Evaluation on Antisperm Antibody in Infertile Men with Oligoasthenoteratozoospermia

Yi-chao XU1, Jing LI2, Wei-bao LIANG1, Wei-jie ZHU3

1. Department of Urology, the First Affiliated Hospital, Jinan University, Guangzhou 510630, China
2. Department of Pathophysiology, Medical College, Jinan University, Guangzhou 510632, China
3. Department of Developmental and Regenerative Biology, College of Life Science and Technology, Jinan University, Guangzhou 510632, China

Objective To evaluate the level of antisperm antibody (ASA) in infertile men with oligoasthenoteratozoospermia (OAT).

Methods Forty-six infertile men with OAT were enrolled into this study. Sperm samples were screened by the direct immunobead test for ASA type IgG and IgA according to the WHO laboratory manual.

Results Of the 46 patients with OAT assessed for ASA-IgG, 2 had immunobead binding which were 22% and 27%, respectively, and the sub-positive rate was 4.3%. No case had the clinical positive level according to the WHO criteria (≥ 50% of the motile sperm with immunobead binding). ASA-IgA was not detected in all cases.

Conclusion A significant incidence or high level of ASA could not be found in infertile men with OAT, which suggests that ASA is not associated with the pathogenesis of infertile men with OAT.

Key words: antisperm antibody (ASA); sperm; oligoasthenoteratozoospermia (OAT); infertility

Infertility has become a public health problem since last decades in most countries, which affects approximately 10% to 15% of married couples. In particular, the male factor is reportedly up to 50% of infertile couples[1-3]. Male infertility may be caused by complex and multiple causes, which are associated with disturbance or defects of anatomy, physiology, endocrinology or genetics, etc.

Oligoasthenoteratozoospermia (OAT) is common severe abnormalities of semen
quality\(^4\). For infertile men with OAT, sperm parameters show very low sperm number, poor motility, and high sperm abnormal morphology. These abnormal sperm parameters reflect aberrant alternations of functions in both testes and epididymides, which demonstrates that infertile men with OAT have complicated etiology\(^5\)–\(^8\). Recently, Pelliccione et al.\(^9\) reported that an activation of the immune system was associated with idiopathic OAT in infertile men. In addition, sperm from OAT-patients showed increased expression of secretory actin-binding protein\(^10\). However, sperm auto-immunity whether related to the etiology of OAT has not been fully understood. In the present study, the antisperm antibody (ASA) level in infertile men with OAT was determined by means of the immunobead test (IBT), aiming to elucidate the role of ASA in the pathogenesis of OAT.

**Materials & Methods**

**Subjects and semen samples**

A total of 46 infertile men with OAT, aged 26–41 years, who had applied for treatment of primary infertility at the First Affiliated Hospital of Jinan University were included in the study. OAT samples were selected according to the criteria described by the WHO manual (WHO, 2010)\(^11\): 1) total sperm number <39 \times 10^6 per ejaculate; 2) progressive motility <32%; 3) ratio of sperm normal forms <4%.

Semen samples were produced by mastrubation after 3–7 d of sexual abstinence. Semen examination was performed according to the WHO manual. Sperm morphology was assessed after slide staining with the Papanicolaou-staining method.

**ASA assay**

The direct immunobead test was used to determine the ASA level of sperm samples according to the WHO manual and as previously described\(^11\)–\(^14\). In brief, a volume of 5 \(\mu\)l washed sperm suspension was mixed thoroughly with 5 \(\mu\)l drops of each type of immunobead (IgG and IgA; Irvine Scientific, Calif., USA) on a glass microscope slide and was examined with a phase-contrast microscope. Motile sperm which had one or more adherent immunobeads were scored wherever the beads were binding. Sperm samples from males predetermined to be ASA-positive and ASA-negative were set as positive controls and negative controls, respectively. A threshold value of 50% was adopted to indicate a significant ASA positivity according to the WHO criteria, 20%–50% motile sperm that had adherent particles were deemed to be sub-positive.

**Results**

Of the 46 patients with OAT assessed for ASA-IgG, 2 patients had immunobead
binding which were 22% and 28%, respectively, and the sub-positive rate was 4.3% (2/46). No case had the clinical positive level according to the WHO criteria (50% or more of the motile sperm with immunobead binding). ASA-IgA was not detected in all cases.

Discussion

OAT is the most frequently seen phenotype in male infertility, and has a variety of etiologies, including age, non-inflammatory functional alterations in post-testicular organs, infective agents, alterations in gamete genome, mitochondrial alterations, environmental pollutants[4-8,15-18]. In addition, varicocele, oxidative stress, “subtle” hormonal alterations and nutritional factors could contribute to defective spermatogenesis and sperm maturation, which lead to poor quality of sperm and even OAT[19-23].

Male infertility and ASA are often related[24-26]. In certain pathological situations such as reproductive tract obstruction, infection, or trauma, testicular blood-testis barrier or/and epididymal blood-epididymis barrier could be damaged, which lead to disturbance of auto-immune system and production of ASA[27-30]. It is generally accepted that ASA may contribute to decrease male fertility[31-33]. However, the present study failed to demonstrate a significant incidence of ASA in the infertile men with OAT. Besides, those 2 cases with sub-positivity did not reach the clinical positive level according to the WHO criteria[11]. These data indicate that ASA is not associated with the pathogenesis of OTA-related infertility. Probably, disordered spermatogenesis and abnormal epididymal function result in poor sperm parameters, leading to OAT. On the other hand, 2 cases had ASA sub-positive levels in this study, which meant that an immune response against sperm antigens could be still developed although OAT-patients had very low spermatogenesis.

In conclusion, this study revealed that a high incidence or high levels of ASA could not be found in infertile men with OAT, which excluded the possibility that sperm auto-immunity was responsible for the OAT etiology. On the other hand, as a screening test for the etiology of male infertility, the detection of ASA should be still performed in OAT population even if the etiologic factor for OAT is not related to ASA.

References


(Received on February 3, 2014)
Conference Information

9th Gynaecological & Early Pregnancy Ultrasound Workshop & 10th ISUOG Outreach Pre-Conference Workshop
May 3rd Singapore / Singapore Obstetrics/Gynecology, Radiology / Imaging
Contact: Ms Jessica Leong, Postgraduate Medical Institute, Singapore General Hospital
Phone: 011-65-6321-4071
Fax: 011-65-6223-9789
E-mail: pgmi.courses@sgh.com.sg

23rd European Congress of Obstetrics & Gynaecology
May 7th to 10th United Kingdom / Glasgow Obstetrics / Gynecology
Contact: Anna Botto or Stefano Ricco, Organizing Secretariat, M.A.F. Servizi SRL - Part of GL Events Group
Phone: 011-39-1-150-5900
Fax: 011-39-1-150-5976
E-mail: ebcog2014@mafservizi.it
Website: http://www.ebcog2014.org/

MRCOG Final Preparation: Intensive OSCE
May 8th to 9th United Kingdom / London Obstetrics / Gynecology
Contact: Royal College of Obstetricians & Gynaecologists
Phone: 011-44-20-7772-6245
E-mail: events@rcog.org.uk
Website: http://www.rcog.org.uk/events/mrcog-final-preparation-intensive-osce

46th Annual Society for Obstetric Anesthesia & Perinatology (SOAP) Meeting
May 14th to 18th Ontario / Toronto Anesthesiology, Obstetrics / Gynecology
Contact: SOAP
Phone: 414-389-8611
Fax: 414-276-7704
E-mail: soap@soap.org
Website: http://soap.org/AM.php

Clinical Directors' Forum
May 15th United Kingdom / London Obstetrics / Gynecology
Contact: Kim Helm, Royal College of Obstetricians & Gynaecologists
Phone: 011-44-20-7772-6468
E-mail: khelm@rcog.org.uk
Website: http://www.rcog.org.uk/events/clinical-directors-forum-2