Clinical Neurology and Neurosurgery 115 (2013) 1927–1942

Contents lists available at ScienceDirect

Clinical Neurology and Neurosurgery

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

Review

Intracavernous internal carotid artery mycotic aneurysms: Comprehensive review and evaluation of the role of endovascular treatment

Michael George Zaki Ghali [a,*], Emil Zaki Ghali [b,c]

[a] Department of Neurobiology & Anatomy, Drexel University College of Medicine, Philadelphia 19129, USA
[b] Department of Medicine, Inova Alexandria Hospital, Alexandria 22304, USA
[c] Department of Urological Surgery, El Gomhoureya General Hospital, Alexandria, Egypt

ARTICLE INFO

Article history:
Received 24 December 2012
Received in revised form 14 June 2013
Accepted 20 July 2013
Available online 15 August 2013

Keywords:
Cavernous carotid artery
Mycotic
Aneurysms
Endovascular
Review

ABSTRACT

Mycotic aneurysms may arise in the setting of many local or systemic infections. Those of the intracranial circulation are especially worrisome due to their potential to compress vital neural structures and their propensity for rupture with consequent hemorrhage. Mycotic aneurysms of the intracavernous internal carotid artery (ICA) represent an exceedingly rare clinical entity, described in less than fifty published cases. Typically presenting as a cavernous sinus syndrome with signs and symptoms of the underlying infection, they are often missed initially, with diagnosis and treatment commencing for the triggering infection or confused with cavernous sinus thrombophlebitis, which may be additionally coexistent, confounding timely diagnosis of the aneurysmal disease. Compared to non-mycotic aneurysms of the intracavernous ICA, which typically have a benign course, the infectious etiology of the mycotic variety increases their tendency to rupture, precludes surgical clipping as a viable treatment option, and requires institution of prolonged antibiotic therapy prior to definitive intervention. Their critical location, friability, and propensity to occur bilaterally result in an unpredictable risk of rapid neurological decline and death, making the timing and specific nature of treatment a unique dilemma facing the treating physician. This review seeks to discuss the natural history of and management strategies for mycotic aneurysms of the intracavernous ICA with special emphasis on the role, safety, and efficacy of endovascular therapies.

Contents

* Corresponding author at: Department of Neurobiology and Anatomy, Drexel University College of Medicine, 2900 Queen Lane, Philadelphia 19129, USA. Tel.: +1 703 577 4848; fax: +1 703 933 3837.
E-mail address: mgg26@drexel.edu (M.G.Z. Ghali).

0303-8467/$ – see front matter
http://dx.doi.org/10.1016/j.clineuro.2013.07.025

1. Introduction

1.1. Overview

Aneurysms arise in areas of vessel weakness resulting from a congenital, traumatic, or inflammatory (e.g., atherosclerotic or infective) etiology. When they occur in the systemic circulation, their main symptomatic presentations include rupture, thrombus formation, and thromboembolism [1]. When occurring in the intracranial circulation, they may cause symptoms by virtue of mass effect producing focal neurologic deficits, by irritation of pain-sensitive neural support structures as a result of expansion causing headache, by gliosis of adjacent parenchyma producing seizure foci, by thrombus formation and subsequent thromboembolism resulting in transient ischemic attacks and ischemic cerebral infarctions, and by rupture with consequent hemorrhage (i.e., subarachnoid, intraparenchymal, intraventricular). Any segment of the carotid artery may be affected by aneurysmal disease and aneurysms of the intracavernous internal carotid artery (ICA), or cavernous carotid artery aneurysms (CCAAs), have historically represented 5% [2] of all intracranial aneurysms with the reported proportion in recent years ranging from 8.3% [3] to 12.8% [4]; approximately 5% of these lesions are bilateral. CCAAs in turn represent 20–25% of cases of cavernous sinus syndrome [5–7]. Mycotic aneurysms account for approximately 2.5–6.2% [1,8–12] of intracranial aneurysms and, of these, less than fifty cases have been reported occurring in the intracavernous ICA.

1.2. Mycotic cavernous carotid artery aneurysms: a unique clinical entity

Several qualities distinguish intracavernous mycotic aneurysms from related cerebrovascular aneurysmal disease and confer significant clinical implications on feasibility and favorability of different management strategies. First, *non-mycotic* CCAAs traditionally exhibit a benign course [13] and typically have a lower rate of rupture than those elsewhere in the cerebral vasculature, with the rate of rupture mainly related to size (0% if < 12 mm) [3]. In contradistinction, the rupture risk of *mycotic* intracavernous aneurysms is less predictable and the clinical course potentially more catastrophic than the non-mycotic variety [14–18]. Also noteworthy is the relative infrequence of endocarditis as an etiologic agent in pathogenesis of mycotic CCAAs (Table 1), with non-endocarditic mycotic cerebral aneurysms possessing a special predilection for the cavernous ICA [19]. Moreover, on account of being located in a region densely packed with neurovascular elements, mycotic aneurysmal disease in the cavernous ICA is often missed initially with the patient diagnosed with the presenting syndrome in isolation (e.g., cavernous sinus syndrome, sinusitis) [20,21]; delay in diagnosis and treatment may increase patient morbidity (e.g., permanent ophthalmoplegia; [22,23]) and mortality.

Due to both their location in the cavernous sinus and friability, surgical clipping for these lesions is not a viable option and treatment is thus more challenging [2]. Given their frequent bilateral occurrence (Table 1) [24–30] (a likely consequence of left-right cavernous sinus venous communication, *see below*), the use of selective aneurysmal exclusion (i.e., via coil/balloon occlusion of the aneurysmal sac or stenting across the parent vessel) is far preferable to surgical ligation or endovascular complete carotid occlusion, and endovascular strategies have proven promising in this regard [29]. Interestingly, although cerebral mycotic aneurysms typically possess fusiform morphology [12], those involving the cavernous ICA are often saccular, facilitating the use of aneurysm-selective endovascular therapies. Finally, given the presence of infectious vasculitis, definitive treatment should be delayed until antibiotic coverage has been given ample time to clear the infectious process in order to (1) avoid colonization of the foreign body and (2) decrease the risk of iatrogenic rupture; but this also must be weighed against the potential need for urgent/emergent intervention [16,18,21], which is often difficult to predict in cases of mycotic CCAAs. The dynamic interplay of these factors makes intracavernous mycotic aneurysms an especially complex management dilemma, but at the same time, uniquely amenable to endovascular intervention.

2. Background

2.1. Pathology and pathogenesis

Karsner describes three broad classes of etiopathologic origins of bacterial intracranial aneurysms: embolic, extravascular, and primary/cryptogenic [31]. Embolic causes are typified by entities including infective endocarditis. Extension of meningitis, osteomyelitis of the skull, sinusitis, or cavernous sinus thrombophlebitis to vessels constitutes extravascular causes whereby infection spreads to and through the vessel wall contiguously. Primary/cryptogenic bacterial intracranial aneurysms are those for which no local or systemic source of infection can be detected clinically nor found on appropriate microbiologic studies. Another pathophysiologic mechanism embraced more recently involves lodging of microemboli in the *vasa vasorum*, causing weakening of the arterial media and aneurysmogenesis [32], analogous to the mechanism of *Treponema pallidum* in producing aneurysms of the aortic arch associated with endarteritis obliterans.

Elucidating the pathophysiology of extravascular etiologies of intracavernous ICA mycotic aneurysms is an autopsy study by Weisman [33] of cavernous sinus thrombophlebitis. In these individuals, neutrophil infiltration of the adventitia and media was evident, along with (reactive) intimal proliferation, suggestive of an associated subacute/chronic inflammatory focal angiitis that may predispose to vessel weakness and subsequent aneurysmal formation. Post-mortem examination of mycotic CCAAs reveals analogous histopathology, with neutrophil infiltration of the arterial media and destruction of elastic fibers [14,18]. Heidelberger et al. more clearly describe pathogenesis of this entity with respect to the clinicopathological time-course and development of associated meningitis [34]. They describe two general mechanisms of *known* pathogenesis: extrinsic (extravascular) and intrinsic (septic