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A Case of Miliary Tuberculosis Developed After an Urgent Operation of a Rupture of an Abdominal Aortic Aneurysm

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We report a case of miliary tuberculosis developed after an urgent operation of a rupture of an abdominal aortic aneurysm. We could not detect a pulmonary lesion except old pleurisy on the chest radiological examination from the abdominal aortic aneurysm operation. As tuberculous aortic lesion is very rare at present, we must pay attention to the possibility of a tuberculous aortic lesion even if we do not detect any risk factors.

Key words: miliary tuberculosis, abdominal aortic aneurysm

INTRODUCTION

A tuberculous aneurysm of the aorta is exceedingly rare. However, as this disease is very serious, we need to diagnose it correctly in the early stages and treat these patients appropriately. The highest survival rates have occurred with combined medication and prompt surgical intervention. We report a case of miliary tuberculosis which developed after an urgent operation of an abdominal aortic aneurysm without further medical treatment.

CASE REPORT

On August 24, 2004, an 83-year-old man presented with a 15-day history of anorexia, high grade fever and slight cough. He was referred by a local doctor for further examination of an abnormal shadow on a chest imaging study. The patient had previously been in another hospital with a rupture of an abdominal aortic aneurysm (Figure 1a), and had received an urgent operation of replacement of an artificial blood vessel on 26 May, 2004. A chest radiograph on that admission had shown no abnormal shadow except left old pleurisy (Figure 1b). Although the details were not known, he had suffered from left tuberculous pleurisy when he was 19-year-old. The specimen of the resected aortic lesion had shown caseous necrosis with epithelioid histiocytes and Langhans giant cells,

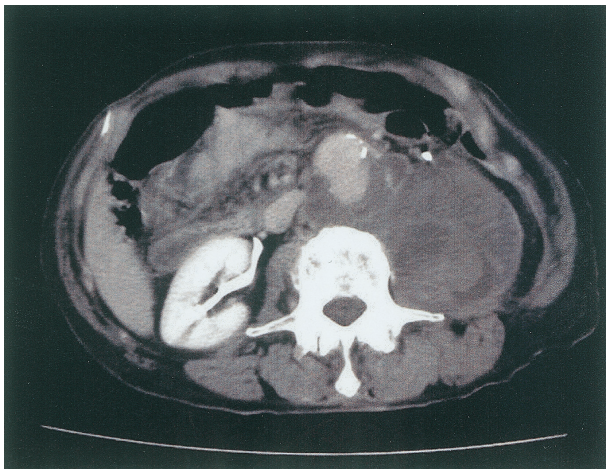

Fig. 1a. Abdominal CT in another hospital admission showing a rupture of an abdominal aortic aneurysm.

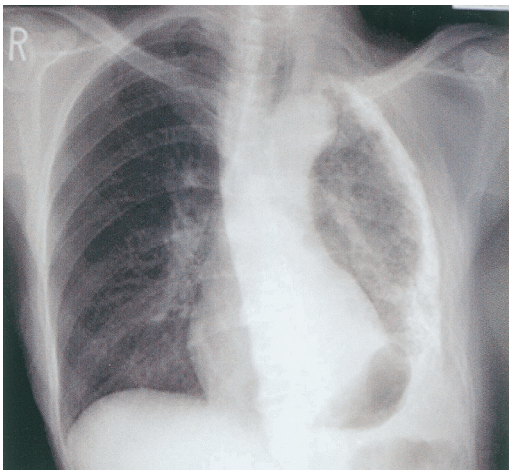

Fig. 1b. Chest radiograph in May 2004, showing left old.

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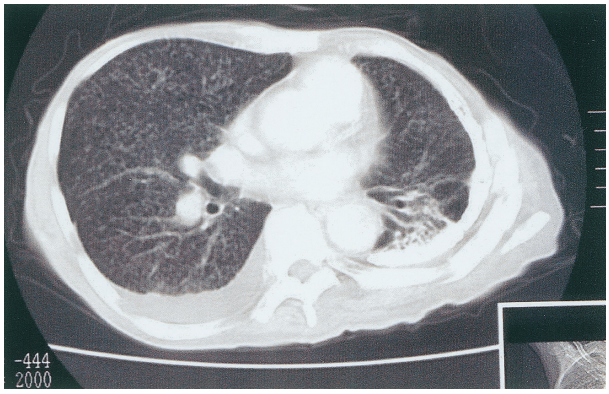

Fig. 1c. Chest CT in August 2004, showing a diffuse miliary nodular shadow.

and the lesion was suspected to be due to *Mycobacterium (M.) tuberculosis* (Figure 2a,b). However, the doctor had not treated it with antituberculous drugs.

On examination at admission in our hospital, the patient was thin with a blood pressure of 136/86 mmHg, pulse 80/min, respiratory rate 14/min, and temperature 36.0 ℃. On admission the white blood cell count was 4,500/mm^3. Serum C-reactive protein was elevated (7.96 mg/dl). The serum total protein level was decreased to 5.5 g/dl. Urinalysis was negative for protein, occult blood, sugar, cells and casts. Sputum culture yielded *M. tuberculosis*, although sputum smear and PCR was negative. Chest CT scanning revealed a diffuse miliary nodular shadow with left old pleurisy (Figure 1c). He was treated with rifampicin, isoniazid, ethambutol and pyrazinamide in our hospital admission. A low grade fever continued for 6 days and then disappeared. After 3 weeks of the

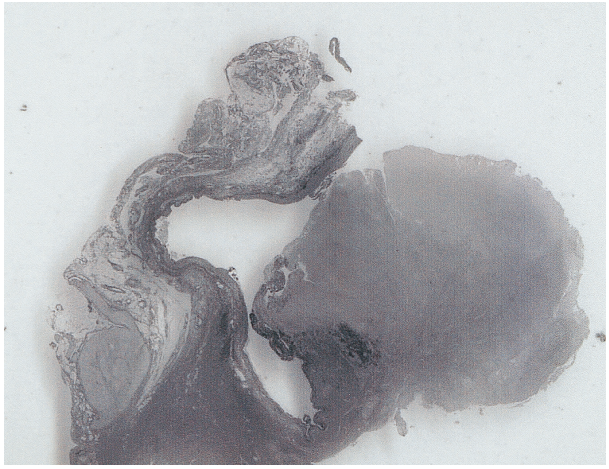

Fig. 2a. Macroscopic view of the ruptured abdominal aneurysm shows a destruction of internal elastic lamella.

Fig. 2b. The specimen of the resected aortic lesion shows caseous necrosis with epithelioid histiocytes and Langhans giant cells.

treatment, the patient complained of an erythematous skin lesion in his bust on 10 August. The lymphocyte stimulation tests by drug for these antituberculous drugs were negative. However, as we could not perfectly deny adverse drug reactions, we stopped all antituberculous drugs. After the skin lesion improved, we started rifampicin and isoniazid from 25 mg each with a full dose of ethambutol, and gradually increased this every three days to the full dose. We stopped pyrazinamide after the skin lesion episode. He was treated with full dose of these three drugs from 23 September. After then he had no complaints and good progress.

DISCUSSION

A tuberculous aneurysm of the aorta is exceedingly rare and since its first description by Kamen (1) in 1895, until December 1999, only 88 such cases could be traced in the literature (2). Before 1950, most of the reported cases were from autopsies; in later years, antemortem diagnosis and successful treatment became more frequent (2). The high risk of sudden rupture in the absence of appropriate treatment in the past has proven uniformly fatal (3). Erosion of the aortic wall by a contiguous focus occurred in 75 % of the cases (adenopathy in 63 %), and in 25 %, direct seeding of the intima, adventitia, or media was the mode of entry. Disseminated tuberculosis was present in 46 % of patients. Most aneurysms were saccular and false and equally distributed between the

thoracic and abdominal aortas (3). In our case, the peritoneum surrounding the ruptured aorta showed a chronic granulomatous lesion with clots. The aneurysm was saccular and false. The majority of aortic aneurysms are arteriosclerotic in origin, although mycotic aneurysms, as described by Osler in 1885 (4), are not rare (5). Tuberculous aneurysms of the aorta, however, are rare types of mycotic aneurysms and surgical resection is exceedingly uncommon (5). The term mycotic aneurysm is a misnomer that has nevertheless been generally adopted to describe aneurysms that occur secondary to the infectious destruction of an arterial wall. Because the appearance of the lesion resembled a fungal growth, Osler used the term mycotic aneurysm (6). There is considerable evidence to indicate that the miliary dissemination is often the result of the mycotic aneurysm, rather than its cause (5). However, in this case we could not detect a pulmonary lesion except old pleurisy on the chest radiological examination from the abdominal aortic aneurysm operation. Therefore, *M. tuberculosis* in the aortic lesion or old pleuritic lesion might be disseminated to the whole body after operation.

Tubercle bacilli may reach the aortic wall in one of three ways (6): 1. The bacilli may implant directly on the internal surface of the vessel wall. 2. The bacilli may be carried to the adventitia or media by the vasa vasorum. 3. Involvement of the vessel wall may occur by direct extension (or indirectly via the lymphatics (7)) from a contiguous focus such as a lymph node or paraspinal abscess. We suspect the third etiology in this case, because the specimen of the peritoneum surrounding the ruptured aorta had shown a granuloma due to *M. tuberculosis*. And the aortic wall was invaded by *M. tuberculosis* from the adventitia.

The patient had a past history of pleurisy tuberculosis without effective drug treatment. Therefore, he might have had a latent tuberculous lesion in the peritoneum. The highest survival rates of the rupture of aortic aneurysm (87 %) occurred with combined medication and prompt surgical intervention. There was no survival with a single intervention or no therapy (6). As tuberculous aortic lesion is very rare at present, we must pay attention to the possibility of a tuberculous aortic lesion even if we do not detect any risk factors.

Many tuberculous cases in Japan as in other industrialized countries, are thought to be the endogenous relapse. This case also seems to be endogenous relapse from old tuberculous pleurisy. It is necessary to fully treat the patient with antituberculous drugs, if there is some possibility of the exacerbation of the tuberculous lesion, since these old people did not receive the sufficient tuberculous drugs in their primary tuberculous infection.

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