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Comment on: First clinical manifestation of Brugada syndrome during pregnancy

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We read with great interest the case report by Dr. Prochnau et al. titled “First clinical manifestation of Brugada syndrome during pregnancy” [1]. Along with our congratulations to the authors for their comprehensive description and clinical sagacity, we would like to highlight some elements of this case that should be considered in pregnant women suspected of having Brugada syndrome.

The first consideration to keep in mind is that pregnancy modifies the anatomical situation of the heart in the chest, displacing it to a more “horizontal” position. This change in heart orientation modifies the morphology of the electrocardiogram (ECG) limb leads but also affects the precordial leads, which adopt a lower relative position with respect to the heart. This could lead to “concealment” of the electrocardiographic features of Brugada syndrome. The effect of the anatomical displacement of the heart induced by pregnancy and its consequences on the morphology of a right ventricular outflow tract (RVOT) ventricular tachycardia was recently described by Caldwell et al. in a very clarifying case report [2].

Indeed, in ECG no. 1 of the article by Prochnau et al., r' waves can be clearly seen in leads V₁ and V₂ and even a slight elevation of the ST segment in V₁. Consequently, our recommendation is to record leads V₁ to V₃ one or two intercostal spaces above the standard position in

every woman in her last months of pregnancy who are suspected of having Brugada syndrome. Now, if we compare ECG no. 1 and no. 3 (baseline of the ajmaline challenge) we can see that in the latter, the QRS axis is lower than in the former, which supports our point.

Another interesting element is the effect of ajmaline and the repolarization of the premature ventricular contractions (PVCs) that were recorded after the administration of the drug. Firstly, it is noteworthy that only 25 mg of ajmaline produced such a considerable widening of the QRS complex, mainly at the expense of forces directed upward, ahead, and to the right, suggesting a marked conduction delay at the level of the RVOT, the site of origin of the PVCs in this patient. Secondly, the first RVOT PVC shows a certain degree of fusion with the sinus QRS complex and the ST segment is more elevated than in the sinus beats, particularly in lead V₁ but to a lesser degree also in V₂. This simulates a phenomenon recently described in several articles about the unmasking of Brugada ECG pattern in the presence of right bundle branch block (RBBB) by early pacing-induced depolarization of the right ventricle. This generates more “normal” QRS complexes, avoiding the masking effect created by the RBBB [3–5]. In this case, it is possible that the considerable delay in the regional activation of the RVOT may imitate in part the effect of a “classic” RBBB and that the anticipated activation (PVC) of this delayed region makes the ST-segment elevation more manifest.

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Compliance with ethical guidelines

Conflict of interest. P. A. Chiale, A. Enriquez and A. Baranchuk state that there are no conflicts of interest.

The manuscript does not include studies on humans or animals.

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