



Penetrating Atherosclerotic Ulcers of the Abdominal Aorta: A Case Report and Review of the Literature

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Penetrating atherosclerotic ulcers (PAUs) of the aorta are defined as atherosclerotic lesions with aortic intima and media ulceration, which may lead to a complete rupture of the adventitial wall. The present article aimed to report an unusual case of a surgically treated patient with abdominal aorta PAU with an illustration of the key features and to review and analyze the existing literature data. PAUs typically develop in elderly and hypertensive patients and in patients with advanced atherosclerosis. Although originally described for the descending thoracic aorta, a similar clinicopathological entity also occurs in the abdominal aorta. Patients with symptoms of a PAU should be treated immediately if they are fit for surgery. Exceptive observation by imaging modalities is necessary in patients with asymptomatic small (<2 cm) PAU, with or without focal dissection.

Key Words: Penetrating atherosclerotic ulcer, Abdominal aorta, Acute aortic syndrome

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INTRODUCTION

A penetrating atherosclerotic ulcer (PAU) is an atherosclerotic lesion frequently observed in the descending thoracic aorta (DTA) and abdominal aorta (AA) in severe atherosclerotic patients. PAUs were first reported by Shennan in 1934 [1] and is currently included in the category of acute aortic syndrome (AAS) including overt aortic dissection (AD), PAU, and intramural hematoma (IMH) with a prevalence of 2% to 7% of AAS [2-4].

Although the exact pathogenesis is largely unknown, a PAU is thought to be formed secondary to aortic plaque rupture, which could cause an aortic intimal tear or aortic rupture. Several factors may be involved with the entrance-tear formation in AAS including the aortic root motion, the geometry of the aorta, wall stress, intrinsic aortic tissue abnormalities, and aortic plaque rupture [5-8].

PAUs are asymptomatic and confined to the intimal layer, and progressive penetration into the elastic lamina and the media may lead to a great spectrum of clinical conditions with a variety of behaviors. A PAU may be self-limited to the aortic intima due to the fibrotic component of the atheromatous plaque with the development of a small, circumscribed area of ulceration, while in other cases, the lesion may extend up to the outer aortic wall, causing focal AD. Moreover, one of the complications of PAUs is IMH formation that can result to pseudoaneurysm or saccular aneurysm (SA) with a risk of aortic rupture [4,9].

The natural history of PAUs remains elusive, and the optimal therapeutic regimen is controversial. While asymptomatic cases of PAU may be managed conservatively with close follow-up, endovascular repair is actually the gold standard for complicated PAUs of the DTA. At the level of the AA, a PAU is often managed by open repair. We present

a case of surgically treated PAU at the level of the infrarenal aorta. An extensive literature review (1974 to 2015) is conducted to discuss the management of PAUs of the AA.

CASE

A 69-year-old male patient was referred to vascular division complaining of sharp pain specifically located at the right lumbar area for the past 1 month. On physical examination, the patient had intact and symmetrical pulses in the upper and lower extremities and no signs of palpable abdominal mass. His personal medical history revealed arterial hypertension and hyperlipidemia. The patient had a 45 pack-year smoking history. The laboratory test revealed a slight increase in white blood cells (WBCs, 11,500/ μ L; normal range, 5,000/ μ L-10,000/ μ L) and C-reactive protein (CRP, 3 mg/dL; normal range, <0.5 mg/dL) and slight decrease in hemoglobin (Hb, 13 g/dL; normal range, 13.8-17.2 g/dL).

Contrast-enhanced computed tomography (CT) scan images from the abdominal and thoracic area revealed the presence of a SA in the anatomical region of the AA mimicking a windsock (Fig. 1) and a large IMH and aortic wall thickening and calcification, findings compatible with a PAU (Fig. 2, 3). Considering the relatively good health status of the patient, we decided to perform open surgery. The aortic repair consisted of the replacement of the diseased aortic segment by a tubular Dacron graft (Fig. 4). The patient had an uncomplicated postoperative course and was discharged from the hospital on the 5th postoperative day. According to the histopathological report, there was diffuse ulceration of the intima and thrombus underneath the ath-

erosclerotic intima and media layers (Fig. 5).

DISCUSSION

AAS was defined as a clinical entity by Vilacosta et al. [10] and Vilacosta and San Román [11], who described three different pathophysiological mechanisms presenting in a similar clinical manner. This disease occurs mainly in elderly patients, between the sixth and eighth decades of life. PAUs occur mainly in the DTA, and it is usually associated with

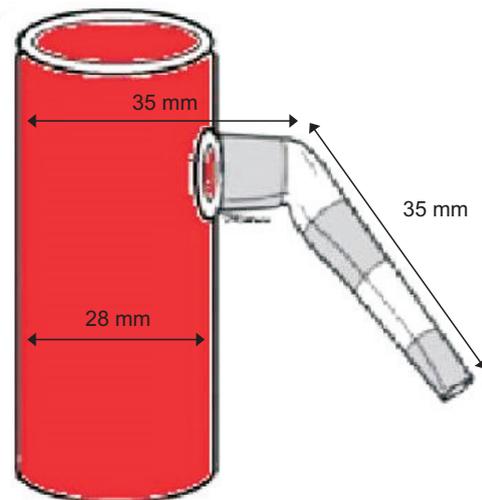


Fig. 1. The dimensions of the penetrating atherosclerotic ulcer mimicking a windsock as assessed by computed tomography imaging.

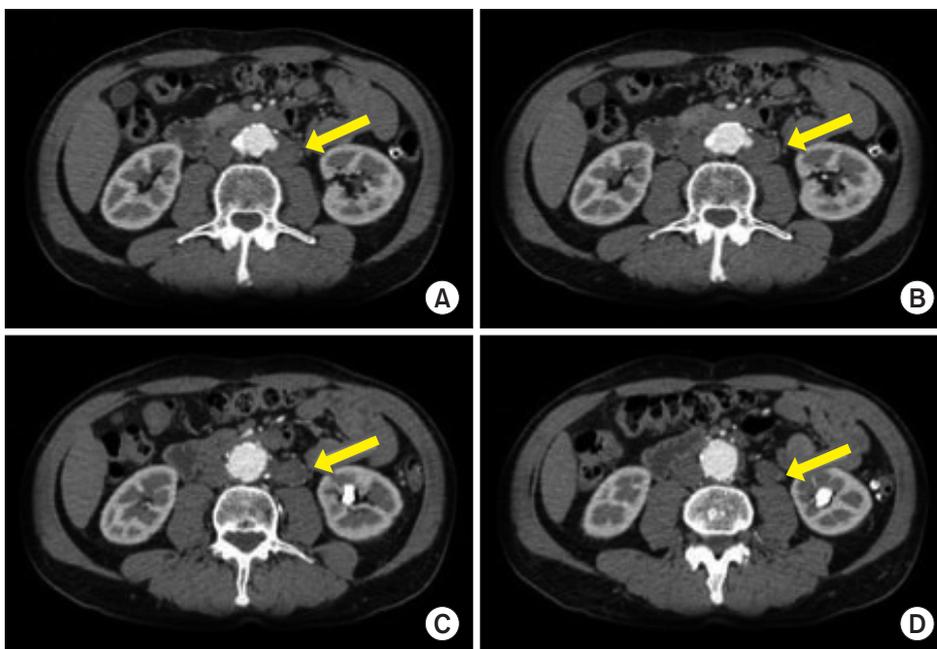


Fig. 2. (A-D) Contrast-enhanced computed tomography images of the abdominal and thoracic area revealed the presence of a saccular aneurysm (arrows) and a large intramural hematoma, findings compatible with a penetrating atherosclerotic ulcer.



Fig. 3. (A-C) Axial images as assessed by computed tomography imaging of the abdomen revealed the presence of a saccular aneurysm in the anatomical region of the abdominal aorta mimicking a windsock.

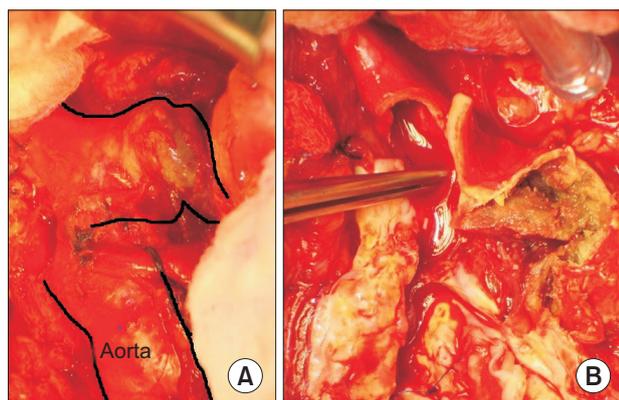


Fig. 4. (A, B) Intraoperative images of the penetrating atherosclerotic ulcer.

hypertension (approximately 94% of cases) and smoking habit. Most of these patients have other atherosclerotic disease manifestations, and half of these patients have a history of thoracic aortic aneurysm or abdominal aortic aneurysm. The clinical picture mimics the symptomatology of a classic AD, with anterior chest pain in the ulcers of the ascending aorta and interscapular or back pain in lesions located at the DTA. Unlike classical dissection, ischemia or embolization in the limbs or other organs is less common [12,13].

However, a PAU exhibits a greater risk of rupture [12-14]. On CT and magnetic resonance imaging, PAUs are characterized by a contrast-filled crater that communicates with the aortic lumen [15]. The usual description on computed tomography angiography (CTA) includes the measurement of the maximum aortic diameter at the ulcer site, the depth of the ulcer, and the length of the intimal defect (width) at the ulcer site, while the growth rate in abdominal PAU is about 3 mm/y [15,16]. A coexisting extra-aortic hematoma, which measures >20 mm in width or >10 mm in depth or which progresses into a total abdominal aortic diameter, all are considered complicated PAUs [9,15,16]. Some authors extend the definition of PAUs to ulcerative lesions not ex-

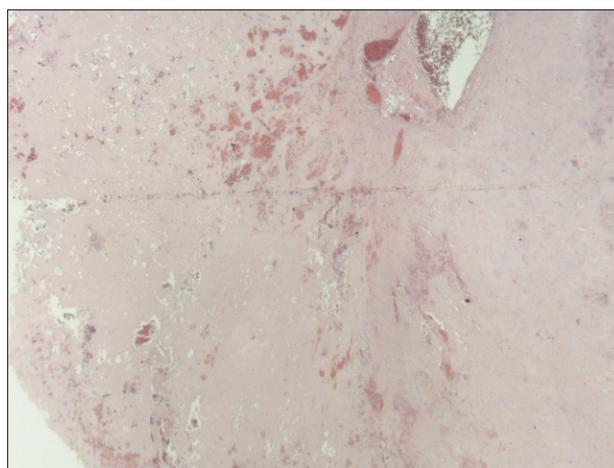


Fig. 5. Histopathological image revealed arterial wall with atherosclerotic lesions with diffuse ulceration of the intima and thrombus underneath the atherosclerotic intima and media layers (H&E, $\times 20$).

tending beyond the aortic wall [17,18].

Nevertheless, according to the Mayo Clinic Classification, the three radiological diagnostic criteria for PAUs on contrast-enhanced CT scans are a transmural rupture with extra-aortic hematoma, a well-defined ulcer crater in the aortic wall, and a subadventitial SA extending beyond the wall of the aorta [2]. Vorp et al. [19] reported the importance of the shape and symmetry, in addition to the size, in evaluating the risk of rupture.

Despite these efforts, there is still a paucity of predictors of the natural history and the behavior of PAUs, and no standard treatment has been established [13].

A PAU may progress into SA, focal AD, or aortic rupture. At the level of the AA, SA formation is observed more often than focal dissection. Hollier et al. [20] recommended surgical intervention for asymmetrical SAs, irrespective of their size, considering the risk of rupture. It has been recommended that treatment should be performed as early as possible in relatively young and fit patients.

Table 1. Cases and/or series of PAUs stratified according to the year of publication, number of patients, age, sex, risk factors, and treatment characteristics

Study (reference)	Number of patients	Mean age (y)	Sex (male/female)	Risk	Treatment
Lagaay [21]	3	58	3/0	HTN-CAD	ABI (2)-tube (1)
Cornud et al. [22]	1	53	1/0	CAD	Tube
Barbier et al. [23]	1	73	0/1	NA	ABI
Aschraft et al. [24]	1	60	0/1	HTN-PAD	Tube
Nora and Hollier [25]	1	56	1/0	HTN	Surgical interposition graft
Barba et al. [26]	1	37	1/0	NA	Surgical interposition graft
Rothwell and Lane [27]	1	72	1/0	HTN	Tube
Harris et al. [13]	10	74	10/0	HTN-CKD-CAD	Medical (3), surgical interposition graft (7)
Origuchi et al. [28]	4	59	3/1	HTN (1)	ABI (3)-tube (1)
Goldstein et al. [29]	1	67	1/0	HTN-CAD	Dacron aorto-iliac graft
Moriyama et al. [30]	1	74	1/0	HTN	Woven Dacron aorto-iliac graft
Taylor and Kalman [31]	4	73	4/0	HTN-CAD-PAD	Tube Dacron (2)-Dacron patch (2)
Hayashi et al. [32]	1	73	1/0	NA	Medical
Quint et al. [33]	1	NA			Medical
Toda et al. [34]	2	70	2/0	HTN-CAD-PAD	Surgical interposition graft
Farooq et al. [35]	2	68	2/0	HTN (2)-PAD	ABI (1)-tube (1)
Ganaha et al. [12]	31	71	15/16	HTN	Interposition stent graft
Tsuji et al. [17]	4	79.5	4/0	HTN (4)-CAD (2)	EVAR (4)
Vasquez et al. [36]	1	81	1/0	HTN (1)	Stent graft
Saiki et al. [37]	1	81	0/1	HTN (1)	Resection graft (AFB)
Feld et al. [38]	1	68	1/0	CKD	EVAR (1)
Minor et al. [39]	2	55	2/0	HTN-CAD-PAD	ABI (2)
Ventura et al. [40]	1	68	0/1	HTN (1)	ABI
Takagi et al. [41]	1	64	1/0	NA	Surgical interposition graft
Batt et al. [18]	8	70	7/1	HTN (4)-CAD (3)-PAD (6)	Surgical interposition graft (5) (tube 3-Dacron aorto-iliac graft 2)- EVAR (3)
Chernenkov et al. [42]	1	80	1/0	HTN	Tube
Tanigushi et al. [43]	1	82	0/1	CAD	Hemashield tube, left aortorenal bypass
Sensi et al. [44]	11	76.7	10/1	HTN (9)-CKD (1)-CAD (6)	ABI (4)-tube (1)- EVAR (6)
Eggebrecht et al. [45]	22	69.1	16/6	HTN (2)-CKD (9)-CAD (12)	EVAR (22)
Thalheimer et al. [46]	1	57	1/0	HTN-CAD-PAD	Tube (1)
Dalainas et al. [47]	2	68.6	2/0	HTN (2)-CKD (2)-CAD (5)	EVAR (2)
Piffaretti et al. [48]	13	73	12/1	HTN (13)-CKD (2)-CAD (2)-PAD (6)	EVAR (13)
Fyntanidou et al. [49]	4	67	4/0	HTN (4)-CKD (1)-CAD (1)	EVAR (4)
Lindblad et al. [50]	19	72	16/3	NA	EVAR (19)
Afifi et al. [51]	1	76	0/1	HTN-CAD-PAD	EVAR (1)
Hyhlik-Dürr et al. [52]	20	72	20/0	HTN (19)-CKD (4)-CAD (8)	EVAR (20)
Kutsukata et al. [53]	1	64	0/1	HTN	Dacron aorto-iliac graft (1)
Ferro et al. [54]	1	64	0/1	HTN-CAD	EVAR (1)
Kainuma et al. [55]	1	67	1/0	HTN	EVAR (1)
Nathan et al. [56]	20	NA			EVAR (10)-open repair (10)
Sadeghi-Azandaryani et al. [57]	1	76	1/0	HTN-CAD	EVAR (1)

Table 1. Continued

Study (reference)	Number of patients	Mean age (y)	Sex (male/female)	Risk	Treatment
Kazan et al. [58]	3	71	2/1	HTN (1)-CAD	EVAR (3)
Palombo et al. [59]	3	67	3/0	HTN (3)-CKD (2)-CAD (4)-PAD (2)	EVAR (3)
Papazoglou et al. [60]	1	69	1/0	HTN	EVAR
Georgiadis et al. [61]	19	70	18/1	HTN (18)-CKD (8)-CAD (11)-PAD (9)	EVAR (19)
Total	230	49/164 (29.9%) ^a			115/164 (70.1%) ^b

PAUs, penetrating atherosclerotic ulcers; HTN, hypertension; CAD, coronary artery disease; ABI, aortoiliac graft; NA, not available; PAD, peripheral arterial disease; CKD, chronic kidney disease; EVAR, endovascular aneurysm repair; AFB, axillary bifemoral bypass; IMH, intramural hematoma; SA, saccular aneurysm.

^aSurgical treatment, ^bendovascular repair.

Isolated dissection of the AA is rarely observed (0.4% to 2% of all ADs) [19]. The presence of a PAU may favor the development of focal abdominal AD. The onset of this dissection is often acute, with abdominal and low back pain, though some cases remain asymptomatic and are incidentally detected.

Fluid-structure interaction analysis concluded that low wall shear stress (lower than 1.5 N/m²) and high shear stress oscillation (oscillatory shear index=0.13) over the cardiac cycle in the PAU region could be considered key hemodynamic factors that result in the rupture of the PAU [20].

The differential diagnoses include inflammatory abdominal aortic aneurysm (IAAA) and inflammatory aortitis, such as Takayasu arteritis, giant cell arteritis, Behçet disease, Kawasaki disease, rheumatoid arthritis, sarcoidosis, Cogan syndrome, ankylosing spondylitis, systemic lupus erythematosus, and Wegener's granulomatosis.

An IAAA is defined by an unusually thickened aneurysm wall, shiny white peri-aneurysmal and retroperitoneal fibrosis, and dense adhesions of the adjacent intra-abdominal structures. The differential diagnoses are established by negative bacterial blood cultures, negative skin test, negative serological tests, the localization of the AA, and the typical anatomical features on CTA, while biopsy may be warranted to exclude malignancy. The patients with inflammatory aortitis must be prescribed with immunosuppressive therapy, which is based on the risk of rupture related to aneurysm dimension and the presence of secondary organ vascular insufficiency related to fibrotic stenosis, before and after the surgical intervention.

Invasive treatment (open surgery or endovascular stent grafting) is indicated in acute or symptomatic cases, while course observation using periodic evaluation by imaging techniques is recommended for asymptomatic or chronic cases of small (<2 cm) PAUs [20]. Open surgical repair with graft interposition is associated with higher operative morbidity and mortality due to the patient's poor general

condition and comorbidities. On the contrary, endovascular stent grafting is a less invasive procedure and is more appropriate for such frail patients. Since a PAU is a focal lesion, an adequate landing zone is almost present. Therefore, the risk of migration or endoleak is low, although the presence of IMH appears to delay the effectiveness of endovascular therapy. Furthermore, an open surgery may be preferred for younger, healthier patients in whom there is much less difference in operative risk between the two strategies and for patients whose long-term durability is important. Herein, we report a distinctive case of an open surgically treated patient with AA PAU, considering the parameters that support open surgical intervention.

1) Literature review

To identify clinical studies reporting cases and/or series of patients with a PAU, an electronic MEDLINE, Scopus, and Google Scholar literature search was performed from 1974 (first publication of penetrating aortic ulcer) to 2015 using the keywords "penetrating atherosclerotic ulcers," "abdominal aorta," and their combinations, and the search was summarized according to age, sex, risk factors, clinical manifestations, and treatment. Due to the extreme heterogeneity of the studies, no weighted estimations, comparisons, and correlations have been performed.

Literature review revealed mainly case reports of PAUs at the level of the AA and a rare series of greater than 10 cases. We collected 298 published cases of PAUs of the AA (Table 1) [12,13,17,18,21-61].

The median age of the patients was 72 years, and PAUs are almost dominated by males (90.3%). Hypertension (80%) and concomitant coronary or peripheral arterial occlusive disease (30.7%) are very common. Asymptomatic PAUs are incidentally detected in 40.3% of the reported cases. Most patients with a PAU of the AA complain of pain (43.8%). A minority (11.9%) of patients present with a hypovolemic

shock, secondary to the rupture of a complicated PAU. Most authors reported endovascular stent graft repair as the treatment of choice (62%), while conservative management was chosen only for 2.6% of the patients. Open surgical repair was performed in 35.4% of patients.

In conclusion, PAUs usually develop in the thoracic aorta but can similarly occur in the AA and may progress into focal AD, SA, or aortic rupture. Symptomatic PAU; large, profound (>2 cm) PAU; and complicated PAU of the AA (evolving to a SA or focal AD) require surgical treatment if the patient is fit for surgery. In this case, we performed an open surgery due to younger age and good general condition of the patient; the type of the graft or bifurcated tube depends on the PAU location and the condition of the arterial wall. The endovascular approach is a less invasive alternative to open surgery, with excellent short-term outcomes according to the recent literature. However, currently there are insufficient long-term studies for PAU.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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