Journal of Community Medicine and Public Health Reports ISSN: 2692-9899
Letter to the Editor Volume 2 Issue 3

Is the low AMH level associated with the risk of cardiovascular diseasein Obese pregnants?

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Received date: 10 July 2021; **Accepted date:** 15 July 2021; **Published date:** 20 July 2021

Citation: Shalal H, E. Phillip HE. Is the low AMH level associated with the risk of cardiovascular disease in Obese pregnants? J Comm Med and Pub Health Rep 2(3): https://doi.org/10.38207/jcmphr20210088

Cardiovascular disease is the leading cause of death for women, it is responsible for more than 33 % of mortality. Menopause does not cause cardiovascular diseases. However, there is an overall increase in heart attacks among women 10 years after menopause. This could be attributed to a decline in the natural ovarian hormones estrogen and progesterone.

For this reason, we read with interest the paper by Başak Güler et al immediately contemplating the value of such a tool in rendering efficient antenatal and postnatal care to women. We were specifically drawn to the conclusion that the decreased AMH levels in obese patients are a risk factor for cardiovascular disease. We found it interesting that the paper was not clear on the timing (the trimester in which the AMH values were checked), this we believe is important not only because it would lend itself to the repeatability of the study; but there is evidence to suggestthat AMH values are the highest in the first trimester and then declinesas pregnancy advances **[1,2]**.

AMH is a marker of ovarian reserve, in terms of follicular numbers not necessarily the quality of the follicles. During ovarian aging, there is a gradual decline in both the quality and quantity of the ovarian follicle pool which can be influenced by genetic, autoimmune, vascular, and toxic factors, **[3,4]** and parity. 64.9 % of the patients in the obese group were multiparous, hence large differences in the ovarian reserve arelikely to exist in women of the same age.

In the study, the authors indicated that Age and Systolic blood pressure had a significantly high predictive value for cardiovascular disease in both the obese and non-obese pregnant patient cohort. In the obese group, a low AMH level was associated with the risk of cardiovascular disease **[5]**. Although pregnancy can be considered a vascular stress test that may unmask a woman's tendency to develop cardiovascular disease later in life **[6]**, this is a brave conclusion. Our concern is based on several issues (a) in the obese patient, the AMH value ranged from1.29±0.91ng/ml whilst in the non-obese group the AMH value ranged from 1.74 ±1.00 ng/ml., clearly revealing an overlap in the values. The measure of significance was defined as a p-value < 0.05. No correlation coefficient value was given to indicate the strength of the significance, but a glimpse of the strength may be appreciated by reviewing the p- value associated with the logistic regression analysis. Multivariate logistic regression analysis in the obese group revealed a p-value of

0.001 when looking at the impact of age on cardiovascular disease, a p-value of 0.002 was reported for systolic blood pressure but a p-value of only 0.049 for a low AMH. Conclusions drawn from multivariate analysis are more accurate and nearer to real-life situations. The predictive value of a low AMH and its association with the risk ofcardiovascular disease in obese patients is uncomfortably weak but it may be an indication of cardiovascular disease. Obesity impacts negatively on granulosa cell function **[7]**, as evidenced by increasing numbers of anovulatory cycles among the obese female population. The resulting dysfunction in the granulosa cells leads to a reduction in AMH production but simultaneously, obesity is associated with cardiovascular risk factors which may interfere with the ovarian circulation **[8]**, and the insult would enhance ovarian aging, leading to a further reduction in AMH levels.

Would it therefore not be more accurate to consider a low AMH to be an indication (maybe early) of cardiovascular disease as opposed to a risk factor for cardiovascular disease? Should this be so, it would allow for a relatively timely intervention in terms of lifestyle changes to delay a potential cardiovascular event, and herein lies the value of this study.

We advise caution in drawing this conclusion that a low AMH level is associated with the risk of cardiovascular disease in obese pregnantpatients based on this study, The significance of a low AMH and thepurported risk of cardiovascular disease in this study is weak, the samplesize is small, there is a risk of bias, the absence of a universallyagreeable definition of a low AMH and the lack of clarity/standardizatio n of the timing during pregnancy when the AMH value was determinedconcerns us. It is also unfortunate that this study was undertaken in a non-universally extrapolatable state, pregnancy, even among women,since it limits its usefulness.

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