Abdominal aortic aneurysms

Abdominal aortic aneurysm is considered to be one manifestation of arteriosclerosis. Several observations support this: microscopic investigations of the aneurysm show arteriosclerotic changes; the patients have risk factors in common with those showing obstructive arteriosclerotic lesions; arteriosclerotic changes in other vessels are seen significantly more often in patients with abdominal aortic aneurysm; systematic surveillance of patients with various types of arteriosclerotic symptoms (such as intermittent claudication or carotid arterial disease) have an increased prevalence of abdominal aortic aneurysm. Further support is given in the paper by Lee et al. where subjects in the Edinburgh Artery Study were carefully analysed. The main results are that atherosclerotic disease is associated with abdominal aortic aneurysm and that smoking is identified as an independent risk factor for the development of abdominal aortic aneurysm.

New information has been published in recent years which will influence our knowledge of the pathogenesis of abdominal aortic aneurysm. There is a clear genetic pattern in the occurrence of abdominal aortic aneurysm: it is significantly more common among brothers and sons of patients with abdominal aortic aneurysm; prevalences of about 30% have been found. Although congenital aneurysms are extremely rare, their development later in life may be genetically determined. Thus, screening should be seriously contemplated in first-degree relatives.

Pathogenetic discussions have been dominated by various aspects of the atherosclerotic process, possibly in combination with the relative lack of nutritional vasa vasorum in the abdominal aorta, which may give rise to ischaemic aortic wall alterations, atrophic changes and widening. This process, in combination with haemodynamic stress, especially in the elderly with elongated aorta, can lead to the development of an abdominal aortic aneurysm. Much new evidence, however, indicates that biochemical events within the aortic wall are factors of primary importance for aneurysmal development and also for rupture. To summarize, there seems to be a decreased amount (and/or quality) of elastin which causes the aneurysm to expand and increased collagenolysis which gives rise to rupture. There are also experimental data supporting these clinical observations.

Kuivaniemi et al. focusing their research on a genetic analysis of collagen, have found clear evidence for an independent genetic defect in a majority of abdominal aortic aneurysms. An extreme genetic abnormality of collagen with aneurysmal development and arterial rupture and significantly reduced survival is seen in patients with type IV Ehlers-Danlos syndrome. Further genetic studies have suggested a recessive inheritance pattern.

One risk factor in the study by Lee et al. which may be common to the development of atherosclerosis and abdominal aortic aneurysm is smoking. In the present study smoking was independent of the degree of arteriosclerosis. We also found smoking to be an important risk factor for the development of abdominal aortic aneurysm in a population-based study. Moreover, we observed an association between abdominal aortic aneurysm and chronic obstructive lung disease. One possible explanation is
that smoking stimulates enzymatic degradation of the elastin matrix of connective tissue in patients with a genetic predisposition, and this degenerative process may occur simultaneously in the abdominal aorta and the lungs.

At present, abdominal aortic aneurysm is a more enigmatic disease than when it was simply considered an arteriosclerotic manifestation. The combination of epidemiological, genetic and biochemical research will continue to increase our knowledge. Abdominal aortic aneurysm could be one common manifestation of more than one aetiology.

In some patients the development of arteriosclerosis destroys the nutritional circulation of the aortic wall, giving rise to dilatation and an abdominal aortic aneurysm. The aneurysmal wall is then part of the arteriosclerotic process as indicated in Fig. 1.

In perhaps the majority of patients, however, there is another scenario: a genetic abnormality in the aortic wall structure, which at a certain age, gives rise to dilatation. Parallel to this process, arteriosclerotic risk factors and especially smoking induce generalized arteriosclerosis and development of an abdominal aortic aneurysm in the dilated aorta. The aneurysm is a locus minoris resistentiae for further arteriosclerotic progress and the end stage is an arteriosclerotic abdominal aortic aneurysm (Fig. 2).

Although much information is still lacking on the biology of abdominal aortic aneurysm, it can be concluded that the disease process is complex and that it is not just one of several arteriosclerotic manifestations.

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References

Intravenous digoxin: still the drug of choice for acute termination of atrial fibrillation?

See page 643 for the article to which this Editorial refers

Introduced by Withering, digitalis glycosides were first used in atrial fibrillation by Bouillaud in 1835[1]. Several reports still present digoxin as the drug of choice for acute termination and/or for control of rapid ventricular response in atrial fibrillation[1,2]. Digoxin has a direct effect on the conduction system of the heart and an indirect effect mediated by the autonomic nervous system. Oral digoxin exerts a direct effect on atrioventricular nodal properties (atrioventricular conduction and atrioventricular nodal refractoriness), but intravenous digoxin does not. The predominant effect of digoxin is through the autonomic nervous system, as shown by the absence of effect in the transplanted heart[3].

The study by Falk et al.[4] showed that intravenous digoxin was no better than placebo in restoring sinus rhythm in patients with recent onset atrial fibrillation. The results of Jordansen et al.[5] in this issue are consistent with those of Falk et al.[4]. Conversion to sinus rhythm occurred in nine of 19 patients on digoxin (47%) and eight of 20 patients on placebo (40%). Additional information is provided. The study shows that in non-converters there is an