COMPARATIVE STUDIES OF THE LIPIDS AND ACID MUCOPOLYSACCHARIDES IN THE WALL OF AORTA AND PULMONARY TRUNK IN EXPERIMENTAL ATHEROSCLEROSIS IN RABBITS

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The interactions between the lipids (L) and acid mucopolysaccharides (AM) in the arterial wall are of certain interest in the progress of the atherosclerotic process. The purpose of our present work is to study experimentally these interactions in two arterial vessels of conducting type — aorta (A) and pulmonary trunk (PT).

Material and methods

The experiments were carried out on mature male rabbits divided into two groups: I^{st} — 7 animals receiving to the food 200 mg/kg cholesterol daily and II^{nd} — 7 controls. The study covered the ascendent part of A and PT both. L were presented by using the staining with Sudan-schwarz and oil-red, and AM — with alcyan-blue and toluidin-blue at pH 5,0. The structural changes of the vascular wall were examined on preparations stained by haematoxilin-eosin, orcein and after van Gieson.

Results and discussion

There are certain structural alterations of the wall of both A and PT presented mainly by proliferation of the connective-tissue elements in the subendothelial intimal layer which causes its considerable enlargement. In the thickened subendothelial layer a large amount of elastic fibers, longitudinally orientated smoothmuscle cells and xanthomic cells forming fibrous plaques can be observed. Because of destructive alterations their elastic component diminishes strongly. The internal elastic membrane is destroyed to fragments, splits up and forms several single lamellae moved apart from each other (fig. 1). The elastic structure of the media is also altered. The elastic lamellae under the plaque become thinner and slighter stained by orcein. In PT wall the splitting and fragmentation of the inner elastic membrane is slighter expressed, the plaques are richer in elastic fibers and more deficient in xanthomic cells.

The total AM amount in the wall of both A and PT is increased in the experimental animals as compared with that of the controls. In experimental animals there is a strongly positive reaction in fibrous plaques within the thickened intima and in the underlying $^1/_3$ of the media which diminishes gradually towards the adventitia while in control ones a reaction like this is established on the borderline between the intima and media only. There is an exception in case of abundantly sedimentation of L and destruction signs when the intensity of the reac-

tions for AM decreases considerably in the plaque and underlying part of the media. In PT wall this intensity is less expressed (fig. 2 a, b).

The wall of both A and PT is affected by the process of L infiltration in any experimental animals which is more expressed in the wall of A. A granular

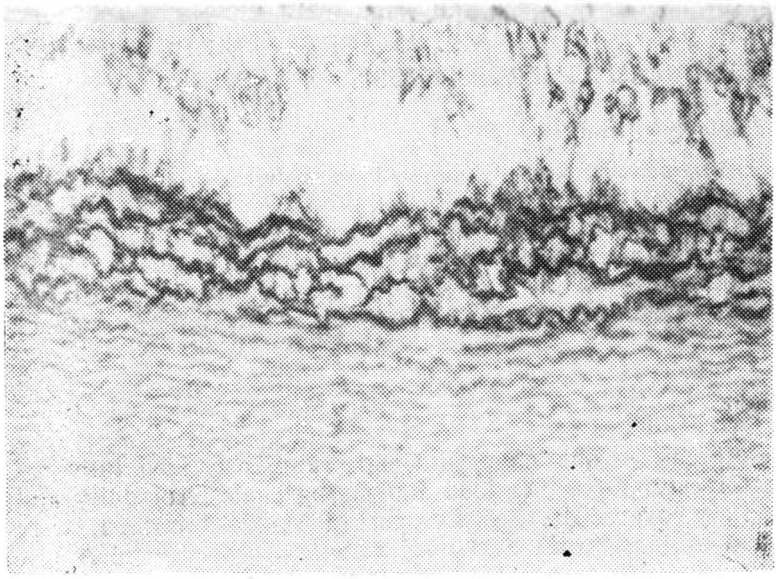

Fig. 1

L infiltration of the thickened and structurally altered int ma is found out. Certain plaques with various size can be observed. Their L sedimentations are grosser and agglomerate-like. On the border between the intima and media the strongest intensity of staining with oil-red and Sudan-schwarz is established. A continuous layer covering almost the whole lumen is formed here. It is noteworthy that this layer exists also under relatively slightly thickened intima on the grounds of which we can accept that the primary L sedimentation is exactly in the cited internal elastic membrane. A finer L infiltration reaching sometimes the outer $^1/_3$ of A and the middle $^1/_3$ of PT can be seen between the elastic lamellae of the inner part of the media. In any cases L sedimentations are more diffuse in the wall of PT. The well-formed L layer between the intima and media is absent (fig. 3 a, b).

Our investigations show that there is definite correlation between the structural changes, AM distribution and the location of L sedimentation in the wall of both A and PT in experimental atherosclerosis. The structural alterations, especially of the elastic skeleton, are less expressed as regards the PT wall in comparison with those concerning the A one. It is known from the literature that the atherosclerotic changes of the A wall are accompanied by disappearing of the elastic fibers. The slighter alteration of the elastic skeleton of the PT wall could be explained by the smaller intramural compression resulting from the lower blood pressure in this vessel. The biggest amount of AM is in the thickened intima and underlying layers of the media. In view of the fact that the aforemen-

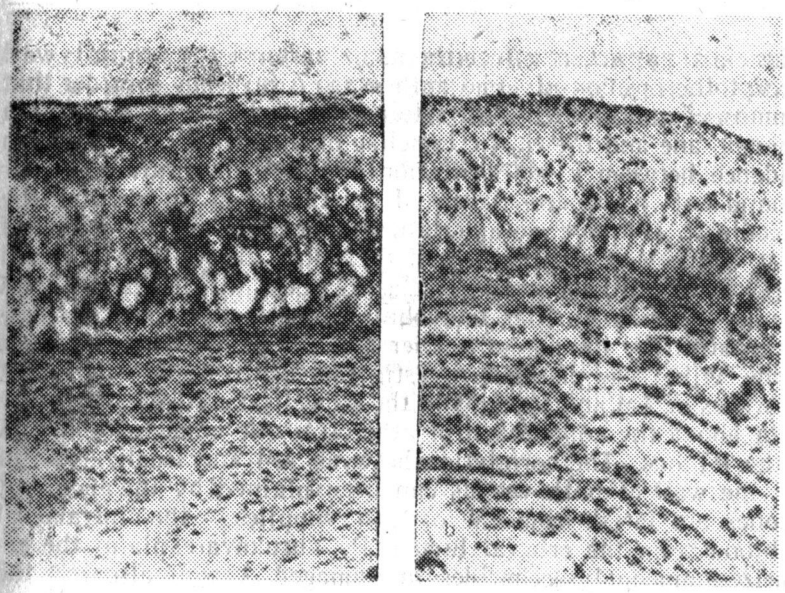

Fig. 2a, b

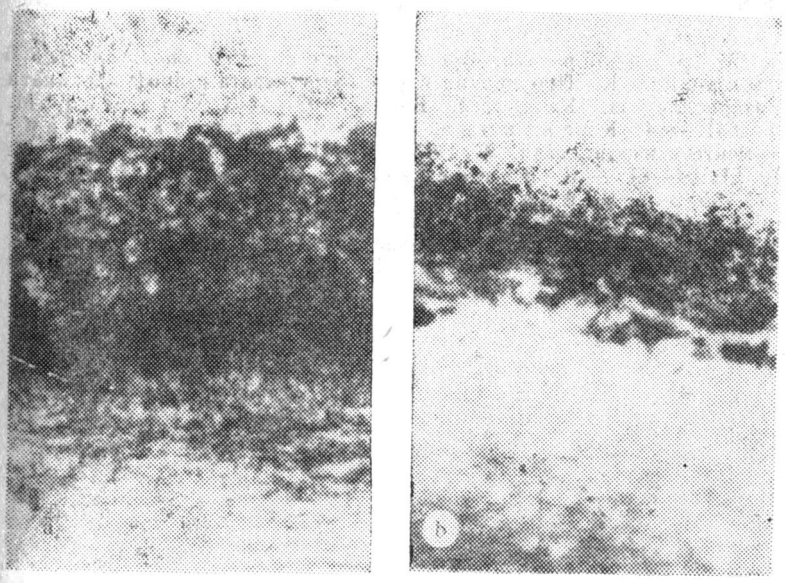

Fig. 3a, b

tioned changes of the elastic structure effect the same parts of the arterial wall we would like to accept the opinion of some authors (6, 7, 8). They consider that the accumulation of mucopolysaccharides in the arterial wall is due to previous lesion of the inner elastic membrane and the elastic lamellae of the media. We establish a definite correspondence between L sedimentation and AM distribution. Their biggest amount is found out in the deep layers of the thickened intima and the inner parts of the media, i. e. where we ascertain the most severe alterations of the elastic component of the wall of both A and PT. Most likely, the increased amount of AM is due to the disappearance of elastic fibers which liberate AM during their transformation from fibrillar to globular organization. The increased L level can be explained by the absent barrier function of elastic membranes.

Our data coincide with those of other investigators (2, 3, 4, 9) who report certain similar regularities of AM and L levels in the A wall. However, there are no bibliographic data about the comparison of atherosclerotic changes of the A wall with those of the PT one. The correlation between AM and localization of L sedimentations in the wall of both vessels can be explained by the fact that their predilection zones are farest from the oxygen supplies — the intramural capillaries and the blood from the arterial lumen. P. Yurukova explains the accumulation of sulfurized mucopolysaccharides in damaged A wall also by the increased oxygen requirements.

Our comparative studies show that the process of L infiltration affects more severely the A wall than the PT one in experimental atherosclerosis in rabbits. In this respect our data support our previous results (1) concerning normal conditions in man. They can be explained by the more abundant vascularization of the PT wall and the poorer possibilities for transmural infiltration because of the lower blood pressure in PT than that in the aorta.

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СОПОСТАВИТЕЛЬНЫЕ ИССЛЕДОВАНИЯ ЛИПИДОВ И КИСЛЫХ МУКОПОЛИСАХАРИДОВ В СТЕНКАХ АОРТЫ И ЛЕГОЧНОГО СТВОЛА КРОЛИКОВ ПРИ ЭКСПЕРИМЕНТАЛЬНОМ АТЕРОСКЛЕРОЗЕ

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РЕЗЮМЕ

В работе описывается некоторые структурные изменения и распределение липидов и кислых мукополисахаридов в стенке аорты. Проводится их сопоставление с легочным стволом в условиях экспериментального атеросклероза у кроликов. В утолщенной и со структурными изменениями интиме внутренней части медии стенки обоих сосудов устанавливается липидная инфильтрация, находящаяся в коррелятивной связи с количеством и распределением кислых мукополизахаридов. Структурные изменения и накопления липидов наблюдаются в большей степени в стенке аорты по сравнению с теми же изменениями стенки легочного ствола.