Editorial

Mechanism of Lipid Deposition on Arterial Wall

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Since the success of the experimental formation of atherosclerosis by cholesterol administration (Anitschkow), the role of lipids in the pathogenesis of arteriosclerosis was considered as an important one. However, more recently lipids are recognized not to play a primary role but to play a rather secondary role as a modifier in the formation of arteriosclerosis. For the study of arteriosclerosis it is important to investigate the mechanism of lipid deposition, since almost all sclerotic regions contain such deposition.

Penetration of plasma contents into the arterial wall was observed recently by radio-isotope experiments. Authors reported that ß-lipoprotein and fibrinogen can penetrate into the arterial wall by in vitro study. Antisera and fluorescent antibodies of both substances are used to prove the presence of ß-lipoprotein and fibrin in the arterial wall. Amount of both substances in arterial wall increase proportionally to the grade of sclerosis.

On the other hand, an increase of acid mucopolysaccharides in the arterial wall is demonstrated either histochemically or by the measurement of SO₄ radical. Where there is lipid deposition one can observe almost always metachromatic substances, and not vice versa. Rabbit experiment shows that significant metachromasia surrounds the sponge implanted subcutaneously. Subsequent administration of lanolin causes marked deposition of lipid in the same region, indicating a close relation between acid mucopolysaccharides and lipid for the lipid deposition. When acid mucopolysaccharides are added to the solution of ß-lipoprotein precipitation was reported to occur. Acid mucopolysaccharides obtained from human aorta also cause same white precipitation in this system.

Similar reaction may happen in the human body as was observed in vitro study. Interestingly enough fibrinogen also reacts with acid mucopolysaccharides, producing white precipitation.

It is not yet concluded that lipid does deposit as ß-lipoprotein or not. Administration of non-ionic surface active agent such as triton or tween produce lipemia, however, in this situation only moderate plaque can be produced. Administration of surface active agent in the lanolin-fed rabbit followed by the additional administration of I¹³¹ labelled triolein causes only slight uptake of I¹³¹ into the aorta when compared to the group without surface active agent.

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agent administration. Also addition of detergents to serum decreases the production of white precipitation with acid mucopolysaccharides, however precipitin titer for anti-β-lipoprotein serum remains unchanged. These are considered to be due to the change in lipid fraction of β-lipoprotein by surface active agent not accompanied by the change in protein fraction. These observations would lead to the conclusion that lipids are more easily deposited as β-lipoprotein into the arterial wall.

Presence of fibrin in arterial wall was described above. Like β-lipoprotein, fibrinogen reacts with acid mucopolysaccharides and produces white precipitation, suggesting the similar mechanism of deposition between β-lipoprotein and fibrinogen. Fluorescent antibody study shows the close presence of fibrin and β-lipoprotein in the tissue. Paper-chromatographical study of affinity among lipoprotein, fibrinogen and acid mucopolysaccharides show that the co-existence of these three substances facilitates the deposition of lipid. Therefore, one can conclude that fibrinogen also play another role in the lipid deposition.

In conclusion, eventhough Eucrustation theory is still acceptable for the pathogenesis of lipid in arterial wall, one can add additional important mechanisms such as described in this paper.