Microbes and Infectious Diseases 2023; 4(2): 415-417

Microbes and Infectious Diseases

Journal homepage: https://mid.journals.ekb.eg/

Letter to the Editor

Viruses and male infertility: Where we are now?

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LETTER INFO

Letter history:
Received 3 November 2022
Received in revised form 14 November 2022
Accepted 19 November 2022

To the Editor

Infertility has the ability to affect around 15 percent of reproductive partners globally, infertility in men accounting for half of all infertile cases. Major etiological causes include infectious and various inflammatory disorders in the reproductive system [1].

Many viruses, can infect testicular cells, including human immunodeficiency virus (HIV) and the mumps virus, causing orchitis, which may cause infertility and testicular cancer in males [2]. Over 30 viruses have been shaded into sperm [3]. In the perspective well-known viruses such as mumps virus, HIV, zika virus (ZIKV), and hepatitis viruses are famous examples of viruses that spread via viremia that may penetrate the barrier of blood–testis, producing epididymitis, orchitis, and alterations in sperm number or their quality [3,4].

Studies have revealed the SARS-CoV-2 might be implanted in the male genital system in some situations. The fact that viremic people can shed viruses into their sperm, 27 viruses have been detected [3], including the ZIKV, which can be found in the sperm of symptom-free males for up to a year after recovery, supports this theory [5]. Men's sperm characteristics are affected by ZIKV infection, and replicative viruses may be identified in motile spermatozoa [6].

Hepatitis B or C, herpes simplex virus, mumps virus, coxsackie virus, papillomavirus, influenza, HIV, and SARSCoV-1 are just a few of the viruses that can cause infertility in men [6].

Viruses have been discovered to infect the testes directly. Men reproductive tract has the ability to provide immunosuppressive conditions due to the blood barrier in the testis, which may have a protective role from immune surveillance. Mumps viruses, have a strong affinity for testes, causing orchitis in about 20- 30% of cases that may cause testicular tissue destruction. The main causes of testis destruction in mumps are still being researched. However, the most widely accepted explanation is that testis degenerations are clearly induced by increasing in testicular temperature as a result of the inflammatory environment [7].

Xu et al. studied testicular autopsy samples from 6 people who died from SARS-CoV-1 disease. SARS-CoV-2 is very linked to SARSCoV-1 with about 85 percent similarity. All patients had orchitis. The researchers observed a WBC infiltration toward SARS-CoV-1 infected testis, which might impair Leydig cell function, destruction of testis barrier, and direct destruction of the seminiferous epithelium. In addition, no orchitis symptoms were seen or reported clinically in these people. SARS-CoV-2 appears to be targeting the male urogenital system in all these ways [7].

DOI: 10.21608/MID.2022.172710.1410
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Viral entrance techniques aren't confined to certain viral epitopes, according to studies. Non-specific mechanisms include viral load, immunological mediators affecting the testis barrier, testicular immunosuppression which shields some viruses from immune surveillance, and pyrexia [8-10]. Orchitis has been connected to a variety of viremia-causing viruses. When paired with orchitis, mumps orchitis is a very well viral infection that can induce testicular atrophy and infertility in males [11].

Any sort of flu as the Zika virus may induce a state of infertility [12]. Based on new global research, SARS-CoV-2 looks to be substantially more offensive than ordinary influenzas in aspects of severe disease, mortality, and morbidity. The common influenza viruses enhance oxidant-pathways in the testis that cause the activation of pathological conditions that are oxidative. Due to a reduction in spermatozoa motility and a contemporaneous rise in oxidative condition, enhanced oxidative has been associated with male infertility [13]. Sperm DNA fragmentation has risen [14]. SARS-CoV-2 demonstrated to fragment sperm DNA, which can reduce fertilization potential. SARSCoV-2 may also have an influence on oocyte performance and improve oxidative stress via mechanisms. Oxidative stress is linked to alterations in DNA methylation [15].

Conclusion

Previous research has shown that a wide range of different viruses have a preference for the male reproductive, notably the testes. Understanding pathophysiology can aid in determining how SARSCoV-2 acts, which may be similar to or somewhat changed from other viruses linked to male reproductive system disorders.

Conflict of interest: None.

Funding source: None.

Ethical approval: Not applicable.

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Al-Khikani FHO. Viruses and male infertility: Where we are now? Microbes Infect Dis 2023; 4(2): 415-417.