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Inflammatory abdominal aortic aneurysms: a disease entity?

Histological analysis of 60 cases of inflammatory aortic aneurysms of unknown aetiology

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Summary. Sixty inflammatory aortic aneurysms of unknown aetiology were examined by serial sections. The histological findings failed to reveal significant differences in either thoracic or abdominal aneurysms with or without marked adventitial fibrosis. Their identical morphology does not favour the existence of a special disease entity of so-called inflammatory abdominal aortic aneurysms (IAAA). Absence or existence of giant cells of any type, occurrence of plasma cells, eosinophils, granulomas, fibrinoid necrosis and adventitial fibrotic thickening cannot be considered as variables which help in differentiation. IAAA are characterized by a marked predominance of male patients and a rather benign clinical course. They usually affect the age group around 60 years. They are not rare and do not seem to be restricted to certain races. Their aetiology, like that of the cases affecting the thoracic aorta (Takayasu's disease, non-specific aortitis) remains unknown, although autoimmune diseases, the retroperitoneal fibrosis of Ormond and arteriosclerosis may be related. However, on the basis of the present evidence we cannot consider them to be one of these diseases. There are no morphological findings which would justify the separation of IAAA from Takaysu's disease.

Key words: Aortic aneurysms – Inflammatory aortic aneurysms of unknown aetiology – Inflammatory abdominal aortic aneurysms – Non-specific aortitis – Takayasu's aortitis

Introduction

The general frequency of aortic aneurysms in the autopsy material varies between 1% and 6% (Carlsson and Sternby 1964; Gore and Hirst 1973; Brindley and Stembridge 1956; Parkhurst and Decker 1955; Zimmermann and Leu 1975; Kunz 1980). Of these, only 2.5–10% are of inflammatory origin (Leu and Jülke 1984; Penell et al. 1985; Zimmermann and Leu 1975). They may be due to syphilitic arterial disease, tuberculosis, rheumatic or rheumatoid diseases, generalized giant cell arteritis, non-specific bacterial infections and miscellaneous disorders such as Behçet's disease, scleroderma, disseminated lupus erythematodes, relapsing polychondritis etc. However, the aetiology of a considerable number of inflammatory aortic aneurysms cannot be established. They are often attributed to non-specific aortitis (von Albertini 1944; Doerr 1961, 1987) or to so-called Takayasu's aortitis (Takayasu 1908). The latter is frequent in India (Vinijchaikul and Blackburn 1969; Sen 1973; Kinare 1975) and in Japan (Nasu 1963) but rare in Europe and the United States. Originally it was believed to be localized mainly to the thoracic aorta of young females, but Sen (1973) has shown that it occurs with equal frequency in all segments of the aorta. Its prognosis is poor and life expectancy among some populations is as low as 1–2 years (Sen 1973). Histology reveals a chronic inflammation of the media and adventitia with marked enlargement of the latter, focal destruction of the elastic fibres, lympho-plasmacytic infiltrates, and occasional granulomas with giant cells of the foreign body or the Langhans type (Nasu 1963; Nasu and Mamiya 1966; Sen 1973; Hall et al. 1985; Cohle and Lie 1988). A close relationship with Ormond's retroperitoneal fibrosis has been assumed (Hardmeier and Hedinger 1964; Darke et al. 1977; Lepor and Walser 1979; Crawford et al. 1985; Mitchinson 1986; Cohle and Lie 1988). Coronary involvement has been considered as frequent by some authors (Rosen and Gaton 1972; Ratner et al. 1975; Veyre et al. 1976; Hall et al. 1985; Cohle and Lie 1988) and as rare by others (Sen 1973).

During the last two decades a subgroup of inflammatory aortic aneurysms in the abdominal aorta of older male patients has been distinguished and named inflammatory abdominal aortic aneurysms (IAAA) (Walker et al. 1972; Darke et al. 1977; Penell et al. 1985; Savar-

ese et al. 1986; Crawford et al. 1985; Nadjafi 1985; Hofmann et al. 1989). These cases are characterized by a benign clinical course after surgical repair without removal of the affected tissue and without further medical treatment. Histology of these cases, however, is identical to that of Takayasu's disesase (Steiner et al. 1973; Leu 1976). Unfortunately no clinical examinations or laboratory tests exist that permit a differentiation between IAAA and Takayasu's disease. Only the existence of multicentric lesions in the aorta and extra-aortic vessels, especially of the supra-aortic branches, is suggestive of Takayasu's disease. However, even this finding is not conclusive unless substantiated by histopathology.

In this study we have re-examined our cases of inflammatory aortic aneurysms of unknown aetiology of the last two decades by serial sections. Our aim was to detect any characteristic histological findings in favour of a distinct disease entity of IAAA and to discuss the possible differences between IAAA and Takaysu's disease.

Materials and methods

In the period from 1969 until March 1990, 60 cases of inflammatory aortic aneurysm in 59 patients were recorded at our institute with the diagnosis "aortic aneurysm apparently of inflammatory origin (non-specific aortitis)". This number does not include any cases with an established aetiology (syphilitic arterial disease, tuberculosis, bacterial infections, collagen tissue diseases etc.).

The material was re-examined by serial sections (10–20 sections/paraffin block). Staining procedures included hematoxylin and eosin, van Gieson and elastin stains with addition of special stains such as periodic acid-Schiff, alcian blue, Berlin blue, Ziehl-Neelsen, Gram, Giemsa and Grocott depending on the clinical information.

Thoracic and abdominal aneurysms were examined separately. In each group, cases with or without marked adventitial involvement (Marked adventitial fibrosis is considered typical of Takayasu's disease) were distinguished. Special consideration was given to the presence of granulomas, giant cells, fibrinoid necrosis, plasma cells, and eosinophils. The files were also re-examined. Age, sex and localization were noted.

The outcome after inpatient treatment remained unknown in the majority of cases (the patients came from a vast area, including many countries). Information concerning extra-aortic manifestations of the vascular disease was rare and was not based on biopsy findings. In the 3 cases that died as inpatients, the autopsy was performed at our institute and the findings were recorded.

Table 1. Age, sex and localization of aneurysms

No. of patients: 59				No of aneurysms: 60	
Men	Women	Thoracic aneurysms: 21		Abdominal aneurysms: 39	
		Male	Female	Male	Female
51	8	15	6	37	2
Mean age (years)					
61 (34–79)	43.8 (19–69)	58.6	48	61	32.5
Ratio: 6.5:1		2.5:1		18:1	

Thoracic aneurysms				Abdominal aneurysms			
Adventitial fibrosis				Adventitial fibrosis			
No		Yes		No		Yes	
Male	Female	Male	Female	Male	Female	Male	Female
8	3	7	3	12	0	25	2
Mean age (years)							
62	64	61.5	32	63	–	60	32.5

Table 2. Racial origin of 36 patients with inflammatory aortic aneurysms with marked adventitial fibrosis

	Total	Abdominal		Thoracic	
		Men	Woman	Men	Woman
Central Europeans	21	17	0	4	0
Southern Europeans	13	7	1	3	2
Coloured races	2	0	1	0	1

Table 3. Histopathological findings

	Thoracic aneurysms Adventitial fibrosis		Abdominal aneurysms Adventitial fibrosis	
	++ 10	+/− 11	++ 27	+/− 12
Giant cells (foreign body type)	0	0	1	0
Giant cells (Langhans type)	0	0	2	0
Granulomas without central necrosis	1	0	4	2
Granulomas with central necrosis	0	0	3	0
Follicles with germinal centres	8	0	20	0
Plasma cells +++	3	5	2	3
Eosinophils +++	0	0	1	0
Wall dissection	5	4	1	0
Wall rupture	0	0	2	0
Death during hospitalization	1	1	1	0

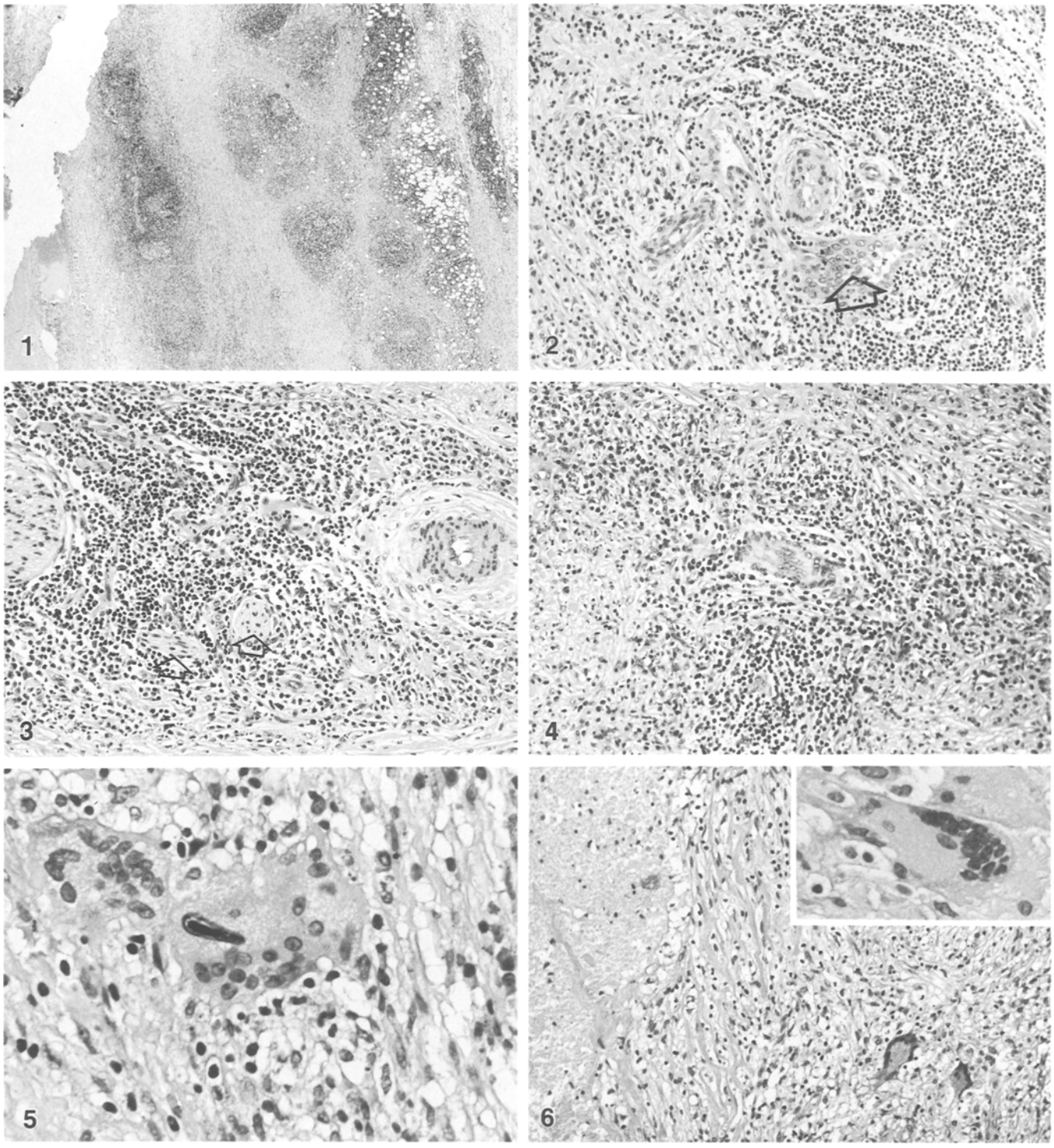

Fig. 1. Aortic wall in inflammatory abdominal aortic aneurysm. The adventitia is expanded; the fibrous scar tissue involves the para-aortic fatty tissue. Several lymphoid follicles with germinal centres are scattered throughout the media and adventitia. H & E, ×18

Fig. 2. Giant cell (*arrow*) in association with a small vessel surrounded by focal inflammation. H & E, ×125

Fig. 3. Focal lymphocytic inflammation around small nerves (*open arrows*). At right a small artery with intimal proliferation. H & E, ×125

Fig. 4. Giant cell of the foreign body type within focal inflammation. H & E, ×125

Fig. 5. Giant cell of the foreign body type with inclusion of an elastic particle. H & E, ×400

Fig. 6. Granuloma with central necrosis and giant cells of the Langhans type. H & E, ×125. *Inset*, Giant cell of the Langhans type. H & E, ×400

Results

The 60 aneurysms studied were from 59 patients, including 51 men of 34–79 years (mean age 61 years) and 8 women of 19–69 years (mean age 43.8 years) with a ratio of 6.5:1. Twenty-one were localized in the thoracic aorta (15 men, 6 women) and 39 in the abdominal aorta (37 men, 2 women), the ratio being 2.5:1 in the thoracic and 18:1 in the abdominal aorta. A marked fibrotic enlargement (considered as a characteristic feature of both IAAA and Takayasu's disease) was absent in 11 of the thoracic and in 12 of the abdominal aneurysms and present in 10 of the thoracic and in 27 of the abdominal aneurysms (Table 1).

The racial origin of the 36 patients (37 aneurysms) with marked adventitial fibrosis (Table 2) did not reveal any pecularities. The majority of patients were of Central and Southern European origin. Only 2 of the female patients were coloured (1 African, 1 Indian).

The histopathological features of thoracic and abdominal aneurysms were identical (Table 3).

The intima was either normal or thickened by arteriosclerotic alterations with or without apposition of thrombotic material. Inflammatory cell infiltrates were rare or of minor degree. The media contained vasa vasora in increased numbers; they were frequently dilated and surrounded by lympho-plasmo-histiocytic infiltrates. The elastic fibres were focally destroyed. The smooth musculature was partly preserved, mostly replaced by fibrous tissue. The adventitia was variably preserved in some of the cases, but mostly increased three- or fourfold in thickness and inseparable from the media. It contained inflammatory cells (lymphocytes, plasma cells, histiocytes) in diffuse distribution or in follicle-like arrangement (Fig. 1). Granulomas composed of histiocytes or epithelioid cells in palisade-like arrangement, without or with central necrosis, were occasionally observed.

Around granulomas, giant cells of the Langhans or of the foreign body type were observed in rare cases. Giant cells of the Langhans type always occurred in connection with granulomas with central necrosis. Giant cells of the foreign body were few. In serial sections single giant cells of the foreign body type were found in association with granulomas, in focal lymphocytic infiltrates or in the liposclerotic para-aortic fatty tissue, occasionally also in connection with vascular proliferations, similar to those found in histiocytomas (von Albertini 1974; Smolle et al. 1989) (Fig. 2). Small nerves or occluded capillaries within focal infiltrates frequently simulated giant cells (Figs. 3–6).

Focal lymphatic infiltrates within the thickened adventitia contained germinal centres in the majority of the cases. Plasma cells occurred quite frequently in moderate or in abundant numbers, either as single cells or within focal infiltrates (Fig. 7).

Eosinophils were rarely found in large numbers (1 case). The larger adventitial vasa vasora were frequently altered by lymphocytic wall infiltrates or by intimal proliferation with or without obliteration of the lumen.

Fibrinoid necrosis, often in combination with haemorrhage, was also a common finding. It was usually of limited extent. Fibrinoid necrosis of the walls of the vasa vasora was also observed.

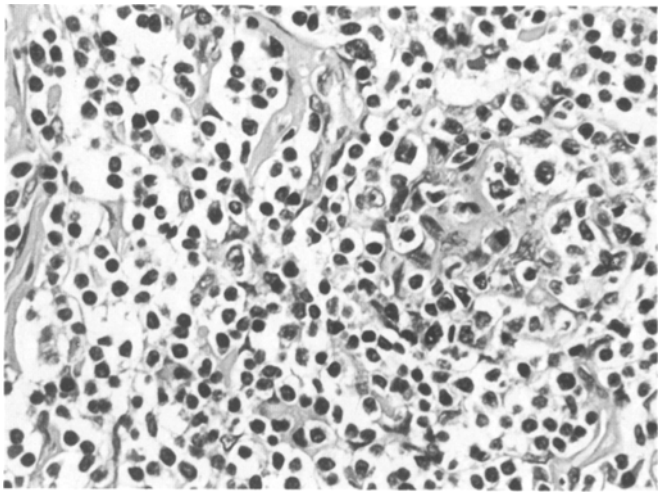

Fig. 7. Focal inflammatory infiltrate of lymphocytes and plasmacells around destroyed vasa vasorum. H & E, ×400

The cases containing granulomas were thoroughly investigated for tuberculosis, autoimmune diseases, nonspecific bacterial infections and parasites. However, special stains, serology, immunological examinations and clinical investigations were negative in all cases.

Dissection of the aortic wall was noted in 10 cases (16.6%), and aortic rupture in 2 cases. Death during inpatient treatment occurred in 3 cases. In 2 patients it was due to acute myocardial infarction caused by arteriosclerosis. No inflammatory vascular lesions of extra-aortic vessels could be detected at autopsy. The third case is very interesting. This sudden death of a 24-year-old women was due to an inflammatory occlusion of the aorto-coronary ostia and was considered to be an acute case of Takayasu's disease. Several inflammatory vascular lesions were observed in extra-aortic vessels (supra-aortic branches). However, in this case the adventitia was hardly enlarged. Sudden death in an early stage of the disease may have prevented the development of an adventitial fibrosis.

The extent of the adventitial fibrosis varied. In cases with profound thickening of the aortic wall, the para-aortic structures such as fatty tissue, lymph nodes, nerves, lumbar ganglia and ureters were frequently included within the fibrous proliferation. The findings in these cases were identical to those of retroperitoneal fibrosis.

Comparison of the four examination groups (Table 3) did not reveal any histological findings that could be attributed to a certain group. A differentiation between IAAA and Takayasu's disease on a morphological basis was therefore not possible.

Discussion

According to earlier investigations (Zimmermann and Leu 1975; Leu and Jülke 1984) inflammatory aortic an-

eurysms of varying aetiology are more frequent in the thoracic (17%) than in the abdominal aorta (6%). However, this is not valid for aneurysms due to non-specific aortitis with a ratio of 2:1 in favour of the abdominal localization.

A predominance of male patients was noted for all types of aortic aneurysms: 4:1 overall, 6.5:1 for medionecrosis, 5:1 for inflammation of any aetiology and 2.5:1 for arteriosclerotic aneurysms (Leu 1986). For aortic aneurysms apparently of inflammatory origin but unknown aetiology the ratio is 3.5:1 in the thoracic, but 18:1 in the abdominal aorta. This marked predominance of the male sex in IAAA is in accordance with reports in the literature (Penell et al. 1985). In classical Takayasu's aortitis the ratio is different, with a predominance of women in most reports (Hall et al. 1985; Robbs et al. 1986; Takagi et al. 1986).

The mean age of patients with aneurysms due to non-specific aortitis (thoracic and abdominal) is around 60 years in both localizations. This is higher than in inflammatory aneurysms of all aetiologies (53.9 years) or in aneurysms due to medionecrosis (51.3 years) but lower than in arteriosclerotic aneurysms (65.9 years) (Leu and Jülke 1984).

Racial predominance could not be detected in our material. Our male patients were all white and of Central or Southern European origin. Of the 5 women, 2 were coloured (1 Indian, 1 African). They probably belong to the group of classical Takayasu's disease, although their clinical course did not differ from that of the other cases. The number of female patients in our series is too small for comparison.

Histology cannot distinguish between Takayasu's disease and the subgroup of IAAA of older male patients. Giant cells of all types are rare in IAAA. However, the only two cases with giant cells of the Langhans type belonged to the subgroup of IAAA, whereas in our cases of thoracic non-specific aortitis of young females (suspected Takayasu's aortitis) we did not detect any giant cells at all. The occasional occurrence of singular giant cells of the foreign body type is not a relevant finding. Such cells occur in many inflammatory vascular diseases, especially when elastic fibres are destroyed. Absence or presence of giant cells of any type is therefore not a useful variable in differentiating between aortic aneurysms apparently of inflammatory origin. Granulomas without and with central necrosis may occur in Takayasu's disease as well as in IAAA. The same applies for plasma cells. Even in abundant numbers they are no indicator of a syphilitic aetiology.

Lymphoid follicles with germinal centres are often observed in cases with adventitial involvement and adventitial fibrosis is typical though not essential for diagnosis in both thoracic and abdominal aneurysms. Its extent may be dependent upon the duration of the disease and in early cases (such as our case of a young woman with sudden coronary occlusion) it may not yet have developed. About half of our cases of thoracic and abdominal aneurysms did not show fibrous involvement of the adventitia of notable degree.

Multicentric segmental inflammatory lesions in vessels of various calibre suggest a systemic disease (such as Takayasu's disease). However, the inflammatory nature of segmental alterations can only be established by histology. Many reports of multiple occlusions in the literature are not substantiated by biopsy findings. It must be kept in mind that multiple aneurysms and segmental arterial occlusions are also a common finding of arteriosclerosis. In an earlier investigation we found multiple arteriosclerotic aneurysms in 11% of the cases (Zimmermann and Leu 1975). In 2 of our 3 autopsy cases the coronary lesions of apparently inflammatory nature which were responsible for the lethal outcome were arteriosclerotic and not inflammatory. The only one of our cases with histologically verified inflammatory lesions of extra-aortic vessels was that of a young woman with Takayasu's disease of the aortic arch and sudden death due to inflammatory occlusion of the aorto-coronary ostium. In general, the findings on dissection and rupture agree with those reported in the literature (Crawford et al. 1985; Kaschner et al. 1985; Downs and Lye 1986; Savarese et al. 1986; Hill and Charlesworth 1988; Sterpetti et al. 1989; Moosa et al. 1989).

It is important to note that the histopathology of inflammatory vascular diseases in general is of low specificity (Churg and Churg 1989). This does not facilitate the differential diagnosis of aneurysms of inflammatory origin. In our cases, the clinical findings and the laboratory results did not help to establish the aetiology.

It has been suggested that IAAA may be a special type of arteriosclerosis (Rose and Dent 1981; Mitchinson 1984; Savarese et al. 1986). A certain degree of secondary inflammation is common in arteriosclerotic aneurysms (Uehlinger 1975; Doerr 1987; Adams 1989) but is usually limited to slight focal or diffuse accumulation of lymphocytes. Atrophy and disappearance of elastic fibres may also occur in arteriosclerosis but differs from the patchy and mothbite-like focal destruction in mesaortitis.

The possibility of an autoimmune disease, related to Takayasu's disease, has also been discussed. The histolopathology would fit with this opinion. The occurrence of lymph follicles with germinal centers is a well-known finding in many autoimmune diseases (rheumatic synovialitis, Hashimoto's disease etc). Several authors (Nasu 1963; Martorell 1967; Siebenmann et al. 1988) have pointed out that the elevated blood sedimentation rate, the loss of weight, the painful sensations and the higher operative mortality in comparison to arteriosclerotic aneurysms (Penell et al. 1985) speak in favour of an autoimmune disease. However, an elevated blood sedimentation rate was also found in arteriosclerotic aneurysms (Uehlinger 1975; Parums 1990) and laboratory findings and systemic disease manifestations have so far not been reported. The benign clinical course after surgical repair without removal of the affected tissue and without medical treatment is also not in accordance with an autoimmune disease.

A special variant of Ormond's retroperitoneal fibrosis has also been considered (Mitchinson 1984) since the histopathological findings are identical (Penell et al. 1985; Wagenknecht 1978). Age and localization are in

the correct range but the favourable clinical course without medical treatment in IAAA differs from that in Ormond's disease (Wagenknecht 1978).

An autoimmune response to lipids infiltrating through arteriosclerotic plaques into media and adventitia has recently been postulated by some authors (Parums 1990; Routledge 1990). This theory could explain many of the features of IAAA and would also explain the aetiology of Ormond's disease (Routledge 1990), but does not account for the different clinical course of IAAA and Ormond's disease or for the occurrence of such aneurysms in young females at localizations where arteriosclerosis is not common.

The mean age of around 60 years lies between that for arteriosclerotic aneurysms and inflammatory aortic aneurysms of all aetiologies. The preference for the male sex and for the abdominal aorta correlates with the arteriosclerotic aneurysms.

These aneurysms may, of course, represent a disease entity of unknown aetiology. The benign clinical course in comparison with Takayasu's disease and many cases of Ormond's disease favours this opinion. There is no histopathological evidence for a special disease entity and we do not possess any clinical findings or laboratory data which would substantiate such an opinion.

The term inflammatory abdominal aortic aneurysms is misleading. All that can be said is that among aortic aneurysms a subgroup is of inflammatory origin. This subgroup can be divided into (a) aneurysms due to a specific infection (syphilis, tuberculosis) and (b) aneurysms due to a non-specific infection. A further number of aneurysms is apparently of inflammatory origin but of unknown aetiology and pathogenesis. This group includes aneurysms due to Takayasu's aortitis (mostly affecting young females) and abdominal aneurysms of older, generally male subjects. A distinction between these two types is based solely on the clinical appearance and cannot be substantiated by histopathological findings. The morphological features do not even permit a differentiation into primary inflammatory disease or inflammation occuring in, say, atherosclerosis. It may well be that the characteristic histological findings of a chronic mesaortitis with or without fibrous periaortic involvement can be caused by different aetiological factors. In any case the morphological alterations in the so-called IAAA do not justify the assumption of a new and distinct disease entity. The term abdominal (or thoracic) aortic aneurysm apparently of inflammatory origin suffices as a descriptive pathological diagnosis until the aetiology and pathogenesis of this disorder are further elucidated.

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