Abstract

Positive sperm-cervical mucus interaction has been known as a major cause of infertility which can not be managed easily. At present, IUI is considered as the treatment of choice for this condition, though its success is debatable.

The female genital tract has a potential space which is filled by fluid. The fluid in different parts of the female genital tract (eg. cervical canal, uterine cavity and fallopian tube) differs in composition which changes with the phases of the menstrual cycle.

Currently available infertility investigation techniques can only assess the sperm-cervical mucus interaction to find out the hostility of the cervical mucus. But there is a possibility of IUF being hostile to the spermatozoa in couples with hostile cervical mucus. In such a situation, IUI can not be considered as the treatment of choice as spermatozoa deposited can get immobilized in the uterine cavity. That may be the reason for low success rates reported in IUI as a treatment for a patient with hostile CM.

However there is no proper method currently available to assess the sperm-IUF interaction (hostility).

Though it is controversial, accountability of ASA on CM hostility has been reported by several authors. To my knowledge, the effect of ASA on sperm-IUF interaction has not yet been studied anywhere in the world.

Chlamydial infection and its effects on CM hostility is also controversial. The effect of chlamydial infection on sperm-IUF interaction is not known.

In this study, a simple and effective method to assess sperm–IUF interaction was developed. The correlation between sperm–CM interaction and sperm–IUF interaction was also studied in order to make a new diagnostic criteria and treatment modalities for the infertile couples with CM hostility. The effects of chlamydial infection and ASA on CM hostility and sperm-IUF too were studied.

All the couples who were referred to RBL for sperm-CM interaction test were screened and the couples who fulfilled the inclusion criteria were selected to the study.

Seminal fluid samples were collected from male partners for seminal fluid assessment, sperm-CM interaction test, sperm-IUF interaction test and direct sperm-MAR test.

Aspiration of CM and IUF were done in the mid cycle of the female partners. CM aspiration was done by a tuberculin syringe without a needle. IUF was aspirated by a sterile infant feeding tube. Urethral swabs and cervical swabs from male and female partners were collected for chlamydia screening.

The mean age for males and females of the study sample were 36.7 and 33.7 years.

The total sample was 274. 168 (61.3%) of the total sample had hostile CM. 48 (28.6%) of couples with CM hostility had hostile IUF while 120 (71.4%) had non hostile IUF. There were 106 (38.7%) with the non hostile CM. 7 (6.6%) out of them had hostile IUF while 99 (93.4%)

had non hostile IUF. We could not observe an agreement between CM hostility and IUF hostility (Kappa = 0.184).

The incidence of chlamydial infection among the females and males were 12% and 10.2% respectively. No statistically significant correlation between male and female chlamydial infections and CM hostility was found. But the correlation between male chlamydial infection and IUF was statistically significant ($p=0.00001$).

The incidence of ASA-IgG positive males of the study sample was 9.29%. No statistically significant correlation was found between ASA and hostile CM. There was a highly significant correlation of ASA in the males with hostile IUF ($p<0.0001$).

Spermatozoa interact differently with CM and IUF and behavior of spermatozoa in IUF cannot be predicted by the behavior of spermatozoa in CM. IUI should be recommended only for the females without hostile IUF. If IUF is hostile ART might be the treatment of choice. If both fluid phases are non-hostile normal coitus might be sufficient.

As such CM hostility cannot be considered as the determining factor for IUI for the patient with hostile cervical mucus. The determining factor for IUI should be a non-hostile of IUF.

ASA may affect sperm-IUF interaction probably via IgG. Male chlamydial infection may influence ASA IgG formation.