VASCULAR DEMENTIA AND ITS PREVENTION

An Essay
Submitted for partial fulfillment of
Master Degree
in Neuropsychiatry

By

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6/6/89
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1992

ACKNOWLEDGMENTS

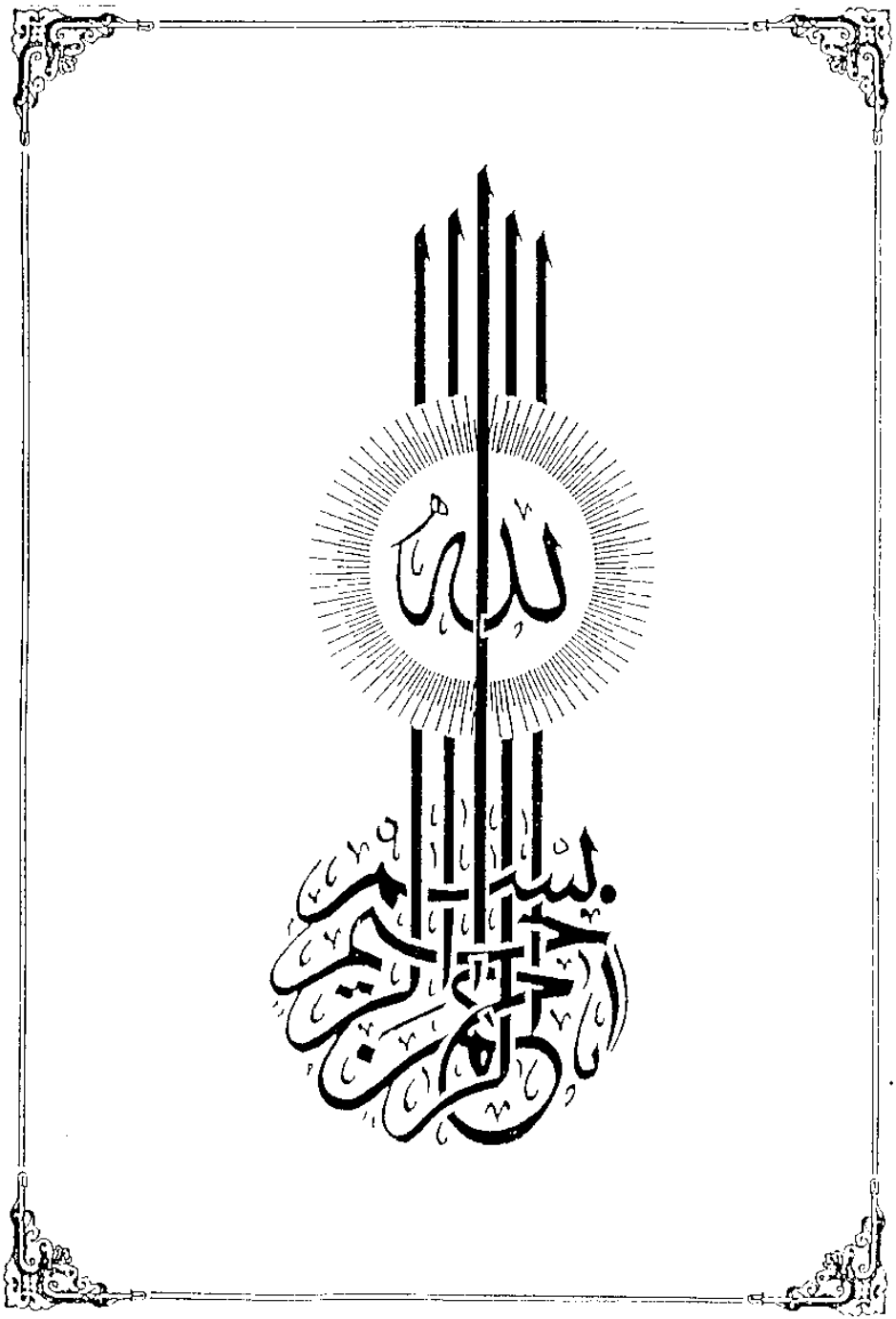
I would like to express my deep gratitude to **Prof.Dr. Youssef Abu Zeid**- Professor of Neurology, Ain Shams University- for his great help and generous supply of the recent references.

I would like also to express my appreciation to **Prof.Dr. Adel Sadek**- Professor of Psychiatry, Ain Shams University- for his enthusiastic support and encouragement

I also would like to thank **Prof.Dr. Ahmed Okasha**- Professor and Chairman of Neuropsychiatric Department, Ain Shams University- for his guidance and support.

My appreciation goes as well to Dr. **Taha Kamel**- Lecturer of Neurology, Ain Shams University- who revised the manuscript several times and gave highly comments that made this work more meaningful.

My thanks and gratitude extend to **Dr. Osama Abdel-Ghani and Dr. Hani Aref**, who helped me prepare the part of this work concerned with hemorheology and vasculitis.



to my family

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INTRODUCTION

* Geriatric disorders are a major concern to clinical medicine. Dementia is the most serious neuropsychiatric disorder of the old age. The essential features of dementia are intellectual deterioration, an impaired memory especially for recent events, disorientation and personality changes without clouding of the level of consciousness. Vascular dementia is the most frequent type of reversible dementia. It can also be prevented when early detected. It occurs with diffuse vascular cerebral lesions or circumscribed lesions in particular areas of the brain. It is clinically characterized by stepwize progression, fluctuating course and predominant deterioration of intelligence with relative preservation of personality.

The main mechanisms leading to cerebral ischemic lesions are atherothrombotic, cardiogenic embolic, lacunar infarcts and cerebral hypoperfusion. Yet, hypertension remains the most important risk factor for vascular dementia.

Since the mortality rate from cerebrovascular disease is decreasing, increasing numbers of elderly people are living, with subsequent cerebral ischemia. Thus, the improvement of new investigating measures and treatment of vascular dementia are of growing concern to medical research.

AIM OF THE WORK

The aim of this work is to study vascular dementia, its diagnosis and differentiation from other causes of dementia and the etiological mechanisms involved in its causation which will be a great help for primary prevention, early detection and subsequent management of new cases.

Literature Review

Definition of vascular dementia

The term vascular dementia is generally used to describe the dementias that are thought to be caused by thromboembolic cerebral vascular diseases (*Scheinberg, 1988*).

Multi-infarct dementia (MID) indicates a dementia disorder primarily caused by multiple cerebral infarcts which is considered a subdivision of vascular dementia.

Since other pathogenic mechanisms cause vascular dementia, a recent study defined the whole group of vascular dementia as dementia disorders with a history of focal or acute signs/symptoms referable to disturbed cerebral circulation (*Parnetti et al, 1990*). In our opinion, this is the most effective definition as it mentions the general pathogenic mechanism which classically causes vascular dementia.

Erkinjunti et al (1988) subdivide the whole group of vascular dementias into two subgroups based on clinical and cerebral CT scan data:

1- Multi-infarct dementia (MID)

2- Haemodynamic vascular dementia (HVD).

In MID the onset of dementia was temporarily related to TIA or stroke events. HVD was considered when disturbances of the systemic circulation e.g. cardiac arrhythmias were related to the development of dementia and without obvious symptoms of cerebral infarcts.

The history of evolution of the concept of vascular dementia and its pathogenesis

The history of the effort made to study the pathologic correlates for vascular dementia and its relation to the clinical picture is interesting and helpful to recognize the deficient points which need further research.

In 1894, *Binswanger* pointed out that dementia could be related to arteriosclerosis. He observed the diffuse areas of patchy demyelinization in the hemispheric white matter of most patients with arteriosclerotic dementia.

In 1912, *Ladame* classified arteriosclerotic dementia; due to multiple infarcts; into three types: 1) cortical, 2) subcortical, and 3) mixed. He also described the correlation between lesion location and clinical symptoms.

In 1937-1956, *Rothschild* studied the clinical differentiation of senile and arteriosclerotic psychosis. He recognized the presence of hypertension, cardiac or renal disease, the abrupt onset of dementia, hemiplegia, pseudobulbar speech, and dizziness, syncope or seizures in the syndrome of vascular dementia. He concluded that he failed to identify anything other than the crudest of correlation between the degree of intellectual impairment and histologic changes and that pure forms of senile and arteriosclerotic changes occured less frequently than the mixtures of both.

In 1968-1970, *Tomlinson et al* published a series of clinical-pathologic studies to determine if there are specific pathologic markers of dementia, degenerative or vascular. They found that patients who had >14 senile plaques per high-power microscopic field were always demented, but those who had fewer plaques may not have been demented. They also found that patients who had >100 ml of infarcted brain tissue were invariably demented. Their studies clearly demonstrated that the pathologic changes in the brains of demented old persons are of mixed senile and vascular etiology in at least 40%. They recognized that the pathologic markers of both degenerative and vascular dementia were quantitative and qualitative.

In 1974, *Hachinski et al* argued that cerebral arteriosclerosis itself never causes dementia and that, when dementia does appear, infarcts are always multifocal. Since this report, the term multi-infarct dementia (MID) has become widely used. It seems now that this concept is very selective and overestimated in clinical practice as the majority of vascular dementia deal with clinical entities in which diffuse cerebral, mainly subcortical damage plays a pre-eminent ethiopathogenetic role.

In 1987, *Elovaara et al* investigated the intrathecal synthesis of immunoglobulin G (IgG) in vascular dementia.

Two years later, *Wallin et al* studied the association between IgG index and hypertension and that the increased IgG index was not mainly a consequence of brain infarctions, but a reflection of a small vessel disorder (lacunar state in the subcortical arteries) supposed to underlie a number of vascular dementia cases in the absence of vasculitis and CNS infection.

In 1990, they also found that altered blood brain barrier permeability found in patients with vascular dementia can be attributed to chronic small vessels disturbances.