Takayasu’s Arteritis with Heart Failure Due to Atherosclerosis

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An autopsy case of an elderly man with Takayasu’s arteritis and atherosclerosis is presented. Ischemic signs and symptoms including anginal attack were aggravated with activity of arteritis despite administration of prednisolone throughout the entire course. The autopsy revealed severe atherosclerosis over the aorta and its branches, and arteritis in the fibrotic stage in localized area. Vessels of vital organs including heart and kidneys, did not show Takayasu’s arteritis but were atherosclerotic. This case highlights the numerous problems encountered in the treatment of elderly patients with vasculitis such as Takayasu’s arteritis.

Key words: Ischemic heart disease, Steroid therapy

Takayasu’s arteritis usually affects young or middle-aged women (1–4), however, recently elderly patients with this disease have been described (1, 5–7). Heart failure which is a major complication of this unique disease appears to be a consequence of systemic hypertension and aortic regurgitation (1, 4). Ischemic heart disease which is commonly due to coronary atherosclerosis in the elderly, is also reported to occur due to coronary arteritis or atherosclerosis in patients with Takayasu’s arteritis (3–14). One can expect that advanced coronary atherosclerotic lesions overlap and/or coexist in the elderly patients with Takayasu’s arteritis. However, to our knowledge no such case has been previously described with clinicopathological details in the literature. In the present paper, a patient with this rare occurrence is described.

CASE REPORT

A 68-year-old Japanese male, who had been suffering from hypertension and hypercholesterolemia for 15 years, was admitted to Keio University hospital with a complaint of blood pressure (BP) difference between both arms in August 1987. The patient was of medium stature and was alert and oriented. Temperature was 36.8°C, respiratory rate 14/min, pulse rate 60/min and regular. The systolic BP was 120 mmHg on the left arm, 140 mmHg on the right arm, 160 mmHg on the left leg, and 170 mmHg on the right leg. The conjuctiva was not anemic. Arteriosclerotic change was found on retinal vessels. The lungs and heart were normal. There was no hepatosplenomegaly. Vascular bruits were heard on bilateral sides of the neck. Pulselessness was noticed on the left radial artery. Laboratory data on admission included the erythrocyte sedimentation rate (ESR) of 42 mm/h and 100 mm/2h. The urine had a specific gravity of 1.005, positive for protein but the sediments were normal. The white blood cell count was 10.2 x 10^3/mm^3 with normal differential. The blood urea nitrogen level was 39.9 mg/dl, serum creatinine 2.1 mg/dl, total cholesterol 180 mg/dl, triglyceride 381 mg/dl, potassium 5.5 mEq/l, and C-reactive protein 1.15 mg/dl. Plasma renin activity (PRA) was 1.3 ng/ml/h. Autoantibodies were negative. Electrocardiography (ECG) showed left ventricular hypertrophy.

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Aortography revealed stenotic lesions in bilateral subclavian arteries (Fig. 1) and the diagnosis of Takayasu’s arteritis was made. Steroid therapy [prednisolone (PSL) 30 mg daily] was started. Laboratory data related to the appearance of inflammation immediately subsided and he was discharged at the same dose of PSL. PSL was tapered off to 15 mg daily, ESR increased gradually and general malaise, dyspnea on exertion and intermittent claudication appeared. The dose of PSL was increased back to 30 mg daily but it did not control these symptoms completely. Melena was noticed and he was admitted a second time to our hospital in December 1987. ECG showed left ventricular hypertrophy and ischemic change which had not been previously found. The endoscopic studies of the upper gastrointestinal tract revealed multiple gastric ulcers. Aortography showed that the stenosis was extended to bilateral subclavian arteries, left common carotid, right renal and left internal iliac artery. Hypertension continued despite administration of multiple antihypertension drugs including a calcium channel blocker and a beta-blocker. Dilation of renal arterial stenosis by percutaneous transluminal angioplasty was performed, however, systolic BP remained in the range of 180 to 160 mmHg. The dose of PSL was gradually decreased according to reduction of the levels of ESR.

In January 1988, the patient suddenly developed chest pain which was disclosed as acute subendocardial infarction. At that time ESR showed a marked elevation with the maintenance dose of PSL of 15 mg daily. After the episode of acute subendocardial infarction, the level of serum creatinine began to raise. Due to continuous elevation of ESR, the dose of PSL was increased to 30 mg daily. In August 1988, second attack of acute subendocardial infarction occurred. Consequently cardiac function deteriorated and the patient died on September 4th, 1988. Autopsy was performed with the permission of his family.

The autopsy revealed severe atherosclerosis over the aorta, its branches and cerebral arteries. The proximal segments of bilateral subclavian arteries, common carotid arteries, superior mesenteric artery, right renal artery, and bilateral internal and external iliac arteries were severely (90-95%) stenotic. An aneurysm (2.0 cm × 1.5 cm) at the proximal region of the left subclavian artery was found. The heart was hypertrophic and weighed 439 g. Subendocardial fibrosis was found in the lateral wall. Both kidneys were atrophic. Light microscopic examination revealed severe atherosclerotic changes with calcification of the whole aorta and its branches. In addition, in the proximal region of brachiocephalic artery, bilateral subclavian arteries, common carotid arteries, and internal and external carotid arteries, these changes were accompanied by considerable splitting and disappearance of medial elastic fibers, particularly those of the outer media, fibrosis of the outer media and the adventitia; there was non-specific chronic inflammatory cellular infiltration in the damaged regions. In the aorta, both atherosclerotic and inflammatory changes were found from the orifice of the left subclavian artery to the level of bilateral renal arteries (Fig. 2). The other region of the aorta exclusively showed atherosclerotic change (Fig. 3). Subendocardial necrosis and fibrosis in the septum and the lateral wall of the heart were evidence of recent myocardial infarction. Atherosclerosis was seen in the examined coronary arteries but changes due to inflammatory process were not observed. In the kidneys, remarkable arteriosclerosis progressed to the peripheral arterioles. Glomerular sclerosis and the tubulo-interstitial change were also noted without inflammatory reaction.
DISCUSSION

The clinicopathological observation of the present patient illustrates the problems encountered in treating elderly patients with vasculitis and atherosclerosis. Recently, as the prognosis of the patients with systemic lupus erythematosus (SLE) has been improved with the introduction of PSL, the association of collagen-vascular disease and atherosclerosis has been intensively focused upon (15–19). The atherosclerotic lesion found in SLE is thought to be basically dependent on the adverse effects of PSL, alteration of both fat metabolism and coagulation-fibrinolytic system and hypertension (15–18, 20). In Takayasu’s arteritis, the coexistence of arteritis and atherosclerosis has been reported (1, 4–14, 21) and some of these cases showed remarkable atherosclerotic lesions in the coronary arteries as well as in other vessels (6, 7, 12–14). However, in some cases the patients were relatively young and a definite conclusion that PSL was the cause of atherosclerotic lesions could not be determined (12–14). This fact suggests that the participation of PSL in the atherogenesis in patients with Takayasu’s arteritis does not always occur. Although the true mechanism of the direct effects of the arteritis on atherogenesis is still unknown, several hypotheses have been proposed (22–25). It is therefore possible that the atherosclerotic lesions of the present patient, which could have been established before Takayasu’s arteritis developed, were accelerated by the arteritis itself and the adverse effects of PSL which was administered to control the arteritis. In the present case, a relatively large dose of PSL was administered to control the symptoms which were initially thought to be the outcome of the activity of the arteritis. Eventually, despite administration of PSL, the symptoms, particularly those related to the ischemia, were not ameliorated.

Our case demonstrates that Takayasu’s arteritis can occur in elderly patients with advanced atherosclerotic lesions. When this coexistence is found, it is critical to be very careful with the usage of PSL in the treatment regimen.

In conclusion, PSL is useful in the management of patients with vasculitis, but such therapy may lead to complications which could be almost as serious and difficult to control as the underlying disease.
itself. The determination of the proper use of PSL in the management of vasculitis in the elderly is an important problem which requires further consideration.

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