

Rupture of a Pseudo Aneurysm of the Abdominal Aorta in a Patient with Human Immunodeficiency Virus Infection

Takashi Ando, MD,¹ Haruo Makuuchi, MD,¹ Yosuke Kitanaka, MD,¹
and Hiroataka Koizumi, MD²

Human immunodeficiency virus (HIV) infection has an impact on all systems of the body, including the cardiovascular system. A 54-year-old man presented with abdominal pain. Enhanced computed tomography revealed rupture of a pseudoaneurysm of the abdominal aorta. After surgery, the patient tested positive for HIV. Histological examination of the resected aorta showed leukocytoclastic vasculitis, a characteristic feature of HIV-related vasculitis.

Key words: human immunodeficiency virus, leukocytoclastic vasculitis, pseudoaneurysm

Introduction

Vasculitis is a serious, though uncommon, manifestation of Human Immunodeficiency Virus (HIV) infection occurring in medium to small visceral arteries,¹ which rarely involves large vessels.² We present a surgical case of a ruptured pseudoaneurysm of the abdominal aorta in a patient with HIV infection.

Case Report

A 54-year-old man with acute abdominal pain lasting several hours was transferred by ambulance to St. Marianna University hospital. The patient reported a general malaise and 10-kg weight loss over six months. On admission, his body temperature was 37.4°C, blood pres-

sure was 100/70, and heart rate was 100 beats/min with a regular rhythm. He had a pulsatile mass in the middle of the abdomen. The physical examination was otherwise unremarkable. Hematological data indicated both anemia and a rise in an inflammatory reaction: WBC, 7,600/mm³; Hb, 11.7 g/dl; ESR, 89 mm/h; CRP, 10.4 mg/dl. Biochemical tests of the liver and renal function were within normal limits. Chest and abdominal X-rays showed no abnormal findings. Enhanced computed tomography (CT) showed a pseudoaneurysm (maximum diameter 45 mm) in the abdominal aorta, indicating an occurrence of rupture (**Fig. 1a** and **1b**).

He subsequently underwent an emergency laparotomy. We suspected an inflammatory lesion around the abdominal aorta, given that the paraaortic lymph nodes were markedly enlarged with strong adhesion to the surrounding tissues. The pseudoaneurysm was replaced with a 14-mm straight Hemashield Gold Graft (Boston Scientific, TM). A 3-0 polypropylene suture was used to perform anastomosis in a continuous fashion. To prevent infective inflammation, we wrapped the tube graft with an omental pedicle. Histological examination of the resected aorta revealed not only atherosclerotic changes but also the presence of leukocytoclastic vasculitis, indicating severe inflammation in the media and adventitia. High-power view demonstrated features of leukocytoclastic vasculitis; namely, vascular damage with neutrophilic infiltrate, eosinophilic fibrinoid change in the

¹Department of Cardiovascular Surgery, St. Marianna University School of Medicine, Kawasaki, Kanagawa, Japan

²Department of Diagnostic Pathology, St. Marianna University School of Medicine, Kawasaki, Kanagawa, Japan

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Corresponding author: Takashi Ando, MD. Department of Cardiovascular Surgery, St. Marianna University School of Medicine, 2-16-1, Sugao, Miyamae-ku, Kawasaki, Kanagawa 216-8511, Japan

Email: taka@marianna-u.ac.jp

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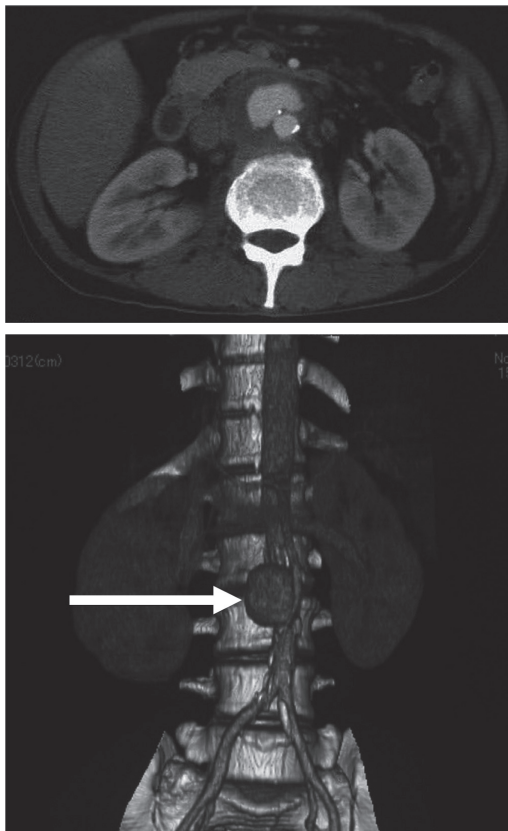


Fig. 1 **a:** Enhanced CT scan of a pseudoaneurysm in the infra renal abdominal aorta, indicating the occurrence of rupture. **b:** 3-D image. Site of excised aneurysmal wall in the pathological specimen (white arrow). CT, computed tomography

vessel wall, and scattered nuclear debris. Such features are characteristics of viral vasculitis (**Fig. 2a** and **2b**).

The patient tested positive for HIV during routine screening, though he had no memory of how he might have become infected. He showed no signs of opportunistic infection (**Table 1**). His CD4 count was $23/\text{mm}^3$; HIV load, 6.9×10^5 RNA copies/ml (**Table 2**). We then started highly active anti-retroviral treatment (HAART), consisting of triple drugs, and also administered four kinds of germicidal drugs (**Table 3**). After two weeks of HAART administration, CD4 count recovered to $62/\text{mm}^3$ (**Table 2**). On postoperative day 14, the patient showed no complications and was discharged.

Three days later, however, he was re-admitted to the hospital with a complaint of abdominal distention due to chylous ascites. Four weeks of intravenous hyperalimentation and no oral intake resolved the lymphorrhea. There was no recurrence of abdominal distention. Fur-

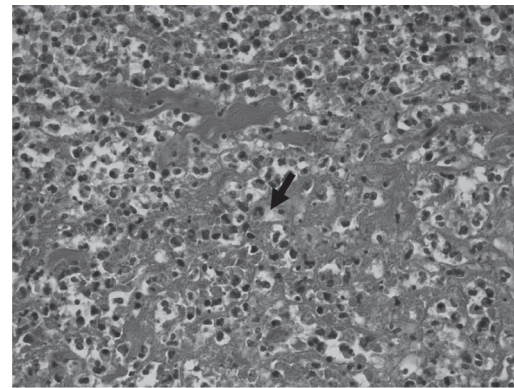
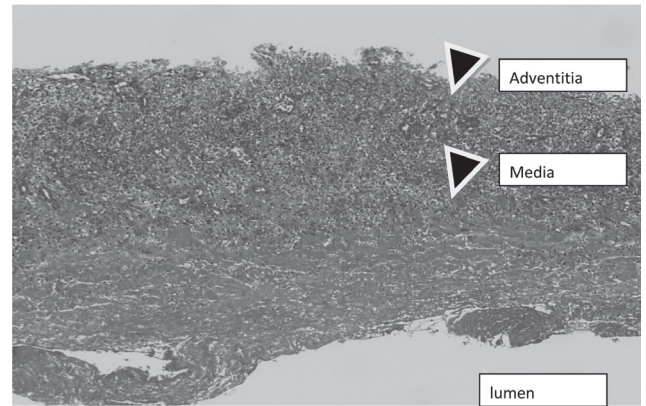


Fig. 2 Histological findings of the pseudoaneurysm. Inflammation in the media and adventitia areas of the aneurysmal wall. (**a**: $\times 80$: Hematoxylin-*es*osin staining) (black arrow).

High-power view of leukocytoclastic vasculitis. Vascular damage with neutrophilic infiltrate, eosinophilic fibrinoid change in the vessel wall (black arrow), and scattered nuclear debris (**b**: $\times 650$: Hematoxylin-*es*osin staining).

thermore, 2 more months of HAART therapy, most likely, dramatically decreased the HIV viral load to $< 4.0 \times 10^2$, though the CD4 count remained at $57/\text{mm}^3$ (**Table 2**). The patient was finally discharged on the postoperative day 80. He visits the outpatient clinic for HAART therapy and has had no symptom of Acquired Immune Deficiency Syndrome (AIDS). There has been no recurrence of either pseudoaneurysm or chylous ascites for four years since the operation.

Discussion

Viruses can be responsible for various types of vasculitis such as polyarteritis nodosa or hypersensitivity vasculitis. HIV-related large vessel vasculitis preferentially

Table 1

HBsAg	negative
HCAb	negative
TPHA	negative
Tb	negative
MAC	negative
VZV	negative
CMV	negative
EBV	negative

Table 2

	Admission	2 weeks later	Discharge
CD4 count (/mm ³)	23	62	57
Viral load (copies/ml)	6.9×10^5	No data	$<4 \times 10^2$

Table 3

HAART	zidovudine	300 mg/day (orally)
	nelfinavir mesilate	2250 mg/day (orally)
	lamivudine	300 mg/day (orally)
Antibiotics	cefazopran hydrochloride	2 g/day (drip infusion)
	azithromycin	1200 mg/week (orally)
	sulfamethoxazole	1600 mg/day (orally)
	trimethoprin	320 mg/day (orally)

HAART, highly active anti-retroviral treatment

affects small and medium-sized extracranial arteries. It also affects large arteries in approximately 10% of cases. However, its exact etiology remains unknown.^{3, 4)} The rarity of involvement of the aorta in AIDS patients was reported by Marks and Kuskov,¹⁾ who showed atypical aneurysms in 16 young adult patients of which 12 were HIV-infected. Javed⁵⁾ reported giant cell aortitis in a HIV-positive case. Chetty⁶⁾ showed leukocytoclastic vasculitis of vasa vasorum in their analysis of HIV-related vasculitis that was characterized by neutrophils and nuclear debris. Plasma cells and lymphocytes were present in the surrounding adventitial tissue. Eosinophils are thought to play a crucial role in hypersensitivity. The present case also showed arteritis in the aneurysmal wall (**Fig. 2a** and **2b**). The pathological findings lead us to suggest that an inflammatory response against HIV infection may develop pseudoaneurysm formation.

The patient had chylous ascites, an extremely rare complication of aortic surgery, which seems to be associated with mycobacterium infection and Kaposi's sarcoma in patients with AIDS.⁷⁾ In the case reported here, there was difficulty in mobilizing the aorta with tape during surgery, which could have caused leaking of chyle. Furthermore, because HIV infection may disrupt the healing process of the aorta, we speculate that it was not only leakage but also an occluded chyle duct that were respon-

sible for the chylous ascites.

In conclusion, we demonstrated the successful repair of a pseudoaneurysm in a patient with HIV infection.

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