Case Reports and Reviews

Case Report

COVID-19 and Male Fertility: Current Evidence on Testicular Involvement and Reproductive Outcomes

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Abstract

COVID-19, caused by SARS-CoV-2, has been linked to multiple systemic complications, including potential effects on the male reproductive system. Evidence suggests that the virus may impair spermatogenesis, alter hormone levels, and disrupt testicular architecture through direct viral entry, immune-mediated mechanisms, and oxidative stress. While many studies report transient effects, particularly in mild cases, severe and long-COVID may result in prolonged dysfunction. This review summarizes the biological pathways by which SARS-CoV-2 may impair testicular function, highlights clinical and experimental evidence, and discusses implications for fertility preservation, reproductive counseling, and therapeutic interventions.

Introduction

Viral infections have long been associated with male reproductive dysfunction. Pathogens such as human papillomavirus (HPV), herpes simplex virus (HSV), and human immunodeficiency virus (HIV) are known to impair sperm quality and testicular function. Since the onset of the COVID-19 pandemic, there has been growing concern regarding the potential effects of SARS-CoV-2 on male fertility. This review consolidates recent evidence regarding testicular involvement, semen quality, hormonal disruption, and clinical implications, providing an integrated perspective on short- and long-term reproductive risks in men affected by COVID-19.

Mechanisms of Testicular Involvement

SARS-CoV-2 gains entry into host cells through the angiotensin-converting enzyme 2 (ACE2) receptor and the transmembrane protease serine 2 (TMPRSS2). Both proteins are highly expressed in testicular tissue, including spermatogonia, Sertoli cells, and Leydig cells [1,2]. Their expression in seminiferous epithelium and interstitial compartments makes the testes a plausible viral target.

Postmortem histopathological examinations have confirmed germ cell loss, lymphocytic infiltration, and disruption of the seminiferous epithelium [3]. These changes may arise not only from direct viral invasion but also from cytokine-mediated inflammation, vascular injury, and oxidative stress [4]. Elevated levels of interleukin-6 and tumor necrosis factor-α in systemic circulation during acute COVID-19 episodes may exacerbate testicular damage and impair spermatogenesis.

Evidence from Clinical and Experimental Studies

Histopathological and molecular examinations in deceased COVID-19 patients revealed SARS-CoV-2 presence in testicular tissues, with associated spermatogenesis damage [5,6]. Experimental models further demonstrated testicular injury and hypogonadism induced by viral infection, which could be attenuated by anti-inflammatory interventions [7].

Clinical studies corroborate these findings. Prospective and longitudinal investigations demonstrate reductions in sperm concentration, motility, and morphology, as well as increased sperm DNA fragmentation during and after COVID-19 infection [8–10]. In some cases, semen quality remained compromised for several months following recovery. Endocrine alterations have also been reported, including reduced serum testosterone and elevated luteinizing hormone (LH), consistent with primary hypogonadism [11].

Recovery and Reversibility

While severe cases appear to cause lasting testicular impairment, some studies suggest recovery of semen parameters and hormone profiles within three to six months post-infection, particularly in patients with mild or moderate COVID-19 [12,13]. Improvements in sperm motility and concentration have been documented, although full restoration to pre-infection levels may not always occur.

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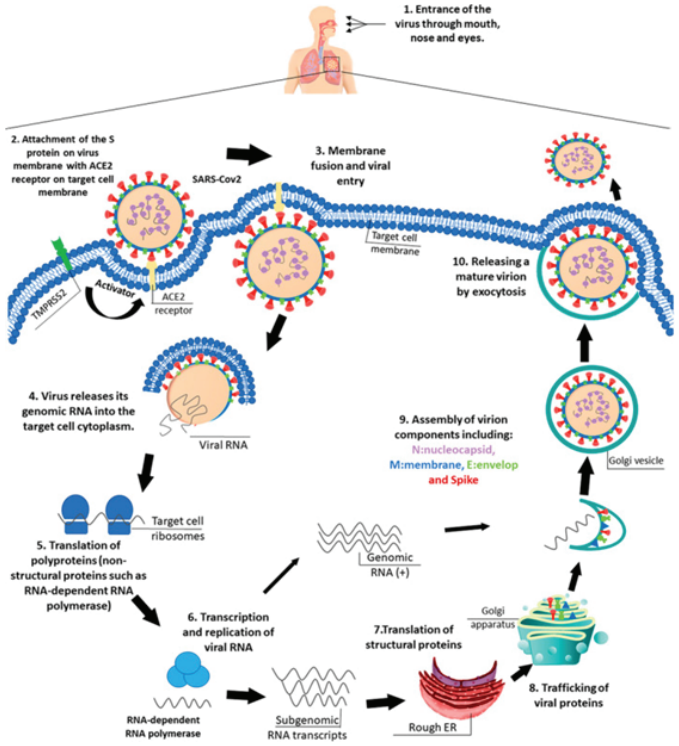

Figure 1. *Schematic representation of SARS-CoV-2 entry into the testes. The virus binds ACE2 receptors on spermatogonia, Sertoli cells, and Leydig cells, with TMPRSS2 facilitating membrane fusion. Resulting inflammation, oxidative stress, and vascular injury contribute to testicular dysfunction.*

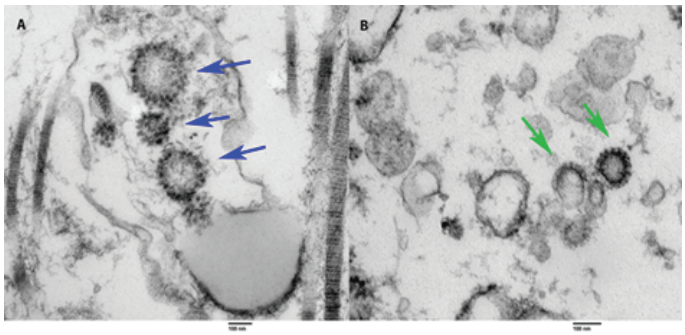

Figure 2. *Electron microscopy images of testicular tissue containing SARS-CoV-2 viral particles. Left panel (A): tissue from a COVID-19-positive male patient. Right panel (B): testicular tissue collected postmortem from a man who died of COVID-19. Blue and green arrows indicate spike-coated viral particles (Chu et al., 2020).*

The degree of recovery may depend on age, pre-existing comorbidities such as obesity or diabetes, and severity of the initial infection. Persistent testicular dysfunction has been described in patients with long-COVID syndromes, characterized by ongoing fatigue, inflammation, and hormonal imbalance [14].

Clinical Implications

Given the potential for reproductive impairment, fertility counseling is recommended for men recovering from COVID-19, particularly those with severe or persistent disease. Follow-up semen analysis and hormonal testing should be considered for men planning conception.

Cryopreservation of sperm may be advisable in cases of severe disease or when ART (assisted reproductive technology)

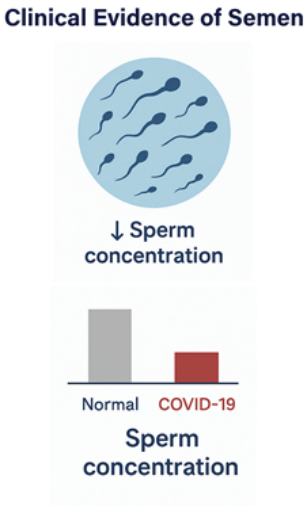

Figure 3. *Summary of clinical findings on COVID-19 and semen quality. Graphical overview showing reduced sperm count, motility, and morphology, increased DNA fragmentation, and alterations in hormone levels (testosterone ↓, LH ↑).*

is anticipated. Research into antioxidant and anti-inflammatory therapies, such as vitamin E, coenzyme Q10, and selective cytokine inhibitors, may provide strategies to mitigate reproductive damage [15].

Long-term monitoring is essential to clarify risks associated with long-COVID and severe infection. Multicenter prospective studies with large cohorts are needed to determine the prevalence, persistence, and reversibility of COVID-19-related male infertility.

Conclusion

SARS-CoV-2 can affect the male reproductive system through direct viral invasion, inflammatory responses, and endocrine disruption. While many patients experience transient reductions in sperm quality and hormonal alterations, severe and prolonged COVID-19 may result in persistent testicular dysfunction. Clinicians should consider reproductive health as part of comprehensive post-COVID care. Early counseling, semen testing, and fertility preservation strategies may help mitigate long-term consequences for male reproductive potential.

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