

A population-based study of the diabetes paradox in stress cardiomyopathy

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Three reports have suggested that there is a decreased rate of stress cardiomyopathy (broken heart syndrome) in patients with diabetes.¹⁻³ There is speculation that early diabetic autonomic neuropathy could protect against stress cardiomyopathy by impairing the autonomic nervous system that links the brain and the heart. This possible protective effect of diabetes in the face of its many negative effects has been termed the diabetes paradox. We undertook a New Zealand population-based study exploring the incidence of stress cardiomyopathy in diabetics and non-diabetics.

All patients aged >40 years registered in the Canterbury, Pegasus and Rural Canterbury Primary Health Organisations (PHOs) are invited for a cardiovascular health check that includes diabetes screening. From this screening a central registry of incident diabetes is maintained. Diabetes is defined as HbA1c of >50mmol/mol. Three major earthquakes have precipitated case clusters of stress cardiomyopathy in Christchurch.^{4,5} Since 2010 we have prospectively maintained a registry of earthquake and sporadic cases meeting modified Mayo diagnostic criteria.⁶ We cross-referenced the two databases, restricting to women aged >65 years as stress cardiomyopathy predominantly occurs in post-menopausal women.⁶ Approval was obtained from the Health and Disability Commission Ethics Committee, reference number URA/11/07/033/AM03.

From our registry of 160 cases of stress cardiomyopathy, 26 women were excluded for being too young and 35 were excluded as they had not undergone the primary care diabetes screening. Ninety of 39,402 non-diabetic patients had stress cardiomyopathy, a

rate of 0.0023. Nine of 5,093 diabetics had a stress cardiomyopathy, a rate of 0.0018. The p value for rejecting the null hypothesis of a 20% difference was 0.51. Thus the rates of stress cardiomyopathy among non-diabetics and diabetics were similar.

The previous studies were retrospective and had no formal or consistent definition of diabetes. Additionally, in the earlier studies the background population diabetes rates used were not specific or age matched to the case population. For instance, one publication has compared the rate of diabetes in a collection of global cases reports of takotsubo patients with the rate in an unmatched United States National Health and Nutrition Examination Survey.⁷ Such a comparison is almost meaningless. In our study we applied prospective definitions for diabetes and stress cardiomyopathy to a defined population of over 44,000 women. The rates of stress cardiomyopathy in diabetics and non-diabetics were very similar, but with wide confidence intervals. In our New Zealand data we have found no evidence for the diabetes paradox, but note that our data does not disprove the hypothesis. A limitation of our work is that we have no data on the type and severity of the diabetes, nor knowledge of any therapy or presence of neuropathy. This weakness arises by nature of the study design that uses the available prospective community diabetes database. That same design gives the study a unique strength in this area of research, prospective and uniform classification of diabetic status. This highlights the difficulty of this type of research and that further study will be required to determine if the paradox is indeed real.

Competing interests:

Nil.

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REFERENCES:

1. Bill V, El-Battrawy I, Behnes M, Baumann S, Becher T, Elmas E, et al. "Diabetes Paradox" in takotsubo cardiomyopathy. *Int J Cardiol.* 2016; 224:88–9.
2. Dias A, Franco E, Rubio M, Koshkelashvili N, Bhalla V, Amanullah S, et al. Takotsubo syndrome: does it matter if you have diabetes? *Int J Cardiol.* 2016; 224:398–9.
3. Madias J. Particulars of diabetes mellitus may matter in patients with takotsubo syndrome. *Int J Cardiol.* 2017; 229:47.
4. Zarifeh JA, Mulder RT, Kerr AJ, Chan CW, Bridgman PG. Psychology of earthquake-induced stress cardiomyopathy, myocardial infarction and non-cardiac chest pain. *Intern Med J.* 2012; 42:369–373.
5. Chan CW, Troughton R, Elliott J, Zarifeh JA, Bridgman PG. One-year follow-up of the 2011 Christchurch Earthquake stress cardiomyopathy cases *The New Zealand Medical Journal.* 2014; 127:15–22.
6. Templin C, Ghadri J, Diekmann J, Napp L, Bataiosu D, Jaguszewski M, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. *N Engl J Med.* 2015; 373:929–938.
7. Madias JE. Low prevalence of diabetes mellitus in patients with Takotsubo syndrome: A plausible 'protective' effect with pathophysiologic connotation. *Eur Heart J. Cardiovasc. Care.* 2016; 5:164–170.