Role Of Routine Neurovascular Imaging In Endocarditis Patients

Thesis Submitted For Partial Fulfillment of Master Degree In Cardiology

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List of abbreviations	
ABE	Acute Brain Embolization
ACA	Anterior Cerebral Artery
CKD	Chronic Kidney Disease
CoNS	Coagulase negative staph.
CNE	Culture Negative Endocarditis
CRP	C-Reactive Protein
CT	Computed Tomography
CTA	CT- Angiography
CVS	Cerebro-Vascular stroke
DSA	Digital subtraction Angiography
ESR	Erythrocyte Sedimentation Rate
ICMA	Intra Cerebral Mycotic Aneurysm
HAI	Health Care Associated Infection
IVDU	Intravenous Drug User
ICMA	Intracranial Mycotic Aneurysms
IE	Infective Endocarditis
OSSA	Oxacillin Sensitive staph. Aureus
MCA	Middle Cerebral Artery
MRI	Magnetic Resonance Imaging
MRA	Magnetic Resonance Angiography
MRSA	Methicillin Resistant staph. Aureus
NBC	Negative Blood Culture
NVE	Native valve endocarditis
PCA	Posterior Cerebral Artery
PVE	Prosthetic Valve Endocariditis
RF	Rheumatoid Factor
SLE	Systemic Lupus Erythematosus
TIA	Transient Ischemic Attack
TEE	Trans Esophageal Echocardiography
TTE	Trans Thoracic Echocardiography

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Introduction

Introduction

Infective endocarditis (IE) is a disease caused by microbial infection of the endothelial lining of intracardiac structures and is invariably fatal if untreated. Infection most commonly resides on one or more heart valve leaflets, but may involve mural endocardium, chordal structures, myocardium, and pericardium. The disease may also occur within septal defects or on the mural endocardium. Infections of arteriovenous shunts and of arterioarterial shunts (patent ductus arteriosus) as well as infection related to coarctation of the aorta can also be included in this definition because of their similar clinical manifestations.

Despite significant advances in the diagnosis and treatment of IE, 6-month mortality rates still approach 25 %. Changes in both patient demographics and microbial biology have challenged conventional wisdom of competing such a disease. Prompt recognition, triggered by a high index of clinical suspicion in susceptible patients, early diagnosis, and aggressive treatment are the critical components of a successful management strategy. Combined medical and surgical interventions can lead to improved outcomes for selected patients.

Embolization is a one of the most dreaded complication of IE. Central nervous system (CNS) involvement is most common; stroke comprises up to 65 % of embolic events and may be the presenting sign of IE in up to 14 % of cases.

CNS embolization can present with subtle neurologic abnormalities, as seen with microembolization, or with sudden hemiplegia and obtundation, as seen with a ruptured mycotic aneurysm and intracranial hemorrhage or with a large embolic stroke. Up to 90 % of CNS emboli lodge in the distribution of the middle cerebral artery, and carry a high mortality rate. Either due to massive cerebrovascular strokes, massive intracranial hemorrhage, or any of their sequelae. Any patient with suspected or definite IE who develops neurologic symptoms should be considered to have CNS embolization until proved otherwise.

Mycotic aneurysms represent a small, but extremely dangerous subset of embolic complications. They occur most frequently in the intracranial arteries and have a particular predilection for the middle cerebral artery and its branches. They result from septic embolization to the arterial vasa vasorum, or through direct intraluminal septic embolization with subsequent spread of infection to the arterial wall layers and weakening of the vessel wall. Mycotic aneurysms tend to develop at arterial branch points, which are a common site of embolic impaction.

The overall mortality rate among IE patients with intracranial mycotic aneurysms is 60 % and approaches 80 % if rupture occurs. The presenting symptoms of intracranial mycotic aneurysms (ICMAs) are highly variable and can range from headache (as seen with a small sentinel bleed) to dense neurologic deficits resulting from sudden intracranial hemorrhage.

In such patients, a contrast-enhanced CT will provide useful information as it is highly sensitive for the detection of intracranial hemorrhage and may thus indirectly identify the location of the mycotic aneurysm. Magnetic resonance angiography is another good technique for the detection of intracranial mycotic aneurysms, although its sensitivity for small aneurysms remains inferior to conventional four-vessel cerebral angiography. Although many intracranial mycotic aneurysms often heal with medical therapy, a subset may rupture unpredictably. Given the complex risks of prophylactic neurosurgical intervention, decisions concerning medical versus surgical therapy must be individualized. Percutaneous neuroradiologic intervention is preferred whenever allowed by the anatomic characteristics of the mycotic aneurysm.

Aim Of Work

To study the real incidence of infective endocarditis related ICMA [Intra Cranial Mycotic Aneurysms], because recent observations and studies revealed that large proportion of ICMA were silent - at least early - and may either remain silent, undiagnosed or accidentally discovered, or recognized when complicate by intracranial hemorrhage in a patient - in many cases - is fully anticoagulated for a prosthetic valve.

This tragedy is the main principle which guided our thinking towards developing a newer policy for early detection of those at risk of developing ICMA & thus modifying the plans of treatment for those affected before massive complications occur, either before or after valve replacement that can necessitate anticoagulation.

Moreover it would be of value to find predictors of developing ICMAs and their effect on the progress of IE patients according to:

- Causative organism.
- Time of detection of ICMA from the time of first IE manifestation (early versus late ICMA detection).
- Response of the IE episode to Antimicrobial therapy.
- March of the inflammatory markers.

Review of Literature.

Review of Literature

Chapter 1

Infective Endocarditis

"The Grim Intracardiac Infection"

Sir "William Osler" first described ' subacute bacterial endocarditis ' in 1885, although "Riviere" described post-mortem findings of vegetations in 1723[1].

Infective endocarditis (IE) is a condition resulting from microbial infection of the endothelial lining of intracardiac structures and is highly fatal if untreated. Infection most commonly resides on one or more heart valve leaflets, but may involve mural endocardium, chordal structures, myocardium, and pericardium[2]. The disease may also occur within septal defects or on congenitally malformed structures. Infections of the great vessels, arteriovenous shunts and of arterioarterial shunts (like patent ductus arteriosus) as well as infection related to coarctation of the aorta can also be included in this definition because of their clinical and pathological similarity.[2]

The vegetation, the characteristic lesion of infective endocarditis (IE), is a variably sized amorphous mass, formed of platelets aggregations and fibrin with abundant enmeshed microorganisms and moderate inflammatory cells. And according to the American college of cardiology (ACC) definition, vegetation as described by echocardiography is mobile, echodense masses attached to valvular leaflets or mural endocardium.[3]

It is no longer termed "subacute bacterial endocarditis", because other non-bacterial organisms are increasingly causing the condition.

In USA, the incidence of IE remained relatively stable from 1950 through 2000 at about 3.6 to 7.0 cases per 100,000 patient-years.[4,5] The incidence may be biased in some areas or some intervals due to external factors. In France, the IE incidence in 1991 and 1999 was 3.1 and 2.6 per 100,000 populations, respectively.[5] In the UK there are about 1500–2000 cases annually. However the task force of the European society of cardiology "ESC" stated IE incidence as a range 3 – 10 per 100,000 populations per year. [6,7]

Both the ACC/AHA & ESC recent reports agreed that in the past 10 years the epidemiologic characterization of Infective Endocarditis is being altered, probably due to population longevity with associated increased prevalence of sclerodegenerative valvular diseases and the decrease in Rheumatic heart disease incidence in developed countries.

Also increasing use of intracardiac devices and prosthesis and the increase in Intra-Venous Drug Abuse "IVDA" might be incriminated as well in such deviation. Another issue is the increasing prevalence of Staphylococci aureus as a leading causative organism rather than viridans Streptococci, and the wide spread use of broad-spectrum antimicrobials rendering staph. Strains into being resistant to penicillins then even to vancomycin.[8,9,10] All these facts had a major role in the dramatic change in incidence, prognosis and outcome for Infective endocarditis in the recent past years.

In all epidemiological studies of IE, the male: female ratio is 2:1, though this higher proportion of men is poorly understood. Furthermore, female patients may have a worse prognosis and undergo valve surgery less frequently than their male counterparts.[11]

This fulminant disease, whose salient features have been known for centuries, continues to pose major diagnostic and therapeutic challenges. It predominantly leads to local valvular and perivalvular destruction with subsequent regurgitation in the affected cardiac valves. Embolization, especially to the brain, is the most feared extracardiac complication. Diagnosis as widely accepted, relies on obtaining positive blood cultures and demonstration of vegetations by echocardiography.

A wide range of microorganisms can cause IE, but only a few species account for the vast majority of cases.[12] Streptococci and staphylococci are the cause of more than 80% of IE cases in which a responsible organism is identified. Streptococcal species were historically the most common group of pathogens, but more recent data identify S.aureus as the present most frequently isolated agent worldwide.[13] Moreover, the rate of antibiotic resistance among causative organisms is increasingly encountered.[3]

Related Definitions and Classifications [2]

Table 1: IE according to localization of infection and presence or absence of intracardiac material
• Left sided native IE • Left sided prosthetic IE (PVE) *Early PVE* → < 1year after valve surgery *Late PVE* → > 1year after valve surgery • Right sided IE • Cardiac Device Related IE (CDRIE)
Recurrence
• Relapse → repeat IE episode by same organism in < 6 months. • Reinfection → infection by a different organism, Or repeat IE episode by same organism in > 6 months