Clinical Imaging 40 (2016) 256–262

Contents lists available at ScienceDirect

Clinical Imaging

journal homepage: http://www.clinicalimaging.org

Review Article

Diagnosis and management of mycotic aneurysms☆

Amy R. Deipolyi [a], Jun Rho [b], Ali Khademhosseini [c,d,e], Rahmi Oklu [c,f,*]

[a] Division of Vascular and Interventional Radiology, Department of Radiology, New York University Medical Center, New York, NY
[b] Massachusetts General Hospital, Harvard Medical School, Vascular and Interventional Radiology, Boston, MA
[c] Biomaterials Innovation Research Center, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Cambridge, MA
[d] Harvard-MIT Division of Health Sciences and Technology, Massachusetts Institute of Technology, Cambridge, MA
[e] Wyss Institute for Biologically Inspired Engineering, Harvard University, Boston, MA
[f] Mayo Clinic, Division of Vascular and Interventional Radiology, Scottsdale, AZ

ARTICLE INFO

Article history:
Received 18 August 2015
Received in revised form 4 November 2015
Accepted 23 November 2015

Keywords:
Mycotic aneurysm
Endovascular
Infection
CT imaging

ABSTRACT

Mycotic aneurysm (MA) is a focal dilation of an infected arterial wall. This uncommon disease follows an aggressive, unpredictable clinical course with significant mortality and presents unique diagnostic and therapeutic challenges. This review discusses the pathogenesis and the diagnostic challenges of MA.

© 2015 Elsevier Inc. All rights reserved.

1. Historical perspective

Virchow first described a saccular outpouching along an arterial wall corresponding to an embolic source in 1847 [1]. Mycotic aneurysms (MAs) were initially considered a physical change secondary to mechanical pressure of emboli against the artery surface [2]. An infectious etiology was first hypothesized by Goodhart in 1877 [3]. The term 'mycotic' originates from Osler's Gulstonian Lectures in 1885 in which he associated aortic aneurysms resembling a fungus growth with a severe or "malignant" form of endocarditis and coined the term "mycotic aneurysms" [4]. The term "mycotic" is a misnomer suggesting fungal infection, though it was originally intended to refer to all microorganisms [5]. Osler established a clear connection between endocarditis and its bacterial origin ("micrococci") through histology using Gram staining, novel at that time in medical practice. He defined the mycotic process in the setting of endocarditis as the transference of microbes from the growth of the valves to distant parts. In 1887, Langton and Bowlby corroborated Osler's findings with observations of numerous bacteria derived from the heart valves in MA walls [6].

2. Prevalence

MA is rare, constituting only 1% of all surgically corrected aortic aneurysms [7–9] and 1–4% of all patients with intracranial aneurysms [10,11]. Like its noninfected counterpart, MA is more prevalent in men [7,12,13]. In one postmortem study of over 22,000 patients, 9 aortic MAs (0.04%), 6 of which ruptured, were encountered in 338 aortic aneurysms [12]. In another postmortem study of over 20,000 patients, 6 MAs (0.03%), 4 of which ruptured, were encountered out of 178 abdominal aneurysms [14]. These large studies collectively demonstrate the rarity of MAs and highlight the importance of accurate diagnosis as they are prone to lethal rupture.

MAs usually involve the aorta most frequently and the femoral, visceral (superior mesenteric, splenic, and hepatic), and cerebral arteries [15–17]. Interestingly, intracranial MAs were more likely to be present at more than one site in an individual. In a more recent study of 243 MAs, the femoral artery was the most prevalent site followed by abdominal aorta, superior mesenteric artery, brachial artery, iliac artery, and carotid artery [15]. Although the latter study did not include thoracic MAs, changing anatomic distribution of MAs may reflect evolving etiologies.

Most of the earlier MAs were associated with endocarditis, as noted in 187 of 217 cases (86%) in 1923 [17]. Since the introduction of organism-specific antibiotics, the prevalence of endocarditis and its complications has drastically decreased. However, increasing intravascular procedures and intravenous drug abuse has led to greater predominance of MAs caused by arterial trauma [15]. Similarly, the bacteriology of MAs reflected that of endocarditis including β-hemolytic group A streptococci, pneumococci, and *Haemophilus* influenza before the antibiotic era [5]. In

☆ Conflict of interest disclosure: The authors declared no conflicts of interest and have no financial disclosures.

* Corresponding author. Mayo Clinic, Division of Vascular and Interventional Radiology, 13400 E Shea Blvd, Scottsdale, AZ 85259, USA. Tel.: +1-480-342-1650.
E-mail address: oklu.rahmi@mayo.edu (R. Oklu).

http://dx.doi.org/10.1016/j.clinimag.2015.11.011
0899-7071/© 2015 Elsevier Inc. All rights reserved.

the postantibiotic era, *Staphylococcus* and *Salmonella* are the most common infecting organisms.

3. Pathogenesis

3.1. Arterial wall degeneration and extracellular matrix degradation

As with noninfected aneurysms, the degeneration of the arterial wall plays an important role in the development of MA [17,18]. The rapid progression of MAs as opposed to noninfected aneurysms is a result of acute inflammatory processes in response to bacterial infection, histologically demonstrated by numerous polymorphonuclear neutrophils in the arterial wall [12] (Fig. 1). Bacterial infection stimulates proinflammatory mediators attracting neutrophils into the arterial wall. There is characteristic neutrophilic infiltrate on histopathology, typically accompanied by with profound vascular injury [19,20]. In turn, neutrophils and microbial factors synergistically activate various elastolytic and collagenolytic enzymes leading to the focal breakdown of the arterial wall and saccular dilation of the vessel wall [21–23]. During acute phases, a focal granulomatous reaction to infecting organisms may be seen histopathologically. Chronically, lymphoplasmacytic infiltrates and fibrosis may be noted. Chronic Gram-positive bacterial infections may induce plasma cells IgG4 expression [20].

MAs have a characteristic saccular appearance likely due to the rapid focal degeneration of the arterial wall in the absence of adequate remodeling (Fig. 1A). In animal models, elastase as opposed to collagenase has been shown to induce the formation of saccular aneurysms [24,25]. Others have pointed out the importance of collagenase activity as a critical factor leading to the rupture of aneurysm [26,27]. Given the high frequency of rupture in MAs, both elastase and collagenase are likely key players in the pathogenesis of MAs and their subsequent rupture. Recent data show that elastolytic matrix metalloproteinases are associated with aneurismal rupture and could in the future provide prognostic information regarding risk of rupture in patients with aneurysms [28].

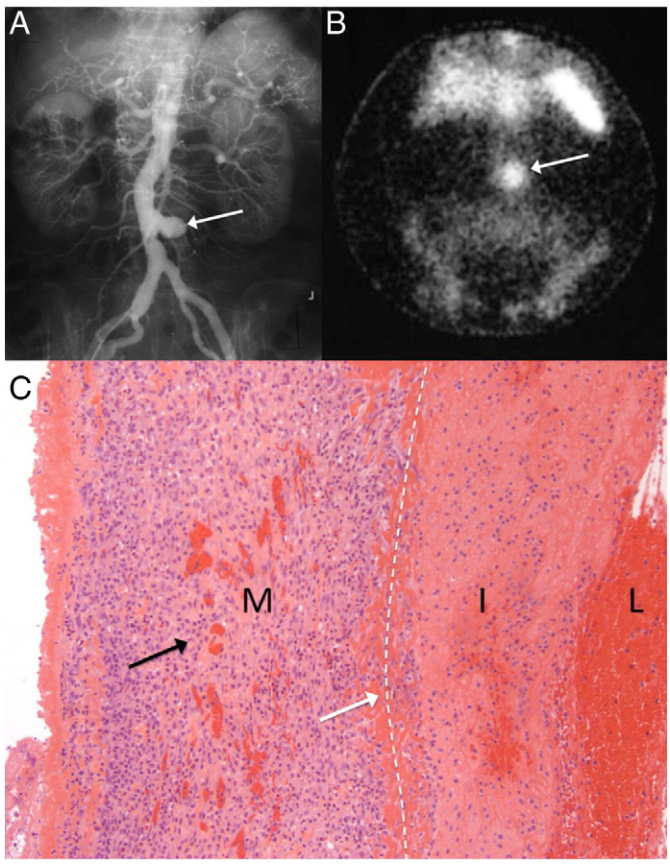

Fig. 1. Saccular MA in the abdominal aorta. (A) Abdominal aortogram demonstrates a large saccular aneurysm (white arrow). (B) ^{111}In-labeled WBC study shows a focal hotspot (white arrow) within the saccular MA in (A). Following postsurgical repair, hematoxylin and eosin staining of the resected aneurysmal tissue reveals extensive infiltration of WBCs, obliteration of the internal (white arrow) and external elastic membranes, and foci of hemorrhage (black arrow) within the media layer (M) of the arterial wall. I, intima; L, lumen; magnification, ×100.

3.2. Etiology of infection

The source of infection is clear in direct bacterial inoculation of the vasculature. A direct arterial injury and subsequent bacterial infection may result from self-inflicted needle injection, medically iatrogenic, or accidental physical trauma. Similarly, contiguous infection from an adjacent extravascular infection extending to the arterial wall may occur [29,30].

Two possible routes of arterial wall infection from a distant infectious focus are septic embolism and bacteremia. The development of MAs from septic embolism at the site of vasa vasorum is well described in the setting of endocarditis [4]. The histology typically shows neutrophils infiltrating from the adventitia, where the vasa vasorum are prominent, through the muscularis media and toward the internal elastic membrane [18,31]. Infection typically begins at a nidus in the vasa vasorum or on ulcerated atherosclerotic plaque luminal surface, and aneurysms usually form at a site of preexisting vascular dilation [20]. The fact that the veins do not develop MAs despite their denser vasa vasorum also supports septic emboli as a major source of MA. Potentially infected emboli are often filtered by the capillary beds of large organs such as the lungs, liver, and spleen where vessels are less than 10 microns in diameter.

MA formation in the setting of bacteremia alone constitutes a smaller portion of cases and therefore a rare clinical entity [15,17]. Considering how frequent bacteremia is, the low prevalence of MAs suggests a resistance of arterial walls to bacterial infection. The intact endothelial lining of the tunica intima is an effective barrier against blood-borne invasion. Also, the high velocity of arterial flow makes bacterial colonization difficult unless there is significant turbulence or jet lesion at the site of valvular insufficiency, coarctation, or fistula [5]. Immunologically, the reticuloendothelial system rapidly clears microorganisms from circulation. Taken together, direct colonization of bacteria on the intact large vessel lumen is extremely rare or nonexistent.

3.3. Vasa vasorum disruption

MA formation in the setting of bacteremia may involve infiltration of the vasa vasorum, which has been described in syphilitic aortitis as well as other MAs [8,12,15,17]. The obstruction of vasa vasorum followed by bacteremia, but not bacteremia alone, results in degeneration of the aortic wall [18]. Vasa vasorum are likely more susceptible to bacterial colonization owing to smaller lumen size and decreased flow rate compared to larger arteries; when disturbed, they are more likely to serve as an infectious nidus. Vasa vasorum tend to be easily disturbed, for, by example, rheumatic fever, cholesterol deposition, nicotine use, and vasospasm due to hypertension, emotional stress, or normal aging [16,32,33].

The vasa vasorum therefore appear to play a crucial role in the pathogenesis of MA in the setting of septic emboli or bacteremia. The vasa vasorum are more pronounced in larger arteries due to insufficient oxygen and nutrient diffusion from the lumen to the outer vessel layer. Consequently, MAs are seen more often in larger arteries or those rich with the vasa vasorum. Interestingly, the inflammatory process in the medium and large vessel vasculitis is also mediated through the vasa vasorum. However, vasculitis is predominantly a T-cell-mediated process that typically does not show severe degeneration of elastic lamina leading to aneurysms [34].

Download English Version:

https://daneshyari.com/en/article/4221203

Download Persian Version:

https://daneshyari.com/article/4221203

Daneshyari.com