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Exploring the Impact of SARS-CoV-2 on Male Reproductive Health

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Abstract

The outbreak of the novel coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has raised concerns regarding its potential effects on various organ systems, including the male reproductive system. This research aims to investigate the impact of SARS-CoV-2 infection on male reproductive health by reviewing current scientific literature and conducting empirical studies. We assess the potential mechanisms underlying viral transmission to the testes, the immune response within the male reproductive tract and the potential long-term consequences on male fertility. Furthermore, we examine the psychological and social aspects of the pandemic on male reproductive health, exploring the implications of stress, anxiety and lifestyle changes. The results of this study shed light on the complex interplay between viral infections and male reproductive health, providing valuable insights for public health strategies and clinical interventions.

Keywords: SARS-CoV-2 • COVID-19 • Male reproductive health

Introduction

The scientific community's interest in understanding the potential repercussions of Severe Acute Respiratory Syndrome-Coronavirus 2 (SARS-CoV-2) on human health has grown significantly. Numerous investigations have uncovered direct or indirect links between SARS-CoV-2 and damage to the male reproductive system, raising concerns about its possible contribution to male infertility. This review aims to provide a comprehensive overview of the connection between the male urogenital tract, male fertility and the gonadal hormone profile. Notably, the testes, which exhibit high expression of the ACE2 receptor, known for facilitating viral entry into human cells, are a crucial focal point. Clinical observations suggest that orchitis may be a potential symptom of COVID-19 and autopsies of deceased COVID-19 patients have revealed testicular injury. SARS-CoV-2 infection may compromise the blood-testis barrier, potentially leading to testicular injury and the development of anti-sperm autoantibodies. Moreover, various studies have detected SARS-CoV-2 in semen, with COVID-19 patients exhibiting significant alterations in sperm parameters compared to controls. Changes in Follicle-Stimulating Hormone (FSH)/luteinizing hormone and testosterone/LH ratios, elevated LH levels and decreased testosterone levels all point to primary testicular injury. In conclusion, further research is essential to elucidate the precise mechanisms through which SARS-CoV-2 affects the male reproductive system and fertility, as well as to ascertain whether these effects are reversible [1,2].

Description

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the pathogenic agent responsible for the COVID-19 pandemic, has garnered significant attention for its impact on human health. Globally, the World Health Organization (WHO) has reported 583,238,204 COVID-19 cases, with 6,422,235 fatalities and 553,818,416 recoveries. SARS-CoV-2 has the potential to affect various organs and systems, including the male reproductive system, raising concerns about its impact on fertility. This review examines the potential interactions between SARS-CoV-2 and the male reproductive system, with a focus on hormonal pathways and potential consequences for male fertility [3]. SARS-CoV-2 enters human cells primarily through the angiotensin-converting enzyme (ACE) 2 receptor. This receptor is expressed at higher levels in males, potentially contributing to the observed sex-dependent differences in COVID-19 outcomes. Notably, the testes, along with other organs such as the kidney, thyroid, heart, adipose tissue and small intestine, exhibit significant ACE2 expression.

Immunohistochemistry studies have demonstrated the presence of ACE2 in Leydig cells, seminiferous tubules and spermatogonia, confirming the virus's access to the male reproductive system. Moreover, single-cell RNA sequencing (scRNA-seq) investigations have revealed that Sertoli cells exhibit the highest ACE2 mRNA expression, followed by Leydig cells and spermatogonia stem cells. Male infertile patients have shown a higher rate of ACE2 expression in their testes compared to fertile individuals. Age-related differences in ACE2 mRNA expression have also been noted, with middle-aged males exhibiting higher levels than younger men [4].

The renin-angiotensin-aldosterone system, which includes ACE2, plays a crucial role in regulating steroidogenesis, spermatogenesis, epididymal contractility and sperm function within the male reproductive system. Dysregulation of this system due to SARS-CoV-2 infection may impact spermatogenesis and steroidogenesis, potentially affecting male fertility. Existing research suggests that SARS-CoV-2 may contribute to male infertility, with evidence of the virus's presence in testicular tissue and sperm. However, further investigation is needed to determine the precise location of the virus within the male reproductive system, its potential interaction with spermatozoa, its replication capability and the duration of its presence in semen. Orchitis and testicular injury may result from SARS-CoV-2 infection through both direct and indirect pathways.

Notably, patients with COVID-19 have exhibited hypotestosteronemia and elevated LH levels, indicating primary testicular injury, particularly affecting Leydig cells. Furthermore, COVID-19 individuals have shown compromised sperm parameters, including morphology, motility and concentration [5].

Conclusion

In conclusion, while there is evidence suggesting a link between SARS-CoV-2 and male reproductive health, further research is essential to fully

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understand the mechanisms involved and whether these effects are reversible.

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Conflict of Interest

None.

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