Case Report

Tuberculous abdominal aortic pseudoaneurysm with renal and vertebral tuberculosis: a case and literature review

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Abstract

Tuberculous pseudoaneurysm of the aorta is rare and exposes patients to a very high risk of unpredictable rupture. To our best knowledge, only 32 cases have been reported related to all arterial systems from 1993 to 2013 in the literature. We report a 44-year-old male who presented with an aortic pseudoaneurysm and tuberculosis of the kidney and vertebrae. He underwent endovascular repair and antibiotic therapy for tuberculosis, combined with a bare stent implanted to seal endoleaks after endograft stenting. The postoperative course was uneventful and the patient recovered and lived well afterwards. Epidemiology, pathogenesis, presentation, management, and mortality of this entity were reviewed and discussed.

Key words: pseudoaneurysm; tuberculosis; infection; treatment.

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Case Report

A 44-year-old man who was admitted for gross hematuria and lower back pain with radiation down to both legs for six months, without accompanying fever, night sweat, or weight loss. He was not a smoker and had no history of angina, hypertension, dyslipidemia, ischemic stroke, or chronic renal failure. Vertebral body biopsy revealed acid-fast bacilli with bone destruction and granulomatous formation (Figure 1),and there was no bacteria by general bacterial culture of pus with aerobic and anaerobic conditions. Urine culture was positive for tuberculosis without other bacteria. These findings well supported the diagnosis of tuberculosis of the lumbar vertebrae and right kidney. Ultrasound showed a 39×38 mm low-echo area of the right kidney, and a 22×15 mm nodule adjacent to abdominal aorta (Figure 2A, 2B). A computed tomography angiography (CTA) scan simultaneously revealed a right renal abscess (Figure 2C) and a pseudoaneurysm sized 20×12×20 mm, which involved the posterior wall of the infrarenal abdominal aorta (Figure 2D-F). In addition, a left psoas abscess (Figure 2D) with vertebral bodies L3-L5 deteriorated (Figure 2F) was found.

After a six-month anti-tuberculosis treatment with isoniazide (0.3g per day), rifampin (0.45g per day), ethambutol (0.75g per day), and pyrazinamide (0.5g per day), the case was referred to us for digital angiographic evaluation, which revealed a saccular pseudoaneurysm of the infrarenal abdominal aorta. Therefore, endovascular abdominal aortic aneurysm repair (EVAR) was performed by implanting a straight (80 mm long and 20 mm diameter) endograft (Endurant Stent Graft, Medtronic, CA, USA), but endoleaks of the left posterior were observed after implantation. Another 80 mm long and 16 mm diameter bare stent (Sinus-XL Stent, Optimed, Ettlingen, Germany) was implanted and the endoleaks were sealed.

The patient was discharged on day 5 following interventional therapy and was instructed to take anti-tuberculosis therapy with isoniazide, rifampin, ethambutol, and pyrazinamide at the same dose for 12 months; he would afterwards require an operation for posterior stabilization of the lumbar vertebrae. On the six-month follow-up, the patient was without lumbar pain or any complication. CTA scan follow-up with contrast was performed at six months (Figure 3A-C), displaying a marked reduction of the pseudoaneurysm.

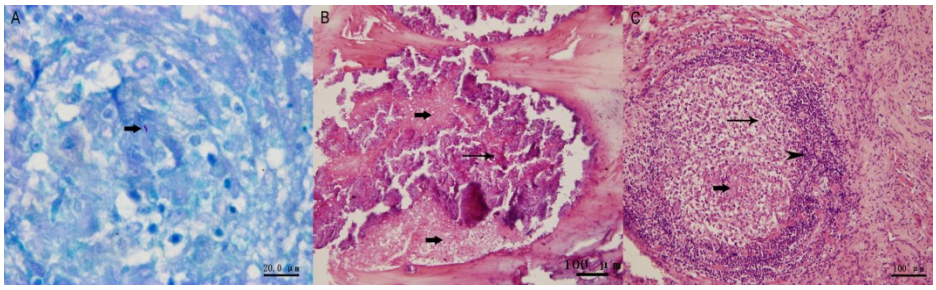

Figure 1. Pathological changes in lesion. **A:** Acid-fast bacilli (thick arrow) were detected in the vertebral body from the granulomatous lesion (acidfaststain, original magnification 100×); **B:**Histopathological slide of vertebral body revealed bone destruction (thin arrow), and caseation necrosis (thick arrows); **C:** Multiple cell granulomas in the lymph node. From center out: pink section – necrosis of initially formed (thick arrow); white section – epithelioidcells (thin arrow); purple section – lymphocyte (arrow head) (hematoxyline-eosin stain, original magnification 20×).

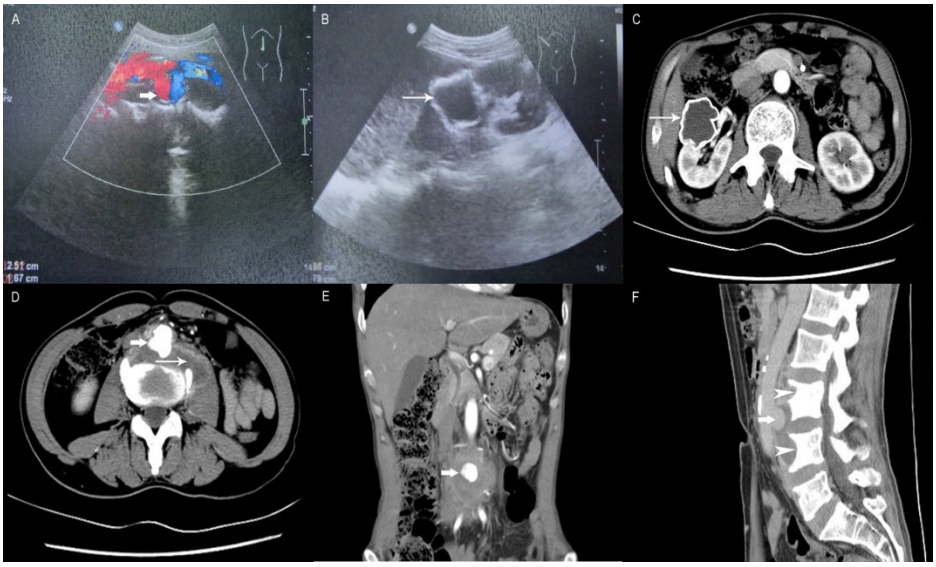

Figure 2. Imaging before treatment. **A, B:**Abdomenultrasound showed two low-echo areas of abdominal aorta (thick arrow) and the right renal (thin arrow); **C:** Abdominal and pelvic CTA revealed a low-density area measuring 24×30×25-mm involving the right kidney (thin arrow); **D, E, F:**Three-dimensionalreconstruction CTA revealed a pseudoaneurysm measuring 20×12×20 mm involving the posterior wall of the infrarenal abdominal aorta (thick arrow) and a low-density area of the left psoas (thin arrow) with deteriorated vertebrae bodies (arrow head).

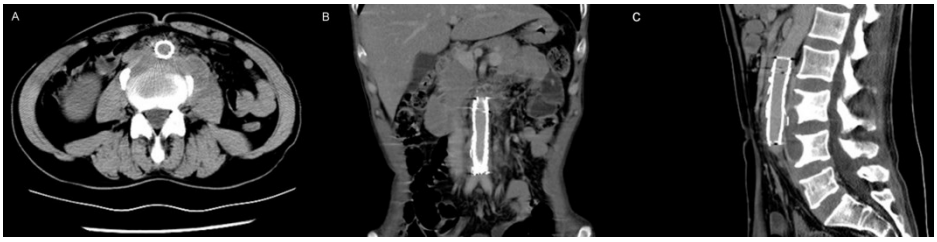

Figure 3. Imaging after treatment. A, B, C: Abdominal and pelvic CTA obtained six months after the initial CT examination, displaying both endo-graft and stent in the original position and marked reduction of the pseudoaneurysm.

Table 1. Review of literature abouttuberculouspseudoaneurysm and treatment

Year	Study	Age (years)/ Gender	Location (artery)	Follow-up (months)	Anti-TB treatment (months)		Pseudoaneurysm therapy	Comments
					Before operation	After operation		
1995	Cross *et al*. [6]	31/Male	Carotid A	2M	-	-	Endovascular stenting	
1996	Haginoet *al*. [7]	80/Male	Ab AA	6M	-	RIE/10M	Extra-anatomic bypass	Prostate cancer with radiotherapy
1996	Ikezawaet *al*. [8]	47/Female	Th AA	11M	RIES/7M	RE/4M	In-situ reconstruction	Pu TB
1998	Bojar et al. [9]	34/Male	Th AA	3M	RIEP/1M	-	In-situ reconstruction	Pu TB, HIV history
1999	Long *et al*. [10]	67/Male	Ab AA	-	RIPE/nearly 2M	Full course	In-situ reconstruction	-
		77/Female	Th AA	2M	RIPS/nearly 1M	Full course	In-situ reconstruction	-
1999	Goizarianet *al*. [11]	27/Female	Th AA	10M	-	RIP/10M	In-situ reconstruction	-
1999	Kao *et al*. [12]	88/Male	Femoral A	18M	-	RIE/12M	In-situ reconstruction	Ruptured femoral aneurysm
2000	Liu *et al*. [13]	42/Female	Ab AA	24M	RIP/2M	Anti-TB/7M	EVAR	-
		41/Male	Ab AA	18M	-	-	EVAR	Ruptured aneurysm
2000	Deshmukhet *al*. [14]	12/Male	Gluteal A	8M	-	RIPE/6M	Endovascular embolization	-
2001	Choudharyet *al*. [15]	5 patients	Th/Ab AA	18-36M	RIPE/2M and (or) RI/4M		In-situ reconstruction	Pu TB, pericardium TB
2004	Satokawaet *al*. [16]	77/Female	Celiac A	-		RI,LVFX/12M	Extra-anatomic bypass	Recurrence after reconstruction
2005	Kim *et al*. [17]	25/Male	Splenic A	-	-	-	Endo-embolization,gastrectomy	Gastric TB, Pu TB
2005	Shikataet *al*. [18]	76/Male	Ab AA	6M	-	RIPE/6M	In-situ reconstruction	Pu TB
2006	Corby *et al*. [19]	12/Female	Mesenteric A	9M	-	RIPE/9M	Endo-embolization	Ruptured, intestinal TB
2006	Sirvanciet *al*. [20]	65/Male	Th AA	-	-	Anti-TB/-	In-situ reconstruction	Recurrence after reconstruction
2006	Bavunogluet *al*. [21]	16/Male	Mesenteric A	Died	-	-	Endovascular embolization	Intestinal TB
2006	Lecceseet *al*. [22]	69/Male	Femoral A	18M	-	RIP/12M	In-situ reconstruction	Pu TB, cutaneous TB
2007	Lohet *al*. [23]	63/Male	Th AA	6M	-	RIPE/6M	TEVAR	Stroke after EVAR
2007	Labrousseet *al*. [24]	68/Male	Th AA	12M	RIPE/2M;	RIPE/2M;RI/10M	TEVAR	Pu TB, renal cancer with nephrectomy
2008	Keeling *et al*. [25]	40/Male	Pu A	Died	RIP/0.5M	-	Endovascular embolization	Pu TB, Rasmussen aneurysm, cardiac arrest
2008	Stephen *et al*. [26]	2 patients	Carotid A	6M	-	Anti-TB/-	Stenting/in-situ reconstruction	-
2010	Bachmeyeret *al*. [27]	38/Male	Illiac A	1M	-	Anti-TB/6M	In-situ reconstruction	-
2012	Li *et al*. [28]	62/Male	Th AA	12M	RIPE/1M	RIPE/5M	TEVAR	Ruptured, Pott's disease
2012	Nakayama *et al*. [29]	84/Male	Th AA	-	No anti-TB	No anti-TB	TEVAR	Pu TB
2013	Villegas *et al*. [30]	72/Male	Ab AA	36M	-	Anti-TB/12M	EVAR	Pu TB

Th AA: thoracic aortic artery; Ab AA: abdominal aortic artery; Pu: pulmonary; R: rifampicin; I: isoniazid; P: pyrizinamide; E: ethambutol; S: streptomycin; (T)EVAR: (thoracic) endovascular aortic repair

Discussion

The etiology of aortic and peripheral aneurysms is the most clinically relevant classification system related to not only natural history, but also treatment. The main cause of aneurysm formation is medial degeneration, found in 80% of cases; dissection, which causes the aneurysm to form in response to excessive proteolytic enzyme activity and the increase of smooth muscle migration that diminishes the integrity of the arterial wall, is found in 17% of cases; finally, 3% of cases are inflammatory aneurysms such as Takayasu disease and aortitis [1]. In addition, approximately less than 1% of all aortic aneurysms [2] are associated with arterial infection, which often results in the formation of pseudoaneurysms that are differentiated from true ones specifically because they lack all three normal elements of the arterial wall, which are more often saccular than fusiform. *Staphylococcus* and *Salmonella* [1] are the major causes of primary aortic infection; tuberculosis is very rarely described. The original cause of non-tuberculous aneurysms are mainly spinal surgery and trauma, whereas pesudoaneurysms of tuberculosis are induced by the constant stimulation of surrounding tissue with tuberculosis infection. In this study, we reviewed 32 cases describing tuberculous pseudoaneurysm from 1993 to 2013 in 25 studies [Table 1].

The earliest report of a tuberculous aortic aneurysm was by Kamen in 1895 [3]. Yet the first attempt to repair a tuberculous aortic aneurysm was made in 1949, by Herndon and colleagues [4]. The patient died of a massive gastrointestinal hemorrhage on the sixth day after surgery. In 1959, Prophetis [5] reported a successful resection of such pseudoaneurysm in the thoracic aorta. But tuberculous pseudoaneurysms may occur anywhere along the arterial system via different infectious pathways of the arterial wall [6,7,10,12,16,17,19,25,27]. Different positions of tuberculous pseudoaneurysm have distinct names; tuberculous pulmonary pseudoaneurysm is called Rasmussen aneurysm [25], and aortic pseudoaneurysm is called Pott's disease [28]. It is therefore of great importance to know the different approaches through which tubercle bacilli reach the arterial wall: (i) the bacilli may implant directly on the internal surface of the vessel wall; (ii) the bacilli may be carried to the adventitia or media by the vasa vasorum; (iii) infection may reach the vessel wall by the lymphatics of the vasa vasorum; or (iv) the outside of the vessel wall may be affected by direct extension from a neighboring tuberculous lymph node, abscess, or bone. This last approach has been reported in the literature as the most common cause (75%) of infection [10]. In our case, the patient had deteriorated vertebrae bodies (L3-L5) and a left psoas abscess. Biopsy revealed caseation necrosis, acid-fast bacilli in the vertebrae bodies and their surroundings, and a granulomatous formation in the lymph node. These finding are completely in accordance with infection by direct extension from a neighboring tuberculous lymph node, abscess, or bone. Interestingly, all evidence in our case supports tuberculosis as a distinctive etiology with aortic pseudoaneurysm.

Because a tuberculous aortic pseudoaneurysm is exposed to a very high risk of unpredictable rupture with serious hemodynamic consequences and mortality (40%) [10], surgical resection with perioperative anti-tubercular therapy offers the only chance for the patient's survival. At present, surgery of choices include open surgery (in situ reconstruction [8-11] or extra-anatomic bypass[7,16]) and interventional therapy (embolization[14,17,21,25], stenting[26], or endovascular aneurysm repair [13,23,24,28-30]) along with anti-tuberculosis therapy. The most common operation is resection of the infected arterial segment, debridement of the surrounding tissues, and revascularization of the lower extremities using grafts brought through uninfected tissues remote from the infected site (extra-anatomic bypass). However, treatment of such lesions involving the juxtarenal aorta or visceral aorta is much more complicated. In our case, the best options (aortic debridement or direct revascularization) were limited for open surgery because the lesion was located near the opening of renal arteries, and the patient needed secondary orthopedic surgery for vertebra fixation. In addition, a review of the literature showed that surgical mortality ranged from 14% to 20% [10,15], and that there was some surgical morbidity, such as recurrent tuberculous psedoaneurysm [16] and risk of graft infection [20]. Therefore, open surgery was not appropriate for this case. Percutaneous endovascular interventional therapy offers shorter hospitalization and convalescence periods, and reduces the morbidity and mortality associated with open surgery [23-26,28-30]. There are other options to resolve pseudoaneurysms than surgery, such as embolization, stenting or EVAR (TEVAR), or a combination of these treatments. But it must be assumed that the primary infection can be controlled with long-term antibiotics (pre-operation and post-operation) in these circumstances. In our case, we combined EVAR and stenting to avoid endoleaks; this resulted in no prosthesis infection during the six-month follow-up.

We also considered EVAR (TEVAR) a temporizing option to treat acute complications from infected aortic aneurysms, such as uncontrolled bleeding.

Despite the use of modern chemotherapy and imaging technology, this disastrous complication still occurs, which reinforces the need for early suspicion, diagnosis, surgical resection, and anti-tubercular therapy along with close postoperative follow-up to prevent recurrence. The entire body should be examined in order to make early diagnosis and to effectively treat this life-threatening infection.

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