

INFECTIOUS ABDOMINAL AORTITIS: A REVIEW OF THE LITERATURE

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Abstract- Infectious abdominal aortitis is a clinical entity rarely seen today in the era effective antibiotic therapy. It usually occurs with aneurysm formation, though non aneurysmal aortitis also occurs.

The most common causative bacteria are E coli, Strep pneumoniae, Staphylococcus aureus and Salmonella species. Fever and abdominal or back pain are the most common clinical features. CT angiogram is usually the first choice imaging modality, Treatment is with antibiotics, removal of the infected aortic segment followed by revascularization. This is a brief review of the epidemiology, pathogenesis, clinical features, diagnosis and management of infectious aortitis and its complications.

Keywords- Abdominal aortitis, mycotic aneurysm, endovascular repair, aortic grafts

Introduction

Infectious abdominal aortitis or mycotic aneurysms are a rare entity in the current era of antibiotics. The term mycotic aneurysm was coined by Osler in 1885 to describe an aneurysm associated with endocarditis [1]. They represent 0.5-1.3% of all operated aneurysms [2].

Infectious abdominal aortitis and mycotic or infected aneurysms represent two extremes of the same disease. If left untreated, it is likely that an infected non-aneurysmal aorta will develop an aneurysm. Conversely, an abdominal aortic aneurysm has a greater chance of becoming infected than a normal aorta.

Epidemiology

Studies have shown a male preponderance in the incidence of abdominal aortic aneurysms. The male to female ratio varies from 30:1 to 6:1 depending on the study and the incidence is higher in smokers [3].

Pathogenesis

The aortic intima is generally resistant, however if this barrier is disrupted by atherosclerosis or trauma, it is more prone to infection. The mechanism of infection is usually in one of four ways. The most common mechanism of infection is hematogenous with bacteremic seeding of an aorta weakened by pre existing atherosclerosis, infection or aneurysms. The primary source of infection may be pneumonia, endocarditis, urinary tract infection, osteomyelitis, cellulitis or intravenous (IV) line sepsis. Mycotic aneurysms may also occur from direct introduction of bacteria into the vasculature through

intravenous drug use, intravascular interventions and monitoring. They may also occur through direct spread from a contigious source like osteomyelitis or mediastinitis. The fourth mechanism of spread is through the vasa vasorum releasing infected emboli into the aortic wall [4].

Other definite risk factors are infectious endocarditis, abdominal aortic manipulation during endovascular procedures, arterial trauma, infected prosthetic bypass grafts [5], congenital aortic anomalies and medical conditions causing immmuno supression like Human immunodeficiency virus (HIV) infection, long term steroid or immunosuppressant use, malignancy alcoholism, chronic renal failure, diabetes and stroke [6]. Radiation and Graft versus host disease have also been associated as risk factors [7].

Microbiology

The most common organisms that cause infectious abdominal aortitis are Gram negative rods (54%) and Gram positive cocci (27%). The most common pathogens are Salmonella (35%), Staphylococcus and streptococcus pneumoniae [11]. Salmonella aortitis usually occurs in patients with pre existing vascular disease [8].

However, aortitis with *Neisseria gonorrheae*, *Clostridium septicum* [26], *Streptococcus agalacticae* [6], *Klebsiella pneumoniae* [10] *Legionella pneumophilia* [11] *Enterobacter aerogenes* [12] and *Campylobacter coli* [24] have also been reported. Gram-negative aortitis has been shown to be a more virulent form of the disease then that caused by gram-positive organisms, with a higher rate of rupture (72% vs 25%) and death [8].

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Streptococcus agalacticae

Source of Infection: Genital tract, osteomyelitis gastrointestinal tract, inguinal lymphadenitis, Infected epidermal inclusion cyst.

Patient Characteristics: 69 years old male with diffuse abdominal pain and constipation.

Antibiotic Rx: Penicillin G 2.4 million units/day for 2 weeks Ceftriaxone 2 gm IV /day with Moxifloxacin 400 mg/day 4 weeks and low dose aspirin plus Po Moxi for 6 months [6].

Salmonella sp

Source of Infection: Gastroenteritis, Spondylodiscitis

Patient Characteristics: 55 years old male with atherosclerosis.

Antibiotic Rx: IV Ampicillin 3 weeks Ceftriaxone or Ciprofloxacin [8].

Streptococcus pneumonia

Source of Infection: Pneumonia.

Patient Characteristics: 74 years old male with fever, pelvic pain , anorexia and weight loss.

Antibiotic Rx: Gatifloxacin for 6-12 weeks after surgical debridement and clearance of blood cultures [12].

Staphylococcus aureus

Source of Infection: Unknown source, Possibly urinary tract

Patient Characteristics: 74 years old male with fever and back pain.

Antibiotic Rx: MSSA: Oxacillin 6-8 weeks IV antibiotics plus 4-6 weeks oral Abx [15]; MRSA: vancomycin plus daptomycin

Escherichia coli ESBL

Source of Infection: Urinary tract infection, Meningitis

Patient Characteristics: 59 year old male with headache, nausea vomiting and abdominal tenderness.

Antibiotic Rx: Meropenam 6 g/day and Ciprofloxacin 1.2 g/ day [14].

Campylobacter coli

Source of Infection: Spondylodiscitis and pre vertebral and paravertebral abscess.

Patient Characteristics: 72 year old male with lower back pain and fever.

Antibiotic Rx: Cephalosporin and gentamicin followed by prolonged course of Ciprofloxacin [24].

Clostridium septicum

Source of Infection: Source unknown, possibly intra abdominal.

Patient Characteristics: 68 year old male with abdominal pain, nausea and vomiting.

Antibiotic Rx: Penicillin G IV followed by oral Amoxicillin plus Clindamycin [26].

Aortic infection by mycobacterium tuberculosis is a rare cause in developed countries but is more common in countries where tuberculosis is endemic, usually occurring from a direct extension from an infected lymph node or lung lesion [2].

Clinical Features

The most common clinical manifestations of infectious abdominal aortitis are fever (73%), abdominal pain (33%), back pain (50%)

testicular pain (10%) and a pulsatile abdominal mass (3%) [13]. Patients with infectious aortitis alone, without aneurysm formation are less likely to be symptomatic. They may also present with compressive symptoms such as dysphagia, dyspnea, hoarsness, cough and rarely, superior vena caval (SVC) syndrome, though these are more likely with thoracic aortitis. The natural progression of aortitis is usually rapid dilatation of the aorta with aneurysm formation. This may progress to aneurysmal rupture and bleeding, intravascular thrombosis, aortic dissection, aortic insufficiency, emphysematous aortitis septic embolisation or acute coronary syndromes. Anuria may be a rare presentation of abdominal aortitis [29].

Diagnosis

It is essential to diagnose acute abdominal aortitis early as there is a high mortality rate associated with it. Non-specific symptoms and signs make it a difficult to diagnose this condition and a high index of suspicion is required. Leucocytosis is usually present with an elevation in acute phase reactants like C reactive protein (CRP) and Erythrocyte sedimentation rate (ESR).

Blood cultures are positive 50-85% of the time. The most common organisms isolated are Gram negative bacilli (around 60) more than half are caused by non typhoid Salmonella and Gram positive bacilli (35%).

Transesophageal echocardiogram is often the first imaging modality to be performed since it is usually done to rule out infectious endocarditis in the setting of positive blood cultures. If the heart valves do not show any evidence of endocarditis, it should prompt examination of the aorta for an aneurysm and /or vegetations [18]. Doppler can improve structural identification and distinguish between an intramural hematoma, aneurysm or pseudoaneurysm.

Abdominal angiogram using computerized tomography (CT) is the most sensitive imaging. Several signs such as thin aortic wall with inhomogeneous contrast enhancement, increased wall diameter, sacciform aneurysm, abscess, and sometimes aortic rupture suggest aortic wall infection. The presence of air in the aortic wall is pathognomonic of infected aortitis. Other signs like peri-aortic lymphadenopathy, nodularity, irregular enhancement of the aortic wall and peri vascular inflammation or a contiguous focus of infection seen on CT will aid in the diagnosis [9]. There may also be a thin hypodense rim in the aortic wall suggesting edema or necrosis which helps to helps localize the pathologic process to the aorta rather than to the retroperitoneum [28].

Magnetic resonance Imaging (MRI) with gadolinium contrast may also be used as a method of diagnosis. The advantages are a view of the entire aorta with excellent resolution. The major limitations are the lack of availability and the inability to perform an MRI in an unstable patient.

Invasive aortography is considered the gold standard and is usually reserved for cases where the diagnosis cannot be made by non invasive methods because it carries the risk of rupture of the aortic wall. Imaging is limited to only the aortic lumen and not the wall [9].

Management and Treatment

The management of infectious aortitis involves 3 parts: treatment with appropriate antibiotics, removal of the infected aortic segment and revascularization.

As soon as the diagnosis of infectious aortitis is suspected, broad spectrum antibiotics should be initiated. However, treatment with antibiotics alone is not sufficient and case reports have shown that

World Research Journal of Medicine Volume 1, Issue 1, 2013 all patients treated with antibiotics alone without aortic replacement died [16,17]. Antibiotic coverage should be extended for six to twelve weeks after surgical excision and clearance of blood cultures [18].

There are two main surgical approaches for the repair of infectious abdominal aortic aneurysms : extra-anatomic bypass (EAB) and in situ replacement. An EAB with ligation of the aorta has been considered the traditional standard management. Extra-anatomic bypass has the advantage of reducing the risk of graft infection. However, EAB has several disadvantages compared with in situ bypass, including a relatively high incidence (20%) of aortic stump disruption, and a higher rate of amputation (20-29%) because the procedure has a long bypass and patency is poor. Moreover, the procedure leads to a compromised blood supply to the pelvis and sigmoid colon and rectum [20-22].

Recent studies have reported that the in situ replacement may be a more appropriate choice for IAAA. During in situ replacement, various graft materials are used to repair the aneurysm. The procedure has been shown to have acceptable results, including lower reinfection and mortality rates. Various graft materials have been used, including cryopreserved arterial allografts, superficial-femoral popliteal veins rifampicin impregnated polyester prosthesis and silvercoated polyester. However, prosthetic Grafts should be avoided in significantly virulent or antibiotic resistant strains of bacteria because of the risk of re infection of the prosthetic graft. In such cases, a homograft from the aorta or the deep veins of the legs may be preferred [17]. For thoraco abdominal and suprarenal aortic aneurysms an endovascular repair is preferred, since the mortality is much lower than with an open surgical procedure. However, for infra renal aneurysms, both kinds of procedures may be performed. Endovascular repair is also preferred among elderly patients and is associated with less morbidity and mortality than an open repair [23].

Prognosis

The prognosis of infectious abdominal aortitis seems to depend on the treatment, survival varies between 84% [25] and between 70-100% for aggressive treatment with both surgery and antibiotics. [27] Medical or surgical treatment alone can have a high mortality rate of 90-100% [25]. Early resection of an infected aortic segment before aneurysm formation and rupture is likely tolead to a better prognosis [19]. Long term follow up is important, and if the patient develops abdominal pain, fever leucocytosis or an elevated CRP, suspicion for reinfection should be high [17].

Conclusion

In conclusion, given its high mortality, a high index of suspicion is required for diagnosing infective abdominal aortitis. It should be suspected in a patient presenting with abdominal or back pain and persistent fever, especially elderly males with atheromatous disease. Is suspected All patients should get an abdominal CT angiogram immediately to evaluate for aortic disease and be rescanned if the symptoms persist, even if the first scan is negative [17].

Conflicts of Interest: None declared.

References

- [1] Osler W. (1885) British Medical Journal, 1(1263), 522.
- [2] Revest M., Decaux O. and Cazlets C. (2007) *Rev Med Int.*, 28, 108-115.

- [3] Von Allmen R.S. and Powell J.T. (2012) The Journal of Cardiovascular Surgery, 53(1), 69-76.
- [4] Rabitsch W., Brugger S.A., Trubel W., Keil F., Greinix H.T. and Kalhs P. (2002) *Transplantation*, 74(7), 1048-1050.
- [5] Rozenblit A., Bennett J. and Suggs W. (1996) Abdominal imaging, 21(6), 512-514.
- [6] Chandrikakumari K., Giot J.B., de Leval L., Creemers E., Leonard P., Mukeba D., Moutschen M., Frippiat, F. (2008) *International Journal of Surgical Pathology*, 16(3), 314-319.
- [7] Lopes R.J., Almeida J., Dias P.J., Pinho P. and Maciel M.J. (2009) Clinical Cardiology, 32(9), 488-490.
- [8] Cicconi V., Mannino S., Caminiti G., Cuoco L., Gasbarrini A., Vecchio F. and Gasbarrini G. (2004) Angiology, 55(6), 701-705.
- [9] Kario K., Mizuno Y., Kanatsu K., Tankawa H. and Ikeda M. (1991) Clinical Imaging, 15(4), 261-264.
- [10]Narang A.T. and Rathlev N.K. (2007) The Journal of Emergency Medicine, 32(4), 359-363.
- [11]Guyot S., Goy J.J., Gersbach P., Jaton K., Blanc D.S. and Zanetti G. (2007) *Transplant Infectious Disease*, 9(1), 58-59.
- [12]Rondina M.T., Raphael K., Pendleton R. and Sande M.A. (2006) Journal of General Internal Medicine, 21(7), C1-C3.
- [13]Cartery C., Astudillo L., Deelchand A., Moskovitch G., Sailler L., Bossavy J.P. and Arlet P. (2011) *Annals of Vascular Surgery*, 25(2), 266-e9.
- [14] Johnstone J.K., Garcia-Toca M., Slaiby J.M., Marcaccio E.J. and Chong T.T. (2012) *Journal of Vascular Surgery*, 55(6), 1779-1781.
- [15]Stephens C.T., Pounds L.L. and Killewich L.A. (2006) Angiology, 57(4), 506-512.
- [16] Ioannidis J., Merino F., Drapkin M.S., Lew M.A. and Cohn L.H. (1995) Archives of Internal Medicine, 155(15), 1678-1680.
- [17]Worrell J.T., Buja L.M. and Reynolds R.C. (1988) *Am. J. Clin. Pathol.*, 89, 565-568.
- [18]Lopes R., Almeida J., Dias P., Pinho P. and Maciel M.J. (2009) Cardiology Research and Practice, 2009.
- [19]Weyrich P., Ettahar N., Legout L., Meybeck A., Leroy O. and Senneville E. (2009) Ann. Clin. Microbiol. Antimicrob., 11(4).
- [20]Gornik H.L. and Creager M.A. (2008) *Circulation*, 117(23), 3039 -3051.
- [21]Maeda H., Umezawa H., Goshima M., Hattori T., Nakamura T., Umeda T. and Shiono M. (2011) Surgery Today, 41(3), 346-51.
- [22]Oderich G.S., Panneton J.M., Bower T.C., Cherry Jr K.J., Rowland C.M., Noel A.A. and Gloviczki P. (2001) *Journal of Vascular Surgery*, 34(5), 900-908.
- [23]Pol R.A., Reijnen M.M. and Zeebregts C.J. (2011) Surgery, 149 (6), 855-856.
- [24]Lemaire X., Dehecq C., Cattoen C., Garnier L.D., Bournet B.S., Yazdanpanah Y. and Senneville E. (2010) Annals of Clinical Microbiology and Antimicrobials, 9(1), 8.
- [25]Nijs A., Vandekerkhof J. and Cartuyvels R. (2002) Eur. J. Microbiol. Infect. Dis., 21, 389-392.
- [26]Annapureddy N., Agarwal S.K., Kanakadandi V., Sabharwal M.S., Ammakkanavar N., Simoes P. and Nadkarni G.N. (2012) *Journal of Infection and Chemotherapy*, 18(6), 948-950.

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- [27]Cani C.S., Arena G.O. and Fiture A.O. (2001) J. Vasc. Surg., 33, 861-867.
- [28]Rozenblit A., Bennett J. and Suggs W. (1996) Abdominal Imaging, 21(6), 512-514.
- [29]da Gama A.D., Martins C., Pedro L.M., Evangelista A., Almeida P., Gimenez J.L. and Rodriguez J.M. (2003) *Revista portuguesa de cirurgia cardio-toracica e vascular: orgao oficial da Sociedade Portuguesa de Cirurgia Cardio-Toracica e Vascular,* 11(3), 155-159.