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Coronary arteritis and aortoarteritis in the elderly males

A report of two autopsy cases with review of the literature

Akira Tanaka [1]*, Masashi Fukayama [1], Nobuaki Funata [1], Morio Koike [1], and Ken Saito [2]

1 Department of Pathology, Tokyo Metropolitan Komagome Hospital, 3-18-22 Honkomagome, Bunkyo-ku, Tokyo 113, Japan
[2] Department of Pathology, Jichi Medical School, Tochigi, Japan

Summary. Two elderly males died suddenly of acute myocardial infarction. Autopsy disclosed extensive and severe coronary arteritis, associated with aortitis. In most of all of the epicardial coronary arteries, fibrous thickening of the adventitia was remarkable and muscle layers of the media were nearly destroyed by infiltration of lymphocytes and plasma cells. The aorta showed adventitial thickening with destruction of the elastic layers of the outer media. In the literature, similar extensive coronary arteritis has been reported in five cases of Takayasu's arteritis. All cases, including ours, were elderly or middle-aged males who died suddenly of myocardial infarction without the antemortem detection of the underlying arteritis. These aspects differ from the typical Takayasu's arteritis and we suggest that these cases are considered as a separate entity.

Key words: Aortitis – Coronary arteritis – Myocardial infarction – Elderly male

Introduction

Infammatory diseases of the coronary artery are rare and they occur as an isolated angitis or as a phenomenon in generalized angitis, such as polyarteritis nodosa, giant cell arteritis, and Takayasu's arteritis (Doerr 1979). Polyarteritis nodosa and giant cell arteritis have pathognomonic histopathological features. Takayasu's arteritis is an aortitis of unknown aetiology, which frequently affects young females (Nasu 1963; Ueda 1968). The pathologic features of Takayasu's arteritis are relatively not so pathognomonic: intimal proliferation and adventitial fibrous thickening of the aorta and its major branches. Although the involvement of the proximal portions of the coronary artery is not infrequent in Takayasu's disease (Rose and Sinclair-Smith 1980), extensive coronary involvement resulting in myocardial infarction is extremely rare. Only five cases have been reported. We presented here two cases of extensive coronary arteritis and aortitis histopathologically somewhat like Takayasu's arteritis, and then, stressed the unique aspects of these reported cases comparing with known systemic arteritis, including typical Takayasu's arteritis.

* *Present address*: Department of Pathology, Osaka University School of Medicine, Osaka, Japan

Offprint requests to: M. Fukayama

Case report

Case 1. A 62-year-old man was admitted to the hospital because of severe dyspnoea. He had been well until three months previously, when exertional dyspnoea developed. This became worse with cough, sputum and chest discomfort. On admission, he was very dyspnoeic with a blood pressure of 108/52 mmHg, a pulse rate of 120 beats per min, and a temperature of 37.2° C. On physical examination, neck veins were distended and the liver edge was felt 1 cm below the right costal margin. Fine crackles were audible at the bases of both lungs. Chest roentgenogram showed pulmonary congestion and bilateral pleural effusions. Sinus tachycardia with left ventricular hypertrophy and left axial deviation was observed on electrocardiogram (ECG). The white blood cell count was 13800 and the serum levels of cardiac enzymes were normal. Serological tests for syphilis were negative. In the evening of the day of the admission, he felt severe chest discomfort and vomited. Abnormal Q waves were present in v1, v2 and v3 leads of ECG at that time. He died 13 h after the admission in spite of therapy for myocardial infarction.

Case 2. A 64-year-old man had been admitted to a hospital because of dysphagia and chest discomfort. The diagnosis of oesophageal carcinoma was made by the roentgenographic and endoscopic examinations. He was transferred to our hospital for intensive therapy for the cancer. On admission, he was passive and supine with a blood pressure of 130/70 mmHg and a pulse rate 72 beats per min. Physical examination was normal.

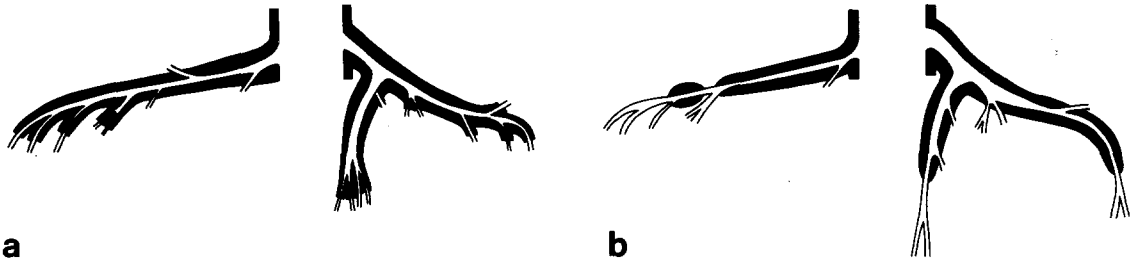


Fig. 1. The scheme of the distribution of coronary arteritis ((**a**) Case 1, (**b**) Case 2). The *thick black lines* show the adventitial fibrosis. The main coronary branches were diffusely affected in both cases

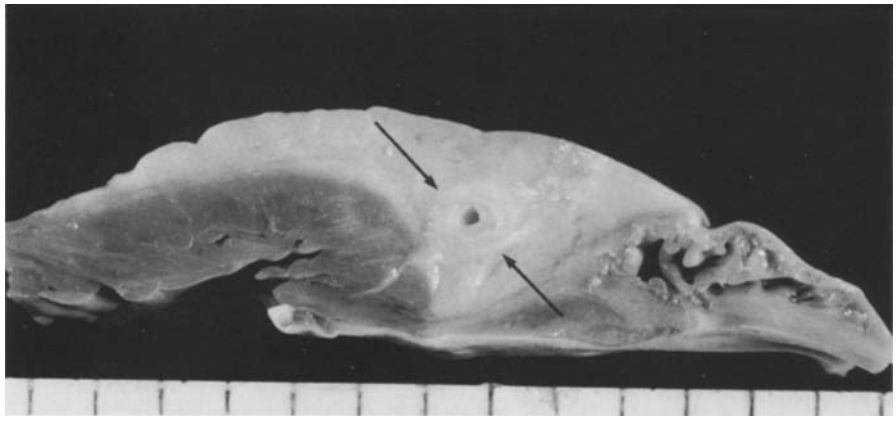


Fig. 2. Macroscopical view of the right coronary artery of Case 1. Concentric fibrosis of the adventitia was prominent (*arrows*)

There were no abnormal findings in routine laboratory data including negative serological tests for syphilis. Chest roentgenogram showed mild enlargement of the cardiac silhouette. An electrocardiogram was normal. Roentgenographic and endoscopic examinations revealed ulcerated carcinoma in the middle of the oesophagus. A cardiac echogram showed small amount of pericardial effusion without remarkable changes in the ventricular walls. On the fifteenth day, he was treated with cisplatinum, methotrexate and bleomycin for the carcinoma. Next morning, he appeared well at 7 am, but was found dead in the bathroom one h later.

Results

Case 1

The heart was moderately hypertrophied, weighing 455 g. On the cut surface, a fresh non-penetrating myocardial infarction was found on the anteroseptal wall. Grossly, fibrous thickening of coronary arteries extended from the ostia to the distal portions for about 7 to 8 cm (Fig. 1a and Fig. 2). Intimal fibrosis was also severe, resulting in narrowing or obliteration of the lumina. However, the intramural coronary arteries were normal and the ostia of both coronaries were patent. Microscopically, the most characteristic finding was marked fibrous thickening of the adventitia with infiltration of lymphocytes and plasma cells. The muscular layers of the media were confluently affected and almost completely destroyed by infiltration of lymphocytes and plasma cells (Fig. 3). Intimal fibrosis was also severe, associated with recanalizations of thrombi or fresh fibrin thrombi. There was no giant cell reaction. Lymphocytic infiltration was observed focally in the coronary veins. The aortic and mitral valves had mild lymphocytic infiltration without deformities and adhesions. In the aorta and its major branches, marked adventitial thickening was confluent from the ascending aorta to the lower abdominal aorta, including their major branches. Atherosclerotic changes were mild. There was neither aneurysm formation nor vascular occlusion. Microscopically, all specimens of the aorta (Fig. 4) and its major branches, including the pulmonary trunks (Fig. 5), showed inflammatory changes: fibrous thickening of the adventitia, similar to that of coronary arteries, was marked with infiltration of lymphocytes and plasma cells. Conspicuously dilated capillaries were growing into the outer layers of the media and dissociated the elastic layers (Fig. 4). Granulation tissue and fibrous tissue was scanty in comparison with dilated capillaries. There was no swelling of endothelial cells. There was no giant cell reaction. Vasa vasora showed neither endoarteritis nor onion-skin like fibrosis.

Case 2

The heart showed moderate degree of hypertrophy, weighing 430 g. On cut surface, many foci

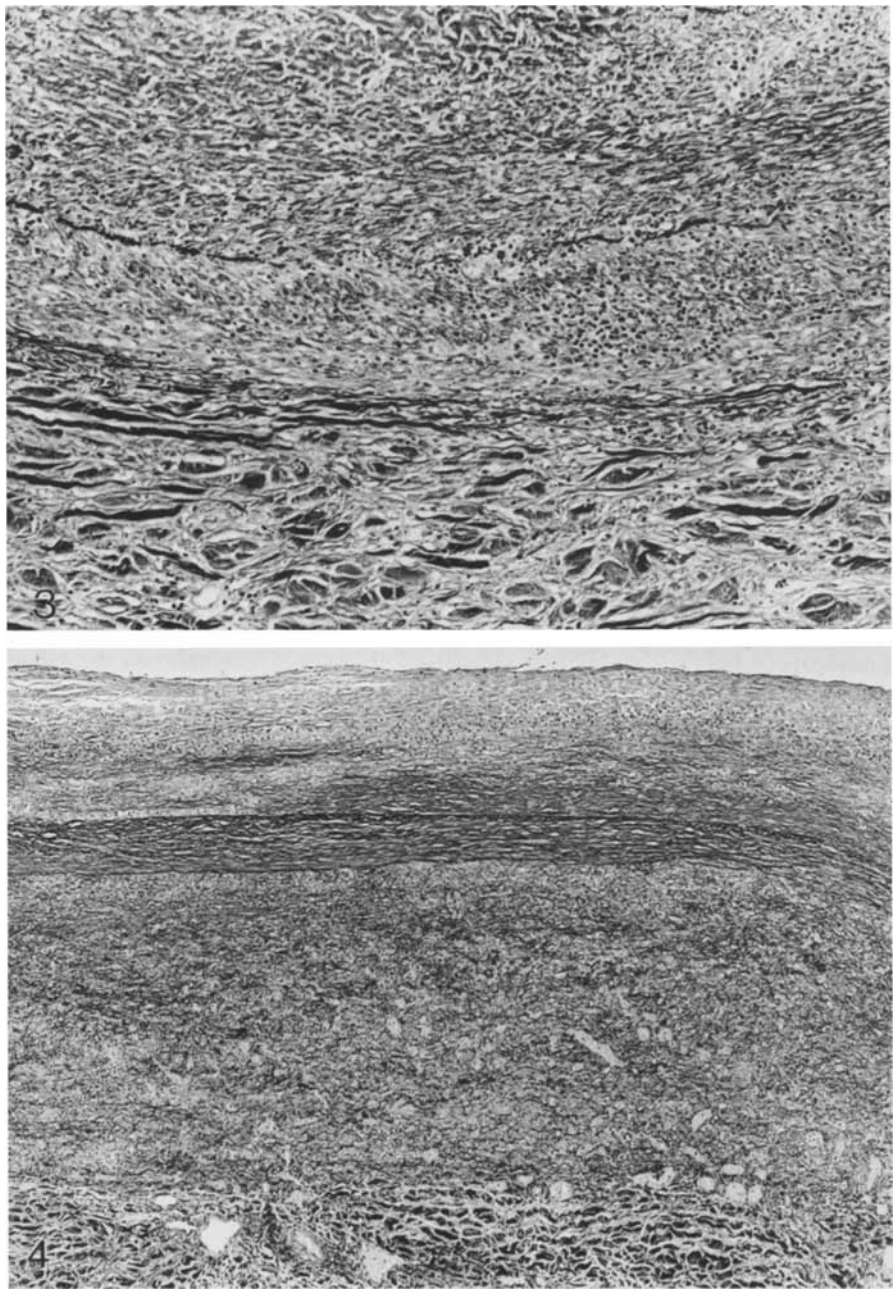


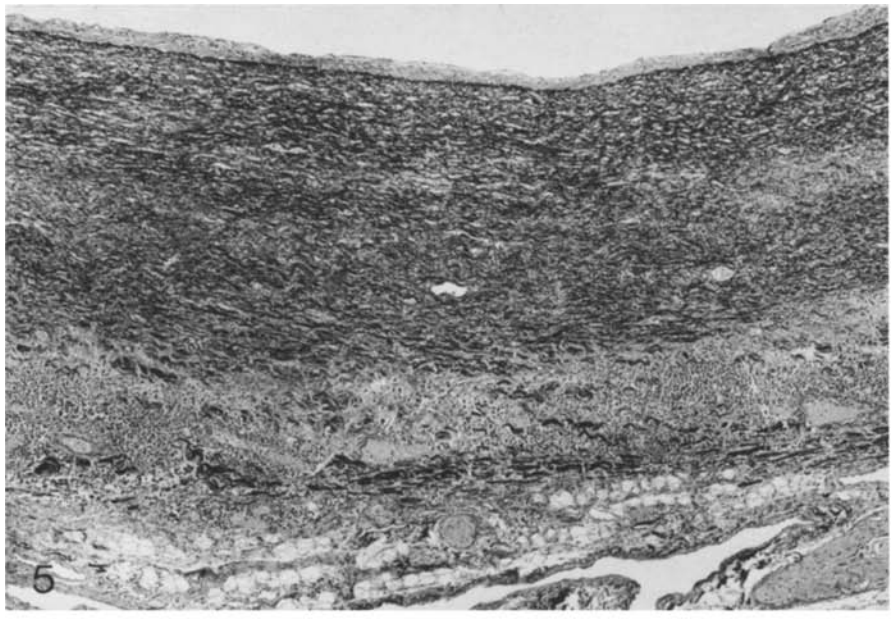
Fig. 3. Coronary arteritis of Case 1. The muscular layers of the media was almost completely destroyed by infiltration of lymphocytes and plasma cells. However, the internal and external elastic laminae remained. Elastica van Gieson (EvG), ×38

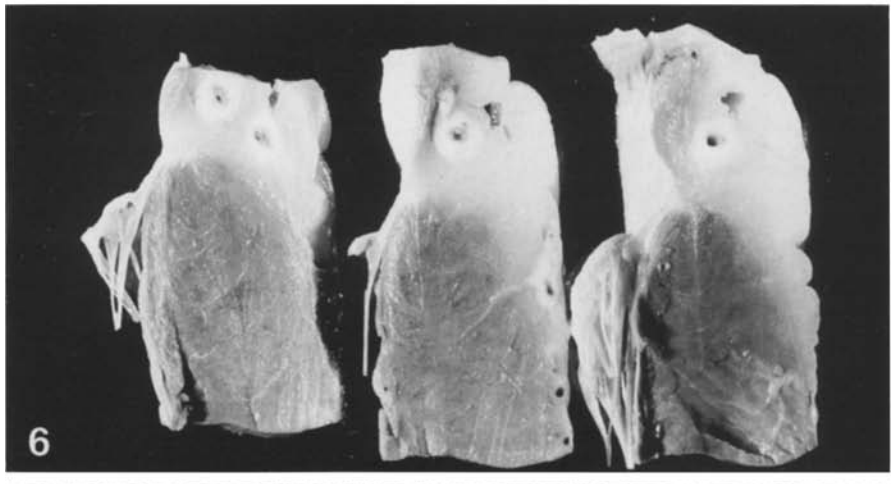
Fig. 4. Aortitis of Case 1. Fibrous thickening of the adventitia was marked with infiltration of lymphocytes and plasma cells. Note many dilated capillaries dissociating the medial elastic layers. EvG, ×15

of fresh myocardial infarction were found in the anterior wall, septum, and posterior papillary muscle. Gross and microscopical findings of the coronary artery were quite similar to those of Case 1 (Fig. 6), although the distribution was more extensive in this case (Fig. 1 b). The gross appearance of the aorta and its major branches was normal. Microscopically, however, inflammatory change identical to that in Case 1 was widely distributed in the aorta (Fig. 7) and its major branches, including the pulmonary trunks. Although adventitial fibrosis was less marked, dilated capillaries were similarly conspicuous in the outer media of the aorta. Other remarkable findings were carcinoma of the oesophagus, metastasizing to the liver, lung, stomach, and retroperitoneal tissue.

Discussion

Both cases showed an unique type of aortoarteritis in elderly males. The inflammatory change was maximal in the coronary arteries. Most of all of





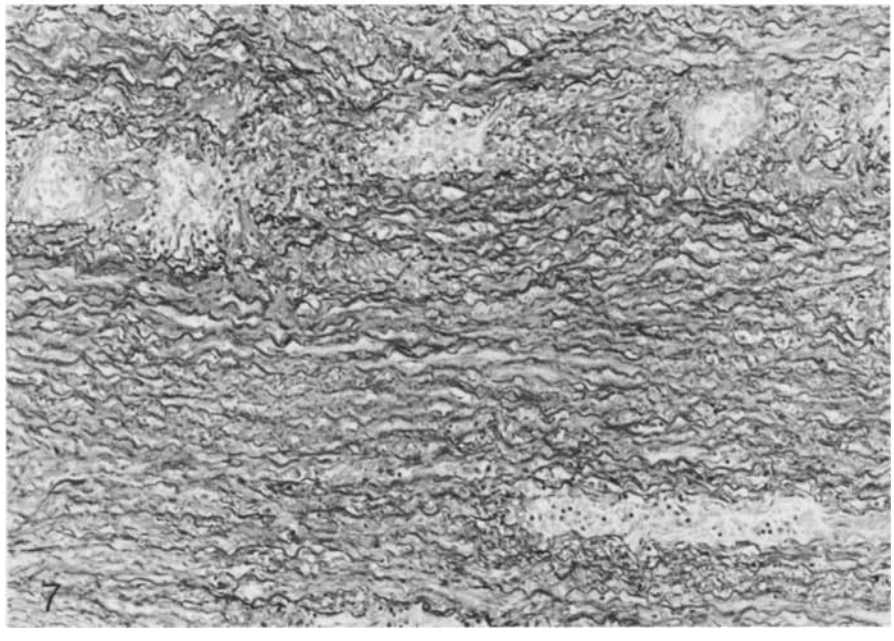


Fig. 5. Arteritis of the main pulmonary artery of Case 1. Inflammatory changes appeared to be similar to those of the aorta. EvG, ×15

Fig. 6. Macroscopical view of the circumflex branch of Case 2, showing severe adventitial fibrosis

Fig. 7. Aortitis of Case 2. Note dilated capillaries dissociating the medial elastic layers. The findings closely resembled those of Case 1. EvG, ×38

Table 1. Summary of the reported cases which showed extensive coronary arteritis resulting in acute myocardial infarction

Case/authors	age	sex	coronary artery	distribution of arteritis aorta, major branches	cause of death
1. Castleman et al. (1967)	45	male	all of the main epicardial branches	entire aorta, most of major branches	myocardial infarction
2. Rosen et al. (1981)	44	male	left: 3.5 cm long right: 2.5 cm long	aortic arch, abdominal aorta, left Sbc.A, C.C.A., S.M.A.	myocardial infarction (sudden death)
3. Aufderheide et al. (1981)	49	male	left main and proximal third of LAD, right: 4 cm long	abdominal aorta	myocardial infarction (sudden death)
4. Aufderheide et al. (1981)	60	male	both main coronary arteries and their primary branches	not detected	myocardial infarction (sudden death)
5. Payan et al. (1984)	71	male	extensive involvement of epicardial coronary arteries	abdominal aorta	myocardial infarction (sudden death)
6. Case 1	62	male	all of the main epicardial branches	entire aorta, most of major branches	myocardial infarction (sudden death)
7. Case 2	64	male	all of the main epicardial branches	entire aorta, most of major branches	myocardial infarction (sudden death)

Sbc.A.: subclavian artery, C.C.A.: common carotid artery, S.M.A.: superior mesenteric artery

Table 2. Summary of the reported cases, which showed coronary arteritis in association with idiopathic retroperitoneal fibrosis or inflammatory aneurysm

Case/authors	age	sex	distribution of coronary arteritis	associated disease
1. Reed et al. (1959)	56	male	2 cm long in the main left and LAD right: segmentally affected.	retroperitoneal fibrosis
2. Hardmeier et al. (1966)	59	female	main left coronary and LAD	retroperitoneal fibrosis
3. Jones et al. (1966)	64	male	small area of LAD	retroperitoneal fibrosis
4. Russo et al. (1967)	66	male	right: 5 cm in length	retroperitoneal fibrosis
5. Mitchinson (1972)	64	male	circumflex branch	retroperitoneal fibrosis
6. Mitchinson (1972)	71	male	6 cm long in LAD	retroperitoneal fibrosis
7. Pereira et al. (1981)	68	male	no description	inflammatory aneurysm
8. Mitchinson et al. (1984a)	65	male	3 cm long in LAD right: 2 cm in length	chronic periaortitis (inflammatory aneurysm)
9. Mitchinson et al. (1984a)	62	male	proximal sites of all main branches	chronic periaortitis (retroperitoneal fibrosis)

LAD: left anterior descending branch

the epicardial branches were diffusely affected with confluent fibrous thickening of the adventitia. The inflammation, in its severe form, involved the whole muscular layer of the media, resulting in its destruction and in thrombus formation in the lumen. The aorta and its major branches also showed fibrous thickening of the adventitia with conspicuously dilated capillaries. We compared these findings with those of the known types of aortoarterities.

Syphilitic, rheumatoid and rheumatic arteritis could be excluded on the basis of the clinical history and laboratory data. Giant cell arteritis, which included temporal arteritis, polyarteritis rheumatica, and polymyalgia arteritica, is known to affect large vessels (Klein et al. 1975) and has a wide spectrum of granulomatous inflammation seen mainly in the media (Oestberg 1972). In our cases, the major sites of inflammation were adventitia and outer media. Those reported cases of giant cell arteritis which caused myocardial infarction with extensive coronary involvement (Morrison and Abitbol 1955; Crompton 1959; Martin et al. 1980; Saeve-Soederbergh et al. 1985; Lie et al. 1986) showed marked giant cell reactions in the media of the coronary artery. Such a finding was

not identified in our cases. Furthermore, there were no clinical manifestations characteristic of giant cell arteritis, such as headache, myalgia and visual disturbance (Huston et al. 1978). Takayasu's arteritis predominantly affects young females: elderly male cases are very rare (Ueda 1968). Pathologically, marked adventitial fibrous thickening of the aorta in our cases is also a common finding in Takayasu's arteritis, but the microscopical finding was different: conspicuously dilated capillaries dissociating the medial elastic layers of the aorta. On review of the literature, five similar cases with extensive coronary arteritis were reported as showing coronary involvement of Takyasu's arteritis (Table 1). Interestingly, all the cases, including ours, were elderly or middle-aged males who died of acute myocardial infarction. All cases escaped ante-mortem detection of the underlying arteritis. These aspects were quite different from those of typical Takayasu's arteritis.

It is also interesting to note that arteritis of epicardial coronary arteries has been reported sporadically in association with idiopathic retroperitoneal fibrosis or inflammatory aneurysm (Table 2): the histology of the aorta in both lesions has some similarities with that of the aorta of our cases. Additionally, these cases also showed male predominance. Mitchinson et al. (1984 b) suggested, that retroperitoneal fibrosis and inflammatory aneurysm were identical conditions and they described the lesions as a unifying entity, "chronic periaortitis and periarteritis". Since the clinicopathological features of "chronic periaortitis and periarteritis" have not yet been clarified, we could not compare our cases with this entity, nor determine whether the lesions are included in the spectrum of a similar disease. However, there might be a type of coronary arteritis associated with an aortitis in the elderly male, which considerably differs from Takayasu's arteritis or giant cell arteritis.

As far as reviewing the reported cases, we suggest that our cases and the reported cases shown in Table 1 should be dealt with separately from Takayasu's arteritis. This type of coronary arteritis may be fatal, presenting as sudden death of myocardial infarction in the elderly men. Further experiences and pathological study would confirm this suggestion.

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