 3). In most cases, MB were observed in these segments of the anterior interventricular branch of the left CA (48.4 and 38.4%, respectively) and in the corresponding

TABLE 1. Incidence of MB in Various Cardiac Pathology and Death Causes

Cardiac pathology	Number of cases	MB incidence			
		total	SCD	acute coronary insufficiency	acute myocardial infarction
IHD	300	118 (39.3)	72 (61.0)	20 (16.9)	26 (22.0)
Hypertension	120	48 (40.0)	28 (58.3)	16 (33.3)	4 (8.3)
Obstructive hypertrophic cardiomyopathy	60	36 (60.0)	32 (88.9)	4 (11.1)	—
Non-obstructive hypertrophic cardiomyopathy	60	18 (30.0)	18 (100)	—	—

Note. Numbers in brackets show percentage of the cases relatively to the number of died persons in each group.

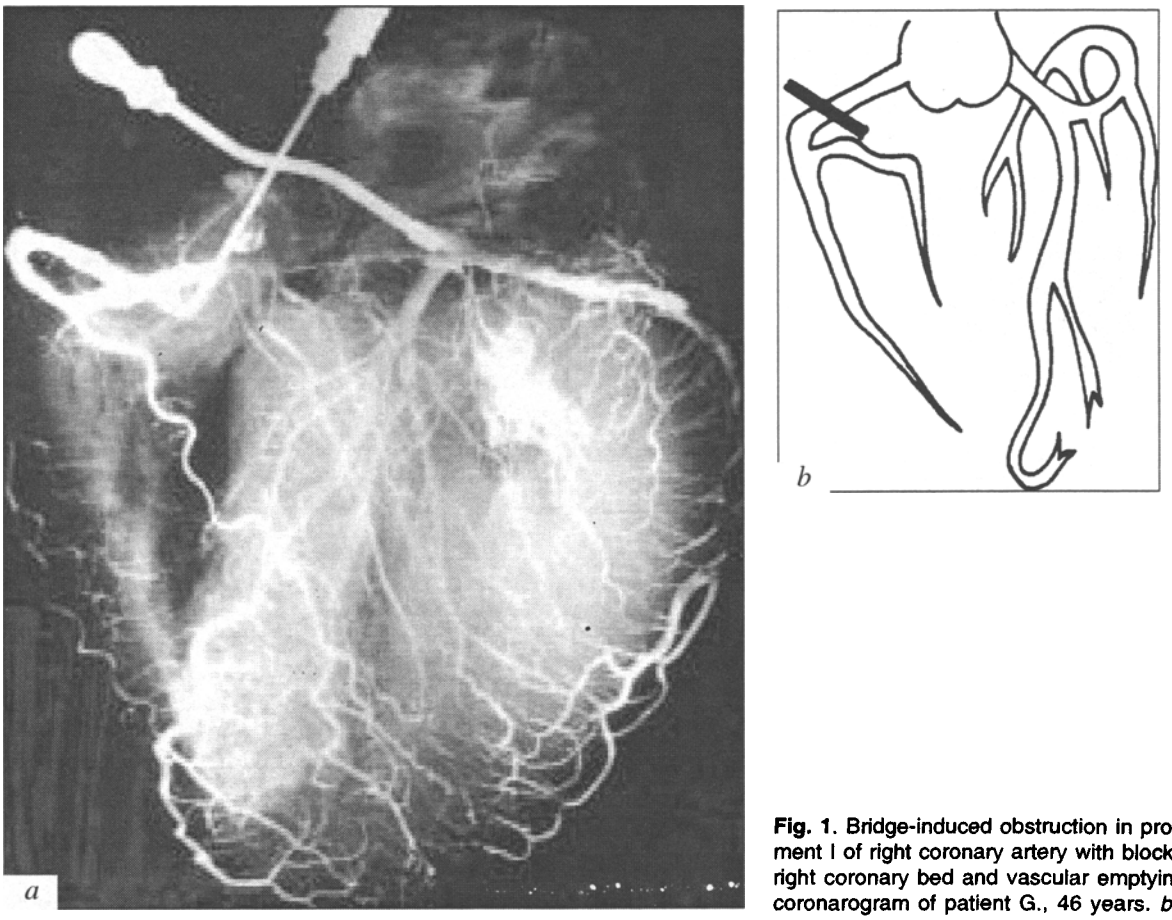

Fig. 1. Bridge-induced obstruction in proximal segment I of right coronary artery with blockade of the right coronary bed and vascular emptying area. *a*) coronarogram of patient G., 46 years. *b*) scheme.

segments of circumflex branch of this CA (45.5 and 33.3%, respectively). Slightly lower incidence of MB was established for II and III segments of the right CA (42.8 and 33.2%, respectively). At the same time, the right CA had maximum number of MB intersections of the first (proximal) segment (16.4%). In the fourth distal segments, the highest incidence of MB (12.1%) was established for the circumflex branch of the left CA. On average, MB were most frequently seen in segment II of all CA, while MB incidence was lower in segment III and far more lower in segments I and IV. Only in one case of SCD (male patient, 44 years, duration of the first cardiac attack 2 h), two MB were found, which crossed the segments II and III of the anterior interventricular branch of left CA in the form of thin muscle bands of equal size (length of 24.4 mm, width of 2.8 mm).

In accordance to segmentary directionality of MB localization in the major CA, typical changes were observed in heart angioarchitectonics in SCD persons. In addition to predominant localization of MB in segment I of the right CA, this artery was characterized by dilation, irregular contours, and pronounced proximal turn. The right coronary vascular bed was clamped, which manifested in vascular emptying (Fig. 1). The development of bridge obstruction observed pre-

TABLE 2. Incidence, Length, and Width of MB in Major CA and Degree of Atherosclerotic Changes in Intersected Segment (*M*±*m*)

CA		Number of cases	Length of MB, mm	Width of MB, mm	Atherosclerotic changes	
					minor	absent
Right		12	20.0±0.4	2.2±0.2	4	8
Left	anterior interventricular branch	102	26.2±0.6	3.0±0.2	22	80
	circumflex branch	36	22.8±0.8	2.4±0.4	10	26

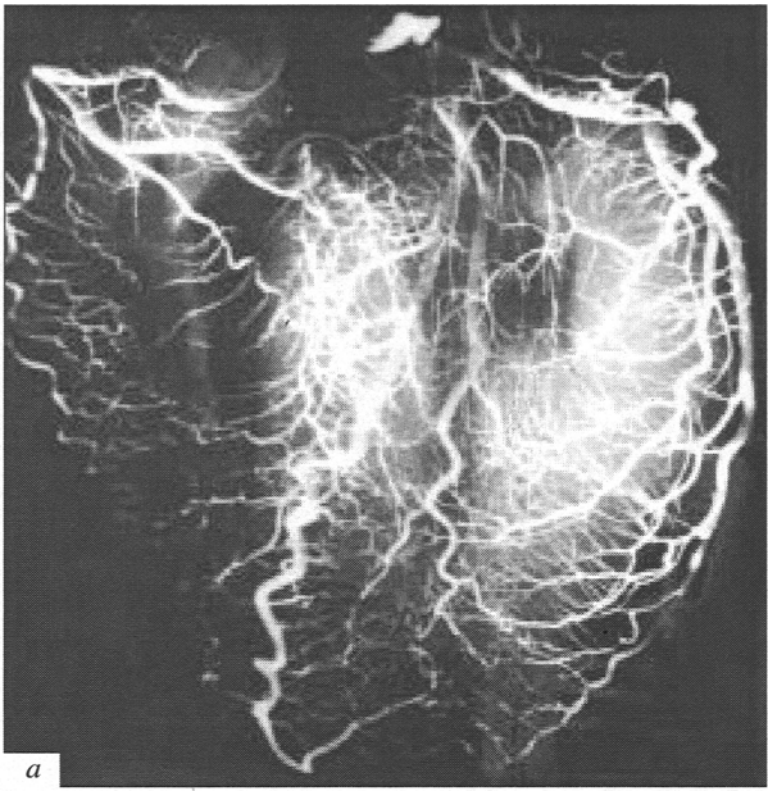

Fig. 2. Bridge-induced obstruction in median segment II of anterior interventricular branch of left coronary artery with manifested dilation, spiral-convoluted shape of the distal portion of this segment, and compensatory remodeling of left coronary vascular bed. *a*) coronarogram of patient N., 48 years. *b*) scheme.

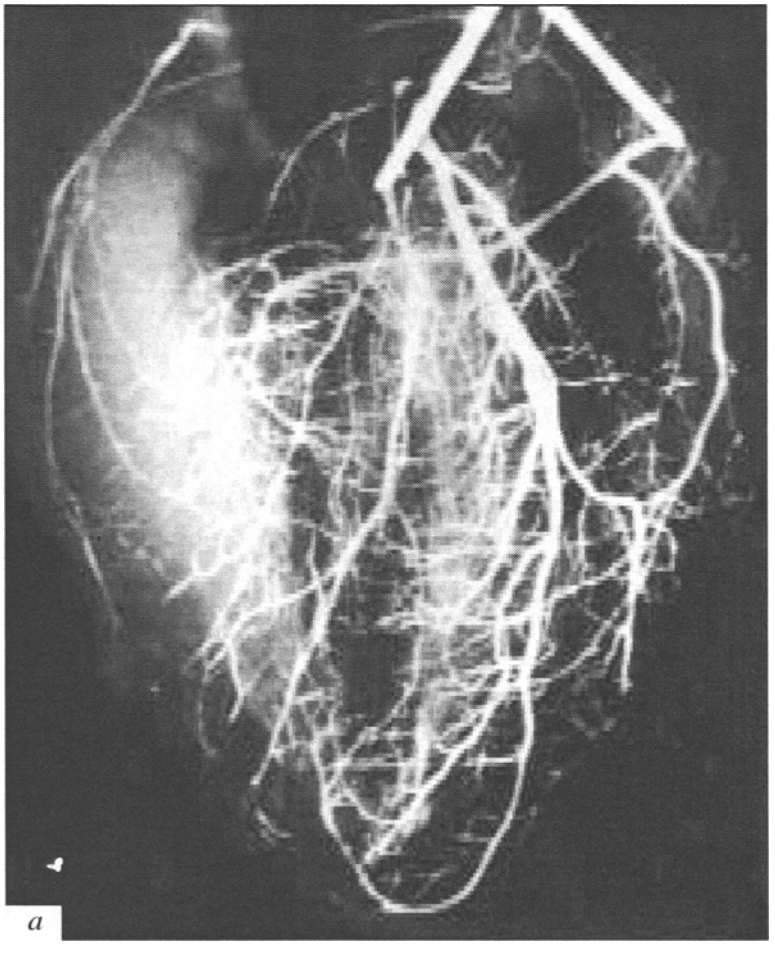

Fig. 3. Bridge-induced obstruction in distal segment IV of circumflex branch of left coronary artery with compensatory remodeling of left coronary vascular bed and the presence of two distinct zones: vascular emptying and compensatory hypervascularization. *a*) coronarogram of patient K., 50 years. *b*) scheme.

TABLE 3. Incidence MB in the Segments of Major CA, Vascular Volume Density in Left Ventricle and Myocardial Blood Supply Index during SCD (*M±m*)

CA	Total number of cases	Segmentary incidence, %				Vascular volume density in left ventricle, %								Myocardial blood supply index, g/mm²	
						anterior wall		posterior wall		lateral wall		septum and apex			
		I	II	III	IV	SCD	control	SCD	control	SCD	control	SCD	control	SCD	control
Right	12	16.4	42.8	33.2	7.6	36.4±1.2	38.8±1.6	35.6±1.2	36.6±1.6	26.2±1.2	28.6±1.6	26.8±1.8	28.8±1.4	18.8	16.8
Left															
anterior interventricular branch	102	6.3	48.4	38.4	6.9	41.4±0.6	42.2±0.6	38.2±1.2	40.4±1.8	31.2±1.2	32.4±1.8	35.8±1.4	36.2±1.2	24.2	17.6
circumflex branch	36	9.1	45.5	33.3	12.1	38.2±1.2	40.2±1.6	36.4±1.6	38.2±1.2	28.4±1.6	30.2±1.4	28.6±1.4	30.6±1.6	20.8	17.8

dominantly in the median segments of the anterior interventricular branch of the left CA resulted in dilation and convolution of the distal portion of this vessel. In addition, compensatory remodeling of the left coronary bed with pronounced marginal collateral-anastomotic plexuses were observed (Fig. 2). Predominant location of bridge-induced obstruction in forth (distal) segment was established in circumflex branch of the left CA. It was accompanied by remodeling of the left coronary bed, which had two zones: 1) vascular emptying and 2) compensatory hypervascularization around the anterior interventricular branch of the left CA (Fig. 3).

On the one hand, the revealed alterations of anginal architectonics in the hearts of SCD patients with differently located bridge-induced obstruction of CA reflect the character of coronary blockade of the corresponding vascular bed, and on the other hand, they indicate the development of compensatory of collateral-anastomotic structures. Being the important criteria of diagnostics, these alterations affect the results of intravital and postmortem examinations of the vascular bed in the hearts of patients without the atherosclerotic lesions in CA, but with suspected availability of MB [6,9,11].

Significance of the revealed alterations in anginal architectonics of the hearts of SCD patients is confirmed by vascularization indices of all subdivisions of the left ventricle (Table 3). In addition to the presence of MB in all segments of CA, a certain decrease in vascular volume density was established in the anterior, posterior, and lateral walls of the left ventricle and in the septum and apex of the heart.

The adequacy of coronary blood flow downstream to MB-induced obstructive area was assessed with myocardial blood supply index (g/mm²). This integral parameter (the ratio of net weight of the cardiac muscle to total lumen area of CA) indicated drop in blood supply to the hearts, where MM were detected in all examined CA (Table 3). A more pronounced relative decrease of coronary blood flow were revealed in patients with bridge-induced obstructions in anterior interventricular branch of left CA (24.2 g/mm² to be compared with the control value of 17.6 g/mm²). These changes indicate pathological level of coronary hemodynamics and they belong to the main factors of thanatogenesis in SCD patients with MB [2,15].

Thus, in dependence on segmentary directionality of bridge-induced obstruction, the characteristic alterations of the vascular architectonics in the heart reflect the degree of coronary deficiency and the level of compensatory manifestations of collateral blood flow. When assessed by myocardial blood supply index, the degree of coronary deficiency during bridge-induced obstructions attests to important role of MB in the

pathogenesis of SCD. In addition, pronounced variability of myocardial blood supply index indicates the compensatory changes of coronary hemodynamics and reveals the leading factors of thanatogenesis in MB-induced SCD.

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