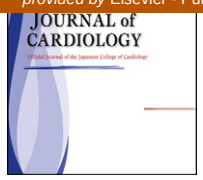
Journal of Cardiology (2012) 59, 123—131

SciVerse ScienceDirect

journal homepage: www.elsevier.com/locate/jjcc



Review

Infected aortic aneurysm and inflammatory aortic aneurysm—In search of an optimal differential diagnosis

Nobukazu Ishizaka (MD, FJCC)[a,*], Koichi Sohmiya (MD)[a], Masatoshi Miyamura (MD)[a], Tatsuya Umeda (MD)[a], Motomu Tsuji (MD)[b], Takahiro Katsumata (MD)[c], Tetsuro Miyata (MD)[d]

[a] *Department of Cardiology, Osaka Medical College, Osaka, Japan*
[b] *Department of Pathology, Osaka Medical College, Osaka, Japan*
[c] *Department of Cardiovascular Surgery, Osaka Medical College, Osaka, Japan*
[d] *Division of Vascular Surgery, Department of Surgery, University of Tokyo Graduate School of Medicine, Tokyo, Japan*

Received 20 September 2011; received in revised form 24 October 2011; accepted 28 October 2011
Available online 2 January 2012

KEYWORDS
Inflammatory aortic aneurysm;
Infected aortic aneurysm;
Differential diagnosis;
Biomarkers

Summary Infected aortic aneurysm and inflammatory aortic aneurysm each account for a minor fraction of the total incidence of aortic aneurysm and are associated with periaortic inflammation. Despite the similarity, infected aortic aneurysm generally shows a more rapid change in clinical condition, leading to a fatal outcome; in addition, delayed diagnosis and misuse of corticosteroid or immunosuppressing drugs may lead to uncontrolled growth of microorganisms. Therefore, it is mandatory that detection of aortic aneurysm is followed by accurate differential diagnosis. In general, infected aortic aneurysm appears usually as a saccular form aneurysm with nodularity, irregular configuration; however, the differential diagnosis may not be easy sometimes for the following reasons: (1) symptoms, such as abdominal and/or back pain and fever, and blood test abnormalities, such as elevated C-reactive protein and enhanced erythrocyte sedimentation rate, are common in infected aortic aneurysm, but they are not found infrequently in inflammatory aortic aneurysm; (2) some inflammatory aortic aneurysms are immunoglobulin (Ig) G4-related, but not all of them; (3) the prevalence of IgG4 positivity in infected aortic aneurysm has not been well investigated; (4) enhanced uptake of 18F-fluorodeoxyglucose (FDG) by 18F-FDG-positron emission tomography may not distinguish between inflammation mediated by autoimmunity and that mediated by microorganism infection. Here we discuss the characteristics of these two forms of aortic aneurysm and the points of which we have to be aware before reaching a final diagnosis.

* Corresponding author at: Department of Cardiology, Osaka Medical College, Takatsuki-shi, Daigaku-machi 2-7, Osaka 569-8686, Japan. Tel.: +81 72 683 1221; fax: +81 72 684 6598.
E-mail address: ishizaka@poh.osaka-med.ac.jp (N. Ishizaka).

0914-5087/$ — see front matter
doi:10.1016/j.jjcc.2011.10.006

Contents

Introduction

Along with greater life-expectancy, the prevalence of aortic aneurysm, a potently life-threatening disorder, is increasing because the aging process promotes aortic remodeling. A spread of imaging modalities, such as computed tomography (CT) and magnetic resonance imaging (MRI), may facilitate the discovery of aortic aneurysm, which may sometimes be asymptomatic, during a medical check-up [1,2]. On the one hand, inflammatory aortic aneurysm represents a specific subset of aortic aneurysm, which is thought to be caused by a pathogenic immuno-inflammatory process, occasionally immunoglobulin G4 (IgG4)-related [3], and corticosteroid and/or immunosuppressive therapies may be effective. On the other hand, for infected aortic aneurysm, another subset of aortic aneurysm, early and timely surgery with perioperative antimicrobial treatment is thought to be mandatory; otherwise, the outcome is in general not favorable. Differences exist between these two forms of aortic aneurysms; however, there are certain similarities in clinical symptoms, laboratory tests, and findings by imaging modalities, which may make differential diagnosis difficult. In this mini review, we discuss these two forms of aortic aneurysm, inflammatory and infected aortic aneurysms, both of which may be under-recognized, underdiagnosed, and sometimes misdiagnosed.

Infected aortic aneurysm and infected aortitis

Prevalence

Since first reported by Osler in 1885 [4], infected aortic aneurysm remains a life-threatening condition [5—7]. 'Infected' aortic aneurysm is sometimes alternatively termed 'mycotic' aortic aneurysm; however, only a minor fraction of this disease is actually caused by fungus [8]. Considering that the term 'mycotic aneurysm' was initially limited to the development of infected aneurysm secondary to infective endocarditis [9], this terminology may lead to slight confusion. The prevalence of infected aortic aneurysm may not be decreasing recently, and it is considered to comprise 0.7—2.6% of all cases of aortic aneurysm [10], although its true prevalence is unknown. Infected aneurysm may develop from a hematogenous spread of infection from microemboli to a preexisting aneurysm, the contiguous involvement of the vessel wall from an adjacent source of sepsis, or direct infectious inoculation of the vessel wall. Atherogenic risk factors, such as hypertension and diabetes, may be present as predisposing conditions [11]; however, infected aneurysm may commonly involve parts of the aorta that are not commonly involved by atherosclerotic aortic aneurysm [5]; about 70% of the infected aneurysms were found to be located in the thoracic and abdominal aorta at or above the renal arteries [12].

Clinical features, laboratory abnormalities, and imaging

The main presenting symptoms are fever and abdominal and severe back, abdominal, or thoracic pain, depending on the location of the aneurysm [7], although some patients with infected aneurysm may be asymptomatic [12]. Blood testing shows evidence of infection, such as leukocytosis, elevated C-reactive protein, and positive blood cultures. In addition to this clinical evidence of infection, the presence of periaortic soft tissue infiltration, as demonstrated by CT or magnetic resonance angiography (MRA) may lead to the diagnosis of infected aortic aneurysm [12]. Although ultrasonography may not be a reliable initial imaging modality for the diagnosis, detection of gas echoes in the aortic wall may lead to suspected infection of the aortic wall [13].

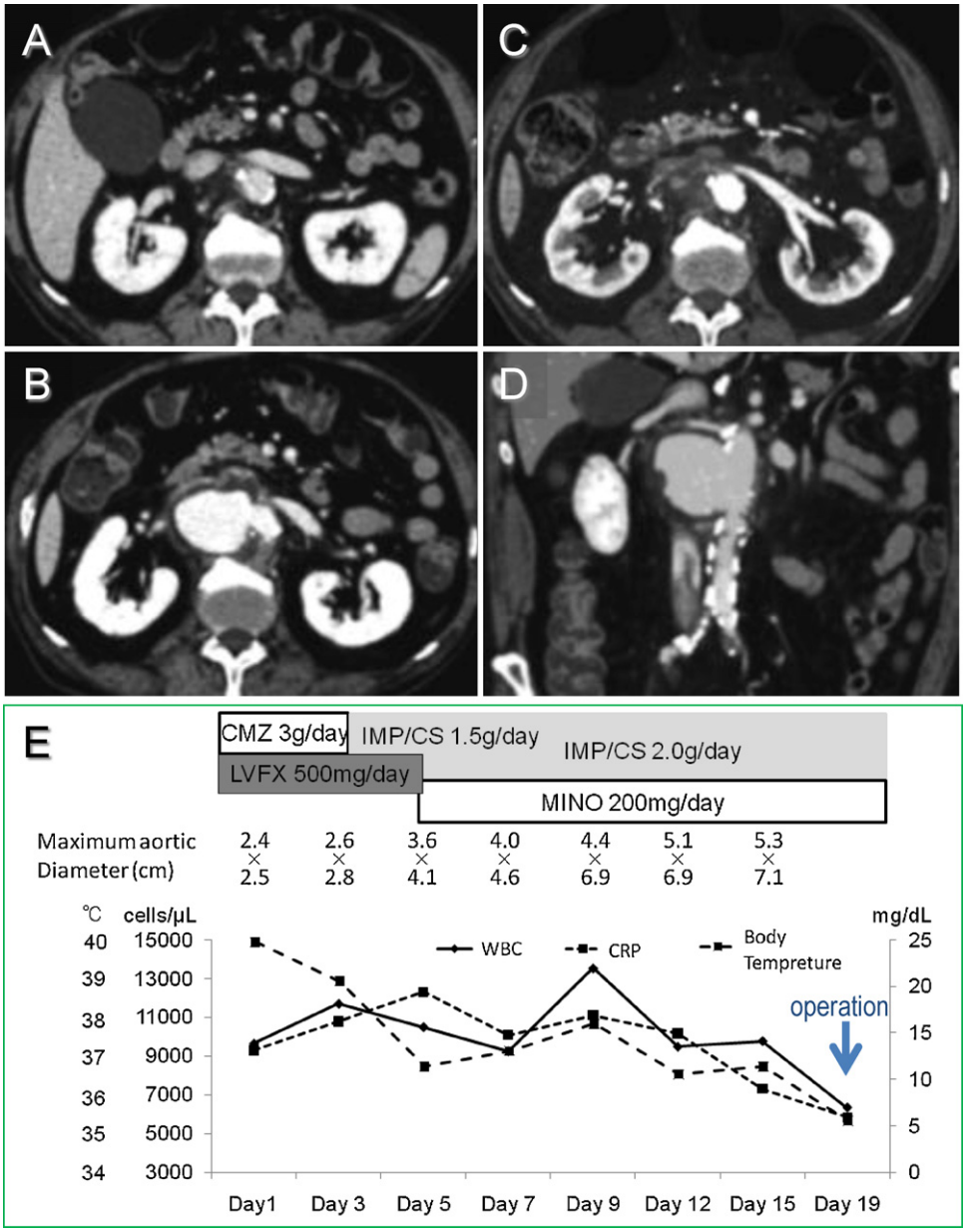


Figure 1 A 78-year-old man who had been diagnosed to have hypertension and hypertrophic cardiomyopathy was referred to our hospital because of high fever (40 °C) and abdominal pain. On day 5 of hospitalization, *Bacteroides thetaiotaomicron* was identified in blood culture. (A—C) Transverse sections of computed tomographic (CT) scanning images. (D) Coronal section of CT image. (A) On the day of admission. (B) 5th hospital day. (C and D) 12th hospital day. The diameter of the aortic aneurysm showed rapid dilatation. (E) Clinical course of the patient. CMZ, cefmetazole; LVFX, levofloxacin; IPM/CS, imipenem/cilastatin; MINO, minocycline; CRP, C-reactive protein; WBC, white blood cell.

Currently, multidetector CT angiography is the imaging modality of choice for the evaluation of suspected infected aneurysms [14]. Gallium scanning and 18F-fluorodeoxyglucose positron emission tomography (FDG-PET) may be used to evaluate disease activity [15]. Infected aortic aneurysm appears as a focal, contrast-enhancing dilatation, which may be multilobulated. A mantle sign-like appearance may be present in infected aortic aneurysm [16], suggesting that this sign is not exclusive to inflammatory aortic aneurysm or idiopathic retroperitoneal fibrosis.

The mortality in infected aortic aneurysm is higher than that in non-infected aortic aneurysm [5]. A bacterially infected aorta may appear to be within the normal-size range in the initial evaluation; however, dilatation of the aorta may progress rapidly over months [17] or even days [15] (Fig. 1), leading to rupture and death without appropriate diagnosis and treatment [7]. On the other hand, identification of infective microorganisms and clarification of their sensitivity to certain antibiotics provide crucial information for both the diagnosis and the choice of

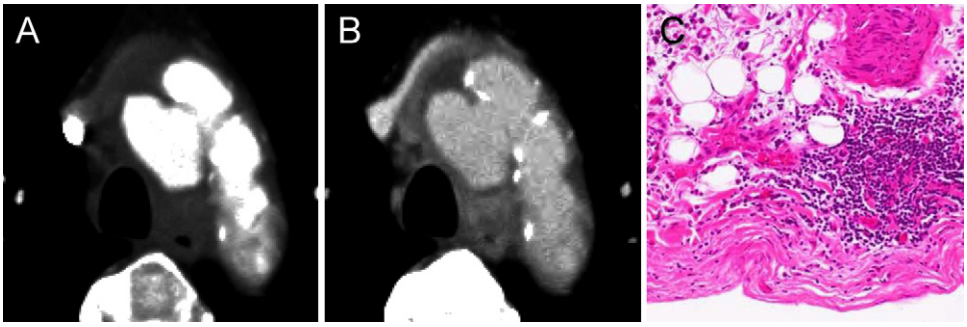


Figure 2 A 62-year-old man experienced chest discomfort and sustained high fever. The C-reactive protein level was elevated (3.68 mg/dL), but the IgG4 level was normal (29.6 mg/dL). Following 2 months of antimicrobial therapy, he was treated surgically. Computed tomography of arterial phase (A) and venous phase (B). Periaortic staining was apparent in the venous phase. (C) Hematoxylin-eosin staining of the aortic section. In the adventitia, there was an accumulation of lymphatic cells, which were mostly negative for IgG4 (data not shown). Original magnification, 200×. Despite repeated cultures, the causative microorganism could not be identified.

antimicrobials. Among the pathogens, *Salmonella*, *Staphylococcus*, *Campylobacter*, and *Streptococcus* species have been identified most frequently [18], but other microorganisms such as *Escherichia coli*, *Mycobacteria*, and *Bacteroides* species (Fig. 1) have been reported. The pathogens detected may show substantial differences according to the region [5,6,10,19], and period surveyed [20,21]. On the other hand, bacterial identification may not always be possible (Fig. 2), because of difficulties in culture or prior antibiotic treatment [10]. Bennett reported that blood cultures may be negative in as many as 47% of patients with infected aortic aneurysm in a paper published in 1967 [22]. In addition, Oderich et al. have reported that infective orgasms were identified in 33 (77%) of 43 patients [5]. Furthermore, Maeda et al. reported that the blood culture was positive only in 1individual among 11 patients with infected aortic or ileac artery aneurysm [20].

Therapy

Infected aortic aneurysm may show rapid growth in size, accompanied by a pseudoaneurysm or a perforated or penetrated aneurysm [5], therefore, diagnostic imaging may have to be repeated if the clinical suspicion persists [23]. Non- or delayed treatment of infected aneurysm often leads to a fatal outcome due to fulminant sepsis, aorto-enteric fistula formation, and rupture [14,24].

Medical management

Purely medical management may be often inadequate because of the possibility of persistent infection and subsequent aneurysm rupture. The conventional strategy for treatment is therefore prompt surgical treatment, followed by long-term suppressive antibiotic therapy. Intensive antibiotic therapy that is started perioperatively and continued for a prolonged period [7,25] is crucial for successful treatment. The required duration of antibiotics has not been well established, however, it commonly ranges from 6 to 8 weeks to lifelong treatment [7]. Although the persistence of infection greatly affects the perioperative outcome [26], whether calming down of the infection can be achieved safely may depend on the condition of each patient.

Surgical management

Survival is clearly dependent on the state of rupture. Thus, timely surgical treatment is mandatory for the management of infected aortic aneurysm; however, surgical risk and perioperative mortality are not negligible [7]. In addition, late postoperative death may occur due to existing comorbidities. Among 15 patients with infected aortitis, Luo et al. reported that, in addition to two perioperative deaths, there were two late deaths due to brain stem hemorrhage and to heart failure [27]. In addition, Moneta et al. reported that, in their series of 17 patients, the late death of 4 patients occurred at 1.3—6.3 years postoperatively, in addition to the perioperative death of 4 patients [28]. Furthermore, Müller et al. reported the outcome of 33 cases of infected aneurysm of either the aorta or the iliac arteriesaortic, among which 12 patients (36%) died perioperatively from cardiac, respiratory, or infectious reasons [7]. In Müller et al.'s series, survival was clearly dependent on the state of rupture. The mortality associated with extraanatomic reconstruction [20] after the primary infection was cured was reported to be 7% [29], and that associated with in situ graft placement was14—36% [7,14,30], although there has been no strict controlled trial. Whether endovascular aortic repair (EVAR) would be feasible for infected aortic aneurysm has been discussed in several previous papers [31]. At the current moment, however, the efficacy of this new therapeutic modality should be analyzed with caution because non-removal of infected nidi may be aggravated by a foreign body [32].

Inflammatory aortic aneurysm

Prevalence

Inflammatory aortic aneurysms present a minor subgroup with an incidence ranging from 2.2% to 18.1% of the total number of abdominal aortic aneurysms [33—35]. Inflammatory abdominal aortic aneurysm is distinguished as a variant of atherosclerotic aortic aneurysm by a thickened aneurysm wall, accompanied by a dense fibrosis involving adjacent structures such as the duodenum, ureter, and inferior vena cava [36]. Although inflammatory aortic aneurysm

is seen mostly in the infrarenal abdominal aorta, it may occur in the ascending and descending thoracic aorta and aortic arch [37—39]. Patients usually present at a younger age than those with atherosclerotic aortic aneurysm [40]. Male sex and smoking are reported to be strongly associated with this form of aortic aneurysm. Data regarding the incidence of inflammatory aortic aneurysms in the whole population are lacking. Considering that the prevalence of newly diagnosed abdominal aortic aneurysm was reported as 65 per 100,000 person-years [41] and that 2—18% of cases might possess inflammatory features [33—35], the prevalence of inflammatory aortic aneurysm might be comparable or slightly higher than that of idiopathic retroperitoneal fibrosis, which has been reported to have an incidence of 0.1 per 100,000 person-years [42]. It should be noted, however, that inflammatory aortic aneurysm, as well as idiopathic retroperitoneal fibrosis, which has similar fibromatosis-like reactive inflammation associated with fibrosis but also aortic dilatation [43], may be unrecognized and thus underdiagnosed [44,45].

Clinical features, laboratory abnormalities, and imaging

Clinical symptoms include abdominal or back pain, weight loss, and low grade fever [40], which are, it may be said, similar to those of infected aortic aneurysm. Yin et al. reported that among 11 patients with inflammatory aortic aneurysm, abdominal pain was present in 10 (91%), lumbar pain in 9 (82%), and fever in 8 (72%) [46]. These symptoms contrast with those of the less-symptomatic atherosclerotic aortic aneurysm [46,47]. The prevalence of heavy cigarette smoking is, of note, very high among patients with inflammatory aortic aneurysm [48].

Elevated inflammatory markers, such as erythrocyte sedimentation rate, white blood cell count, and C reactive protein, are commonly observed [49]. In addition, positivity of anti-nuclear antibody and elevation of IgG4maybe observed in inflammatory aortic aneurysm, suggesting the role of autoimmunity [3,49—51]. IgG4-related systemic disease is a newly recognized disorder that may manifest as inflammatory abdominal aortic aneurysm or retroperitoneal fibrosis [52]; therefore, elevation of serum IgG4 levels and/or infiltration of IgG4-positive plasma cells in the periaortic tissues may help the diagnosis of inflammatory aortic aneurysm. By contrast, Kasashima et al. reported that only about half of inflammatory aortic aneurysm cases are judged to be IgG4-related; however, the clinical picture does not seem to differ substantially between the IgG4-related and non-IgG4-related forms of inflammatory aortic aneurysm [53], indicating that this disease cannot be determined solely on IgG4 positivity and that it may be a heterogeneous disease.

CT scanning displays the aneurysm and the thickened aortic wall with periaortic inflammation and fibrosis, the so-called ''mantle sign'' (Fig. 3). Periaortic enhancement by contrast medium in CT scanning may, however, mimic rupture, acute intramural hematoma, and extravasation of contrast — findings that sometimes require careful dissociation from impending rupture or dissecting aortic aneurysm, especially in the presence of severe abdominal or back pain [54]. FDG-PET scanning may be able to illustrate the active inflammation around the aortic wall [55]. Macroscopically, the appearance of white, glistening, perianeurysmal fibrosis is characteristic of inflammatory aortic aneurysm [36,48].

Therapy

Medical therapy

Corticosteroids [47,56] and immunosuppressive agents, such as methotrexate, cyclophosphamide, and azathioprine have also been reported to be effective [34], however, the ultimate efficacy of the anti-inflammatory approach has not been proven [47].

Surgery

Although inflammatory aortic aneurysm may be less liable to rupture than atherosclerotic aneurysm [57], open surgical repair is one of the therapeutic options aimed at preventing rupture. Surgical results have greatly improved over the past 35 years [58,59], although the mortality for inflammatory aortic aneurysm may remain slightly higher than that for atherosclerotic aortic aneurysm. Although periaortic inflammation can be improved post-operatively, it may show no change or worsening in some patients [35].

Endovascular aortic repair

Although experience of the treatment of inflammatory aortic aneurysm with EVAR has been accumulating, no randomized controlled trials exist comparing open surgical repair and EVAR. Paravastu et al. reported in their systematic review that EVAR is associated with lower 1-year mortality as compared with open surgical repair [60]. On the other hand, both treatments led to regression of periaortic inflammation (73% in the open surgical repair group and 65% in the EVAR group). On the other hand, secondary intervention was found to be required in 22% of patients after EVAR.

The periaortitis seen with inflammatory abdominal aortic aneurysms usually resolves after repair by endovascular techniques such as open surgical repair; however, resolution of the periaortic fibrosis may occur less often after EVAR [59]. Development of de novo retroperitoneal fibrosis after EVAR has been reported, albeit rarely, to occur [61], and EVAR may not offer any benefits for hydronephrosis [62]. EVAR appears to be feasible when the anatomical features are appropriate [59]; however, open surgical repair might be the preferred treatment, especially when inflammatory abdominal aortic aneurysm is complicated by hydronephrosis.

Differential diagnosis between infected and inflammatory aortic aneurysms

As discussed above, there are similarities between infected and inflammatory aortic aneurysms in terms of their clinical features (e.g. abdominal/back pain), laboratory data (e.g. increased C-reactive protein), and imaging findings (e.g. perianeurysmal soft-tissue mass). In addition to the aorta, vessels of various sizes, including vertebral, coronary, and iliac arteries, may be involved in inflammatory

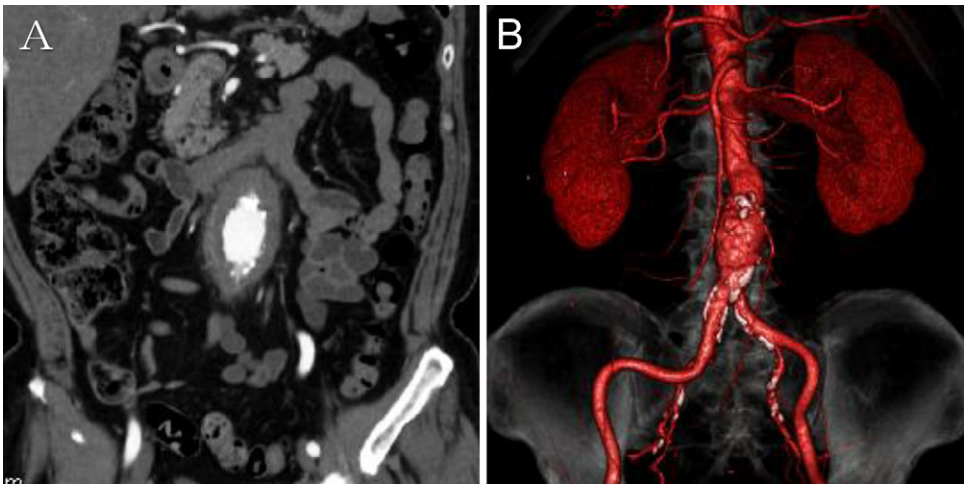


Figure 3 A 57-year-old man presented with repetitive epigastralgia. Coronal sections of computed tomography (CT) scanning images. (A) Dense soft tissue surrounding the mildly dilated abdominal aorta can be observed. (B) 3D reconstruction of the CT images. Calcification of the wall of the aorta and common and internal ileac arteriesis observed. In this patient, corticosteroid therapy reduced clinical symptoms and periaortic soft tissue. Serum IgG4 level was not elevated in this case. Details in this case have been reported elsewhere [77].

aneurysm [51,63,64] and infected aneurysm [65—67]. In addition, similar therapeutic strategies (e.g. open surgical repair, EVAR) may be applied to these two different clinical conditions. There are, however, also differences, such as the indications for medical therapy with corticosteroid or antimicrobial drugs, speed of aneurysm expansion, and perioperative mortality. Considering that periaortic inflammations are becoming more readily and reliably discovered by CT scanning [68], the necessity for a differential diagnosis is increasing.

Imaging

On CT scan, infected aortic aneurysm appears usually as a saccular form aneurysm with nodularity, irregular configuration, or air in the aortic wall [69], in contrast to inflammatory aortic aneurysm, which typically exhibits a fusiform morphology [70], although a saccular morphology is also possible [71]. Calcification within the aneurysm wall may be less common in infected aneurysm [72], although it can be present (Figs. 1 and 2). In inflammatory aortic aneurysm, the degree of calcification in the aortic wall may differ according to the case [1,56,73]. Hydronephrosis may occur also in the infected aneurysm [74].

As mentioned above, FDG-PET is a useful method for assessing the active inflammation and therapeutic effectiveness in patients with inflammatory aortic aneurysm [75—77]; however, increased FDG-uptake may also be demonstrated in infected aortic aneurysm [15,78] as well as in other types of large-vessel inflammation [79], such as Takayasu arteritis [80]. The ability of FDG-PET to provide a differential diagnosis between inflammatory and infected aneurysm may, therefore, be limited. The value of examining FDG uptake for the assessment of aneurysm wall strength, rupture risk [81], and effect of medical interventions needs future studies [82].

Molecular diagnosis

It has been reported that broad-range polymerase chain reaction (PCR) amplification targeting the bacterial 16S rRNA gene, followed by direct sequencing may provide a prompt and facilitated identification of infected bacteria in culture negative cases [83—86].

IgG4

On the one hand, elevation of serum IgG4 and/or periaortic infiltration of IgG4-positive lymphocytes may be observed in inflammatory aortic aneurysm [87]. On the other hand, serum IgG4 may be elevated in a wide variety of disorders, which may include so-called ''IgG4-related disease'', such as autoimmune pancreatitis, Mikulicz's disease, and Riedel's thyroiditis [88]. As discussed above, only about half of inflammatory aortic aneurysm cases may show IgG4-positivity [3]. Of note, Kanemitsu et al. have recently reported infiltration of numerous IgG4-positive plasma cells in the thickened periarterial tissue of the aorta with *Enterobacter* infection [89], suggesting the possibility that IgG4-related immune inflammation might also play a role in infected aortitis and that both inflammatory and infected aortic aneurysm may coexist. Further studies should address the incidence of IgG4-positivity in infected aortic aneurysm, for which little information is currently available. Total IgG may also be elevated in inflammatory aortic aneurysm; however, it may also be elevated in atherosclerotic aortic aneurysm, and thus may be a less potent biomarker for the discrimination of inflammatory aortic aneurysm from the non-inflammatory form [53].

Other biomarkers

As discussed above, inflammatory markers, such as erythrocyte sedimentation rate and C-reactive protein, may be elevated in both inflammatory and infected aortic aneurysms. Procalcitonin, a polypeptide comprising 113 amino acids, is gathering increasing attention because of strong correlation between procalcitonin concentration and extent and severity of bacterial infections [90]. In the case of infected aortic aneurysm we had experienced (Fig. 1), serum procalcitonin level at admission was 1.05 ng/mL, a level that was suggestive of bacterial infection. Procalcitonin is produced ubiquitously by endotoxin or mediators released in response to bacterial infections; thus, utility of inflammatory cytokines [92] and procalcitonin for discriminating inflammatory IgG4-related, or non-IgG4-related, aortic aneurysm from infected aortic aneurysm should be assessed in future studies.

We recently reported that the serum sIL-2R level was elevated in 6 (75%) of 8 patients with chronic periaortitis [91], suggesting that sIL-2R may be another candidate biomarker for discriminating inflammatory from infected aortic aneurysm; again, however, little is known about sIL-2R levels in infected aneurysm. New biomarkers that specifically identify the inflammatory (or infected) aortic aneurysm are now under surveillance.

Conclusions

Here we have briefly summarized the current understanding of both infected and inflammatory aortic aneurysm, and discussed the potential uncertainty for a differential diagnosis. These two clinical entities possess various similarities in clinical manifestation, biomarkers, CT, MRI, and radionucleotide imaging. It is considered that incorrect administration of immunosuppressing agents in infected aortic aneurysm would be hazardous [93] and delayed diagnosis may lead to uncontrolled growth of microorganisms. Caution should be taken to avoid the easy usage of corticosteroid or immunosuppressing drugs before the full diagnosis is established.

Acknowledgments

The work was supported in part by a Grant-in-Aid from the Ministry of Health, Labour, and Welfare (Acute Aortic Syndrome), Meiji Yasuda Life Foundation of Health and Welfare, Rohto Pharmaceutical Company, Mitsui Life Social Welfare Foundation, and a grant from the St Luke's Grant for the Epidemiological Research.

References

[1] Matsuki Y, Sato K, Fujikawa A, Kyoto Y, Hashimoto H, Hakozaki Y. A case of incidentally detected IgG4-related sclerosing disease involving inflammatory abdominal aortic aneurysm and autoimmune pancreatitis. Mod Rheumatol 2010;20:306—10.
[2] Suddleson EA, Katz SG, Kohl RD. Mycotic suprarenal aortic aneurysm. Ann Vasc Surg 1987;1:426—31.
[3] Kasashima S, Zen Y. IgG4-related inflammatory abdominal aortic aneurysm. Curr Opin Rheumatol 2011;23:18—23.
[4] Osler W. The Gulstonian lectures, on malignant endocarditis. Br Med J 1885;1:467—70.
[5] Oderich GS, Panneton JM, Bower TC, Cherry Jr KJ, Rowland CM, Noel AA, Hallett Jr JW, Gloviczki P. Infected aortic aneurysms: aggressive presentation, complicated early outcome, but durable results. J Vasc Surg 2001;34:900—8.
[6] Black PH, Kunz LJ, Swartz MN. Salmonellosis—a review of some unusual aspects. N Engl J Med 1960;262:921—7.
[7] Muller BT, Wegener OR, Grabitz K, Pillny M, Thomas L, Sandmann W. Mycotic aneurysms of the thoracic and abdominal aorta and iliac arteries: experience with anatomic and extra-anatomic repair in 33 cases. J Vasc Surg 2001;33:106—13.
[8] Bisdas T, Teebken OE. Mycotic or infected aneurysm? Time to change the term. Eur J Vasc Endovasc Surg 2011;41:570.
[9] Patel S, Johnston KW. Classification and management of mycotic aneurysms. Surg Gynecol Obstet 1977;144:691—4.
[10] Hsu RB, Chen RJ, Wang SS, Chu SH. Infected aortic aneurysms: clinical outcome and risk factor analysis. J Vasc Surg 2004;40:30—5.
[11] Soravia-Dunand VA, Loo VG, Salit IE. Aortitis due to Salmonella: report of 10 cases and comprehensive review of the literature. Clin Infect Dis 1999;29:862—8.
[12] Macedo TA, Stanson AW, Oderich GS, Johnson CM, Panneton JM, Tie ML. Infected aortic aneurysms: imaging findings. Radiology 2004;231:250—7.
[13] Naganuma H, Ishida H, Konno K, Sato M, Ishida J, Watanabe S. Mycotic abdominal aneurysm: report of a case with emphasis on the presence of gas echoes. Abdom Imaging 2001;26:420—2.
[14] Lee WK, Mossop PJ, Little AF, Fitt GJ, Vrazas JI, Hoang JK, Hennessy OF. Infected (mycotic) aneurysms: spectrum of imaging appearances and management. Radiographics 2008;28:1853—68.
[15] Iimori A, Kanzaki Y, Ito S, Kotani T, Hirano-Kuwata S, Daimon M, Katsumata T, Akagi H, Komori T, Terasaki F, Ishizaka N, Ukimura A. Rapidly progressing aneurysm of infected thoracic aorta with pseudoaneurysm formation. Intern Med 2010;49:2461—5.
[16] Nakamura T, Matsuoka Y, Sakamoto A, Egawa A, Nakajima K, Ishimaru H, Fujimoto T, Yamaguchi T, Itoh M. Japanese title. Jpn J Radiol 1995;28:79.
[17] Murakami I, Kou T, Oda Y, Yazumi S. Rapidly progressing mycotic aortic aneurysm. Intern Med 2011;50:161.
[18] Cina CS, Arena GO, Fiture AO, Clase CM, Doobay B. Ruptured mycotic thoracoabdominal aortic aneurysms: a report of three cases and a systematic review. J Vasc Surg 2001;33:861—7.
[19] Hsu RB, Tsay YG, Wang SS, Chu SH. Surgical treatment for primary infected aneurysm of the descending thoracic aorta, abdominal aorta, and iliac arteries. J Vasc Surg 2002;36:746—50.
[20] Maeda H, Umezawa H, Goshima M, Hattori T, Nakamura T, Umeda T, Shiono M. Primary infected abdominal aortic aneurysm: surgical procedures, early mortality rates, and a survey of the prevalence of infectious organisms over a 30-year period. Surg Today 2011;41:346—51.
[21] Lee NY, Kan CD, Chi CY, Chen PL, Lee HC, Chang CM, Wu CJ, Ko WC. Community-acquired methicillin-resistant Staphylococcus aureus mycotic aneurysm. Intern Med 2010;49:623—5.
[22] Bennett DE. Primary mycotic aneurysms of the aorta. Report of case and review of the literature. Arch Surg 1967;94:758—65.
[23] Oskoui R, Davis WA, Gomes MN. Salmonella aortitis. A report of a successfully treated case with a comprehensive review of the literature. Arch Intern Med 1993;153:517—25.
[24] Gufler H, Buitrago-Tellez CH, Nesbitt E, Hauenstein KH. Mycotic aneurysm rupture of the descending aorta. Eur Radiol 1998;8:295—7.
[25] Hollier LH, Money SR, Creely B, Bower TC, Kazmier FJ. Direct replacement of mycotic thoracoabdominal aneurysms. J Vasc Surg 1993;18:477—84.

[26] Sessa C, Farah I, Voirin L, Magne JL, Brion JP, Guidicelli H. Infected aneurysms of the infrarenal abdominal aorta: diagnostic criteria and therapeutic strategy. Ann Vasc Surg 1997;11:453—63.
[27] Luo CY, Ko WC, Kan CD, Lin PY, Yang YJ. In situ reconstruction of septic aortic pseudoaneurysm due to Salmonella or Streptococcus microbial aortitis: long-term follow-up. J Vasc Surg 2003;38:975—82.
[28] Moneta GL, Taylor Jr LM, Yeager RA, Edwards JM, Nicoloff AD, McConnell DB, Porter JM. Surgical treatment of infected aortic aneurysm. Am J Surg 1998;175:396—9.
[29] Bacourt F, Koskas F. Axillobifemoral bypass and aortic exclusion for vascular septic lesions: a multicenter retrospective study of 98 cases, French University Association for Research in Surgery. Ann Vasc Surg 1992;6:119—26.
[30] Kyriakides C, Kan Y, Kerle M, Cheshire NJ, Mansfield AO, Wolfe JH. 11-Year experience with anatomical and extra-anatomical repair of mycotic aortic aneurysms. Eur J Vasc Endovasc Surg 2004;27:585—9.
[31] Semba CP, Sakai T, Slonim SM, Razavi MK, Kee ST, Jorgensen MJ, Hagberg RC, Lee GK, Mitchell RS, Miller DC, Dake MD. Mycotic aneurysms of the thoracic aorta: repair with use of endovascular stent-grafts. J Vasc Interv Radiol 1998;9:33—40.
[32] Kan CD, Lee HL, Yang YJ. Outcome after endovascular stent graft treatment for mycotic aortic aneurysm: a systematic review. J Vasc Surg 2007;46:906—12.
[33] Stella A, Gargiulo M, Faggioli GL, Bertoni F, Cappello I, Brusori S, D'Addato M. Postoperative course of inflammatory abdominal aortic aneurysms. Ann Vasc Surg 1993;7:229—38.
[34] Vaglio A, Buzio C. Chronic periaortitis: a spectrum of diseases. Curr Opin Rheumatol 2005;17:34—40.
[35] von Fritschen U, Malzfeld E, Clasen A, Kortmann H. Inflammatory abdominal aortic aneurysm: a postoperative course of retroperitoneal fibrosis. J Vasc Surg 1999;30:1090—8.
[36] Walker DI, Bloor K, Williams G, Gillie I. Inflammatory aneurysms of the abdominal aorta. Br J Surg 1972;59:609—14.
[37] Yoshida M, Mukohara N, Honda T, Ozaki N, Shida T. Inflammatory aneurysm of the ascending aorta: report of a case. Surg Today 2007;37:794—7.
[38] Girardi LN, Coselli JS. Inflammatory aneurysm of the ascending aorta and aortic arch. Ann Thorac Surg 1997;64:251—3.
[39] Ishida M, Hotta M, Kushima R, Asai T, Okabe H. IgG4-related inflammatory aneurysm of the aortic arch. Pathol Int 2009;59:269—73.
[40] Rasmussen TE, Hallett Jr JW. Inflammatory aortic aneurysms. A clinical review with new perspectives in pathogenesis. Ann Surg 1997;225:155—64.
[41] Wong DR, Willett WC, Rimm EB. Smoking, hypertension, alcohol consumption, and risk of abdominal aortic aneurysm in men. Am J Epidemiol 2007;165:838—45.
[42] Uibu T, Oksa P, Auvinen A, Honkanen E, Metsarinne K, Saha H, Uitti J, Roto P. Asbestos exposure as a risk factor for retroperitoneal fibrosis. Lancet 2004;363:1422—6.
[43] Ormond JK. Bilateral ureteral obstruction due to envelopment and compression by an inflammatory retroperitoneal process. J Urol 1948;59:1072—9.
[44] Verit A, Yeni E, Unal D, Kafali H, Ozturk A, Ozardali I. Idiopathic retroperitoneal fibrosis mimicking a pelvic tumor: a case of pericystitis plastica. Yonsei Med J 2003;44:548—50.
[45] Meier P, Gilabert C, Burnier M, Blanc E. Retroperitoneal fibrosis, an unrecognized inflammatory disease. Clinical observations and review of the literature. Nephrologie 2003;24:173—80.
[46] Yin MD, Zhang J, Wang SY, Duan ZQ, Xin SJ. Inflammatory abdominal aortic aneurysm: clinical features and long term outcome in comparison with atherosclerotic abdominal aortic aneurysm. Chin Med J 2010;123:1255—8.
[47] Hellmann DB, Grand DJ, Freischlag JA. Inflammatory abdominal aortic aneurysm. JAMA 2007;297:395—400.
[48] Crawford JL, Stowe CL, Safi HJ, Hallman CH, Crawford ES. Inflammatory aneurysms of the aorta. J Vasc Surg 1985;2:113—24.
[49] Vaglio A, Corradi D, Manenti L, Ferretti S, Garini G, Buzio C. Evidence of autoimmunity in chronic periaortitis: a prospective study. Am J Med 2003;114:454—62.
[50] Sasaki S, Yasuda K, Takigami K, Yamauchi H, Shiiya N, Sakuma M. Inflammatory abdominal aortic aneurysms and atherosclerotic abdominal aortic aneurysms—comparisons of clinical features and long-term results. Jpn Circ J 1997;61:231—5.
[51] Vaglio A, Pipitone N, Salvarani C. Chronic periaortitis: a large-vessel vasculitis? Curr Opin Rheumatol 2011;23:1—6.
[52] Stone JR. Aortitis, periaortitis, and retroperitoneal fibrosis, as manifestations of IgG4-related systemic disease. Curr Opin Rheumatol 2011;23:88—94.
[53] Kasashima S, Zen Y, Kawashima A, Endo M, Matsumoto Y, Kasashima F. A new clinicopathological entity of IgG4-related inflammatory abdominal aortic aneurysm. J Vasc Surg 2009;49:1264—71.
[54] Pang YC, Chan YC, Ting AC, Cheng SW. Tender inflammatory infrarenal aortic aneurysm simulating acute rupture. Asian Cardiovasc Thorac Ann 2010;18:180—2.
[55] Kroger K, Antoch G, Goyen M, Freudenberg LS, Veit P, Janicke I, Bockisch A, Forsting M. Positron emission tomography/computed tomography improves diagnostics of inflammatory arteritis. Heart Vessels 2005;20:179—83.
[56] Yabe T, Hamada T, Kubo T, Okawa M, Yamasaki N, Matsumura Y, Kitaoka H, Doi Y. Inflammatory abdominal aortic aneurysm successfully treated with steroid therapy. J Am Coll Cardiol 2010;55:2877.
[57] Lindblad B, Almgren B, Bergqvist D, Eriksson I, Forsberg O, Glimaker H, Jivegard L, Karlstrom L, Lundqvist B, Olofsson P, Plate G, Thorne J, Troeng T. Abdominal aortic aneurysm with perianeurysmal fibrosis: experience from 11 Swedish vascular centers. J Vasc Surg 1991;13:231—7.
[58] Pennell RC, Hollier LH, Lie JT, Bernatz PE, Joyce JW, Pairolero PC, Cherry KJ, Hallett JW. Inflammatory abdominal aortic aneurysms: a thirty-year review. J Vasc Surg 1985;2:859—69.
[59] Tang T, Boyle JR, Dixon AK, Varty K. Inflammatory abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2005;29:353—62.
[60] Paravastu SC, Ghosh J, Murray D, Farquharson FG, Serracino-Inglott F, Walker MG. A systematic review of open versus endovascular repair of inflammatory abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2009;38:291—7.
[61] Vijaynagar B, McMahon GS, McCarthy MJ. Periaortic inflammation after endovascular aneurysm repair. Ann Vasc Surg 2011;25(558):e555—7.
[62] Coppi G, Rametta F, Aiello S, Saitta G, Gennai S, Silingardi R. Inflammatory abdominal aortic aneurysm endovascular repair into the long-term follow-up. Ann Vasc Surg 2010;24:1053—9.
[63] Toyoshima Y, Emura I, Umeda Y, Fujita N, Kakita A, Takahashi H. Vertebral basilar system dolichoectasia with marked infiltration of IgG4-containing plasma cells: a manifestation of IgG4-related disease? Neuropathology 2011;(May) [Epub ahead of print].
[64] Matsumoto Y, Kasashima S, Kawashima A, Sasaki H, Endo M, Kawakami K, Zen Y, Nakanuma Y. A case of multiple immunoglobulin G4-related periarteritis: a tumorous lesion of the coronary artery and abdominal aortic aneurysm. Hum Pathol 2008;39:975—80.
[65] Flye MW, Wolkoff JS. Mycotic aneurysm of the left subclavian and vertebral arteries. A complication of cervicothoracic sympathectomy. Am J Surg 1971;122:427—9.
[66] Yamamoto Y, Ushijima T, Yamaguchi S, Takata M, Takago S, Matsunaga M, Watanabe G. Mycotic aneurysm of the left anterior descending coronary artery after coronary artery bypass graft surgery. Ann Thorac Surg 2011;91:1601—3.

[67] Yu SY, Hsieh HC, Ko PJ, Huang YK, Chu JJ, Lee CH. Surgical outcome for mycotic aortic and iliac anuerysm. World J Surg 2011;35:1671—8.
[68] Iino M, Kuribayashi S, Imakita S, Takamiya M, Matsuo H, Ookita Y, Ando M, Ueda H. Sensitivity and specificity of CT in the diagnosis of inflammatory abdominal aortic aneurysms. J Comput Assist Tomogr 2002;26:1006—12.
[69] Oz MC, Brener BJ, Buda JA, Todd G, Brenner RW, Goldenkranz RJ, McNicholas KW, Lemole GM, Lozner JS. A ten-year experience with bacterial aortitis. J Vasc Surg 1989;10:439—49.
[70] Villareal RP, Howell MH, Krajcer Z. Regression of inflammatory abdominal aortic aneurysm after endoluminal treatment with bare-metal Wallstent endoprostheses. Tex Heart Inst J 2000;27:146—9.
[71] Sharif MA, Soong CV, Lee B, McCallion K, Hannon RJ. Inflammatory infrarenal abdominal aortic aneurysm in a young woman. J Emerg Med 2008;34:147—50.
[72] Gomes MN, Choyke PL. Infected aortic aneurysms: CT diagnosis. J Cardiovasc Surg 1992;33:684—9.
[73] Bartlett P, Woods D, Dobranowski J. Inflammatory abdominal aortic aneurysm: a cause of urinary obstruction and acute renal failure. Can Assoc Radiol J 1989;40:164—6.
[74] Woodrum DT, Welke KF, Fillinger MF. Candida infection associated with a solitary mycotic common iliac artery aneurysm. J Vasc Surg 2001;34:166—8.
[75] Belhocine T, Blockmans D, Hustinx R, Vandevivere J, Mortelmans L. Imaging of large vessel vasculitis with (18)FDG PET: illusion or reality? A critical review of the literature data. Eur J Nucl Med Mol Imaging 2003;30:1305—13.
[76] Takahashi M, Momose T, Kameyama M, Ohtomo K. Abnormal accumulation of [18F]fluorodeoxyglucose in the aortic wall related to inflammatory changes: three case reports. Ann Nucl Med 2006;20:361—4.
[77] Sakamoto A, Okamoto K, Ishizaka N, Tejima K, Hirata Y, Nagai R. 18F-fluorodeoxyglucose positron emission tomography in a case of retroperitoneal fibrosis. Int Heart J 2006;47: 645—50.
[78] Choi SJ, Lee JS, Cheong MH, Byun SS, Hyun IY. F-18 FDG PET/CT in the management of infected abdominal aortic aneurysm due to Salmonella. Clin Nucl Med 2008;33:492—5.
[79] Blockmans D. PET in vasculitis. Ann NY Acad Sci 2011;1228:64—70.
[80] Kobayashi Y, Ishii K, Oda K, Nariai T, Tanaka Y, Ishiwata K, Numano F. Aortic wall inflammation due to Takayasu arteritis imaged with 18F-FDG PET coregistered with enhanced CT. J Nucl Med 2005;46:917—22.
[81] Sakalihasan N, Van Damme H, Gomez P, Rigo P, Lapiere CM, Nusgens B, Limet R. Positron emission tomography (PET) evaluation of abdominal aortic aneurysm (AAA). Eur J Vasc Endovasc Surg 2002;23:431—6.
[82] Truijers M, Kurvers HA, Bredie SJ, Oyen WJ, Blankensteijn JD. In vivo imaging of abdominal aortic aneurysms: increased FDG uptake suggests inflammation in the aneurysm wall. J Endovasc Ther 2008;15:462—7.
[83] Ichimura S, Nagano M, Ito N, Shimojima M, Egashira T, Miyamoto C, Ohkusu K, Ezaki T. Evaluation of the invader assay with the BACTEC MGIT 960 system for prompt isolation and identification of mycobacterial species from clinical specimens. J Clin Microbiol 2007;45:3316—22.
[84] Tanabe M, Nakajima H, Nakamura A, Ito T, Nakamura M, Shimono T, Wada H, Shimpo H, Nobori T, Ito M. Mycotic aortic aneurysm associated with Legionella anisa. J Clin Microbiol 2009;47:2340—3.
[85] Goldenberger D, Kunzli A, Vogt P, Zbinden R, Altwegg M. Molecular diagnosis of bacterial endocarditis by broad-range PCR amplification and direct sequencing. J Clin Microbiol 1997;35:2733—9.
[86] Koo M, Manalili S, Bankowski MJ, Sampath R, Hofstadler SA, Koo J. A ''silent culture-negative'' abdominal aortic mycotic aneurysm: rapid detection of Bartonella species using PCR and high-throughput mass spectrometry. Hawaii Med J 2010;69:68—9.
[87] Ishizaka N, Sakamoto A, Imai Y, Terasaki F, Nagai R. Multifocal fibrosclerosis and IgG4-related disease involving the cardiovascular system. J Cardiol 2012;59:132—8.
[88] Khosroshahi A, Stone JH. A clinical overview of IgG4-related systemic disease. Curr Opin Rheumatol 2011;23:57—66.
[89] Kanemitsu S, Shimono T, Nakamura A, Yamamoto K, Wada H, Shimpo H. Molecular diagnosis of nonaneurysmal infectious aortitis. J Vasc Surg 2011;53:472—4.
[90] Schuetz P, Albrich W, Mueller B. Procalcitonin for diagnosis of infection and guide to antibiotic decisions: past, present and future. BMC Med 2011;9:107.
[91] Sakamoto A, Nagai R, Saito K, Imai Y, Takahashi M, Hosoya Y, Takeda N, Fujihara M, Hirano K, Koike K, Enomoto Y, Kume H, Homma Y, Maeda D, Yamada H, et al. Idiopathic retroperitoneal fibrosis, inflammatory aortic aneurysm, and inflammatory pericarditis — retrospective analysis of 11 case histories. J Cardiol 2012;59:139—46.
[92] Sato Y, Notohara K, Kojima M, Takata K, Masaki Y, Yoshino T. IgG4-related disease: historical overview and pathology of hematological disorders. Pathol Int 2010;60:247—58.
[93] Smith FC, Rees E, Elliott TS, Shearman CP. A hazard of immunosuppression: Aspergillus niger infection of abdominal aortic aneurysm. Eur J Vasc Surg 1994;8:369—71.