



Correspondence

Low serum level of antimullerian hormone and gestational hypertensive disorders



We read with interest the article published in the 2019 March issue of the *Taiwanese Journal of Obstetrics and Gynecology* by Jamil and colleagues, entitled “serum antimullerian hormone and renalase levels in predicting the risk of preeclampsia” [1]. The authors used the cross-sectional study to explore the relationship between two serum markers (antimullerian hormone [AMH] and renalase) and the development of preeclampsia and the results showed that low serum level of AMH was correlated with the increased risk of preeclampsia in pregnant women, and an adjusted add ratio was 1.073 (95% confidence interval [CI] 1.007–1.143, $p = 0.029$) [1]. Based on the above-mentioned finding, the authors further proposed the hypothesis that hypertensive disorders of gestation and ovarian aging were associated with lower serum AMH levels, and furthermore, suggested that AMH has a role in detecting vascular diseases [1]. We congratulated their success in the use of serum markers in the prediction of the risk of preeclampsia and this work was finally published. The following comments were not against the achievement of the authors, but we hope to receive the authors' response.

First, the pathogenesis of preeclampsia is still fully understood, but it is believed that abnormal placentation (impaired spiral artery remodeling, dysregulation of endothelial function, dysregulation of immune function, shallow trophoblast invasion and impaired motility or movement ability of trophoblasts), producing high resistance vessel system between maternal and fetal surface with deteriorating uteroplacental perfusion, resulting in hypoxia and inflammation [2,3]. Therefore, it is supposed that an idea marker should appear in the early pregnancy (the first trimester). However, the serum samples in the current study were collected in gestational age between 14 and 32 weeks. It is uncertain that the alternation of these serum markers appears before or after the development of preeclampsia. If change happens before the development of preeclampsia, it may be much more useful in the prediction of the risk of preeclampsia.

Second, as shown by authors, AMH is a good marker to be a representative of oocyte reservation (ovary aging), and it is believed that AMH did not fluctuate in the menstrual cycles and pregnancy [4,5]. Therefore, the rationale of time to collect samples (gestational age between 12 and 32 weeks) needs explanation. Is there any possibility to provide serum level of AMH in these patients before this pregnancy to explore the relationship between serum level of AMH and occurrence of preeclampsia?

Third, it is relatively interesting to find that serum level of AMH was lowest in women with gestational hypertension (0.73 ± 1.29) when comparing with those in normal controls (1.62 ± 2.29) and women with preeclampsia (0.85 ± 1.07), although the difference

did not reach to the statistical significance. Since the standard deviation (2.29, 1.29, and 1.07) is so wide, and even over the absolute calculated data (mean, 1.62, 0.73, and 0.85), respectively, the clinical significance should be reconsidered. Did the authors have any comment?

Fourth, the adjusted and unadjusted odd ratios for the prediction of the risk of preeclampsia are 1.073 and 1.076 when using serum level of AMH, respectively. Based on little increasing risk of developing preeclampsia, did the authors really think that this prediction is practicable or valuable in the clinical use? Although a recent systematic review to evaluate the role of AMH in preeclampsia proposed the current data are suggestive of the potential predictive value of serum AMH as its levels seem to be lower among women who have preeclampsia, the cut-off value and specificity and/or sensitivity (positive predictive value and/or negative predictive value) during the first trimester of pregnancy is still uncertain [6].

Finally, the role of AMH for the risk of vascular diseases is very interesting. Although the association between AMH and all-cause mortality in women is still investigated, a recent report in 2016 showed an independent and inverse association between serum AMH levels and all-cause mortality in men (hazard ratio [HR] 0.94, 95% CI 0.90–0.98) [7]. In addition, men in the highest quartile had a significantly lower risk of mortality than those in the lowest quartile did (unadjusted HR 0.13, 95% CI 0.07–0.25; adjusted HR 0.36, 95% CI 0.16–0.81) [7].

Could the authors kindly respond to the above-mentioned questions? Thank you.

Conflicts of interest

The authors declare that they have no competing interests.

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