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MALE INFERTILITY: DIAGNOSIS, TREATMENT, OVERCOMING

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ABSTRACT

Major difficulties exist in the accurate and meaningful diagnosis of male reproductive dysfunction, and our understanding of the epidemiology and etiology of male infertility has proven quite complex. Sperm analysis is still the cornerstone in diagnosis of male factor infertility, indeed, individually compromised semen paramaters while adequately address therapeutic practices is progressively flanked by additional tests. Administration of drugs, IUI, correction of varicocele, and, to a certain extent, IVF although they may not be capable of restoring fertility itself often result in childbearing.

INTRODUCTION

In contrast with general medicine, “infertility” belongs to a special category of human ailments. Where a specific disease is present, diagnosis and treatment are closely linked and the successful outcome involves a close binomial relationship between the patient and the medical practitioner. In human infertility, the treatment is focused on the two partners, and the discomfort generated by human infertility is usually not affecting their general health but is linked for the most part to the performance of the female and the male gametes per se.

MATERIALS AND METHODS

While the diagnosis of male factor infertility is the most straightforward the etiology is still unclear. In fact, the underlying cause of oligoastheno-terato-zoospermia (OAT) is still unknown rendering the effectiveness of any conventional treatment for male infertility extremely doubtful. Reports have drawn attention to genetic defects in spermatozoa as a cause of male infertility [3]; however, the availability in the last 20 years of micromanipulation techniques has made it feasible to treat infertility related to issues in the male partner in ways that previously would have been deemed hopeless. These new therapeutic options have opened fresh perspectives, providing ground for critical analysis of the available conventional diagnostic and therapeutic approaches that involve either drugs, varicocele surgery, intrauterine insemination (IUI), IVF, and ICSI.

RESULTS AND DISCUSSION

According to varying surveys, it appears now that the male partner is the culprit in some 25–50 % of infertility cases. Since the focus on human semen quality and its relationship to male fertility began in the late 1940s [4, 5], numerous studies have probed the fluctuations in sperm concentration among human ejacuates in relation to the ability to reproduce [6]. Apart from physiological variations, three elements that appear to potentially affect human sperm production include environmental, genetic, and psychosocial factors. While each of these can act individually they can often compound their effect producing the male infertile state.

In the general population, about 25 % of couples do not achieve pregnancy within 1 year, 15 % seek treatment for infertility, and less than 5 % remain childless. Nonetheless, despite advances in the diagnostic workup of infertile men, in about 50 % of men with compromised spermatogenesis the cause is unrecognized [7–9]. It has been demonstrated that DNA damage in human spermatozoa has been linked with poor semen performance, resulting in poor fertilization rates, impaired embryo development, increased pregnancy loss and a possible health consequences in the offspring, not excluding cancer [10–13]. However, at this stage the origin and role of sperm DNA fragmentation in the male gamete, as currently measured, is still controversial.

The position of andrology in the male infertility workup has been strengthened by the introduction of testicular sperm extraction (TESE) or microsurgical epididymal sperm aspiration (MESA) following appropriate differential diagnosis of infertility causes (obstructive/non-obstructive azoospermia, congenital bilateral absence of the vas deferens) and in some cases, testicular biopsies. The effectiveness of treating varicoceles in terms of pregnancy outcome is still a matter of discussion. However, it seems clear that spermatogenesis can be, at least partially restored, after microsurgical varicocelectomy in some men with cryptozoospermia or secretory azoospermia [31].

While obtaining an inferility history it is important to attempt to identify any risk factors (e.g., cryptorchidism, environmental hazards), that may be of aid in their andrological assessment, unfortunately, the overzealous enthusiasm of the reproductive specialists may drive the excessive use of unconfirmed diagnostic tests. However, these tests that claim to predict fertility status or to indicate the appropriate ART procedure risk to emotionally and financially drain childless men.

In the case of of chromosomal anomalies, these can be numeral, structural, or both. Aneuploidy leading to male infertility may involve the sex chromosomes (e.g., an additional X-chromosome in Klinefelter's syndrome) or autosomes (e.g., trisomy 21). Structural chromosome anomalies (small deletions, translocations, inversions) can lead to male infertility and these may involve both sex or autosomal chromosomes.

The information generated by conventional semen analysis has historically classified patients into categories lacking knowledge of causality and leaving conventional therapy as somewhat empirical and at times ineffective (Table 1). A single condition such as oligozoospermia may involve a multitude of different etiologies. It is not until we resolve the causes of male infertility at a molecular level that we shall be able to achieve the holy grail of diagnosis, treatment, and prevention.

The epidemiology of male reproduction has been evolving quite rapidly in recent years. A better understanding of spermatogenesis and its genetic control has led to the formulation of new hypotheses on the role of the DNA packing and small RNAs.

Apart from a few exceptions, central hypogonadism and some post-testicular forms, the only available treatment option for the large majority of male infertility situations is medically assisted reproductive technologies, represented by in vitro fertilization [4] or in the presence of extreme OAT and various forms of azoospermia, ICSI as the preferred method [1]. ICSI is the most effective means of treating couples with male factor infertility and previous ART fertilization failures. This is consistent with the spermatozoa collected from the epididymis and from the testis achieving comparable fertilization and consistent pregnancy outcomes.

Table 3

Diagnostic categories for the infertile male based on WHO criteria

No demonstrable cause
Idiopathic oligozoospermia
Idiopathic asthenozoospermia
Idiopathic teratozoospermia
Idiopathic azoospermia
Obstructive azoospermia
Isolated seminal plasma abnormalities
Sexual or ejaculatory dysfunction

CONCLUSION

However, ART is a symptomatic therapy which does not address the underlying cause for infertility with the risk of transmitting both identified and concealed genetic anomalies. An increased incidence in chromosomal anomalies and possibly neonatal malformations have been reported especially when the indication for utilizing ART is severe male factor infertility [3]. Apart from the mentioned health consequences of the offspring fathered by a man with severe spermatogenic failure, including the inability to reproduce such as sons of men with Y deletions. In fact, there is still very little known about the long term health conditions of both the infertile man and their offspring [2]. A higher incidence of sperm aneuploidy in infertile men with secretory azoospermia translates to a higher frequency of gonosomal abnormalities in the male progeny, possibly because of meiotic defects surfacing during male germ line maturation.

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