CEOG Clinical and Experimental Obstetrics & Gynecology

Original Articles

Reproductive Biology Section

Absence of blood type A or AB may be associated with diminished oocyte reserve

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Summary

Purpose: To determine if blood type A protects against developing diminished oocyte reserve. *Materials and Methods:* Retrospective evaluation of incidence of blood type A (or AB) in women with normal oocyte reserve (day 3 serum follicle stimulating hormone (FSH) ≤ 11 mIU/ml) *vs*. diminished oocyte reserve (FSH ≥ 18 mIU/ml). *Results:* Five hundred forty-seven of 1,232 (44.4%) women with normal reserve had blood type A or AB *vs.* 33.8% (44/130) with diminished oocyte reserve (p= 0.027, chi-square). *Conclusions:* Lack of blood type A or AB may link to some other gene that may be responsible for premature depletion of oocytes.

Key words: Blood type; Diminished oocyte reserve.

Introduction

A previous study suggested that the presence of blood group A might be an independent risk factor for early onset ovarian hyperstimulation syndrome [1, 2]. If this is true, there exists the possibility that the women with this blood type may be more protected against diminished oocyte reserve.

The aim of the present study was to test the hypothesis that women with type A or AB blood may have a lower incidence of decreased oocyte reserve compared to women with blood type B or O.

Materials and Methods

A retrospective chart review was performed on women aged ≤ 39 years with day 3 serum follicle stimulating hormone (FSH) ≤ 11 mIU/ml (serum E2 < 50 pg/ml) *vs.* those whose serum FSH was ≥18 mIU/ml. The frequency of various blood types were determined in each group.

Results

There were 1,232 women evaluated whose serum FSH was ≤ 11 mIU/ml (normal oocyte reserve group). There were 547 (44.4%) with blood type A or AB.

There were 130 women evaluated with serum FSH ≥ 18 mIU/ml and 44 (33.8%) had blood type A or AB (p = 0.027, chi-square analysis).

Discussion

These results are consistent with the possibility that women with blood type A are more protected against developing diminished ovarian reserve. The next step would be to determine if oocyte quality, as manifested by live delivered pregnancy rates, can be related to blood type.

Corroboration by other centers could lead to a possible greater understanding of mechanisms for diminished oocyte reserve by exploring gene linkages with blood type and other genes that may play a role in folliculogenesis or follicular atresia.

If absence of type A is corroborated to be an independent risk factor for developing premature ovarian reserve, this is one additional piece of information that a single woman could use in determining if she wants to freeze oocytes for the future.

References

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