RETINAL EMBOLI IN CARDIAC PATIENTS

THESIS

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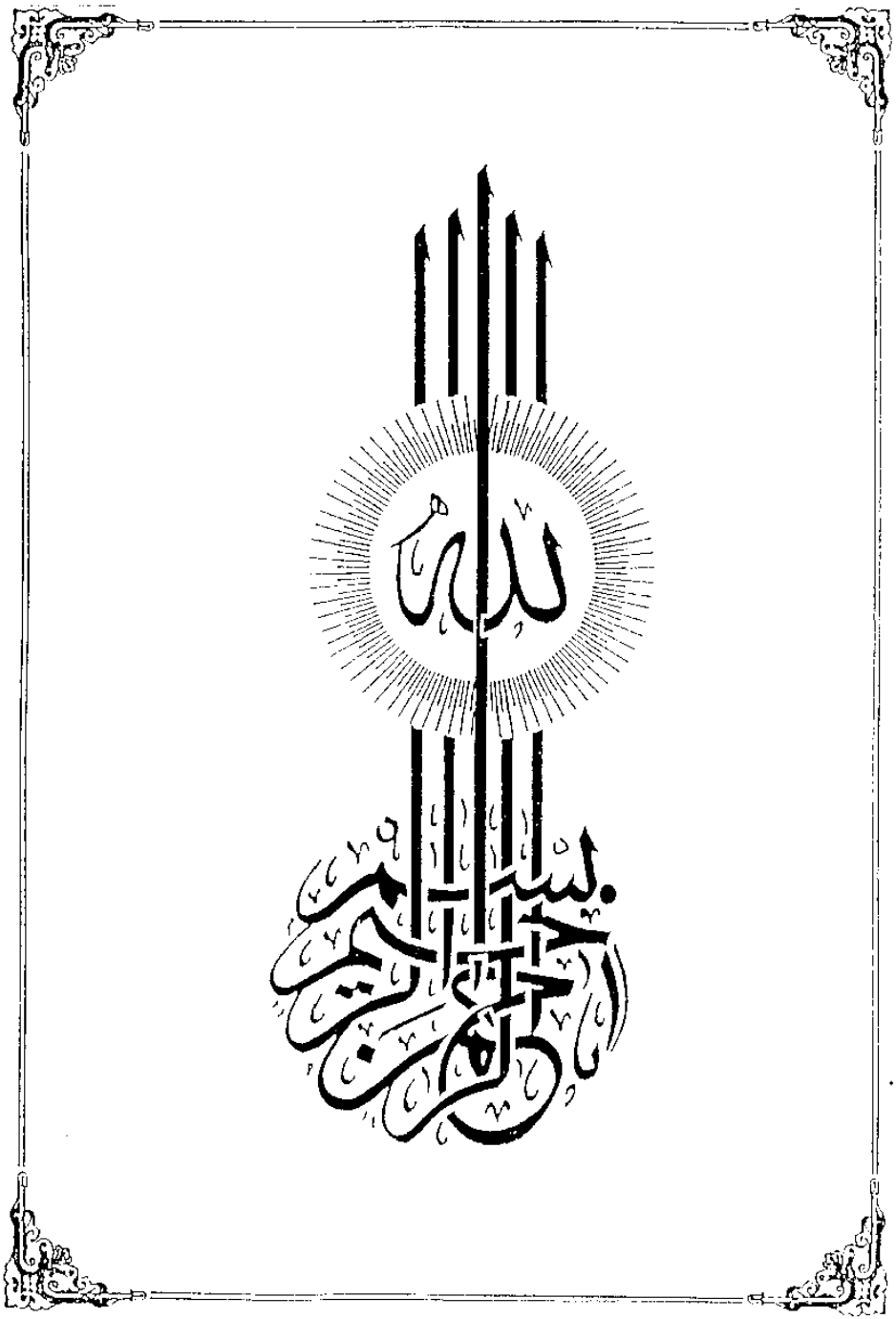

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INTRODUCTION

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Emboli secondary to heart diseases are responsible for retinal occlusive disease. There are a variety of endogenous and exogenous embolic materials to the retinal arterial system.

These embolic materials have been known to enter the retinal arterial tree, block vessels, and cause retinal ischemia, also they may explain an amaurosis fugax in cardiac patients.

Serious embolic events demand a thorough search for an underlying systemic cause and full study of the patient's cardiovascular status. Intensive investigations for the eye are also necessary.

Prophylaxis of emboli in cardiac disorders greatly reduce the mortality and morbidity associated with recurrent embolization as each embolus represents a potential catastrophe.

Most patients may be expected to respond to medical treatment of the cardiac conditions as well as of the retinal arterial occlusion, with prompt resort to surgery where necessary.

NATURE OF EMBOLI

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Arterial embolism is defined as : fragments of centrally located thrombi or atheromatous material that may embolize and occlude large or small peripheral blood vessels (Beeson and McDermott, 1975).

The emboli may be endogenous like cholesterol, calcium, platelets, fibrin, lipids, bacteria, tumour, and amniotic fluid or exogenous like parasites, fungi, silicone, air, mercury, oil, talc, cornstarch, glass beads. These materials have been known to enter the retinal arterial tree and block vessels and cause retinal ischemia (Kresca et al., 1979).

Emboli in cardiac patients usually originate from mural or valvular thrombi in the left side of the heart (atrium or ventricle), less commonly from an atheromatous ulcer in the aorta or a more peripheral artery, and from thrombi in aneurysms.

Paradoxical emboli originate from the right side of the heart and pass through a patent foramen ovale. Most

emboli occur in association with myocardial infarction, atrial fibrillation, mitral valve disease, chronic congestive heart failure, or endocarditis. With the advent of surgical replacement of heart valves, prostheses have become a common source of emboli (Beeson and McDermott, 1975).

Cardiac catheterization and iatrogenic trauma from angiography are infrequent causes of emboli (Wibber and Gutman, 1974). Also embolism of the central retinal artery have been described in association with endocardial myxoma (Manschot, 1959 ; Goodwin, 1963).

Pathophysiology :

Emboli lodge at bifurcation of arteries and at narrowed arteriosclerotic areas. The embolus stops blood flow through the artery and is followed within a few hours by secondary progressive arterial thrombosis before and sometimes beyond the point of obstruction. Secondary vasospasm has been assumed to be an important factor causing ischemia of the affected extremity.

Pathology :

Emboli from the heart or aneurysms show the same pathology as the parent thrombi. Emboli lodged in arteries usually organize by the ingrowth of connective tissue, and later recanalization may occur. However, fragmentation of the embolus before organization is not uncommon with fragments lodging in more distal vessels (Beeson and McDermott, 1975).

Types of retinal emboli :

1. Cholesterol emboli : (Hollenhorst plaques) :

They arise from an atheromatous plaque in the carotid artery and consists of cholesterol and fibrin. They lodge at the bifurcation of retinal arterioles, are refractile, and may appear larger than the vessel that contains them (Vaughan et al., 1977).

Cholesterol crystals may arise also from ulcerated atheromata of the aorta (Ball, 1966). They appeared sometimes during the operative or post-operative period, they were seen to travel within the vessels and they

A cholesterol embolus at the optic disc, which is refractile and appears larger than the vessel which contains it. A collateral vessel is seen at the lower border of the disc. (Vaughan et al., 1977).

occasionally appeared in showers (McBrein et al., 1963).

Cholesterol embolization of the retina can occur spontaneously as well as in patients who have had arteriography. They are bright orange-yellow in colour , show heliographic reflections, mobile within the retinal vascular tree (with and without massage of the globe), and retinal infarction seldom visible clinically (Penner and Font, 1969).

They can be dislodeged in the distal vessels, do not obstruct blood flow, and therefore are asymptomatic and normally disappear within 2 to 3 weeks (Wibber and Gutman, 1974).

Microscopic examination of atheromatous emboli reveals cholesterol crystals, occasionally surrounded by giant cells, in the lumen, usually attached to the vessel wall, the intima being relatively intact. The arteries with cholesterol emboli may develop progressive sclerotic changes in their walls adjacent to the site of the impaction. There were subintimal deposits of hyaline which

lead to a reduction in the caliber of the lumen (Brownstein et al., 1973).

The emboli had undergone variable degrees of cellulr organization. The retinal pathology included microinfarcts at the posterior pole and a zone of extensive peripheral cystoid degeneration in the area of distribution of an involved arteriole (Ball, 1966). The cholesterol crystals incite an inflammatory response which leads to fibrosis and complete vessel obstruction (Beeson and McDermott, 1975).

2. Calcific emboli :

These emboli originate from damaged cardiac valves. They lodge within the arteriole, producing complete occlusion and infarction of the distal retina. They are solid and calcified and occur in younger patients with a variety of cardiac lesions (Vaughan et al., 1977).

Calcific emboli from rheumatic valvular disease occur late in the course (Kellay and Randall, 1979).

A calcific embolus is easily seen, lodged in the inferior branch retinal artery just proximal to its bifurcation within the optic disc. There is heavy white retinal edema distal to the occlusion. The inferior retinal arterioles are markedly narrow and irregular in character with sludging of blood flow.
(Chumbley, 1981).