Background: LVH and alterations in LV geometry are associated with cardiovascular risk and prognosis. Among those at greatest risk are individuals suffering from hypertension and/or T2D, with the co-existence of these conditions suggested to increase risk. The authors state that there is a lack of data from large scale studies assessing cardiac features in diabetic and non-diabetic hypertensive subjects, in particular in the rural population of China, where hypertension is prevalent. The authors aimed to investigate the impact of hypertension with or without diabetes and its possible gender-dependent effect on LV hypertrophy, cardiac geometry and function in northeast rural Chinese population.

Methods: 10270 participants in total consisting of 4990 normotensive without T2D; 4462 hypertensive without T2D; and 818 hypertensive with T2D. Data collected included questionnaire/interview, basic clinical data, 12 hour fasting blood sample and transthoracic echocardiography. Heart structure, geometry and function were assessed.

Findings: Hypertension associated with LVH and geometric changes in a large rural Chinese population. Risk was increased in female subjects and in subjects with diabetes.
Review:

- Firstly, the authors should be commended for the high response rate of 83.5% in the study as well as the strict training and standards they set for investigators and implementation of committees and subcommittees to monitor the quality of the study.

- Missing data: demographic characteristics from the questionnaire/interview including lifestyle risk factors, dietary habits, family income, evaluation of psychological status, and quality of life. This information either needs to be provided or these variables must be removed from analysis.

- Age is associated with diastolic decline and with increases in LV structural measurements. The study would be strengthen if the authors confirmed that their data set exhibited these same normal associations (as it would increase confidence that results obtained from their sample population are relevant to the wider population).

- Please specify if parasternal short or long axis was measured and if the measurements listed were taken during diastole in the methodology section.

- Hypertension was characterised as SBP ≥140 mmHg and (or) a DBP ≥90 mmHg. Can the authors please provide data that shows how many subjects within each group presented with both systolic and diastolic hypertension, systolic hypertension only with normal diastolic blood pressure and diastolic hypertension only with normal systolic blood pressure. And can the authors comment on why they chose to include subjects with diastolic hypertension with normal systolic blood pressure? As systolic hypertension is considered the main driver of hypertension induced LVH and dysfunction.

- Variables such as fasting blood glucose, cholesterol and pulse pressure have previously been reported to associate with or to predict alterations in LV geometry and function. Did the authors perform statistics to determine whether any of the basic clinical data (Table 1) associated with the observed alterations in LV geometry, structure and function? This analysis may be beneficial to tease out the influence of these variables with the observed alterations. Particularly as the basic clinical data listed in Table 1 varied between groups.
Following on from above, in Table 3. Multiple logistic regression analysis was preformed however established cardiovascular risk factors HDL-C, LDL-C, FBG, SBP were not included in analysis. Do statistical differences remain when these variables are accounted for?

Line 57 the authors state "As a result of adaptation of left ventricle to neurohormonal activation and chronically increased hemodynamic load, there are three abnormal LV geometrical patterns including concentric remodelling, concentric hypertrophy and eccentric hypertrophy". The authors are reminded that neurohormonal signalling and increased hemodynamic load may be contributing factors to alterations in LV geometry and may not be requirement. As such the authors statement is misleading. Alterations in cardiac metabolism for example are of particular interest in the diabetic heart, where an increased reliance on fatty acid oxidation and uncoupling between glucose oxidation and glycolysis are suggested to promote LVH. Please see the following paper for more information Can J Cardiol. 2017 Mar 19. pii: S0828-282X(17)30114-9. doi: 10.1016/j.cjca.2017.03.009.

Line 155, reference 14 is specific to a study that assessed LV geometry in children and adolescents. This reference is inappropriate for the subject demographics in the current study. Please provide a more appropriate reference.

Line 213 the authors state "It indicated that the synergistic effects of hypertension and diabetes with both stress damage and interstitial deposition of advanced glycation end products in cardiac tissue can induce more serious LV hypertrophy and remodelling". I have two comments for this statement, first, the study did not include a normotensive DM group. Normotensive patients with DM have previously been reported to exhibit alterations in LV geometry, structure and function that are comparable to hypertensive patients (Cardiovasc Diabetol. 2017 Apr 20;16(1):53. doi: 10.1186/s12933-017-0535-5). Can the authors please comment on how they are confident that the alterations observed in the study are a synergistic effect between HT and DM, when they do have a DM only group? And secondly, the authors may consider re-wording this statement, they have no evidence from their current study that interstitial deposition and advanced glycation end products in cardiac tissue were present in their populations.

Only 8% of subjects were hypertensive with DM. Can the authors please comment on this limitation?
Are the methods appropriate and well described?
If not, please specify what is required in your comments to the authors.

Yes

Does the work include the necessary controls?
If not, please specify which controls are required in your comments to the authors.

Yes

Are the conclusions drawn adequately supported by the data shown?
If not, please explain in your comments to the authors.

No

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I am able to assess the statistics

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