Amyloidosis of the Aorta

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A review of the literature would suggest that amyloid deposition into the intima of the aorta is an unusual finding. An opportunity to study this condition in a patient with primary amyloidosis is the basis for this report. The suggestion is made that factors responsible for this may involve mechanisms similar to those that participate in the pathogenesis of atherosclerosis.

It is well over a hundred years since Budd in 1845 first used the expression “waxy liver” to introduce to medicine the condition now known as amyloidosis. Virchow in 1858 called this waxy material amyloid. He did this on the basis of some earlier work by Meckel, who in 1853 had shown that this substance has a selective affinity for iodine. Today amyloid is considered to be a particular glycoprotein that may be identified somewhat empirically with a variety of differential stains. It has been described by pathologists as a hyaline intercellular infiltrate that may occur locally or in every organ of the body. It has also been referred to as a substance that may accumulate in intercellular and connective tissue spaces in association with hyperglobulinemia. Generally speaking, cases fall into 2 groups, primary or secondary amyloidosis, depending on the presence or absence of a recognizable antecedent disease.

Textbooks of pathology, both American and foreign, seem to agree that amyloid may be found in every organ in the body, but nowhere in either the older or more recent reviews is there a description of amyloid infiltration into the intima of the aorta. Amyloidosis of large and small arteries, arterioles, capillaries, and veins is common knowledge, but its presence in the intima of the aorta must be a very unusual finding, or it has been generally overlooked, or its presence has simply been taken for granted.

The sole purpose of writing this brief report is to describe our findings in the aorta of a normotensive male patient, 55 years of age, who recently died of renal failure, the result of generalized primary amyloidosis in which the kidneys were seriously affected. Grossly the aorta was the seat of moderately severe diffuse atherosclerosis in which there was nothing to lead one to suspect the presence of amyloid.

In the course of the autopsy thin sections were taken from all levels, from both atheromatosus and healthy areas, and stained in the fresh state with crystal violet; to our surprise amyloid was found in varying amounts in the intima, in almost all sections, while lesser amounts were seen about the vasa vasorum in the adventitia and outer third of the media.

Grossly there may be nothing to indicate its presence in the aorta unless appropriate stains are used, and this may explain in part why it has received so little attention. In histologic sections, unless it were suspected and a number of differential stains employed, as was done in this case, it could very easily be overlooked or possibly mistaken for fibrin. In the intima it was entirely extracellular, simply lying in the ground substance in varying amounts and in varying locations. In atherosclerotic plaques it was found freely mixed with lipid in the depth of these lesions and also superficially adjacent to the endothelial lining. In areas where there was no demonstrable lipid, amyloid was seen in streaks midway between endothelium and media. In no area was there any suggestion of cellular reaction to the amyloid.

The presence of amyloid in the intima of the aorta is not just a collector’s item, nor does its importance in this area depend on its functional significance, for this is probably negligible. On the contrary, its importance lies in the fact that like lipids and lipoproteins, amyloid,
a glycoprotein, can penetrate the endothelium of the aorta, and like them can accumulate in the ground substance of the intima.

When so much attention is being focused on the etiology of arteriosclerosis, particularly in respect to the relative importance of such factors as diet, the physical characteristics of circulating lipoproteins, cholesterol levels, blood pressures, and endothelial permeability, the progressive accumulation of amyloid in precisely the same areas within the intima of the aorta as that of cholesterol may be of some interest to investigators who are concerned with the mechanisms involved in the pathogenesis of that infinitely more important disease, atherosclerosis.

**Summary**

An instance of amyloid deposition in the intima of the aorta is described. Its possible significance is discussed.

**Summario in Interlingua**

Un caso de deposition de amyloide in le intima del aorta es describite. Su possibile signification es discutite.

**References**

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Circulation. 1957;16:268-269
doi: 10.1161/01.CIR.16.2.268

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/16/2/268

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