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Inflammatory Markers And Their Role In Male Infertility.

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Abstract

Background: Inflammation plays a significant role in male reproductive health and has increasingly been recognized as an important contributor to male infertility. Male infertility accounts for nearly 40% of infertility cases worldwide and is influenced by multiple inflammatory and oxidative mechanisms affecting spermatogenesis and sperm function. This review discusses the role of inflammatory markers in male infertility, focusing on cytokines, chemokines, acute phase proteins, and oxidative stress-related biomarkers. Pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferon-gamma (IFN-γ) have been implicated in disruption of the blood–testis barrier, hormonal imbalance, oxidative stress, and germ cell apoptosis. Oxidative stress markers including reactive oxygen species (ROS), malondialdehyde (MDA), and 8-hydroxy-2-deoxyguanosine (8-OHdG) are closely associated with sperm dysfunction and impaired semen quality. The review further explores mechanistic pathways linking inflammation with impaired spermatogenesis, clinical evidence correlating inflammatory markers with semen abnormalities, and the potential diagnostic and therapeutic implications of these biomarkers. Anti-inflammatory therapies, antioxidants, and lifestyle modifications have shown variable improvements in semen parameters and fertility outcomes. Despite growing evidence, substantial gaps remain regarding the causal relationship between inflammation and male infertility. Further research is necessary to establish standardized biomarkers and targeted therapeutic strategies for clinical application.

Keywords: Male infertility, inflammation, cytokines, oxidative stress, spermatogenesis, seminal plasma, biomarkers.

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INTRODUCTION

Inflammation affects various aspects of human reproduction, including fertility, assisted reproduction techniques, miscarriage, and end-pregnancy outcomes [1,2].

This review focuses on male fertility. An estimated 40% of infertility cases worldwide are attributed to male factors. Spermatogenesis transpires throughout male life and constitutes the basis of male fertility. Thus, elucidating spermatogenesis and identifying factors that impact it are key to understanding male fertility. The four main hypotheses positing how inflammation impairs spermatogenesis are as follows: (1) disruption of the blood-testis barrier and/or accessory reproductive tract barriers [15]; (2) interruption of hormonal homeostasis via the hypothalamic-pituitary-gonadal (HPG) axis [16]; (3) increased oxidative stress due to either elevated reactive oxygen species (ROS) production or decreased antioxidant activity [17]; and (4) germ cell apoptosis due to the activation of pro-apoptotic factors or the inhibition of anti-apoptotic factors [18]. The cytokine interleukin 1β (IL-1β) has been well characterized within the first and fourth hypotheses. Furthermore, many inflammation-associated factors have been found to impact spermatogenesis, notably the pro-inflammatory cytokines IL-6, tumor necrosis factor alpha (TNF-α), and interferon gamma (IFN-γ) [19,20]. An additional recent focus is whether inflammation acts as a causal or associative agent in human male infertility. Several conditions associated with male infertility, such as obesity, diabetes, varicocele, and metabolic syndrome, are known to induce, amplify, or prolong inflammation, establishing a direct link between the inflammatory state and male infertility [3,19]. Nevertheless, the confidence that these conditions trigger male infertility through inflammation remains low.

2. Overview of Male Infertility

Infertility is defined as the inability of a couple to conceive after twelve months of having unprotected sexual intercourse [1]. Male infertility affects 10–15% of men of reproductive age. Gametogenesis is a complex process with many steps, and inflammation may play a central role through the secretion of cytokines and the expression of microRNAs. The phenotypes of male infertility can be classified according to the predominant abnormality in the semen. Abnormalities in male semen quality are associated with several factors, including rising environmental pollution and increased exposure to reproductive toxins, which are reported to have detrimental effects on male fertility [3,4]. Globally, pollution by endocrine-disrupting chemicals is at an alarming level. Academic institutions and regulatory bodies need to take action in studying, monitoring, and minimizing their effects on human health and reproduction [5,6].

As an enriched microenvironment for undergoing spermatogenesis, the male reproductive tract represents a site for multifaceted immunological challenge and surveillance. Local generation and systemic circulation of inflammatory substances in the semen and blood, respectively, serve as potential indicators that reflect the inflammatory status of the male reproductive tract [7,8]. In vitro studies demonstrated that intra-testicular injection of pro-inflammatory cytokines impairs the architecture and functional activities of the male reproductive system. Also, inflammation of the male reproductive tract induces the recruitment of inflammatory immune cells into the male reproductive fluid, thereby affecting its constitution. Such local and systemic inflammatory responses may accompany excessive production of pro-inflammatory cytokines, chemokines, acute phase proteins, and indicators of oxidative stress, resulting in alteration of semen quality and the several male semen abnormalities, and excessive and chronic inflammatory responses may further induce apoptosis of spermatogenic cells in the testis [1].

3. Inflammation and the Male Reproductive System

Markers of inflammation influence male fertility by acting on the hypothalamic-pituitary-gonadal axis and by affecting different stages of spermatogenesis [1]. Inflammation can, therefore, have a profound impact on male fertility, yet the exact nature of the relationship between inflammation and male infertility remains to be fully determined. What remains unclear is whether changes in inflammatory markers reflect cause or consequence on the individualisation of male factor infertility. Furthermore, although inflammation can be associated with male infertility, many instances of male factor infertility are not associated with an inflammatory response, leading to a substantial proportion of the general population with male factor infertility remaining with an unknown or idiopathic origin.

Male infertility is often regarded as a silent epidemic that affects 10–15 % of men worldwide [1]. Worldwide studies demonstrate that 35–50 % of men exhibit some form of semen abnormality. Such seminal characteristics are known to negatively impact the quality of the semen and have been linked to the presence of high levels of inflammatory markers. Various countries work to grasp the underlying phenomena surrounding inflammation and male infertility to help tackle this growing concern.

Although the median range of inflammation markers fluctuates regionally throughout Europe, hallmark inflammatory markers have consistently been identified to be linked to male infertility, such as Interleukin-1 (IL-1), Interleukin-6 (IL-6), Tumor necrosis factor alpha (TNF-α), and Interferon-gamma (IFN-γ) to name a few. These markers have had their concentrations measured in seminal plasma [1].

4. Key Inflammatory Markers Implicated in Male Infertility

The pathophysiology of male infertility has spawned speculation about a possible association with inflammation. Investigations to date implicate a variety of etiology-dependent noxa ranging from infectious agents (bacteria, viruses) through proinflammatory damage-associated molecular patterns (DAMPs) (estradiol, heat shock proteins, phosphatidylserine) to the effects of exogenous pollutants (heavy metals, harmful chemicals, drugs) [1]. Of particular importance in this context, however, are infectious agents in general (particularly the human immunodeficiency virus, varicella zoster virus, and hepatitis B virus) and agents associated with sexually transmitted diseases (Chlamydia trachomatis, Neisseria gonorrhoeae, Mycoplasma hominis), which have been implicated in male infertility by several groups, in some cases more than a decade ago. Collectively, these observations support consideration of inflammation as a prominent factor in the male infertility epidemic.

4.1 Cytokines

Cytokines are pro-inflammatory markers associated with certain inflammatory diseases, the immune system, and spermatogenic processes [1]. The key cytokines concerning male infertility include interleukin-1 (IL-1), IL-6, tumor necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ), and transforming growth factor-beta (TGF-β). Cytokines, as biologically active glycoproteins that receive and transmit signals through cell-surface receptors, are secreted by reproductive and non-reproductive cells in the male reproductive tract under physiological and pathological conditions.

The testis exhibits an immune-privileged status, in which the blood–testis barrier and other immune control mechanisms protect Sertoli cells, germ cells, and Leydig cells from both systemic and local inflammatory cytokines. Local factors that concentrate specifically in the testis, such as the inhibitory action of testosterone on TNF-α secretion by macrophages, are

to some extent capable of counteracting the recruitment of systemic pro-inflammatory cytokines. Main sources of pro-inflammatory cytokines in the male reproductive tract include macrophages, Leydig cells, Sertoli cells, and germ cells. Signalling pathways activated by these cytokines, such as NF-κB, the activation of which is induced by TNF-α, are commonly involved in other pathologies in different tissues and organs [1].

4.2 Chemokines

Chemokines comprise a large family of small (8–12 kDa) but structurally related proteins that serve as ligands for cell-surface G protein–coupled receptors [9]. They participate in diverse physiological and pathophysiological processes, including the recruitment of leukocytes during inflammation, and promote a variety of cellular functions such as proliferation, differentiation, and apoptosis [1]. In the context of the male reproductive system, several chemokines and their respective receptors are involved in the normal development and maturation of spermatozoa, while cytokines and chemokines produced by accessory reproductive organs also act in paracrine fashion. In combination with these chemokines, pro-inflammatory cytokines such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor alpha (TNF-α) are amongst the earliest pro-inflammatory mediators produced by testicular and accessory glands during inflammation [1].

4.3 Acute Phase Proteins

Persistent inflammation can significantly affect male reproductive health through various mediators, one such family being Acute Phase Proteins (APPs). CRP and serum amyloid A (SAA) are the most frequently investigated human APPs that correlate closely with systemic inflammation. Complement component C3 has also been studied as an APP, together with the chemokines CXCL8/IL-8 and CCL2 [1]. It is worth mentioning that chronic inflammatory conditions such as chronic inflammation of the prostate increase the frequency of AMP deposition in the spermatogenic epithelium in humans and in animal models, and correlate with affected semen quality. APPs and their associated-pathologies have been correlated with diminished sperm performance in a cohort of men presenting with either unexplained infertility or other pathology-pathologies located in the genital tract. Fertility potential decreases when sperm morphology is affected, such as in AmpaB or C.

Beyond the involvement of APP in sperm action, their measurement in serum has emerged as a potential option for evaluating inflammation in male fertility. The concentration of SAA in the serum of oligozoospermic and azoospermic men is higher than that of fertile men, being considered a potential late diagnostic marker for the assessment of fertility. Detection of the above-mentioned complement component is not feasible in the seminal plasma of oligozoospermic individuals; however, the measurement in serum and its strong correlation with sperm quality suggests its complementary role along with other selected pro-inflammatory cytokines in assessing the status of the male reproductive system. Based on the above, Acute Phase Proteins constitute an important group of factors to be included in further investigations [1].

4.4 Oxidative Stress and Related Markers

Oxidative stress (OS) arises from an imbalance between reactive oxygen species (ROS) production and cellular antioxidant defenses, resulting in redox equilibrium disruption and cellular dysfunction [10]. OS affects male fertility by damaging sperm quality and function [11]. Elevated seminal plasma ROS levels are commonly observed among infertile men, often due to testicular or post-testicular infections, smoking, alcohol consumption, industrial chemicals, medications, and circadian cycles. Seminal leukocytes exacerbate oxidative stress through ROS synthesis, with seminal plasma antioxidant systems that normally protect sperm from OS proving insufficient in pathological conditions. Consequently, high ROS concentrations accelerate sperm apoptosis. Lipid species peroxidation, leading to malondialdehyde (MDA) accumulation, and sperm DNA damage indicated by 8-hydroxy-2-deoxyguanosine (8-OHdG) pose significant preconception risk.

Measuring seminal ROS reflects sperm damage risk [12]. Infertile men exhibit higher plasma lipid peroxidation, sperm ROS levels, and seminal lipid peroxidation, alongside reduced total antioxidant capacity. Malondialdehyde quantification facilitates oxidative stress assessment due to its stable yet toxic nature, while both seminal MDA and 8-OHdG detection are relevant to male preconception care.

5. Mechanistic Links Between Inflammation and Spermatogenesis

Normal spermatogenesis occurs within a unique immune-privileged environment [13]. Sequential inflammatory events interfere with different stages of this tightly regulated process and lead to impaired fertility [13]. Consequently, a variety of etiologies related to male infertility—such as testicular trauma (primary testicular torsion or assisted reproductive technology), genital infection or inflammation, and exposure to harmful agents (mining, working with metals or chemical compounds, smoking, and poor diet)—are associated with inflammatory processes [1]. Although the relationship between these conditions and the origin of male infertility remains uncertain, elevated inflammatory markers in the genital tract reflect a potential role in impaired reproductive function.

Two candidate mechanistic pathways that may link inflammatory processes to spermatogenesis and male infertility are summarized. Males with normal seminal analysis face limited options when undertaking fertility treatments, magnifying the need to fully understand and target the underlying inflammation-related etiology [1].

6. Clinical Evidence: Associations Between Markers and Semen Parameters

Numerous studies have investigated the association of specific inflammatory markers with semen parameters from clinical samples of infertile men to explore the hypothesis that inflammation contributes to male infertility [1]. Such work encompasses a wide variety of measurement approaches and study designs applied to diverse patient cohorts, resulting in a complicated picture. Nevertheless, several reports have proceeded to a sufficient depth of analysis to justify the identification of noteworthy connections among inflammatory markers and semen quality or reproductive success [1]. The overall conclusion is that, notwithstanding the heterogeneity of variables involved, there is substantial intra- and inter-study support for clear-cut, if multifocal, associations between the semen inflammatory milieu and one or more features of reproductive deficiency.

Many of the studies that examine the link between inflammatory markers and reproductive function in males do not transparently indicate whether the objective is to determine solely an association or whether causality is intended to be part of the investigation. Where the focus is more firmly centred on association, limitations on the validity of such interpretations remain pervasive. Primarily, factors that co-occur with exposure to the inflammatory marker under scrutiny are often associated with the poor reproductive function of interest without being explicitly controlled, leaving open the possibility that an observed correlation stems, at least in part, from those alternative sources rather than from the specific inflammatory signature itself [1].

7. Diagnostic and Therapeutic Implications

When focusing on inflammatory markers and male infertility, attention can shift from establishing a link to evaluating diagnostic and therapeutic implications of widely reported markers such as cytokines and oxidative stress indicators [1]. Depending on the clinical context, the presence of inflammatory biomarkers in body fluids may then support the diagnosis of an inflammatory condition or facilitate differentiation between distinct male infertility profiles. Likewise, such markers may serve as therapeutic targets for anti-inflammatory interventions, antioxidants, or lifestyle modifications associated with semen quality improvements.

Diagnostic implications extend beyond the inflammatory history or the symptoms prompting the male fertility assessment, as excess inflammatory signals can be present even in the absence of apparent signs or a limited number of clinical indicators. Therefore, it becomes relevant to explore which inflammatory markers assume considerable value for diagnosis or monitoring purposes. The parameters of clinical interest include their sensitivity, specificity, positive predictive value, and negative predictive value, as well as the panel of markers that may have been proposed for evaluating a specific context and the optimal time-point for their assessment concerning clinical examination and sampling [14].

7.1 Diagnostic Utility of Inflammatory Markers

Numerous studies associate inflammatory markers with male infertility. However, exploration of their diagnostic utility across the spectrum of male-factor subfertility remains limited. Individual inflammatory markers have been evaluated as potential diagnostic tools for male fertility and their union into panels for simultaneous assessment has been proposed. Still, a critical synthesis of the evidence across all studies measuring the association between inflammation and any seminal fluid parameter remains absent [14]. Inflammation can arise in the testis or in reproductive accessory glands surrounding the epididymis. In both cases, seminal fluid characteristics may change and localization influences infiltration by circulating immune cells, seminiferous epithelium involvement, and spermatogenesis disruption. Men with unconfounded normal sperm concentrations develop normal total sperm counts yet often bear semen parameters that indicate poor fertilizing potential. These patients may benefit from evaluation for male inflammatory factors [1].

7.2 Therapeutic Targets and Interventions

Infertility due to inflammation is a complex phenomenon that impacts male reproductive health, and different therapeutic avenues have been proposed, but few include randomized controlled studies that evaluate parameters of fertility or development of offspring. Anti-inflammatory approaches result in improvement of sperm parameters in azoospermic, oligozoospermic, pickled sperm, and asthenozoospermic men [1]. Antioxidants are employed as protective agents of oxidative damage mediated by inflammatory cytokines. The application of coenzyme Q10, vitamin E, and others leads to specific amelioration of sperm parameters, and improvement of fertility was also observed. The extent of improvement in specific sperm parameters was variable among the different antioxidants. A surgical intervention in varicocele treatment yields enhancement of several sperm parameters, and this restoration of fertility is frequently observed. Excessive body weight invokes an inflammatory milieu that affects fertility. Weight reduction is seen to improve fertility in obese patients, the parameters that respond to weight loss vary according to the individuals. Other lifestyle changes that enhance fertility include alcohol restriction, cigarette cessation, and cessation of chemotherapy and anti-cancer agents [1].

8. Methodological Considerations in Research

Inflammatory response constitutes a considerable threat to the male reproductive system, affecting either accessory glands or testis directly through infections or even a systemic inflammatory insult [1]. Spermatozoa production is a complex process influenced by many factors. The testicular environment is delicate and needed to be well protected from any of

such threats. Pathological situations leading to spermatozoa production impairment especially during inflammatory events show a high contribution of cytokines, chemokines, and pro-inflammatory microRNAs in the seminal plasma. The presence and levels of these inflammatory markers and the corresponding injury induced by elevated pro-inflammatory mediators show a considerable correlation with different types of male infertility [14]. The early identification of specific inflammatory markers in the seminal plasma can determine the initial steps of establishing a helpful assay to be used in accordance to common semen analysis, the first step in diagnosing male infertility.

Only a few studies have correlated the presence of different inflammatory markers in the seminal plasma along with routine semen analysis to characterize further the different types of male infertility. An extensive characterization for each type of male infertility is needed, as each can show a different origin leading to specific acute or chronic inflammatory signals. Understanding better and identifying novel different sperm toxicity markers beyond the most classic ones is necessary based on emerging scientific evidence [14]. Inflammation, aging, and male infertility are emerging as some of the hottest topics across the globe, and more efforts and funds should continuously be invested exploring these areas, along with the already covered classic subjects [1].

9. Gaps in Knowledge and Future Directions

Inflammation is a complex physiological process, resulting from harmful stimuli such as pathogens, damaged cells, or irritants. Male infertility, defined as failure to achieve pregnancy after one year of unprotected intercourse, affects approximately 10–15% of reproductive-age couples, with an estimated male contribution of around 50% [1]. Semen analysis, which typically examines sperm count, motility, morphology, and pH, often reveals abnormalities; thus, the condition is often referred to as male factor infertility. In male infertility, a plethora of altered seminal plasma concentrations of biochemical constituents have been reported, some of which correspond to the inflammatory process, mirroring the status of systemic inflammation [14].

CONCLUSION

The investigation of inflammatory markers identified across diverse studies elucidates the multifaceted impact of inflammation on male fertility. Despite evidence of altered levels in various reproductive-related conditions, inflammation in contextualized terms emerges as a potential contributor to diminished fertility prospects. Expansion of the current dataset linking specific markers to objective semen parameters obtained in an unselected cohort aligns inflammatory pathways with exclusive hyperviscosity criteria. Cytokines function as major orchestrators of local and systemic inflammatory responses, with hormones modulators of corresponding signals and spermatogenic rhythm. Collectively, data identify the aberrant rise of distinct inflammatory markers in seminal fluid as one pattern recurrently associated with male infertility contexts displaying specific alterations of spermatozoal parameters [1]. Multiple male reproductive disorders associate with inflammation; yet, differentiating firmly established etiologies signalling evidently altered semen quality from the largely associative imprint inflammation emerges with on still-clinically-anonymous profiles guides a more precise approach.

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