

# Pathomorphology of Atherosclerotic Coronary Artery Aneurysms and Heart Architectonics

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We evaluated pathognomonic incidence of atherosclerotic aneurysms in the anterior interventricular branch of the left coronary artery and proved greatest vulnerability of its first proximal segments. Positive correlations between the incidence of aneurysms in major coronary arteries and their size and bag-like shape were revealed. The characteristic aneurysm-dependent alterations of cardiac angioarchitectonics were found, which underlie pronounced shifts in coronary hemodynamics. Certain criteria for coronarographic diagnostics of atherosclerotic aneurysms were proposed with the methods of their correction. A thanatologic relation of coronary artery aneurysms to the nature of fatal complications is substantiated.

**Key Words:** *atherosclerosis; coronary artery aneurysms; pathomorphology; coronarography*

Acquired aneurysms of coronary arteries (CA) are considered as an infrequent pathology, where the progress in diagnostic is impressive. It became possible due to wide application of selective coronarography into cardiological practice. Among the acquired CA aneurysms, the leading role (54%) is played by atherosclerotic aneurysms (AA). The contribution of other CA aneurysms (congenital, syphilitic, mycotic, and traumatic) is very low.

The mechanism of the development of AA in CA is not clear. The leading role in this process is played by pronounced atherosclerotic alterations developing under conditions of isolated insufficiency of elastic fibers and hypoplasia of muscle elements in the vascular wall, which lead to the formation of aneurysm under the effect of hemodynamic factors. Of certain importance is local mucoid degeneration of the connective tissue in CA wall. In its turn, CA aneurysms disturb cardiac angioarchitectonics and dramatically change coronary hemodynamics. As a result, they ra-

dically aggravate prognosis of basic diseases promoting the development of severe and frequently fatal complications. Growing incidence and insufficient study of CA aneurysms pathology, improvement of the methods of intravital diagnostics, and possibility of wide surgical correction explain actuality of the considered problem.

Our aim was to examine incidence, localization, occurrence, and coronarographic exposure AA in CA, to establish the criteria of pathomorphological diagnostics of this disease, and to assess the effect of this pathology on coronary hemodynamics and thanatogenesis.

## MATERIALS AND METHODS

We carried out postmortem examination of 1000 atherosclerotic hearts. In 15 cases (1.5%, men aging  $54.4 \pm 0.2$  year) coronarography revealed AA in CA. The reference group comprised the other 985 patients died with atherosclerotic hearts and pronounced obstructive atherosclerotic lesions in CA. The control group ( $n=100$ ) comprised persons of the comparable age, which died accidentally and whose pathomorphological examination revealed no atherosclerotic alterations and aneurysms in CA.

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To assess the state of CA, in all experimental and control cases we employed originally modified method of polypositional postmortem contrast coronarography alone or in combination with the routine WHO anatomic method. Reconstructed coronarograms of opened and flattened hearts were used to study three major branches of CA (the right, anterior interventricular [IVB], and circumflex branches). Each branch was subdivided into four segments with equal area of intimal surface preliminary determined by the routine WHO anatomic method. Filling of the coronary system was performed under age-dependent individually calculated pressure.

In parallel to postmortem study of CA, the aneurysms were visualized by detailed macroscopic preparation. The macroscopic study of CA determined maximum diameter, volume, and the state of their content. Then equal half-aneurysms with proximal and distal parts of the segment of CA were examined histologically. The specimens of CA aneurysms were fixed in 10% neutral formalin and embedded into paraffin. The sections were stained with hematoxylin and eosin, picrofuchsin according to Van Gieson, picrofuchsin-fuchselin according to Weigert to reveal the elastic fibers. To determine the degree of coronarographic identification of AA in CA and the character of fatal complications, the clinical, coronarographic, and pathomorphological data were examined in parallel.

## RESULTS

Most frequently, AA were seen in the anterior IVB of CA (Table 1). AA in the circumflex branch of the same CA ranked second. The least number of aneurysms was documented in the right CA. Topographically, the most indicative parts of the examined CA were segments I and II. In proximal segment I, the incidence of AA was equal in all basic CA (one case in each). In segment II, pronounced lesions were observed in the anterior IVB of left CA. The next in incidence was the circumflex branch of this CA. The

minimum number of aneurysms was characteristic of the right CA.

Distal segment III was affected by aneurysmal process only in the anterior IVB of the left CA. In this case, a positive correlation between incidence of AA in basic CA and their maximum diameter was established (Table 1). In addition, it was shown that the most aneurysms in CA were bag-shaped. The maximum number of such alterations were observed in the anterior IVB of the left CA. The aneurysms of cylindrical shape were observed rarely and only in the anterior IVB and in the circumflex branch of the left CA.

Analysis of the mean incidence of A in three major CA showed that AA were in 0.5% patients died with atherosclerotic heart. In these cases, the aneurysmal process took place predominantly in segment II of CA followed in decreasing order by segments I and III. Analysis of the mean maximum diameter of aneurysms showed that that in most cases AA were bag-shaped, while cylindrical aneurysm was observed in only one case. It should be noted that 15 hearts had 16 aneurysms in CA, because one patient had two aneurysms in segment II of anterior IVB of the left CA.

Taking into consideration the fact that each atherosclerotic aneurysm is a local dilation of basic CA and comparing this fact with the data on their localization, size, and anatomic shape, it is important to differentiate them on the basis of coronarographic and pathomorphological data. The revealed aneurysms differed from post-stenotic and trivial dilations of CA and also from their isolated ecstasies, megadolicho arteries, and antioclusion extensions [2]. From differential viewpoint, the state of distal part of CA outgoing from the aneurysm should be taken into consideration together with the character and the degree of intimal lesion.

Each local AA in CA was accompanied by marked alterations in cardiac angioarchitectonics manifested in decompensatory symptoms and compensatory-adaptive shifts. The anterior IVB of the left CA demonstrated peculiar alterations in angioarchitectonics of the left coronary basin, which reflected

**TABLE 1.** Incidence of AA in Segments of Three Major CA ( $n=1000$ )

CA	Number of AA	Segments				Diameter of AA (cm)	Anatomic shape	
		I	II	III	IV		bag-shaped	cylindrical
Right	2 (0.2)	1 (50.0)	1 (50.0)	—	—	1.0±0.2	2 (100.0)	—
Anterior IVB of the left CA	9 (0.9)	1 (11.1)	5 (55.6)	3 (33.3)	—	1.4±0.4	8 (88.9)	1 (11.1)
Circumflex branch of the left CA	4 (0.4)	1 (25.0)	3 (75.0)	—	—	1.2±0.2	2 (50.0)	2 (50.0)
Mean indices of three CA	5 (0.5)	1 (20.0)	3 (60.0)	1 (20.0)	—	1.2±0.3	4 (80.0)	1 (20.0)

**Note.** Brackets mean percentage of cases.

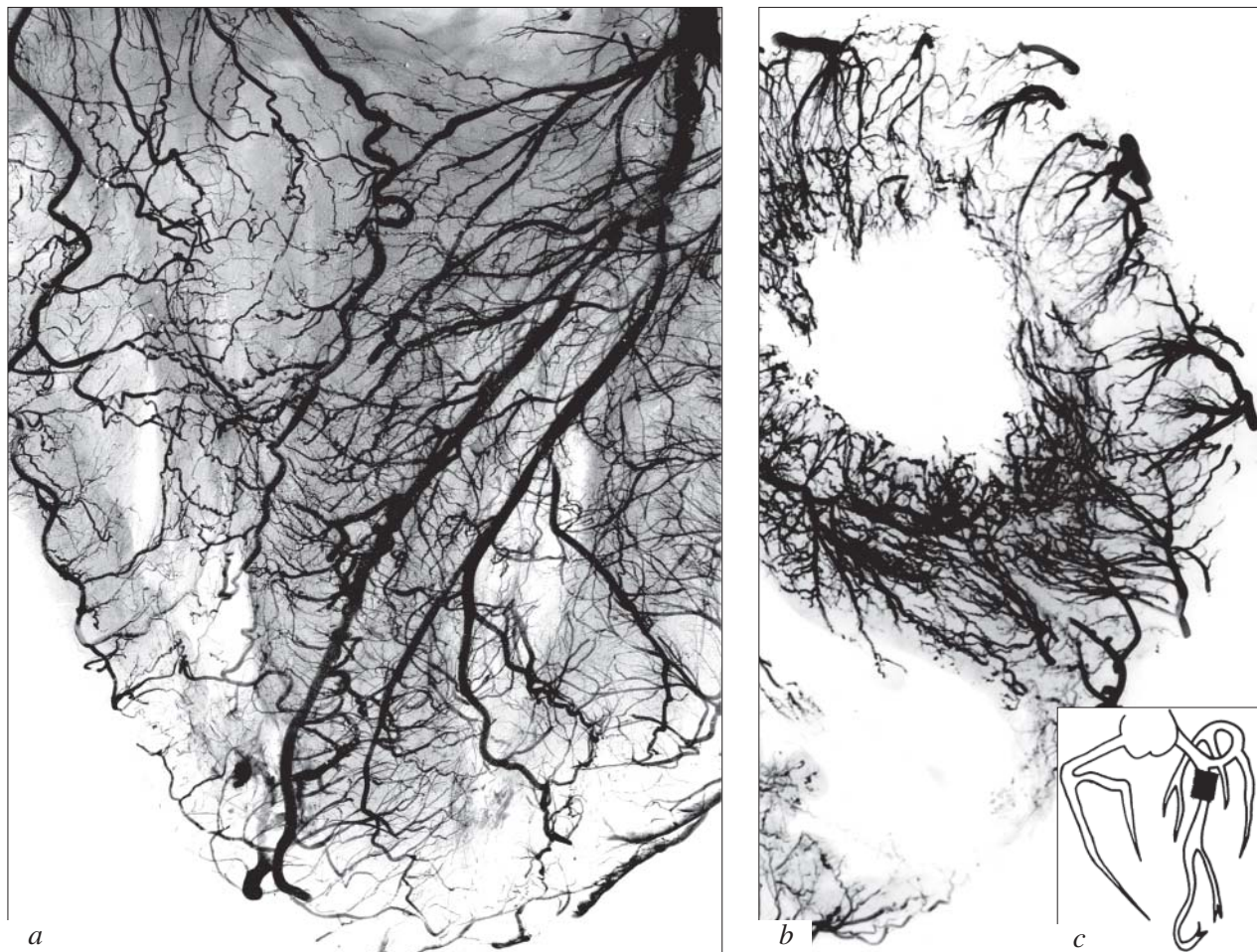
irregular dilation of basic and outgoing primary branches of CA. Some coronary branches had “pathological” involutions transforming to angularity. These alterations of the vascular bed in atherosclerotic heart reflecting the tendency to decompensation were accompanied by compensatory and adaptive heterogeneous hypervascularization of the myocardium. The coronarograms in transversal sections cut across the aneurysmal structures clearly showed isolated contours with apparent manifestation of collateral junctions that formed complicated intramural plexuses (Fig. 1).

Similar manifestations of cardiac vascular network were also observed in cases with AA in the circumflex branch of the left CA. The aneurysms located in the right CA led to pronounced rearrangement of the right coronary basin with the development of the pathways for alternative blood flow. These decompensatory changes were observed against the background of compensatory-adaptive monomorphic hypervascularization of the myocardium. The targeted

coronarograms of the transversal sections demonstrated clear contours of aneurysms accompanied by uniform compensatory-adaptive microvascular reaction (Fig. 2).

The revealed alterations in cardiac angioarchitectonics indicate that during the development of marked obstruction in aneurysm-dependent CA, the peculiar dynamic pressure gradient is created between CA and relatively intact vessels. This gradient becomes the basic driving force of blood flow in the affected coronary collector. The maintaining factors of this pressure gradient are collateral connections in pre- and post-aneurysmal segments of CA. The significantly elevated pressure in the pre-aneurysmal segment increases the volume of circulating blood in the adjacent collaterals and the tangential tension (shear stress) of the vascular wall. Analysis of the disturbances in coronary hemodynamics should take into consideration the actual hydrodynamic factors.

Postmortem coronarographic diagnosis of AA is not absolutely reliable. It is not adequately corroborated



**Fig. 1.** Cylindrical aneurysm in the first segment of the anterior interventricular branch of the left coronary artery with decompensatory rearrangement of the left coronary basin and the compensatory-adaptive heterogenic hypervascularization, which is more pronounced in coronarogram of targeted transversal cardiac section. Shown are the fragments of flatten (a) and targeted transversal (b) cardiac section of patient D. (54 years). The scheme is shown in (c).

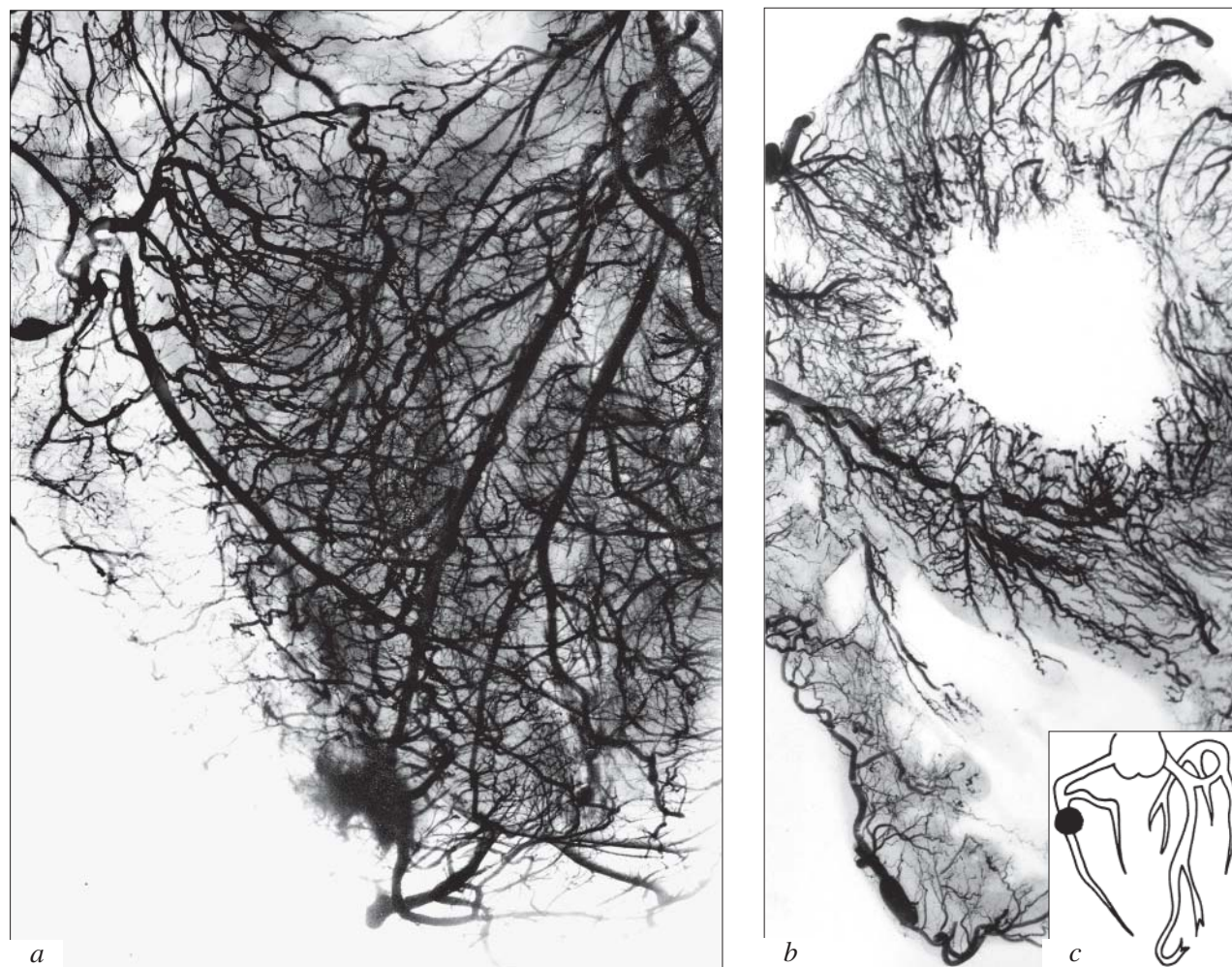


ted by routine anatomic method (Table 2). However, all cases of aneurysms in the right CA were revealed by both methods. In 3 cases, coronarographic diagnosis of aneurysm in the anterior IVB of the left CA was erroneous. Similar mismatch was documented in 2 cases of aneurysms in the circumflex branch of the same CA. In total, in 5 of 15 AA in the major CA, the coronarographic method did not yield reliable diagnostic data in contrast to the following routine anatomic inspection. It is interesting, that in 3 of these 5 cases of diagnostic mismatch, the aneurysms of CA were cylindrical. These data show that the study of coronarogram of flattened hearts, filling of coronary system under individually calculated pressure, and correct projection can decrease the gap within postmortem diagnostic data.

Among the developing fatal complications, the leading role is played by thrombosis of the aneurysms accompanied by obstruction of the affected CA and thromboembolism of its distal segments. The next in

incidence was myocardial infarction and cardiac tamponade. Myocardial infarction developed in cases of complete thrombosis of aneurysm and consequential obstruction of large coronary branch leading to pronounced blockade of coronary basin followed by the development of discirculatory necrosis of the myocardium. The break of AA into pericardium cavity resulted in cardiac tamponade and, as a rule, sudden death. Cardiac insufficiency developed due to marked ischemic myocardial dysfunction was rarely documented as the fatal complication. These data shed light on the peculiarities of thanatogenesis during AA in CA.

Therefore, AA in CA were responsible for a certain part of deaths in patients with atherosclerotic heart. They were characterized by a peculiar pathomorphology, which determines the basic diagnostic criteria. In most cases, AA were revealed in the anterior IVB of the left CA with predominant localization in the first (proximal) segments. The incidence of AA



**Fig. 2.** Bag-shaped aneurysm in the segment II of the right coronary artery with decompensatory rearrangement of the right coronary basin and uniform compensatory-adaptive reaction in small vessels revealed on coronarogram of targeted transversal section of the heart. The fragments of coronarogram of flattened (a) and targeted transversal (b) cardiac section of patient N. (56 years). The scheme is shown in (c).

**TABLE 2.** Identification of AA in CA by Coronarography and Character of Fatal Complications

CA	Number of AA	Coronarographic detection				Fatal complications			
		detected		not detected		thrombosis of aneurysm	myocardial infarction	cardiac tamponade	cardiac insufficiency
		abc.	%	abc.	%				
Right	2	2	100	—	—	—	—	1	1
Anterior IVB of the left CA	9	6	66.7	3	33.3	3	2	3	1
Circumflex branch of the left CA	4	2	50	2	50	2	2	—	—
Total	15	10	66.7	5	33.3	5	4	4	2

in the major CA positively correlated with their size and bag-like appearance. These data form the basis of pathomorphological diagnostics, which makes it possible to differentiate AA from aneurysm-like manifestations in CA.

AA in CA induce pronounced alterations in cardiac angioarchitectonics, leading to marked shifts in coronary hemodynamics characterized by prevalence of decompensatory mechanisms. At the same time, postmortem coronarographic diagnostics of aneurysms is not reliable and necessitates obligatory correction with coronarography of the hearts flatten in a special way under individually calculated pressure in strictly chosen projection. Thrombosis of AA with thromboembolism of distal segments of the affected CA results

in their partial or complete obstruction triggering the development of the most frequent complications, including myocardial infarction included. The break of aneurysm into pericardial cavity leads to cardiac tamponade terminated with sudden death. In addition, the development of progressing cardiac insufficiency is possible because of pronounced ischemic dysfunction caused by decompensation of coronary circulation.

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