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A strobe multicenter descriptive study of 55 infectious aortitis

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Abstract

Infectious aortitis (IA) is a rare and severe disease. The treatment classically associates open surgery with prolonged antibiotic therapy. This study aimed to describe clinical characteristics, medical and surgical supports in a large and current series of IA.

We conducted a retrospective multicenter study of native aorta IA, between 2000 and 2019. Inclusion criteria were the presence of a microorganism on blood culture, aortic sample or any other validated technique and structural anomaly in imaging.

We included 55 patients (85% men), with a median age of 65. Microbiology data substantially differed from previous studies with 12 Gram-negative rods IA, of which only 3 due to *Salmonella spp*., 24 Gram-positive cocci IA of which 12 *Streptococcus spp.*, and 18 IA due to intracellular growth and/or fastidious microorganisms, of which 8 *Coxiella burnetii*, 3 *Treponema pallidum*, and 5 tuberculosis suspicious cases. Fifteen patients (27%) presented with thoracic IA, 31 (56%) with abdominal IA, and 9 (16%) with thoraco-abdominal IA. Eight patients had no surgery, 41 underwent open surgery, only 4 endovascular aneurysm repair, and 2 a combination of these 2 techniques. Nine patients died before 1-month follow-up. There was no difference in the mortality rate between the different types of germ or localization of IA.

The variety of germs involved in IA increases. Positron emission tomography-computed tomography scan is a very useful tool for diagnosis. Surgery is still mainly done in open approach and a prospective multicenter study seems necessary to better determine the place of endovascular aneurysm repair versus open surgery.

Abbreviations: AA = aortic aneurysm, CT = computed tomography, EVAR = endovascular aneurysm repair, GCA = giant cell arteritis, GNR = gram-negative rods, GPC = gram-positive cocci, IA = infectious aortitis, ICGF = intracellular growth and/or fastidious microorganisms, IGRA = interferon gamma release assays, PCR = polymerase chain reaction, PET-CT = positron emission tomography-computed tomography, TB = tuberculosis, TST = tuberculin skin test.

Keywords: cardiovascular, infected aneurysm, infectious aortitis, mycotic aneurysm, Q fever, syphilis, tuberculosis

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1. Introduction

Infectious aortitis (IA) is a rare disease and corresponds to a specific infection of the aortic wall. It can present itself as an isolated aortic wall thickening or more often as an aortic aneurysm (AA). Infection-related AA represent <5% of all AA[1] and there is no epidemiological data concerning non-aneurysmal IA. IA affects men more often than women, probably because of the higher frequency in that population of atherosclerosis which could favor the insemination of a microorganism through structural alteration of the aortic wall; immunosuppression could also be a risk factor for IA but is still disputed.[2,3]

With the improvement of microbiology and imaging techniques, the variety of microorganisms involved in IA has likely increased. The germs most often incriminated are Gram-positive cocci (GPC), with a large majority of *Staphylococcus aureus,* and Gram-negative rods (GNR), mainly *Salmonella spp*. These data stem from a series of 29 patients with IA, the largest before the year 2000.[1] Since then, numerous isolated case-reports or small series have been published and the reported germs are similar to what was described before 2000: 30 cases of *S aureus*,[4–15] 72 *Salmonella spp*.,[4,5,16–18,7,9,19–25] 16 *Streptococcus spp*.,[4–6,26–29,10,30,31] and 6 *Clostridium septicum*.[32–37]

IA is a severe disease, with poor prognosis in the absence of surgical treatment,[2] worse than for non- IA.[38] Open surgery with replacement of the aorta – be it with prosthesis or allograft – has long been considered the gold-standard treatment of infectious AA but numerous case-reports show that there is a growing resort to endovascular aneurysm repair (EVAR) techniques.[19,39]

The aim of this study was to describe the clinical and biological characteristics of patients at the diagnosis of IA, and to report their therapeutic management and evolution in a major multicenter study.

2. Material and methods

This retrospective descriptive multicenter French study included adult patients who presented an IA diagnosed between January 1, 2000 and December 31, 2018.

IA cases were identified through the international classification of diseases 10th revision diagnostic codes for arteritis with a subsequent analysis of each file to select those corresponding to IA, analysis of bacteriology laboratory registers for aortic samples or call for observation to the infectious disease referring doctor of the hospital.

Inclusion criteria were the presence of IA on native aorta. IA was defined as structural anomaly of the aortic wall in imaging (computerized tomography [CT] scan, magnetic resonance imaging, ultrasonography) and/or perioperative finding (wall thickening, ectasia or aneurysm, false aneurysm, dissection, rupture) associated with the identification of a microorganism on blood culture and/or aortic sample, either by conventional bacteriology methods or by polymerase chain reaction (PCR) or serology. Tuberculous aortitis was diagnosed by either the identification of *Mycobacterium tuberculosis* or a positive interferon gamma release assays (IGRA) test or a Mantoux tuberculin skin test (TST), associated with a complete infectious and immuno-inflammatory assessment excluding another etiology.

Patients with aortic structural anomalies and a non-aortic infection liable to explain the microorganism's identification were excluded, as well as patients with prosthesis-based IA or who had undergone aortic surgery up to 1 year before diagnosis.

The study was approved by the ethics committee of the Nantes university hospital, and complied with the requirements of the "Commission Nationale de l'Informatique et des Libertés," in accordance with current French legislation. Patient informed consent was obtained for the study.

Patients were divided in different subgroups for statistical analysis, according to the type of germ (GPC, GNR, intracellular growth and/or fastidious microorganisms (ICGF), and fungi) and the anatomical localization of IA (thoracic, abdominal, and both). Statistical analysis were conducted using Prism software version 5.00 for Windows (GraphPad software©). Quantitative variables are presented as median, minimal, and maximal value. Nonparametric Mann–Whitney test was used for comparing quantitative variables, Fisher exact test for nominal qualitative variables, and the log-rank test for survival analysis based on Kaplan–Meier method. *P*-value was considered statistically significant when <.05.

3. Results

Fifty-five patients were included; their characteristics are summarized in Table 1. The different types of germs could be divided in 4 subgroups with 24 pyogenic GPC (24%) – 12 *Streptococcus pneumoniae*, 9 *S aureus*, 3 other Streptococci (*Streptococcus agalactiae, Streptococcus equi zooepidemicus, Streptococcus dysgalactiae*); 12 pyogenic GNR (22%) – 5 *Campylobacter spp*., 3 *Salmonella spp*., 2 *Escherichia coli*, 1 *Francisella tularensis*, 1 *Haemophilus influenzae* ; 18 ICGF (33%) – 8 *Coxiella burnetii*, 5 tuberculosis (TB) suspicious cases even though only 1 microbiologically proven with *M tuberculosis*, 3 *Treponema pallidum*, 2 *Listeria monocytogenes*; 1 fungus (1.8% – *Candida parapsilosis*). Patients with GNR and GPC aortitis were significantly older than those with ICGF aortitis (*P* = .003 for GNR vs ICGF; *P* = .038 for GPC vs ICGF).

3.1. Microbiological data

The type of germs identified and their means of identification are described in Supplemental Table 1, http://links.lww.com/MD/E959. For pyogenic IA (GNR and GPC), diagnosis was made through culture of blood sample and/or culture of aortic sample:

Blood culture: positive in 8 patients with GNR aortitis (67%) and 17 patients with GPC aortitis (71%)

Aortic sample was available for 9 patients with GNR aortitis and 17 patients with GPC aortitis. It was obtained during the intraoperative act and/or para-aortic/intramural abscess punctures. Aortic culture was positive in respectively 7 (78%) and 15 (88%) of cases and was the sole mean of identification in 4 out of 12 GNR aortitis and 7 out of 24 GPC aortitis due to negative blood sample cultures.

One IA was documented through standard culture of the intraoperative cell salvage system, as part of a blood safety quality control procedure, which was positive for *Campylobacter spp*. – the same germ being incidentally identified on blood culture.

This series reports 8 patients with *C burnetii* IA (14.5%). All were men aged over 50 and lived in rural environment. The abdominal aorta was solely concerned, in the form of an aneurysm.

Only 3 patients (5.4%) developed IA secondary to an infection in another organ, that is, CT made at diagnosis of the initial infection showed no aortitis nor AA. Initial infection sites were: 1 *S pneumoniae* pneumonia with bacteremia, 1 *E faecium* and *E coli* fecal peritonitis, and 1 *S pneumoniae* spondylitis.

Table 1

Clinical, biological, and evolution characteristics of infectious aortitis.

	Total n = 55	GNR n = 12	GPC n = 24	ICGF n = 18	Fungus n = 1
Men	47 (85%)	12 (100%)	17 (71%)	13 (72%)	1 (100%)
Median age [min; max]	65 [21; 88]	72 [52; 88]	67 [33; 75]	60 [21; 74]	62
Tobacco	34 (62%)	7 (58%)	18 (75%)	8 (44%)	1
High blood pressure	31 (56%)	8 (67%)	15 (63%)	8 (44%)	0
Dyslipidemia	21 (38%)	6 (50%)	9 (38%)	5 (28%)	1
Diabetes mellitus	9 (16%)	3 (25%)	5 (21%)	1 (6%)	0
Immunosuppression other than diabetes	18 (33%)	5 (42%)	8 (33%)	4 (22%)	1
Clinical and biological data					
Median max temperature [min; max]	37.8 [36.0; 39.9]	39.0 [37.8; 39.6]	38.4 [36.0; 39.9]	37.0 [36.8; 38.2]	NA
Aortitis related pain	41 (75%)	8 (67%)	22 (92%)	10 (56%)	1
Thoracic pain	9 (16%)	1 (8%)	8 (33%)	0	0
Abdominal pain	21 (38%)	4 (33%)	11 (46%)	6 (33%)	0
Lumbar pain	18 (33%)	4 (33%)	8 (33%)	5 (28%)	1
Altered general condition	20 (36%)	3 (25%)	10 (42%)	7 (39%)	0
Median C-reactive protein (mg/L)	145 [6; 606]	223 [46; 437]	175 [24; 606]	22 [6; 208]	77
Aortitis localization					
Thoracic	15 (27%)	3 (25%)	8 (33%)	4 (22%)	0
Thoraco-abdominal	9 (16%)	3 (25%)	3 (13%)	2 (11%)	1
Abdominal	31 (56%)	6 (50%)	13 (54%)	12 (67%)	0
Aortitis aspect					
Aneurysm	51 (93%)	10 (83%)	24 (100%)	16 (89%)	1
fusiform	15 (29%)	3 (30%)	4 (17%)	8 (50%)	NA
sacciform	28 (55%)	6 (60%)	17 (71%)	5 (31%)	NA
false-aneurysm	21 (38%)	3 (30%)	12 (50%)	6 (38%)	NA
Surgery					
Open surgery	41 (74%)	9 (75%)	18 (75%)	13 (72%)	1
EVAR	4 (7%)	1 (8%)	3 (13%)	0	0
Combined	2 (4%)	0	2 (8%)	0	0
Evolution					
Median follow-up (mon) [min; max]	17 [0; 160]	15 [1; 86]	22 [0; 93]	17 [0; 160]	17
Local relapse	1 (2%)	0	0	1 (6%)	0
Death	16 (29%)	7 (58%)	7 (29%)	2 (11%)	0

EVAR = endovascular aneurysm repair, GPC = Gram-Positive Cocci, GNR = Gram-negative rods, ICGF = intracellular growth and/or fastidious microorganisms, NA = not available.

Seven patients (13%) presented with aorta-neighboring organ infection that was concurrent or subsequent to IA diagnosis: 4 with spondylodiscitis (2 *C burnetii*, 1 *S aureus*, and 1 *S pneumoniae*), 1 with infringement to the right pleura (*S agalactiae*), and 2 with psoas damage (*E coli* and *C burnetii*).

3.2. Tuberculosis suspicious cases

Five patients were suspected of tuberculous IA, even though only 1 was microbiologically proven with *M tuberculosis*. Among the other 4 patients with no identification of *M tuberculosis*, 3 had a positive IGRA test and 1 had positive Mantoux TST (and deigned not receptive for IGRA test due to profound immunosuppression), 3 had multiple thoracic adenopathies with granuloma, 1 had histologically proven granulomatous aortitis (the one with Mantoux TST), 1 had granulomatous uveitis, 1 was immunosuppressed due to kidney transplantation, and another due to human immunodeficiency virus (HIV), and 3 were immigrants from TB-endemic countries. Evolution was positive under appropriate TB treatment regimen for all 4 patients.

3.3. Cardiovascular risk factors

The only statistically significant difference between the different subgroups of patients in terms of cardiovascular risk factors concerned active smoking which was more important in the GPC group compared to the ICGF group (71% vs 39%, $P = .047$).

Only 10 patients (18%) were known for having chronic AA previous to the diagnosis of IA, all of "a priori" atherosclerotic nature – and also secondary to giant cell arteritis (GCA) for one – with a median age of 66 [min 48; max 79] and a median number of 2 cardiovascular risk factors other than age. The microorganisms identified in these IA with previous aneurysm were – contrary to the global cohort – in majority ICGF: 6 patients (60%), 3 GPC (30%), and 1 GNR (10%).

Six patients had no cardiovascular risk factors – 5 men and 1 woman – of which 3 with *T pallidum, 1 S pneumoniae, 1 C burnetii* and 1 *Salmonella dublin*.

3.4. Immunosuppression risk factors

Twelve patients (22%) were immunosuppressed, all men. Three were under immunosuppressant drugs: ciclosporine & mycophenolate mofetil for kidney transplantation eleven years before (n = 1), long-term rituximab regimen for non-Hodgkin lymphoma with accumulative dose of 17,825 mg (n = 1), high intake of Class I super potent topical corticosteroid (up to 3 tubes representing 1.5 g per day of clobetasol for years) for erythrodermic psoriasis (n = 1). Apart from the patient described above with lymphoma, 2 others suffered from hematologic

diseases: refractory anemia (=1) and Waldenström macroglobulinemia (n=1). Two had alcohol liver cirrhosis. Three had active solid cancer. One had chronic HIV infection with 584/mm^3 CD4 count and finally another combined chronic HIV infection (440/mm^3 CD4 count) with past splenectomy.

3.5. Clinical data

Clinical presentation was clearly nonspecific: the majority of patients only suffered from pain (thoracic, abdominal, and/or lumbar), altered general condition, and fever.

We noted a bitonal voice in only 1 patient (2%) and cough with no associated pulmonary disease in 2 patients (4%). Patients with pyogenic IA were significantly more febrile at diagnosis than patients with ICGF IA ($P < .0001$ for GNR vs ICGF; $P = .0005$ for GPC vs ICGF).

3.6. Anatomical and imaging characteristics of IA

There was no statistical association between the type of microorganisms identified or the age of patient and the localization of IA. All save 2 patients had a CT for IA diagnosis. For these 2 patients without CT imaging, 1 was diagnosed by transesophageal echocardiography showing a dissected aneurysm of the ascending thoracic aorta (syphilitic IA), and other by positron emission tomography (PET)-CT. Four patients (7%) had a non-aneurysmal IA, meaning that the aorta presented isolated wall thickening and no aneurysmal distortion. The germs involved in these 4 patients were: *M tuberculosis, T pallidum*, *E coli*, and *E coli* & *E faecium*.

Considering only the 51 patients with aneurysmal IA, median max diameter was 54 mm [min 35; max 84]. Sacciform aneurysm was significantly more frequent for patients with GPC IA compared to ICGF IA (71% vs 31%, $P = .0116$).

For 27 patients (49%) IA extended beyond the sole aorta to neighboring anatomical structures. Presence of gas bubbles in or around the aorta wall was noted for 4 patients (7%) and mural thrombus for 20 patients (36%).

PET-CT was made in 19 patients (35%) at diagnosis: 18 evidenced aortic hypermetabolism compatible with aortitis. Figure 1 shows different pathological findings of IA in CT and PET-CT.

Histological analysis of the aorta was available for 27 patients (49%). It showed lesions of arteritis in 23 cases, of which 9 with pan-arteritis, 9 with granulomas, and 7 with giant cells.

Germs were seen through standard colorations in only 4 patients; all of which had pyogenic IA (3 with *S aureus* and 1 with *H influenzae*).

3.7. Treatments

All patients with IA received empirical antibiotic regimen, if the germ and its antibiogram was not already known at the time of IA diagnosis, with adaptation according to antibiogram when obtained. Regarding ICGF aortitis, treatment durations were ≥36 weeks in case of TB, ≥48 weeks for *C burnetii* except in 2 cases with respectively 2 weeks (death) and 12 weeks, 12 weeks for *L monocytogenes* and finally ≥3 weeks for syphilis. In case of pyogenic IA, median treatment duration was 6 weeks [min 2; max 16].

Eight patients had no surgery: 5 had ICGF IA and 3 had pyogenic IA (1 had non-perforated *E coli* IA due to neighboring psoas abscesses that were drained, and the 2 others died early during management). Therefore 47 patients (85.5%) underwent surgery: 41 had open surgery only (in situ repair with graft), 4 had EVAR and 2 had a combination of these 2 techniques, sequentially for one and in the same operating time for the other. Surgical treatment is detailed in Table 2 depending on the localization of IA. Two patients underwent both EVAR and open surgery, either sequentially or in the same operating time, therefore allowing successful management of rapidly-progressing AA (sequential treatment) or of multiple and complex lesions of which some accessible only through EVAR (thoracic lesions) and others through open surgery (abdominal lesions).

3.8. Mortality and evolution

Only 1 patient relapsed; he had abdominal IA due to *C burnetii* and was initially treated with double antibiotic regimen (doxycycline and hydroxychloroquine) as well as open surgery with prosthetic graft.

Three months later he was still febrile and painful due to an abscess of the aneurysmal aortic wall upon contact of the prosthesis. The prosthesis was removed, an allograft bypass was performed and no systemic development was ensued. The ablated prosthesis was positive for *C burnetii*.

Sixteen patients (29%) died, of which 9 before 1 month follow-up (16%), due to the severity of sepsis, local complication of IA (aneurysm rupture, perforation) and/or complications of aortic surgery: 8 of them had pyogenic IA and 1 had *C burnetii* IA.

Survival curves comparing pyogenic IA and ICGF IA are represented in Figure 2. There is no difference in mortality between these 2 groups ($P = .17$).

4. Discussion

This study reports the largest IA series to our knowledge, and the data remind us that it is a rare entity. Table 3 compares data from the main IA series in the literature after that of Miller et al[1] There seems to exist a patient risk profile to develop IA: it more often concerns old men; immunosuppression appears to be a risk factor to take into account; in this series the presence of at least 1 cardiovascular risk factor was almost constant. Pre-existing anomalies of the aortic wall, like aortic congenital anomalies, and endocarditis are also described as risk factors[3,27,40] but these data are not confirmed by other robust series as well as ours.[1,5]

4.1. Structural anomaly of the aorta

The presence of an AA previous to the development of aneurysmal IA was present in 16% of cases, which leads to suspect that pre-existing AA increases the risk of microorganism seeding of an already altered aortic wall. In this series, similar to that of Miranda et al, pyogenic IA predominantly appears as sacciform aneurysm.[5] However, for ICGF IA, the distribution is more equally divided between fusiform and sacciform aneurysm, suggesting that when the inflammatory process is slower than for pyogenic IA, the alteration of the aortic wall is progressive, more circumferential, and more fusiform than sacciform.

The high frequency of aortic structural anomalies at diagnosis differs from that of non-infectious inflammatory aortitis (idiopathic or associated to GCA) for which, in the study by Espitia et al, the proportion of aneurysmal IA was 20.5% in GCA and 38.6% in idiopathic aortitis.[38] IA appears more aggressive

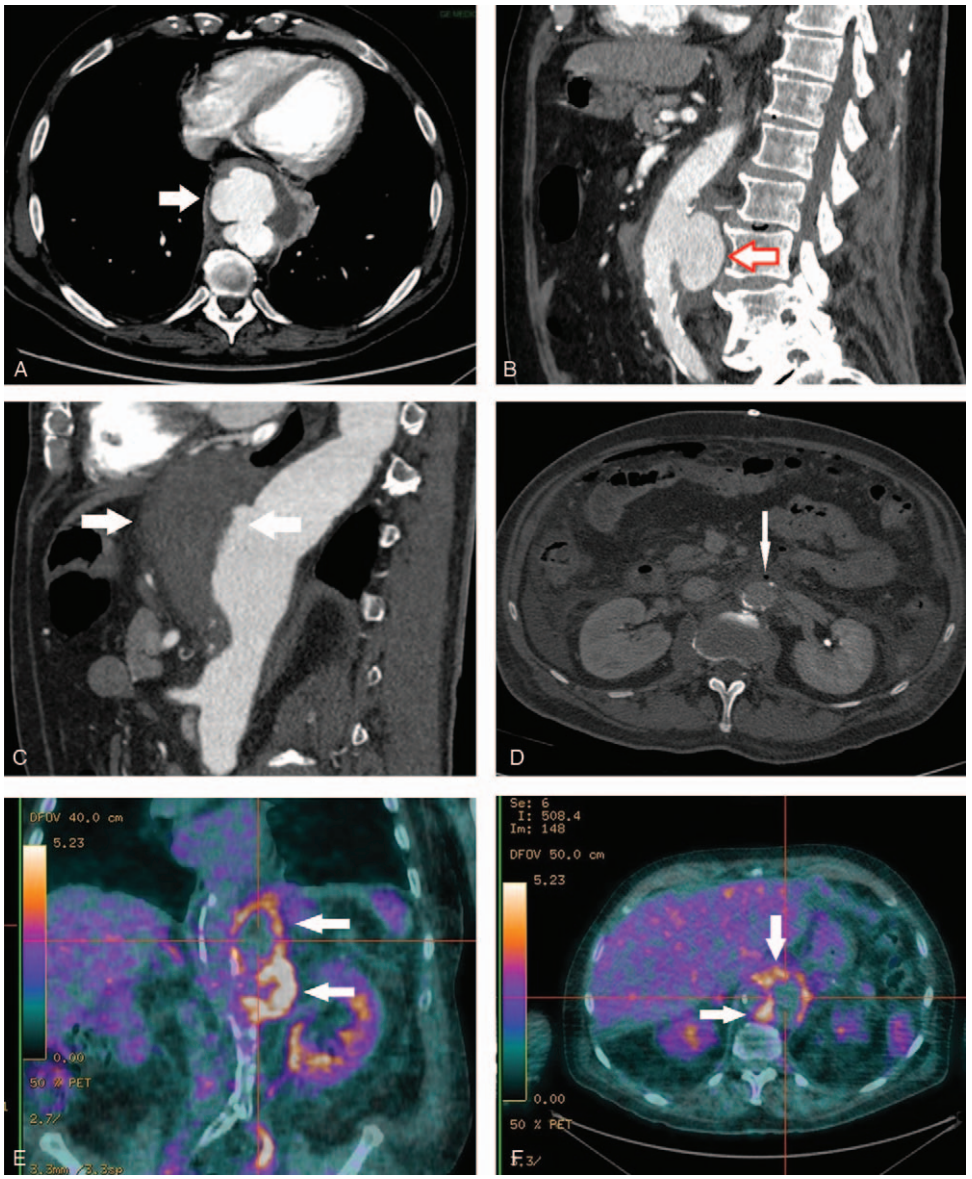

Figure 1. Imaging findings of infectious aortitis. (A) CT axial cut-section of descending thoracic aorta showing false anterior sacciform aneurysm. (B) CT sagittal reconstruction of abdominal aorta showing false posterior sacciform aneurysm. (C) CT sagittal reconstruction of thoracoabdominal aorta showing anterior contained rupture (hematoma). (D) CT axial cut-section of abdominal aorta showing wall pneumatosis (gaz bubble in the anterior segment). (E and F) PET-CT coronal and axial reconstruction showing intense hypermetabolism of the aortic wall. CT = computed tomography, PET-CT = positron emission tomography-computed tomography.

Table 2

Surgical treatment details.

	Thoracic only (n = 15)	Abdominal only (n = 31)	Thoracic & abdominal (n = 9)	TOTAL (n = 55)
Aortic surgery	13 (87%)	28 (90%)	6 (67%)	47 (85%)
Open surgery only	8 (53%)	27 (87%)	6 (67%)	41 (74%)
In situ reconstruction with allograft	3	15	4	22
In situ reconstruction with prosthesis	5	5	2	12
EVAR only	4 (27%)	0	0	4 (7%)
Combined	1 (7%)	1 (3%)	0	2 (4%)

EVAR = endovascular aneurysm repair.

and leads to important and asymmetric deformation of the aortic wall developing into an aneurysm. The sacciform development of the aneurysm could be secondary to the infectious inoculum which alters one side of the aortic wall.

PET-CT allows for positive diagnosis in case of lack of standard CT signs of aortitis, due to greater sensibility, and could be used for the follow-up after antibiotic therapy or vascular prosthesis implanted in septic circumstances.[14,41,42]

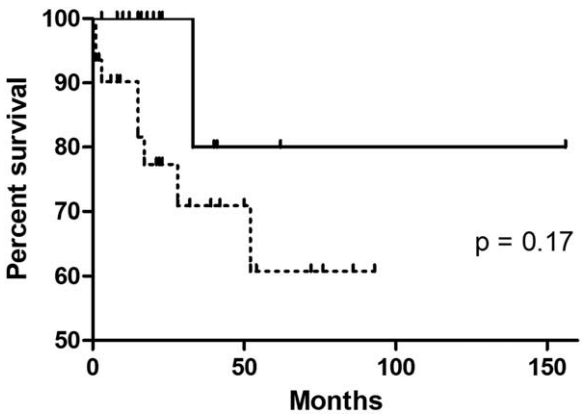

Figure 2. Survival curves of infectious aortitis (IA). Pyogenic and fungal IA curve is represented in solid line and intracellular growth and/or fastidious microorganisms IA curve in dashed line. IA = infectious aortitis.

4.2. Microbiology

The microorganisms' distribution in this series is quite different from others in the literature where *Salmonella spp*. is almost exclusively predominant. Contrary to the 2 major series after that of Miller et al – those of Huang et al (2014) et Dubois et al (2010)[4,19] – this series finds a predominance of *Streptococcus spp*., mainly *S pneumoniae*. This series reports a substantial number of *C burnetii*, higher than that of *Salmonella spp*. Better sanitation may account for the change in microorganisms' distribution and the lower prevalence of *Salmonella spp*.; Miller et al suggested that *Salmonella spp*. IA was encountered more frequently in low sanitation countries than in high-income countries.[1] Furthermore, improvement in microbial diagnostic techniques makes it easier to detect germs like *C burnetii*. Thereby, Broos et al reported a series of 122 cases of Q fever vascular complications: 14 thoracic IA (11.5%), 10 supra-renal abdominal IA (8.2%), and 94 infra-renal abdominal IA (77%); the infection concerned pre-existing prosthesis in 62 (50.8%) of these 122 cases.[43] This germ's reservoir is animal – mainly cows, goats, and sheeps – and; therefore, farmers are the most exposed.

All our patients with *C burnetii* IA were recruited by the 3 hospital centers of Western France which cover both urban and rural populations and where Q fever is endemic due to dense livestock farming, therefore accounting for the relatively high frequency of this germ in our series.

Syphilitic aortitis is now rare, but must still be evoked when facing a thoracic aneurysm,[2] as is TB aortitis. It should be noted that positive TB aortitis is frequently difficult to formally affirm, with only 20% of cases in this series. Delaval et al reported their experience of 11 TB aortitis and already noted that definite microbial diagnosis was available for only 3 (27%). The difficulty in isolating *M tuberculosis* can be explained by low inoculum in the low oxygenated aortic wall and; therefore, the diagnosis usually rests on converging clinical, biological and pathological arguments combined with full recovery under TB treatment.[44]

We had very few IA secondary to an infection in another organ; therefore the infection's mechanism is first and foremost hematogenous. However secondary extension to peri-aortic organs is to be feared (muscular psoas abscesses, spondylodiscitis) and can lead to potentially lethal complications like an aorto-digestive tract fistula[5,9,16,43] with, in the present series, 1 hemorrhagic shock due to an aorto-jejunal fistula.

Table 3
Main characteristics of infectious aortitis (IA) in largest case series of IA.

	Present series	Huang et al (2014)	Dubois et al (2010)	Molacek et al (2014)	Delaval et al (2017)	Luo et al (2018)	Miranda et al (2010)
n= (% of men)	55 (85%)	43 (81%)*	44 (86%)	17 (53%)	11 (27%)	NA	10 (80%)
Age (yr)	Median 65 (range 21–88)	Mean 63.5 (range 41–81)	Mean 67.3 (range 42–84)	Mean 73 (range 58–90)	Median 44.6 (range 16–75)	NA	Mean 61.7 (range 50–82)
Micro-organisms	12 *S pneumoniae* (21.8%)	36 *Salmonella* spp. (81.8%)	13 *Salmonella* spp. (29.5%)	7 *Salmonella* spp. (41.2%)	Tuberculosis	NA	3 *S pneumoniae* (30%)
	9 *S aureus* (16.3%)	8 other / no precision (18.2%)	10 other (23%)	3 no germ found (17.6%)			2 *Salmonella* spp. (20%)
	8 *C burnetii* (14.5%)		9 no germ found (21.4%)	2 Mixed / no precision) (11.8%)			2 *S aureus* (20%)
	5 *Campylobacter spp* (9.1%)		7 *S aureus* (15.9%)	2 *S aureus* (11.8%)			2 other Streptococci (20%)
	5 Tuberculosis (9.1%)		3 *E coli* (6.8%)	1 *K pneumoniae* (5.9%)			1 *E cloacae* (10%)
	3 *Salmonella* spp. (5.4%)		3 *S pneumoniae* (6.8%)	1 *L monocytogenes* (5.9%)			
	3 other Streptococci (5.4%)		3 *Streptococcus* other (6.8%)	1 *C albicans* (5.9%)			
	10 other (18.2%)						
Anatomical lesions	31 abdominal (56%)	10 thoracic (22.7%)	42 abdominal (95.5%)	NA	5 thoraco-abdominal (45.4%)	(10 abdominal aorta	8 abdominal (80%)
	15 thoracic (27%)	6 thoraco-abdominal (13.6%)	2 thoraco-abdominal (4.5%)		4 thoracic (36.3%)	& 4 left common iliac artery)	1 thoracic (10%)
	9 thoraco-abdominal (16%)	28 abdominal (63.6%)*	100% aortic aneurysm		2 abdominal (18.2%)	100% aneurysm	1 thoraco-abdominal (10%)
	51 aneurysm (93%)	100% aortic aneurysm			7 pseudo-aneurysm (64%)		Sacciform in 90% of cases
					4 increased wall thickness and/or stenosis and/or periaortitis (36%)		
Surgical treatment	41 open surgery (74.5%)	29 open surgery (66%)	43 open surgery (97.7%)	14 open surgery (82.6%)	8 open surgery (73%)	100% EVAR	100% open surgery
	8 medical only (14.5%)	11 EVAR (25%)	EVAR = 1 (2.3%)	2 medical only (11.8%)	3 medical only (27%)		
	4 EVAR (7%)	4 medical only (9%)		1 EVAR (5.9%)			
	2 mixed (4%)						
Thirty-day mortality	16%	NA	18.2%	5.9%	0	0	10%

EVAR = endovascular aneurysm repair, NA = not available.
* Forty-three patients for a total of 44 mycotic aortic aneurysm, hence the total of 44 in the microorganism case.

The pathology study contributes greatly to the diagnosis mainly in case of TB aortitis[44] even though granulomatous aortitis with giant cells can also be encountered in idiopathic or ACG-associated aortitis. On the other hand pathology study was not particularly useful for all the other microorganisms' IA of this series, and when Miller et al evidenced 6 different pathological profiles in their study of 29 IA, none was pathognomonic of any microorganism.[1]

4.3. Prognosis and surgical management

The prognosis of IA is severe, particularly in case of pyogenic IA where the absence of surgical management leads to short-term death.[2,5,19] Once surgery has been successful, the type of microorganism involved – pyogenic or ICGF – does not seem to affect mortality. EVAR procedure alone concerned only 4 patients, who were old and suffered from thoracic IA only. Indeed, surgeons shun fixing foreign material upon contact of an infectious source[4] and this could account for the low number of patients treated with EVAR. In urgent circumstances, when facing a fragile patient presenting with thoracic IA – whose surgical approach is more delicate than abdominal IA – EVAR may prevent short-term death by aneurysm rupture or aortic perforation. Prolonged follow-up through imaging is then essential in order to detect any precocious relapse.

4.4. Limits

This study has 2 mains limits: lack of exhaustive data due to retrospective recruitment and difficulty in identifying all IA cases due to absence of specific diagnostic coding and the multiplicity of microbial diagnostic modalities (standard culture, serology, PCR).

5. Conclusion

IA is a rare and serious disease, with surgery in 85.5% of cases and death in 30%. Its prognosis appears more severe than idiopathic or GCA-related aortitis. Pre-existing aneurysm was present in 18% of cases. The abdominal aorta was more usually concerned. In this study, the microorganisms' distribution was quite different from others in the literature with a majority of *S pneumoniae* and *Staphylococcus spp*., and most of all an important proportion of *C burnetii*. TB and syphilitic aortitis are still encountered in IA although they are rare. CT was the key-element for positive diagnosis of IA and blood cultures usually disclosed the microorganism's identification in case of pyogenic IA (staphylococci, streptococci, salmonellae, etc), contrary to ICGF IA (*C burnetii*, syphilis) where serologies or PCR lead to the diagnosis. TB IA's diagnosis remains challenging. Clinical presentation was more severe in case of pyogenic IA compared to ICGF IA, but with no difference in mortality. The great majority of patients underwent conventional surgery and few had EVAR. Studying prospective cohorts of IA could help better define therapeutic managements and notably the positioning of the different surgical procedures, as well as imaging follow-up.

Author contributions

Louis Journeau: study concept and design; acquisition of data; analysis and interpretation of data; drafting of the manuscript. Marine de la Chapelle, Thomas Guimard, Yasmina Ferfar, David Saadoun, Isabelle Mahé, Yves Castier, Philippe Montravers, Xavier Lescure, Damien Van Gysel, Nathalie Asseray, Jean-Baptiste Lascarrou, Yves-Marie Vandamme, MDc, Jérôme Connault, Patrick Desbordes de Cepoy, Julia Brochard, Yann Goueffic, Marc-Antoine Pistorius: acquisition of data and critical review; Chan Ngohou: methodology; David Boutoille analysis and interpretation of data Olivier ESpitia: study concept and design; acquisition of data; analysis and interpretation of data; drafting of the manuscript; supervision

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