Editorial

Diabetic cardiomyopathy-an overview

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The association between diabetes mellitus and major cardiovascular complications including ischaemic heart disease is well known.

Diabetes is an independent risk factor for mortality and morbidity in both symptomatic and asymptomatic heart failure[1]. The symptoms of cardiac failure in diabetic patients are often out of proportion to the measured left ventricular systolic function. One putative explanation for this discrepancy is diastolic dysfunction of the left ventricle, which is a frequent finding in many studies of cardiac function in Type 2 diabetic subjects.[2] There is evidence for the existence of a specific cardiac muscle disease among diabetic subjects in which there is cardiomyopathy in the absence of discernible coronary artery disease, hypertension or valvular disease.[3,4] This condition has been designated diabetic cardiomyopathy and was first postulated in 1972[5] following observations of heart failure in four patients with no other risks factors other than diabetes. This has been confirmed in several small and large scale studies.[6,7,8] Clinically, the most common findings are ECG and pathologic evidence of left ventricular hypertrophy and haemodynamic

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measurements are consistent with diastolic dysfunction.

The exact mechanism for the pathogenesis of diabetic cardio-myopathy remains elusive and management of the condition is also empirical and unsatisfactory. Exercise,[9] and use of beta-blockers and calcium antagonists[10] have been recommended but the beneficial effects of these strategies remain unproven. It is unlikely that significant progress will be made in this direction until there is a better understanding of the pathogenesis of the disease.

Some of the hypotheses proposed include endothelial dysfunction, autonomic dysfunction, metabolic derangement and interstitial fibrosis. There is also a suggestion that abnormalities of the contractile proteins could be responsible for the mechanical cardiac defects.[11] The study of Hamby et al[7] provided evidence for small vessel disease by demonstrating both endothelial and sub-endothelial fibrosis in patent epicardial coronary arteries in three victims of the condition. The possibility of autonomic dysfunction was raised by the demonstration of uneven areas of sympathetic denervation in the hearts of diabetic patients and the

correlation of diastolic function with catecholamine levels.[12] Furthermore 91% of a group of diabetic patients had cardiac autonomic neuropathy and 59% of these had abnormal systolic function compared with 8% in diabetics without cardiac autonomic neuropathy.[13] Fibrosis which is perivascular and interstitial is the commonest histopathological finding in diabetes. This was found in many of the diabetic patients with autonomic cardiac dysfunction. [13]

Dyntar[14] showed that both palmitic acid and glucose in high concentrations promoted loss of myofibrillar organization and sarcomeric disarray which have been reported to occur in diabetic cardiomyopathy. He suggested that gluco- and lipo-toxicity may be important in the development of diabetic cardiomyopathy.

Other studies[15,16] have linked cardiac dysfunction in chronic diabetes mellitus to defective Ca^{2+} handling and decreased sarcoplasmic reticulum. Ca^{2+} uptake and release has been reported in the heart of rats made diabetic by injection of streptozotocin.[14]

In this issue, Howarth and Qureshi advanced these observations and provided experimental data for prolonged Ca^{2+} transient time as well as the relaxation time of cardiac myocytes from rats with streptozotocin-induced diabetes mellitus. These observations, they argued are in support of defective sarcoplasmic reticulum Ca^{2+} uptake in myocytes from diabetic rats.

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