LETTERS TO THE EDITOR

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Diabetic cardiomyopathy: a controversial entity

We read with interest the study by Konduracka et al., which concludes that patients with long-term type 1 diabetes mellitus under intensive insulin treatment do not have echocardiographic, biochemical, or morphological signs of diabetic cardiomyopathy. However, as the authors admit, this is directly opposed to several previous studies which clearly show the presence of myocardial diastolic dysfunction in patients with type 1 diabetes, when compared with control subjects. Importantly, like Konduracka et al., these studies excluded patients with coronary artery disease and hypertension. These co-morbidities may themselves result in diastolic impairment, thus precluding any firm conclusions regarding the specific effect of diabetes on the human myocardium.

We assert that the authors’ explanation for the absence of diastolic abnormalities in their diabetic cohort is unconvincing. They speculate that myocardial overload and increased peripheral resistance after the administration of exogenous insulin may be responsible for the diastolic abnormalities seen in previous studies. However, the diabetic population studied by Konduracka et al. was also treated with intensive insulin therapy. Moreover, as the authors used tissue Doppler imaging to investigate myocardial function, one would expect them to identify changes in left ventricular diastolic properties with much less dependence on loading conditions compared with conventional echocardiography. Since 89% of their cohort had diabetic retinopathy and 45% cardiovascular atherosclerotic neuropathy, the absence of even mild impairment in left ventricular relaxation is very surprising. With such a high incidence of the aforementioned diabetic complications, one would expect increased prevalence of microalbuminuria and diabetic nephropathy, but the authors do not present data on renal function. Moreover, it seems that their cohort did not exhibit ideal glycaemic control over time, and this makes the presence of diabetic cardiomyopathy even more likely. Interestingly, all 17 deceased diabetic patients had, at autopsy, microscopic evidence of small vessel diabetic complications and the majority of them had also cardiac fibrosis. It is therefore surprising that these 17 deceased patients with long-standing diabetes (mean duration >20 years) with evidence of poor glycaemic control (HbA1c 8.4%) and histological evidence of cardiac disease had no abnormality on echocardiography. Another issue that needs further clarification by the authors is whether their diabetic subjects were under any other medications apart from insulin and statins. If the majority of their diabetic population was under treatment with angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers, as one would expect for the treatment of diabetic retinopathy or other microvascular complications, then this may well have influenced the echocardiographic measurements. Furthermore, N-terminal pro-B type natriuretic peptide levels are affected by inhibition of the renin-angiotensin system, and perhaps even the biochemical results should be interpreted with caution.

The study by Konduracka et al. raises major concerns, but we need to admit that any study on diabetic cardiomyopathy poses several difficulties because of the several confounding factors (i.e. hypertension, coronary atherosclerosis, glycaemic control, and medications) that come into play. Finally, we believe that although diabetic cardiomyopathy is a controversial entity, compelling epidemiological and clinical data support its occurrence in humans.

References


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