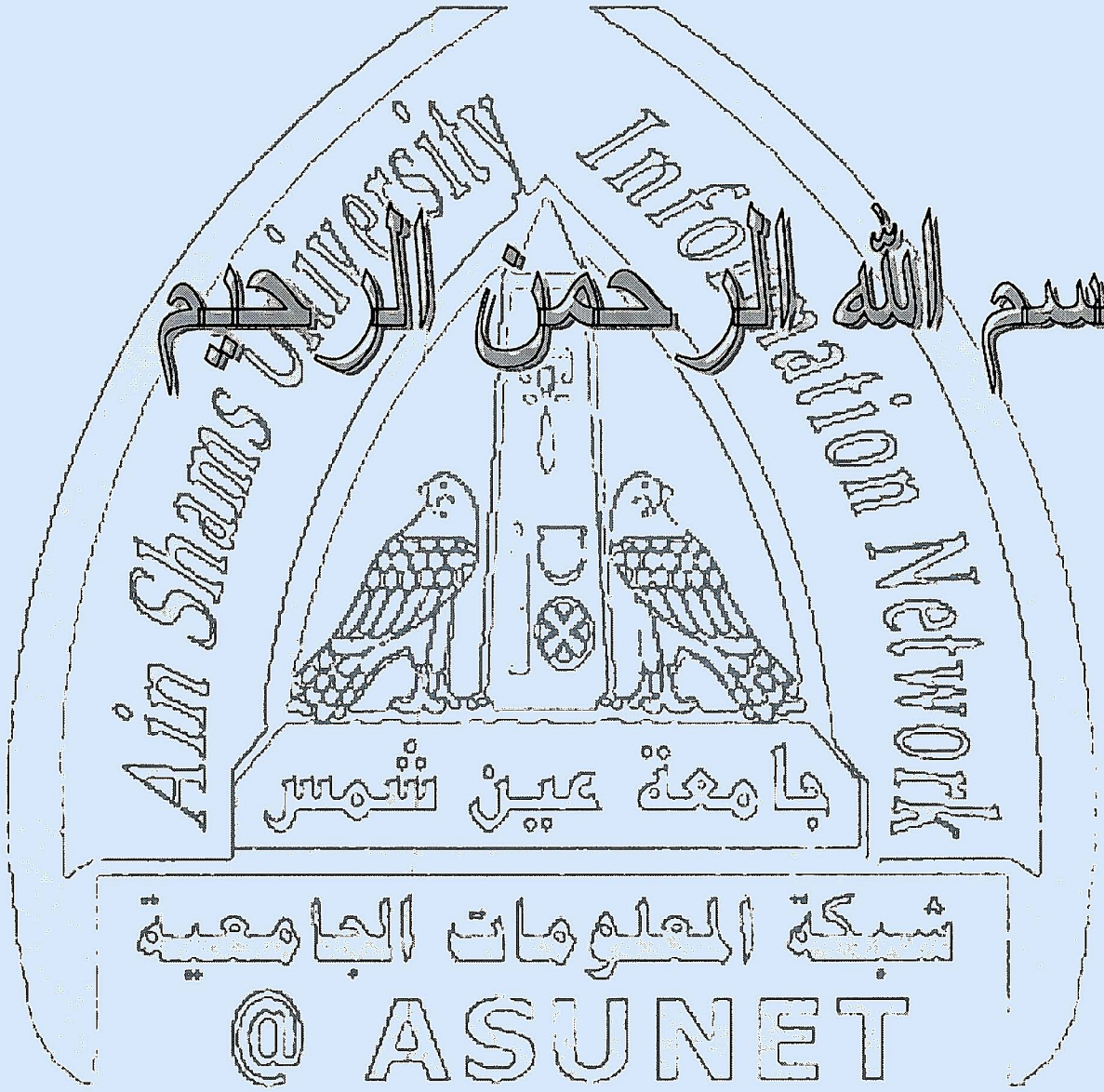
شبكة المعلومات الجامعية



شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

قسم

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شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

بعض الوثائق الاصلية تالفة

ULTRASTRUCTURE OF SPERMATOZOA IN INFERTILE PATIENTS WITH ASTHENOZOOSPERMIA

THESIS

Submitted to the Faculty of Medicine

University of Alexandria

In Partial fulfillment of the requirement of the degree of

MASTER OF DERMATOLOGY AND VENEREOLOGY

By

Shereen Ahmed Hossam - Eldin

M.B.B.Ch. (Alex.)

Dermatology department

Faculty of Medicine

University of Alexandria

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SUPERVISORS

Prof. Dr. Aly Ibrahim El-Said

Professor of Dermatology and Venereology

Faculty of Medicine

Alexandria University.

Prof. Dr. Aly Foad El-Ariny

Professor of Dermatology and Venereology

Faculty of Medicine

Alexandria University.

Prof. Dr. Nabila E. Abdel Meguid

Head of Zoology department

Faculty of Science

Alexandria University.

DEDICATION

To my parents and
their devotions throughout
my life

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CONTENT

Asthenozoospermia

According to the World Heath Organization 1999, asthenozoospermia is defined as spermatozoa motility of less than 50% or more of rapid or sluggish (grade b) progressive motility within 60 minutes of ejaculation ie: (grade a) $\geq$ 25 μm at 37°C. Note that 25 μm is approximately equal to 5 head lengths or half a tail length. It may also be 25% or more with rapid progressive motility within 60 minutes of ejaculation[1] [2]. Abnormal motility (asthenozoospermia) is an isolated disorder, may exist as many as 24% of patients presenting for evaluation of male subfertility and may be significant factor in another 55% of patients with combined defects in sperm density, motility and morphology[3] [4].

Asthenozoospermia, or low motility, generally reflects defects in Spermatogenesis. However in patients with isolated asthenozoospermia, acquired or congenital (genetic) defects in flagellar function are likely [5].

Marchini et al. [6], mentioned that patients with asthenozoospermia had a wide range of decreased sperm motility varying from severe and permanent asthenozoospermia with < 5% progressive motility . Gross malformation of the sperm structure have often been correlated with immotility and a corresponding loss of fertility[4] [7].

Asthenozoospermia, is a common cause of human male infertility and various structural anomalies of the human sperm flagellum have been described in relation to disturbances in sperm motility[8] . Anomalies of the periaxonemal structures, reported to be one of the predictive measures of male infertility status[9], are associated with defects of axonemal complex in patients with severe or total

asthenozoospermia[10]. Isolated anomalies of the periaxonemal elements are related to motility disturbances through a flagellar dyskinesia[11].

Electron microscopy (EM) has been advocated as the most important tool for showing the structural integrity and the potential effectiveness of the sperm[12]. It is already well established that patients with severe (< 10% motile) unexplained abnormalities in sperm motility should, be considered for EM evaluation[13]. Indeed, this was our major reason for performing EM in asthenozoospermic patients [14].

Etiology of asthenozoospermia

Specific causes of asthenzoospermia is to be put into consideration:

1- Artifactual asthenozoospermia

Asthenozoospermia is often secondary to artifacts introduced by collection or analysis techniques. Some plastic containers are spermicidal thus have an effect on motility. The use of soap or saliva during masturbation as a lubricant, motility can be still affected. Exposure of the specimen to extremes of heat may be detrimental prolonged abstinence[13] and prolonged incubation in seminal plasma, leading to decline in motility, owing to specific inhibitors of motility present in the fluid [3].

2- Infection:

Presumed mechanism of infection causing infertility are; bacterial attachment to sperms, immobilizing factor produced by *E. coli*, immune system recruitment and alteration of glandular function[14].

This is established in instances in which organisms produce injury to seminiferous epithelium or epididymal obstruction[15]. Sperm function can be altered at the accessory gland level resulting in infertility. Male accessory gland infection in most of cases is related with increased levels of white blood cells. Several reports documented the association between high levels of WBC in semen and male infertility[16].

The seminal vesicles secrete both compounds that directly stimulate sperm motility and antigens that prevent immune response against spermatozoa. It secrets antigens of IgG-Fc receptor III that protect sperms from IgG-mediated destruction and from antibody-

mediated cellular cytotoxicity[17]. In addition to trophoblast lymphocyte cross-reactive antigens which may induce maternal allogenic reactions implicated in the immunological acceptance and maintenance of pregnancy [17][18].

There are evidence demonstrating that WBC affect fertility. It has been confirmed that men with leukocytes > $1x10^6$/ml had high number of immature germ cells, hypofunction of seminal vesicles, lower sperm vitality and lower sperm motility [16].

It has been demonstrated that asthenozoospermic men with leukocytospermia, activated T-helper lymphocytes and that macrophages showed an activated state toward specific immunoglobulins, supporting the hypothesis that monokines and lymphokines may play a role in sperm motility[19].

Bacterial infection

Microbiological examination should be carried to rule out infection in prostate or/and seminal vesicles. Significant decrease in the sperm motility in patients with bacterial infection may be due to the action of bacterial toxins or due to the damage to sperm organelles or change in milieu, like, seminal plasma. It may be also due to interference with intracellular production of cyclic AMP in spermatozoa[20].

Cablamonne et al. [21], reported that increase in seminal leukocytes (>10^5 while cells/ml of ejaculate) is always associated with decrease of sperm motility. Berger et al.[22], found a definite relationship of % of motility and pyospermia.

It was noted that if ***Escherichia coli***, was ≥ 10^7 colonies/ml, a significant decrease in sperm motility will occur [23]. They can directly interfere with sperm metabolism or sperm tail motility. Paulson and

Polakoski, demonstrated an immobilizing factor produced by *E. coli* that had been found to impair sperm motility in vitro[24]. They also noted that the effect of immobilizing factor was reversed by dilution while incubation of sperm with live bacteria caused an 80% incidence of irreversible necrospermia[25]. Certainly neutrophils recruited in response to infection are known to release reactive oxygen species that can decrease sperm motility[26]. Wolfe et al. [27] investigated, the mechanisms of adherence between *E. coli* and sperm. *E-coli* readily adhered to the agglutinated sperm. The phenomenon was observed at E-coli sperm ratio 1:20. By transmission electron microscope *E-coli* adherence was observed both on sperm heads and tails. Strains of ***Neisseria gonorrhoea***, may be also attached to spermatozoa that can as well interfere with sperm metabolism or sperm tail motility[25].

Mycoplasma (Ureaplasma Urealyticum), has been demonstrated on the surface of spermatozoa in association with coiled tails. and decreased motility[28]. It may contribute to male infertility especially in cases where motility is borderline as a significant improvement in the spermatozoal motility was found in the group infertile men treated from mycoplasmal infection. Toth and Lasses[29], reported that Ureaplasma Urealyticum plays an important role in male infertility by changing semen parameters, including decreased sperm count and decreased sperm motility. Grossegebauer et al., reported that defects in the tail, midpiece segments and binding of the Ureaplasma to sperm could be demonstrated using both light and electron microscopy [30] Bornman et al. [31], reported that the frequency of tail abnormalities in the mycoplasma positive group was greater as compared to normal values.

Chlamydia trachomatis, can be detected in the semen of asymptomatic men with low sperm motility using polymerase chain

reaction technology[32] Erbengi has investigated the significance of chlamydial infection in artificial insemination, using the electron microscopy which revealed the presence of elementary and reticulate body forms of C. trachomatis in spermatozoa[33] Chlamydia not adherent to but also penetrated into the tail structure. Thus affecting functional and morphological forms of trachomatis and can be transmitted by spermatozoa and may cause infertility. Chlamydia was found to be the cause of infection in 67% of men less than 35 years of age who did not have gonorrhoea bacterial infections. Further studies are required to define, the extent to which C. trachomatis may alter the, function of male reproductive organs[34].

3- Varicocele

Varicocele is dilatation, tortousity and incompetence of valves of the pampiniform plexus / internal spermatic venous system that drains the testicle. Varicocele is discovered in approximately one-third (19%-41%) of men being evaluated for infertility[35]. It is usually more apparent on the left more than the right side and is diagnosed by palpation of the standing patient. The veins distend with increase abdominal pressure during the valsalva maneuver. Depending on the results of palpation, the varicocele is assigned to one of the following grades[36]:Grade I; can be palpated only during valsalva maneuver, grade II; can be palpated without a valsalva maneuver, grade III; is a visible distension of pampiniform plexus and grade IV; is atrophy of the testis[35]

Several anatomic features, all of which increase hydrostatic pressure within the left testicular venous drainage system. The left internal spermatic vein drains perpendicularly into the left renal vein at a