Dear Editor,

Thank you for carefully reviewing our manuscript previously titled "**A novel α-galactosidase A gene mutation in a Chinese Fabry Disease family: a case report and literature review**" for possible publication in the *World Journal of Clinical Cases*. We are grateful to you and your reviewers for their constructive ~~critique~~opinions. We have revised the manuscript, highlighting our revisions in red. and have attached point-by-point responses detailing how we have revised the manuscript in response to the reviewers' comments below.

Thank you for your consideration and further review of our manuscript. Please do not hesitate to contact us with any further questions or recommendations.

Yours Sincerely,

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Reviewer Comments:

Reviewer #1:

Comment: The authors should underscore the delay for diagnosis and point out that a lysosomal storage disease should have been suspected years ago in the presence of cardiac hypertrophy, arrhythmias, and renal involvement. Family history (two close relatives with same heart disorder) should have reinforced the suspicion of a genetic nature of disease. Table 1shows that the proband and her 52 years old sister had a normal (<10 mm) thickness of left ventricular (LV) posterior wall in spite of an increased thickness of interventricular septum (16 mm and 15.8 mm, respectively). Thinning of LV posterior wall is a feature of Fabry related cardiomyopathy in the late stage. The authors could address this point in discussion. Please use a galactosidase-α and a galactosidase -β instead of agluberase-α and agluberase-β, on page 11. As concerning proband's son, there is discrepancy between the value of LV posterior wall reported in the text and that shown in table 1. Please check and amend.

Response: We have supplemented Time delays in the diagnosis was 6 years (on page 4) in this paper, and supplemented the literature of Patients may seek help from multiple medical specialists before a correct diagnosis is made, resulting in delayed treatment initiation (on page 11).

Thinning of LV posterior wall is a feature of cardiac manifestation in Fabry disease patients~~related cardiomyopathy~~ in the late stage. ~~You~~ As you

mentioned in your reply, we reviewed the expert consensus on vascular performance published last year in the Fabry and The Report, but the specific case has not yet been found. As myocardial fibrosis develops, the posterior and inferior LV wall can thin and become hypokinetic or akinetic (on page 11-12).

We have modified agluberase-α and agluberase-β instead of a galactosidase-α and a galactosidase -β. Thanks to reviewers for spotting misspellings.

I'm really sorry we should have noted in table 1 that the data were preERT (IVST17.4 mm, LVPWD14.5 mm). The value of LV posterior wall reported in the text was from 6 months after ERT (IVST17mm, LVPWD16.5 mm).