A STUDY OF PATIENTS WITH RECENT MYOCARDIAL INFARCTION ASSOCIATED WITH HIGH VENOUS PRESSURE

THESIS

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C A I R O

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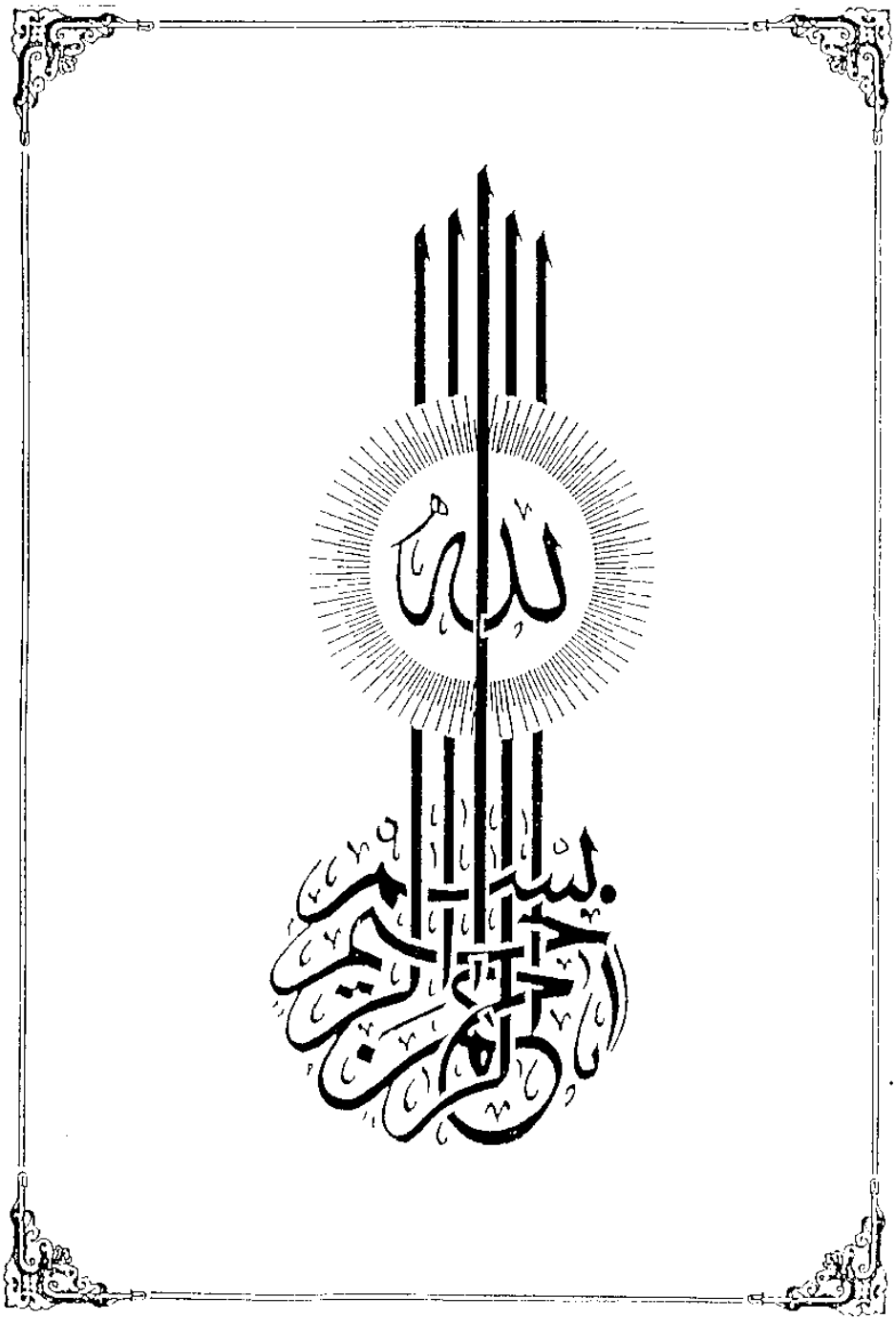

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Ischaemic heart disease is gaining more and more interest in our contemporary world because of its great morbidity and its potentially high mortality (1).

This interest , however , began long time ago , since ischaemic heart disease was already described in the era of the Pharaohs. In Ebber's Papyrus , it is reported: "If thou examinest a man for illness in his cardia, and he has pains in his arm, in his breast (mamma) and in one side of his cardia,...then, thou shalt say thereof : It is (due to) something entering into his mouth, it is death that threatens him." (2)

Later on , the syndrome was better understood and several disease entities were described beginning from simple angina pectoris and ending into the most complicated forms of myocardial infarction.

In the U.S.A. , it was reported in 1979, that around a million people a year suffered an attack of acute myocardial infarction (1). About half this number died, giving a crude mortality rate of about 160/100,000 individuals (1). This rate

dropped to 150/100,000 in 1982 (1).

In Egypt , the overall mortality from ischaemic heart disease,according to death certificates (which are to be taken with caution and reservation) was 19/100,000 individuals in 1972 (3 a). In 1979, it was 15.78/100,000 (3 b) and in 1982, 15.95/100,000 individuals (3 c). The incidence of acute myocardial infarction, however, is not known . A pioneer study could reveal, in 1978, an overall prevalence of ischaemic heart disease by definite electrocardiograhic criteria of 3.7% in sedentary employees, 1.6% in manual workers and 1.3% in farmers (3 a).

That ischaemic heart disease is often the precursor of infarction is obvious to any practitioner . Also , patients with recent myocardial infarction may develop complications, among which right -sided heart failure is by no means rare (4). The latter may be due to various causes e.g. right-sided failure secondary to left-sided failure, associated right ventricular infarction, pulmonary thromboembolism, cardiac tamponade, rupture of the interventricular septum &/or of one of the papillary muscles, and neglected cardiac arrhythmias (4). For proper treatment of right-sided failure associated with recent myocardial infarction, the underlying cause should be promptly diagnosed, as obviously the management of these different situations is different.

The aim of this work is to differentiate between such various conditions with special interest in right ventricular infarction .

Until recently , it was thought that acute myocardial infarction was almost exclusively a left-sided disease. In 1940, Blumgart et al. advocated the theory of the Thebesian system to protect the right ventricle by retrograde flow through its thin wall (5). This theory was discarded , however, and the mechanism of protection is now ascribed to a combination of low metabloic needs of the right ventricle and the effect of low pressure on coronary collateral circulation (6). The assumption that right ventricular damage does not impair significantly cardiac function (7) made of right ventricular infarction more a post-mortem curiosity than a clinical entity. It was not until 1959 that the pathogenesis of right ventricular infarction was first described (8).

The interest in right ventricular infarction has much increased, however, in the last few years. In 1974 , Cohn et al. (9) described its clinical and haemodynamic features: Right ventricular infarction is no more just a post-mortem diagnosis (8 , 10) ,but a clinical entity and a pontentially reversible cause of cardiogenic shock (9,11,12) , so that aggressive diagnosis and therapy are now quite justified (9,11). This urged us to plan this piece of work.

Our aim , again, is to elucidate the clinical and laboratory characteristics of right ventricular infarction, so as to early differentiate the disease from the alike syndromes and allow for prompt management of the patients to get the best possible outcome.

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LITERATURE REVIEW

" I Do Not Know What I May Appear To The World : But To Myself I Seem To Have Been Like A Little Boy , Playing On The Seashore , Diverting Myself And Now And Then Finding A Smoother Pebble, Or A Prettier Shell Than Ordinary, While The Great Ocean Of Truth Lay All Undiscovered Before Me."

(Sir Isaac Newton)

(1642-1727)

INCIDENCE , PATHOGENESIS AND PATHOLOGY OF RIGHT VENTRICULAR INFARCTION

The current concept of the right ventricular infarction (RVI) being a rare event is no more holding true. The incidence of this disease entity varies considerably from one author to another depending on the criteria used for diagnosis. Beam (13) , in 1938 , reported a 1.7% incidence based on post-mortem studies of myocardial infarction patients.

The famous paper of Wartman & Hellerstein (14)in 1948 reported on 2000 consecutive autopsies 22 instances of RVI out of 164 cases of myocardial infarction (13.4% prevalence) of which only 4 showed isolated RVI (2.4%).

Isner and Roberts (15), in 1978 , reported 33 cases of RVI in 236 necropsy patients (13.9%) for infarction , all of which were associated with posterior left ventricular (LV) wall infarction and none was an isolated RVI.

Cintron et al. (16) , in 1981 , evaluated 96 patients with acute myocardial infarction , 44 of whom had a diagnosis of inferior infarction . Of these 44 patients , 16 had a bedside diagnosis of right ventricular(RV) dysfunction.These 16 patients were all surviving 3 months after the infarction. The clinical incidence of RVI is thus

16.7% of the total number of cases of myocardial infarction , and 36.4% of cases of inferior infarction.

Other authors have found a clinical incidence of RVI varying from 3 - 8 % of all cases of infarction (17), whereas pathologic and radionuclide studies have found a 24-45% incidence rate of RV involvement in patients with inferior infarction (15,18,19).

Comparing patients with RVI with other cases of infarction , Isner et al.(20) could not disclose any difference in between as regards patient's age , sex , extent of coronary artery luminal narrowing , presence of RV hypertrophy or RV thrombi , or the length of time of symptoms of myocardial ischaemia. They reported even a post-mortem diagnosis of a patient with dilated cardiomyopathy who sustained RVI in the absence of coronary artery luminal narrowing.

Saffitz et al. (21) reported also a case of coxsackie viral myocarditis causing transmural RV and LV infarction without coronary narrowing . Motro et al.(22) reported a case of RVI caused by mild contusion of the heart as a result of blunt chest trauma without coronary occlusion.

Other investigators disclosed severe coronary artery lesions and/or increased RV mass associated with the infarctions.Wade (8) observed that 18 out of his 19 post-morterm hearts with RVI showed

right coronary occlusion , posterior LV infarction and cardiac hypertrophy . Experimentally induced RV hypertrophy increased susceptibility to infarction (23) .

In a recent paper of Kopelman et al. (24) it was found that patients with RV hypertrophy as a result of chronic lung disease were prone to RVI in the setting of inferior myocardial infarction . Isolated RVI might occur in patients with chronic lung disease , RV hypertrophy and insignificant coronary artery disease . Both increased myocardial oxygen demand and a decreased blood supply might play a role in this relation . There was , however , a high frequency of concurrent inferior LV wall infarction , particulary involving the posterior interventricular septum (IVS) (15,24).In addition , most patients had at least a 75% reduction in the cross-sectional area of the dominant coronary artery supplying the inferior LV wall (15,24).

It was after a post-mortem study that Isner & Roberts (15) reported that cases of RVI showed 75% or more narrowing of the arteries supplying the posterior wall , either the right coronary or the circumflex artery. RV dilatation was more frequent in RVI.They reported also that , due to the fact that the RV free wall was thin, its infarction usually was transmural .They classified the extent of RVI as follows:

Grade I:Less than 50% of the posterior RV wall is infarcted

Grade II :The infarction is limited to the posterior wall but involves more than 50% of it.

Grade III:All the posterior wall and less than 50% of the antero-lateral wall are involved .

Grade IV :The RV posterior wall and more than 50% of the anterior wall are involved.

It seems important at this stage to study briefly the blood supply of the RV. It is mostly served through the RV branch of the right coronary artery (RC) which supplies the RV free wall except for its anterior margin which is supplied by the left anterior descending (LAD) of the left coronary artery (LC) (25).Most important is that the LAD supplies also the upper anterior 2/3 of the septum,most of its inferior surface , and gives rise to the artery of the moderator band; this is a large branch of LAD which passes through the moderator band and is an important source of blood supply to the antero-lateral portion of the RV free wall and papillary muscle (26).

The posterior descending branch of the RC (90%) or of the circumflex (10%) supplies the posterior wall of the RV near the septum,together with the posterior surface of the LV.The acute marginal branch of RC supplies the diaphragmatic surface of the RV and, occasionally , the postero- apical IVS .The lateral wall of the RV is supplied by a marginal branch of the RC(25).