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The Development of the Structure of Atherosclerosis and Aspects of Coronary Heart Disease

Erich Ebner

Hoherweg 2, 99425 Weimar, Germany

ABSTRACT

The genesis of arteriosclerosis and, as a consequence, coronary heart disease is put up for discussion. On the basis of morphological considerations, it appears to be absurd to define the cause of arteriosclerosis exclusively on the molecular level. Cholesterol is a functionally important building block in the body. It is regulated by the body. Long-term observations of statin therapy indicate consequential damage, which makes it necessary to rethink this form of therapy. The cause of arteriosclerosis is the result of a chronic ischemic disorder of the arterial vessel wall.

***Corresponding author**

Erich Ebner, Hoherweg 2, 99425 Weimar, Germany, E-mail: erich.ebner@gmx.de

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Introduction

The centuries-old observations of arteriosclerosis and its consequence have always challenged science. The complexity of the scientific analyzes have not made it easy to classify the clinical picture of arteriosclerosis. Numerous causal association hypothesis were developed to open up therapeutic possibilities. The anatomical-functional view and molecular view are in competition. From the point of view of the ischemic disorder of the arterial vessel wall, the lipid hypothesis should be put up for discussion.

Discussion

The literature on atherosclerosis is extremely extensive and focuses on mechanisms of development and pathogenesis, including biochemical metabolic pathways, of lipid metabolism. The worldwide assignment of the disease and its consequence makes this necessary. But it must be noted that arteriosclerosis is not a modern disease, because since ancient times there have been representations that point to this disease process. In 1552 Fallopius was possibly the first to describe changes in the arterial vessels that could correspond to arteriosclerosis. Further data-appropriate descriptions followed by Harvey, von Haller, Morgagni, Scarpa and numerous other scientists of the modern age. In 1743, for example, Morgagni recorded an exact macroscopic description of coronary sclerosis. The connection of the anatomical findings to the symptoms of angina pectoris was only a consequence of time and was defined by Heberden in 1769 in a day before the Royal College of Physians, just as Burns recognized angina pectoris as ischemic pain in 1809.

The importance of the disease in terms of health policy means that we need to rethink its cause and therapeutic options.

What is arteriosclerosis from the point of view of pathological anatomy? Like every organ, the arterial vessels initially have two functions: the transport of oxygen-enriched blood to the periphery, quasi as a work function, and a nutritive function, supplying the vessel wall via the capillary system of the adventitia. A disruption of the capillary blood flow to the vascular wall must consequently lead to metabolic disorders, tissue defects or necrosis. It is conceivable that intermittent pressure increases, flow reductions at vascular outlets and bends, vascular constrictions due to stress or circulatory disorders of microcirculation lead to these changes. The time factor, the duration of the disruptive mechanisms, plays a not insignificant role. These chronically progressive disorders lead to local ischemic inflammation and necrosis with the resistance to calcification. The disseminated occurrence of this damage to the arterial vessel walls in different stages of development underpins such an interpretation.

The chronic local ischemia, triggered by insufficient capillary blood flow, leads to nutritional disorders of the entire vascular wall and thus to inflammatory injuries to the vascular wall parts, besides tissue defects also to varying degrees to necrosis. But what does this mean? The violation of the normal anatomical tissue structure is associated with a breakdown of the tissue. Depending on the extent and duration, different phases of degeneration are found. Connective tissue proliferation, collagen fiber formation and cellular defense cells of the blood, intra- and extracellular deposits of metabolic products and intermediate products of the metabolism of the damaged cells, including cholesterol and its breakdown products, fats, minerals, carbonates, calcium phosphates, proteoglycans from the extracellular matrix tend to form colliquitation necrosis when the pH value changes, and more. All these processes lead to an expansion of the disturbances to all vascular layers up to the intimate and trigger the known deposits there after the destruction of the intima layer, which ultimately lead to thromboembolic complications. Vascular changes, loss of elasticity, expansion of the diffusion disorders in the area around the plaques and finally the reduction of the overall reduction of blood flow to oxygen deficiency cause the increasing remodeling of the arterial vessels.

The pathological-anatomical considerations were the focus of research in the first half of the 20th century, as Rotter [2] and Lizbachh [1] defined arteriosclerotic plaques as ischemic necrosis. Since the 1930s, however, the outstanding diverse discoveries in biochemistry on the part of the American scientific view have moved away from the anatomical-physiological perspective towards molecular-structural perspectives.

How did the cholesterol hypothesis come about as the cause of arteriosclerosis? The discovery of biosynthesis is associated with the names of Bloch, Lynen and Comforth. This elucidation of the metabolic pathways is important and continues to be the subject of intensive research. But Key's working group, as a nutritionist, believed that cholesterol should be stylized as the basis of a hypothesis in order to create a basis for pharmacological therapy, statin therapy. That was successful, but the studies are blamed for an unmistakable marketing intention. Comparative considerations of studies doubt the seriousness of the interpretations.

The question arises as to how the substances cholesterol and fat are to be classified, and what functional significance they have as the end product of fat metabolism. From a biochemical point of view, cholesterol is a polycyclic alcohol and is functionally responsible in the organism for the stabilization of cell walls and other structures, such as proteins and signal substances for nerve function. The human body's need for cholesterol is 90% produced in the body's own metabolism and only 10% is taken from food. Genetic factors play a role in this. The body's own regulation of cholesterol metabolism [3], largely a negative feedback, is controlled with regard to the intake from food via a protein complex consisting of three proteins that is synthesized in the liver. The cholesterol level therefore largely corresponds to its own regulation and is not dependent on food intake. Fat, on the other hand, is esterified glycerin and functionally an energy store.

In comparison to the cholesterol studies, one should more or less ignore the fact that cholesterol is a basic building block for the organism, so that the following points must be emphasized:

- Cholesterol is vital
- The synthesis metabolism is largely independent regulation,
- A correlation of the cholesterol level with age and gender is doubtful, but individual fluctuations are likely
- Nutritional studies do not lead to a plausible explanation,
- It can be said that statin therapy does not bring any benefit.

The reduction of cholesterol by statin application can provoke significant fatal effects. Wound healing strengthening in general and in particular in dentistry as well as in the neurological field. The influence can lead to disorders such as in Alzheimer's disease or dementia or intensify their development because of the biochemical the central LDL cholesterol level correlates with impaired memory and other cognitive functions[3,4,7], so that a significant loss of memory and attention develops [5,6]. Because cholesterol plays a significant role in the function of the synapses in the brain and its deficiency has fatal consequences. The biochemical reactions in the synaptic area are diverse and in some cases not fully understood. But the reduction in cholesterol has been shown to have negative consequences. The cause of total memory loss due to statin administration has been documented. Even taking cholesterol-lowering drugs early, such as during pregnancy, has a negative influence on the development of the IQ quotient.

If one looks at the scientific activities relating to coronary heart disease in the 20th century, one finds an idea of the ischemic cause that is generally aligned with arteriosclerosis, as shown by the views of Rotter [2] and Linz Bach [1] already mentioned. But also earlier considerations by Warburrg, Schürmann, Hüber and Schoberts saw the arteriosclerosis of coronary heart disease in the same sense as an expression of the local hypoxia of the vascular walls as a result of a chronic hypoxic inflammation through negative impairment of the nutritive function of the adventitia, which progressively the substrate of the inner layers, the muscular and intimate, destroyed. Long-term external disturbances such as high blood pressure can lead to insufficient flow and thus successively cause metabolic changes. From today's point of view, Linzbach's views on microcirculation disruption are associated with a lack of oxygen in the tissue, disruption of the acid-base balance and thus with the disruption of metabolic processes. Also, Rotter saw the disruption of the permeability of the cells as the starting point for the development of arteriosclerosis. With reference to Mönkeberg at the beginning of the 20th century and Burger in the 30s of the 20th century, the idea of the disturbance of the permeability indicating the formation of edema, swelling of the cells and fibrin formation was an expression of the mesenchymal reaction with effects on endothelial and sub endothelial structures. These productive proliferative processes within the vessel walls were compared to bradythrophic tissues.

The advances in the diagnosis and therapy of arteriosclerosis and including coronary heart disease are based on effective apparatus diagnostics and the therapeutic options. Education and nutritional advice as well as reducing the lack of exercise and thus overall a more conscious lifestyle are crucial factors. Cholesterol as a vital component of the organism has nothing to do with arteriosclerosis in a negative sense.

Conclusion

The morphological changes in arteriosclerosis and the associated coronary artery disease do not speak in favor of a purely molecular cause within the metabolic pathway of cholesterol. On the other hand, one can assume a chronic ischemic process in the development of the arterial vessel wall changes, which in the sense of ischemic necrosis explains the often diffuse and locally differently developed findings. Cholesterol, which according to one hypothesis is held responsible for arteriosclerosis, has to be reassessed. As an important building block in the organism, cholesterol is indispensable and a pharmacological influence has to be reconsidered, as long-term effects of a particularly neurological nature become apparent.

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