Peer Review File

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Comment 1. I am a bit concerned about the age of the study group – since it is known that EA which manifests in childhood has a probably worse prognosis than the outcomes among adults, I would not mix up those age groups; if the Authors decide otherwise, it should be depicted in Methodology precisely how many children at what age were included and commented in the Discussion or Limitation section.

Reply 1. *We thank the reviewer for this comment. We added the information in the Methodology (page 5) and Limitation sections (page 12).*

Comment 2. The Authors should provide information on the control group – at least the gender age and if there was no significant difference.

Reply 2. *In the Results section it is specified that "Among control subjects, twenty subjects were males (80%), five were females (20%)"; we then added a specification about the age of controls (page 7). Controls were 25 while patients were 50 totally; clinical data of controls compared to Ebstein's patients are presented in Table 1. As specified in the Methods, it was a historical control group of healthy subjects we used in our retrospective study. Controls were not prospectively enrolled and matched according to patients' data.*

Comment 3. Please provide information in methodology what do you mean by preserved RVEF.

Reply 3. *We added a definition of preserved RVEF in the Methods (page 7).*

Comment 4. I have some concerns when applying the criteria of mild and severe form of EA based on the cutoff point of TV displacement = 16mm/m2 – as far as I know the clinical value of this index is not well established yet; Why the authors chose this parameter instead of others like Celermjer index or R/L index? please provide a comment in the discussion or limitation section.

Reply 4. *We added a comment in the Discussion section (page 10).*

Comment 5. I would suggest consolidating the tables – in my opinion, a lot of data is redundant or repeated; Table 1 could include controls and statistical difference; Table 4 and 5 also could be combined together.

Reply 5. *We consolidated the tables: Table 1 now includes the clinical characteristics with p-value of Ebstein's patients and controls; Table 3 now includes the CMR data of Ebstein patients and controls, we provided data on flow on the tricuspid, pulmonary and aortic valve for Ebstein anomaly.*

Comment 6. A very interesting topic is the adaptive mechanism of RV in the context of pressure or volume overload. It would be very valuable to compare the GCS and GRS in EA and "pure" tricuspid regurgitation; this would give evidence if those parameters in EA are compromised because of the inherent myocardial problem or because of hemodynamic factor which is volume overload; please provide additional comments on that.

Reply 6. *Thank you for this comment. Further comparison studies are needed to evaluate if the reduction of strain values we evidenced in Ebstein's anomaly is purely due to an intrinsic myocardial impairment or could also be a consequence of pure volume overload from tricuspid regurgitation.*

There are currently no data in the literature concerning this and we think it is a very interesting topic we would further investigate. We added a comment on that regarding all strain values (GRS, GCS and GLS) in the Discussion (page 9).

Comment 7. It also seems that RV GLS is not impaired at all – could you discuss it as well? perhaps a comparison with Tricuspid regurgitation would also elucidate this matter; I would also suggest adding this plot to Figure 1.

Reply 7. *Thank you for this comment. We added comments on page 9 and 10. We added to Figure 1 the data of RV GLS.*

Comment 8. I have some serious doubts about the scientific value of the scatterplot Figure 4 and all the dedicated paragraphs; Please explain once again what is the point of this analysis; I feel like this was a part that does not convey any valuable information. I can see that even in 25% controls we can find impaired GRS and GCS; why do you need this tool to discriminate EA from controls? Would you really suggest it as a diagnostic method? Could you find any idea how would that impact medical aspects/management?

Reply 8. *Thank you for the comment. We provided strain values to show that initial alterations of myocardial contraction could be found in EA also in cases where traditional markers of right ventricular dysfunction as RVEF are still normal. Definition of controls is given in the text as subjects without any cardiac disease, at the timepoint of the study. We did not want to prove a clinical impact of strain values, as it should be further addressed in larger studies with correlation with clinical parameters.*

Comment 9. Please doublecheck the style – I suggest shortening some long sentences in order to improve the clarity of the paper (line 64-67, 73-76, etc).

Reply 9. *We modified the length of sentences in the suggested lines of the uploaded manuscript.*

Comment 10. The authors assessed the use of cardiovascular magnetic resonance feature-tracking analysis as an early predictor of right ventricular contractile mechanics in patients with Ebstein's anomaly. 50 EA patients were compared to 25 historical controls. RV GRS and GCS were found to be significantly impaired compared to controls. This represents a useful method to look for RV dysfunction in EA patients before they develop symptomatic RV dysfunction seen on traditional parameters like RVEF. I would like to congratulate the authors for a well-presented manuscript.

Reply 10. *Thank you very much for your comment, we really appreciate.*