Peri-operative management of ruptured cerebral Aneurysms

An Essay submitted for partial fulfilment of the master degree in General surgery

By

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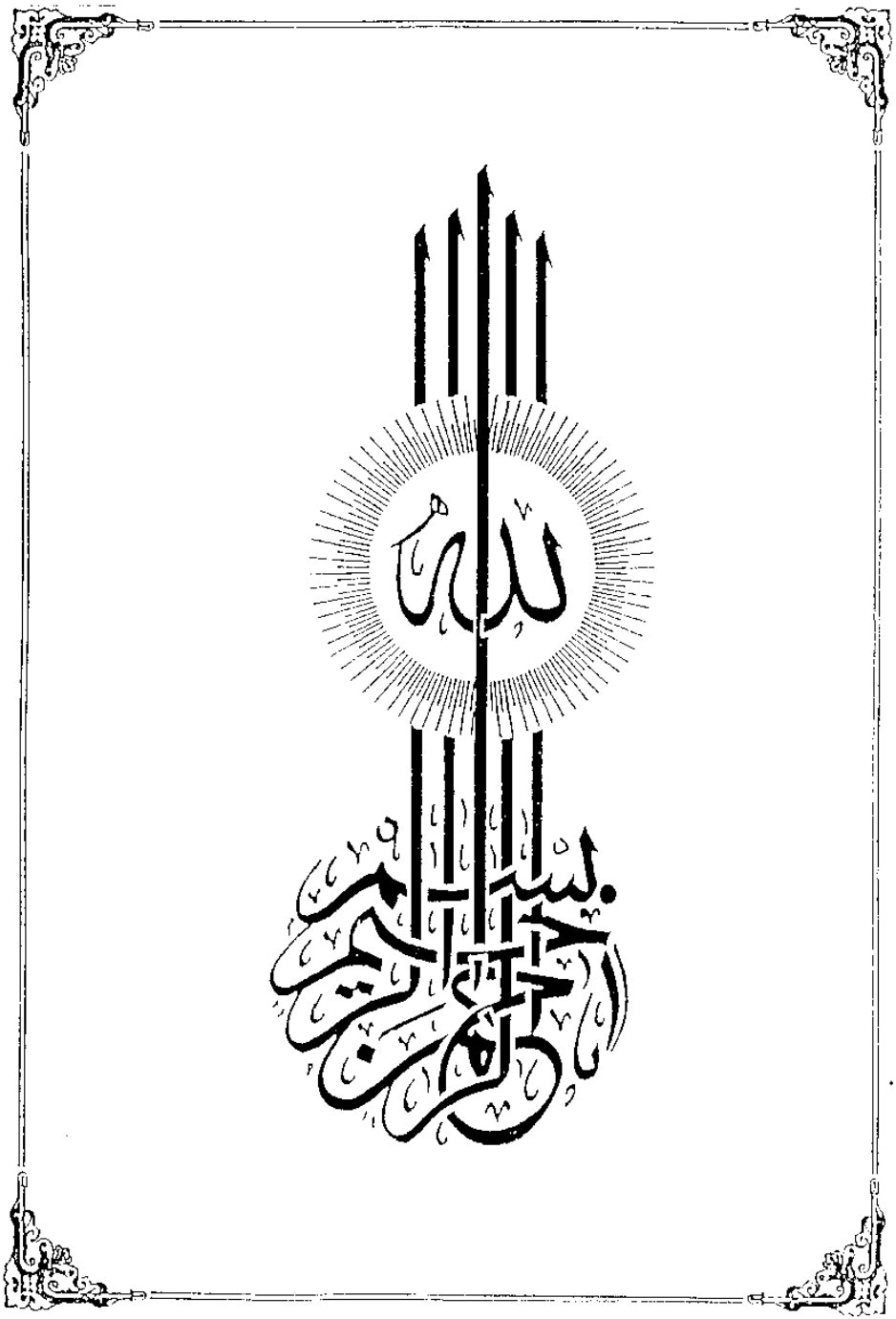
1995



Acknowledgment

It has been a great pleasure for me to have been supervised by professor ***Dr. Ahmed Samir EL-Molla,*** Professor and chairman of Neurosurgery Department Ain Shams university whose wise guidance, inspiration and valuable counselling paved the way for me all through this work.

I'd like to express my deep gratitude to Professor ***Dr. Magdi El-Kalliny,*** Assisstant professor of Neurosurgery, Ain Shams university whose meticulous supervision and encouragement was behind the completion of this work.

My Sincere gratitude to ***Dr. Mohamed Ashraf Gobashy,*** Lecturer of Neurosurgery Ain Shams university for his great help, guidance and precious cooperation which made this work possible.

List of Abbreviations

ACA	:	Anterior cerebral Artery
BBB	:	Blood Brain Barrier
CBF	:	Cerebral Blood FLow
CPP	:	Cerebral Perfusion pressure
CT	:	Computed Tomography
CVR	:	Cerebrovascular Resistance
DI	:	Diabetes Insipidus
EEG	:	ElectroEncephalogram
ICA	:	Internal Carotid Artery
ICP	:	Intra Cerebral Pressure
MABP	:	Mean Arterial Blood Pressure
MCA	:	Middle Cerebral Artery
MRI	:	Magnetic Resonance Imaging
MRA	:	Magnetic Resonance Angiography
OEF	:	Oxygen Extraction Fraction
PCA	:	Posterior Cerebral Artery
PCWP	:	Pulmonary Capillary wedge pressure
PICA	:	Posterior Inferior Cerebral Artery
SAH	:	Subarachnoid Hemorrhage
SIADH	:	Syndrome of Inappropriate Secretion of antidiuretic Hormone
SPECT	:	Single Photon Emission Computerized Tomography
SSEP	:	Somatosensory Evoked Potential
TCD	:	Transcranial Doppler

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INTRODUCTION

Introduction

The existence of intracranial aneurysms, their symptomatology and the results of rupture were recognized with increasing frequency by many far seeing physicians in central Europe from the early 1800's.

In 1920, *Harvey cushing's* first encounter with a cerebral aneurysm was closely followed by *walter Dandy's* first operation on an aneurysm in 1928. *(Cushing, 1923).* Norman McComish Dott from Edinburgh deserves the honour of being the first surgeon to attempt a pioneering direct attack on an aneurysm by wrapping it with muscle. His patient made a full recovery. In 1936 *Walter Dandy* trapped a carotid caverous aneurysm with a silver clip. *(Dandy, 1939).*

In october 1947 *Graeme Robertson* fully recognized the phenomenon of delayed SAH-induced cerebral ischemic deterioration and cerebral vasospasm often accompanying aneurysmal SAH. *(Robertson, 1949).*

The history of intracranial aneurysm recognition covers two centuries from medical management to direct clipping. Surgery had little impact on the outcome until the development of the microscope and improved neuro-anesthetic techniques.

Intracranial saccular aneurysms represent the most common aetiology of non-traumatic subarachnoid hemorrhage. The incidence of unruptured intracranial aneurysms is about 1% in the general population and it peaks in the 7th decade. *(Inagawa et al, 1990).*

Despite major improvements in surgical management, only 60% of patients who reach hospitals with neurosurgical facilities have a favorable outcome. Further improvement may depend on future developments in Endovascular obliteration which has allowed untreatable aneurysms to be successfully treated *(Higashida et al, 1990).*

The main preventable complication is rebleeding which is often disabling or fatal, thus a major aim of treatment is obliteration of the aneurysm. Another problem following subarachnoid hemorrhage is the development of ischemic deficits secondary to vasospasm. *(Kassel et al, 1985).* The development of medical therapy to deal with this complication may improve the outcome in a group of patients.

A subarachnoid hemorrhage is a catastrophe for the patient and his family. A significant number of patients who recover without significant neurological deficits show problems with social and occupational integration. Rehabilitation is the key to maximizing the remainder of the patient's Lifetime.

A positive attitude by the principal physician is important in achieving the best outcome. The management of aneurysms is a rapidly advancing subject with many controversial issues regarding the medical management and the timing of surgical intervention.

EPIDEMIOLOGY

Epidemiology of cerebral Aneurysms

Introduction:

Intracranial saccular aneurysms represent the most common etiology of non-traumatic subarachnoid hemorrhage *(Pakarinen, 1967)*.

The incidence of unruptured intracranial aneurysms is low in the general population and is reported as 1% in large autopsy series. The incidence peaks in the 7th decade. *(Inagawa et al, 1990)*.

Types of Aneurysms:

Saccular aneurysms:

It is an acquired degenerative lesion related to hemodynamic stress. *(Stehbens, 1989)*. It consists of an arterial outpouching and occurs at the bifurcation of proximal arteries at the base of the brain. The mean age for ruptured and unruptured aneurysms is in the mid 50 year range and there is a slight female preponderance.

Multiple aneurysms are noted in 15-30% of cases. Anterior circulation aneurysms occur in 90% cases. *(Hacker et al, 1983)*.

A saccular aneurysm is significant because it may rupture into the subarachnoid space.

Fusiform (arteriosclerotic) Aneurysms:

It is associated with systemic atherosclerosis. It is a tortuous dilatation of a vessel and commonly occurs in the basilar artery. Rupture is unusual and it is more often associated with brainstem compression or embolic events when sympomatic. These fusiform aneurysms are detecteed in 0.1% of autopsies. *(Inagawa. et al, 1990).*

Mycotic Aneurysms:

Usually occurs in the distal aspect of the middle cerebral artery and are related to bacterial emboli, most often from subacute bacterial endocarditis. High dose of antibiotics are the primary mode of therapy and lead to thrombosis. These aneurysms may rupture and have been noted in only 0.05% of autopsies. *(Inagawa et al 1990).*

Traumatic Aneurysms:

These are rare and usually associated with delayed intracerebral hematoma with a poor prognosis.

Neoplastic Embolic Aneurysms:

They are rare and usually result from an atrial Myxoma of the left atruim. They may be fusiform, saccular or the vessel may be completely occluded.

Natural History:

Knowledge of the natural history of a disease process is an absolute prerequisite for evaluating the influence of any treatment modality over the course of that disease. The prognosis of the untreated patients, as

determined by the natural history of the disease, is the yardstick by which the outcome of treatment is measured. There is universal agreement that the Major determinant of the natural history of intracranial aneurysms is whether the aneurysm is intact or has previously ruptured and it influences the course of the disease and the prognosis of the patient.

Unruptured Intracranial Aneurysms:

The natural history is largely derived from follow up studies of subarachnoid hemorrhage patients with multiple aneurysms who underwent craniotomy for treament of the ruptured aneurysm. There is agreement that an intact, angiographically identified, asymptomatic aneurysm will rupture at a rate of 1-2% per year. *(wiebers et al, 1981).*

Ruptured Intracranial Aneurysms:

The natural history of intracranial aneurysms is dramatically altered once rupture has occured. The mortality rate during the first week following subarachnoid hemorrhage was found to be 27% *(Sahs et al, 1969).* The study by *Pakariren et al in 1967*, 4 documented a 15% mortality prior to hospital admission and a mortality rate of 32%, 43%, 65% and 60% at day 1, week 1, month 1 and 6 months respectively. The natural history of ruptured aneurysms is poor. The overall risk of a second ictus within 48 hours is appox 6% with a rate of 1.5% per day for the next 2 weeks. Patients who survive the initial bleed for 6 months, are at a higher risk of rebleeding with an average rate of 3.5% per year for the next 10 years. The second and third aneurysmal subarachnoid hemorrhage carry a mortality of approx 65% and 85% respectively. *(Winn et al, 1977).*

Factors influencing the natural history:

Size:

In general, unuptured aneurysms tend to be smaller than ruptured aneurysms, with asymptomatic aneurysms being smaller than symptomatic aneurysms. *(weir, 1985).* An autopsy study by Crompton noted the critical size for aneurysm rupture to be 4mm in diameter. *Weibers et al, 1987* evaluated 130 patients with 161 unruptured aneurysms with an average follow up of 8.3 years and found that 15 aneurysms had ruptured and were > 10mm in diameter. Aneurysm size may be an important factor in determining the appropriate mode of treatment. It is important to note that aneurysms may enlarge with time, the rate being unpredictable and an expectant policy may be erroneous, in all but the smallest aneurysms (under 3mm).

Age:

The majority of cases of aneurysmal subarachnoid hemorrhage occur between the fourth and sixth decades of life. Recent studies indicate that aneurysms can rupture at any age and the incidence of subarachnoid hemorrhage increases almost linearly with age from 1.6/100,000 per year between 10 and 19 years of age to 49.6/100,000 per year for those over the age of 70 *(Ljunggren et al, 1982).*

Sakaki found that patients over the age of 65 had a significantly worse outcome than younger patients, with respect to both overall management and operative results. This is most probaly due to concomitant illnesses which exert a strong negative influence on outcome in the elderly. *(Sakaki et al, 1989).*

Multiple aneurysms are noted to be more common in the juvenile group, compared to the population at large. Interestingly, aneurysms in the Juvenile age group were associated with hypertension in one third of cases and there was a complete absence of middle cerebral artery aneurysms *(yashimoto et al, 1978).*

Gender:

There is an overall female predominace of intracranial aneurysmal disease. It was also noted that females have a higher incidence of vasospasm with corresponding poorer prognosis *(George et al, 1989).*

Multiplicity:

Patients harbouring multiple aneurysms have shown that having an unsecured additional aneurysm has a poorer long term out look, than having all the aneurysms secured. It was noted that 31% of patients died of recurrent subarachnoid hemorrhage compared to 95.2% good long term outcome in those patients in whom all aneurysms were Ligated. *(Mount et al, 1983).*

Familial influences:

It was found that familial intracranial aneurysms occured in 6.7% of family members. *(Norrgard et al, 1987).* The patients are usually younger at the time of rupture, with an average age of 42 years *(Berg et al,1992).* when familial intracranial aneurysms are the only abnormality the mode of inheritance is usually autosomal dominant *(Bannrman et al, 1970).*