

Morphological Investigations of Type A Aortic Dissection

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Purpose: The aim of this study was to analyze systematically the morphology of aortal segments of Type A dissection.

Methods: Nineteen patients were operated on for Type A dissection in the Department of Thoracic, Cardiac, and Vascular Surgery in Goettingen, Germany, from January 2002 to January 2005. All diagnoses were confirmed by transesophageal echocardiography and computed tomography of the chest. All taken aortic segments were examined by the conventional histological and electron microscopical method.

Results: Besides subadventitial hyperplasia of collagen filaments, the preparations showed hyperplasia of endothelial cells with loose cellular junctions, desquamation of endothelial cells, and morphological changes of endothelial cells with villus development, as well as signs of aortitis.

Conclusion: The present results arouse suspicion of local inflammation of the aortic wall, but with moderate progress under strong hyperplasia. Because of rupture of the intima, the inflammation appears as an acute disease. (*Ann Thorac Cardiovasc Surg* 2010; 16: 331–334)

Key words: histology, scanning electron microscopy

Introduction

In recent years, the number of aneurysm dissections has been increasing, and both the diagnostic skills and the treatment of this disease have greatly advanced.¹⁾ Many factors have been implicated in the etiology of dissecting aortic aneurysms. Systemic hypertension is present in at least three-quarters of reported cases and must be consid-

ered an augmenting factor, but not an etiologic factor.^{2,3)} Cystic medial necrosis has been considered crucial in the pathogenesis. In Marfan syndrome, medionecrosis and fragmentation of the elastic fibers of the aorta are mentioned commonly.^{2–6)} The most common pathological findings for Type A dissection are atheroma dissection, initial fibrosis of the vasa vasorum, medial atrophy, and arteritis.⁷⁾ The aim of the study was to make a systematic morphological analysis of aortal segments of Type A dissection to gain more information about the etiology of this disease.

Material and Methods

For this study, samples from the dissection membrane of human aortic wall were taken from patients undergoing valve and aorta ascendens replacement operations for acute Type A dissection of aorta. Biopsies were performed on 6 male patients (mean age 75 + 1 years) and 3 females (68 + 3).

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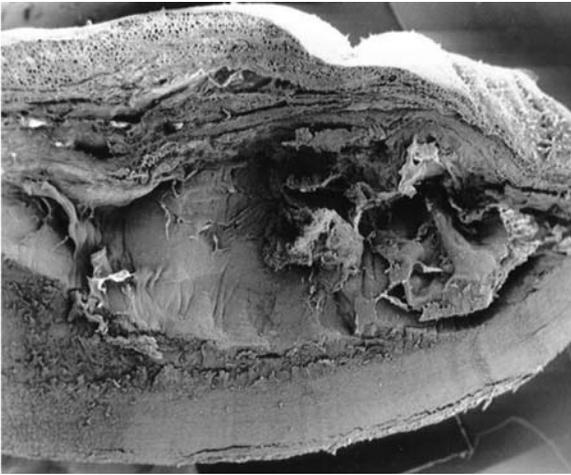


Fig. 1.

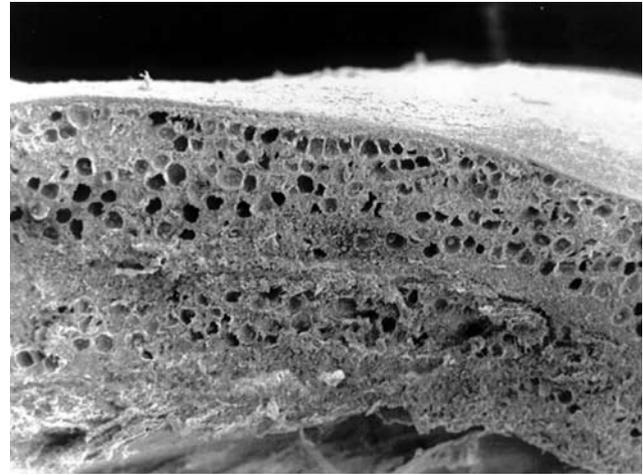


Fig. 2.

Histological investigations

Under sterile conditions, samples of dissected membrane of the aorta by Type A dissection were harvested and fixed in a buffered 3.7% formaldehyde solution for at least 24 hours at 4°C. Specimens were dehydrated in an ascending series of alcohol and embedded in paraplast, using an automatic embedding machine (Histokinette, 2000, Reichert-Jung). Five- μ m-thick serial sections were cut using a Jung Biocut microtome. They were mounted on glass slides coated with albumin-glycerine. The samples were then deparaffinized and stained by conventional technique (hematoxylin-eosin).

Scanning electron microscopy

To study the ultrastructure of the explanted dissection membrane of aorta in Type A dissection, scanning electron microscopy was performed. Specimens from the dissection membrane were fixed for 6 hours in a solution containing 2.5% glutaraldehyde and 200 mMol cacodylate. Samples were dehydrated in an ascending series of alcohol and dried using a critical point drier. All samples were then coated with gold/palladium in a cool sputter. For electron microscopic observation, a digital scanning microscope (DSM 960, Zeiss) was used.

Histological results

A scanning electron microscope revealed the following: The subendothelial tissue is comblike and riddled with blood. The endothelial cells are constructed in a polygonal way and offer a loose connection between each other (Figs. 1 and 2). In destroyed aortic wall segments, collagen fiber bundles are dispersed by fibrin-organized blood.

Endothelial cells are arranged isle-like in the smooth transition between normal and pathological aortic segments. Even macroscopically regular aortic segments miss the endothelium between single endothelial islands (Figs. 3 and 4). These endothelial cells reveal hyperplasia with a development of villus on the surface. At the side are islands with regular endothelium. Remarkable is a subendothelial hyperplasia of collagen in high gear separated from the endothelium through a comblike structure. Under electron microscope, the cavities seem to be lined with endothelium and surrounded by fibrose tissue.

Discussion

Aortic dissection is characterized by separation of the media in a course parallel to that of blood and a continuous transverse intimal and medial tear.^{2,7,8)} Hypertension is present in more than 80% of patients with dissecting aneurysms²⁾ and must be considered an augmenting, but not etiologic factor.⁶⁾ Genetic factors contribute to the genesis of some dissecting aortic aneurysms. Marfan syndrome, Turner syndrome, polycystic renal disease, the Ehlers-Danlos syndrome, and congenital bicuspid deformity of the aortic valve predispose for dissecting aortic aneurysms,^{6,9)} and several mechanisms have been suggested to induce dissecting aneurysms. In nursing rats, beta-aminopropionitrile, aminoacetonitrile, and desoxycorticosterone interact on ground substance and the medial musculature and elastic tissue lead the weakness of the aortic wall. In past years, the increasing importance of elastine for the development of aortic dissection has come to the fore. Dorbin has demonstrated that elastine

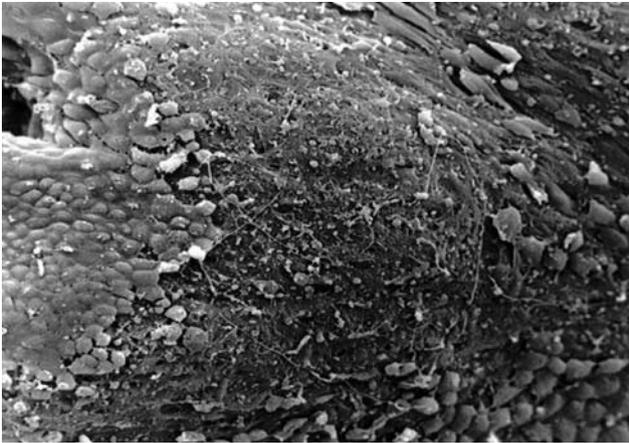


Fig. 3.

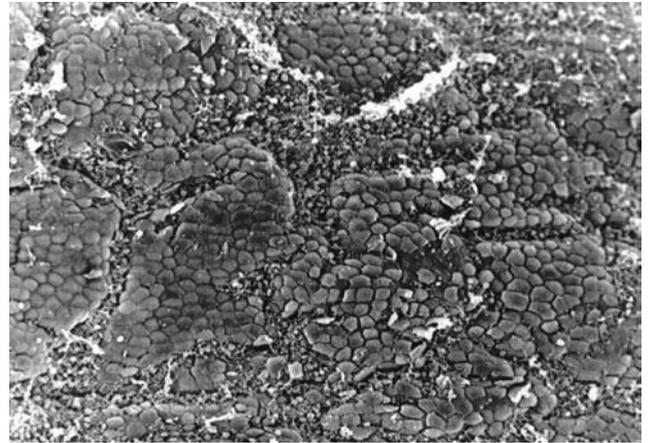


Fig. 4.

and not collagen provides the aortic wall with longitudinal retractive force. The hepatic copper level, metabolism of collagen fibers, and defects in collagen cross-linking may play a pathogenetic role in the mechanism of genetic predisposition. Recklinghausen had already clearly stated in 1864 that the crucial tissue process in the development of an aneurysm was the focal destruction of elastic fibers of media. Helmstedter described this process in detail. Many pathological findings were described in the aortic dissection, e.g., mesarteritis spots in tunica media, giant cells in the aged arterial wall, media macro infarcts in the elastic muscular tissue, and mucoid cysts. But none of these revealed a key finding that would enable full.

With this present study, we can demonstrate the changes of the ultrastructure of the aorta, which also expresses the pathology of acquired changes of valvular heart disease. This includes subadventitial hyperplasia of collagen filaments, hyperplasia of endothelial cells with loose cellular junctions, desquamation of endothelial cells with exposure of extracellular matrix, as well as hyperplasia of subadventitial collagen, which clearly militate against an acute development of this disease. The endothelial cells are hyperplastic with only loose cellular junctions. The macroscopically examined aortic segments are regular and show desquamation of endothelial cells. The islet-like endothelial tissue shows hyperplasia with development of villous surface, and only a few isles exhibit normal structure. The changes of tunica media resembling cystic medianecrosis are of great interest. Under electron microscope, the cavities seem to be lined with endothelium and surrounded by fibrose tissue. Gore and Seiwert recognized two types of medial degeneration. The more-common, muscular type that occurred in people

over 40 was found with loss of muscle and condensation of elastic laminae. The less-frequent type was termed medial degeneration of the elastic tissue type. It occurred in younger people, and there was focal loss of elastic tissue involving varying lengths of laminae, often associated with cystic change of varying degrees. All detected cavities show the same structure. A subadventitial hyperplasia of collagen filaments was viewable, but of normal structure.

Summarizing the above, we ascertained changes of the endothelium, the tunica media, and the collagen in aortic segments of Type A dissection, which ultimately militate against an acute development of this disease. Pathological findings of the aortic wall even in macroscopically normal aortic segments are remarkable. Further examinations seem to be necessary to detect the exact etiology of aortic dissection.

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