



# A Case of Asthenozoospermia Following COVID-19 Infection

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## Abstract

**Purpose** Studies pertaining to the effect of COVID-19 infection on male fertility are scarce. This case report describes a case of transient asthenozoospermia, absence of sperm motility, following a moderately severe COVID-19 infection.

**Case** A couple presenting for infertility treatment due to low ovarian reserve presented for their second intrauterine insemination (IUI). Their first IUI was performed 1 month earlier when the semen parameters were normal. A couple of weeks before the second IUI, the unvaccinated 48-year-old male partner contracted COVID-19 and was admitted to the hospital for several days. He received IV Remdesivir and continuous oxygen by nasal cannula. His hospitalization did not require intubation or intensive care unit admission. He was discharged after 12 days of hospitalization without home oxygen treatment. On the day of the second IUI, the semen analysis showed a normal sperm count with 0% motility. Three months following his COVID-19 diagnosis, a repeat semen analysis showed restored normal parameters with more than 40% motility.

**Conclusion** This aim of this report is to increase awareness that moderate COVID-19 requiring hospitalization could affect, though temporarily, sperm motility and should be considered in the differential diagnosis when male infertility is encountered.

**Keywords** COVID-19 · SARS-CoV-2 · Asthenozoospermia · Male infertility · Case report

## Introduction

The COVID-19 pandemic has been associated with several health consequences pertaining to many bodily organs from the lungs to the brain, to others [1, 2]. Even though it is well established that the COVID-19 vaccine is safe for reproduction [3], there have been a lot of theories pertaining to the fact that COVID-19 infection itself could impact male fertility [4]; however, data confirming this hypothesis are lacking.

To our knowledge, there are recent cross-sectional studies showing that COVID-19 infection is negatively correlated with sperm concentration [5, 6]. Gacci et al. [5] showed a correlation between sperm count and COVID-19, where

the severity of the infection was correlated with low sperm count. Guo et al. [6] found a lower sperm count, more abnormal morphology, and less motile sperm in patients with COVID-19 compared with controls. One of the pitfalls of those studies is that there were no baseline semen parameters on the participants before onset of disease.

Hence, we are reporting a case of a 48-year-old man, unvaccinated to COVID-19, who was known to have normal semen parameters then suffered from moderately severe COVID-19 disease that required hospitalization and led to asthenozoospermia (absence of motile sperm) that recovered 3 months following the onset of the disease.

## Case

A 48-year-old man and his 43-year-old female partner, who had low ovarian reserve, presented for secondary infertility and difficulty conceiving for 10 years. His past medical history was significant for hypertension, dyslipidemia, coronary artery disease, and obesity with a body mass index of 31.9 kg/m<sup>2</sup>. His past surgical history was significant for a knee arthroscopy and a cardiac catheterization with a stent placement. His home medications included Losartan, Clopidogrel,

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and Rosuvastatin. The couple has a 10-year-old child who was conceived with clomiphene citrate and timed intercourse.

Following the couple's counseling, the treatment plan was to perform a series of intrauterine inseminations (IUI) in order to attempt conception. The first IUI was performed on January 21, 2021, where the semen parameters were all normal (based on WHO 6th Edition criteria [7]) with a sperm count of 16.8 million/mL and 86% motility. The IUI was not successful, and the couple wanted to proceed with a second one. A couple of weeks before the second IUI, the male partner COVID-19 infection where he reported symptoms that included fever, chills, cough, and progressive shortness of breath. He was then admitted to the hospital for continuous oxygen by nasal cannula but did not require intubation or admission to the intensive care unit. He received IV Remdesivir during his inpatient stay (200 mg on day 1 then 100 mg daily for 10 days), and his symptoms improved after 3 days and was then discharged after 12 days of hospitalization without home oxygen treatment.

On the day of the IUI on March 3, 2021, his semen analysis showed sperm with normal concentration (> 15 million/mL) with 0% motility, in a total normal volume of 1.5 mL. The IUI was canceled, and he was advised to repeat the semen analysis 3 months following his COVID-19 diagnosis (April 1, 2021), which showed normally restored parameters with a concentration of 32.1 million/mL, 44% motility (progressive motility 72.5%, non-progressive motility 13.5%, immotile sperm 14%), 9% normal morphology, and normal volume (2.6 mL), viscosity and liquefaction.

## Discussion

Our patient exhibited a rare case of transient asthenozoospermia after a moderately severe course of COVID-19 infection. Having his semen analysis before and after his disease, with no other changes in his health, make it clear that COVID-19 was the direct cause of this phenomenon. To our knowledge, there were no other published cases of transient asthenozoospermia after COVID-19 that document a normal semen analysis before onset of disease.

The role of COVID-19 infection on patient sperm count and quality following recovery from the virus is still unclear. One recent study aimed to observe the semen quality of sexually active men status post recovery from COVID-19 [5]. The study included 43 men between the ages of 18 and 65 who had previously been affected by COVID-19 and had two negative PCR test results before sample collection. Semen analysis revealed that eight patients (18.6%) were azoospermic and three (7%) were oligospermic. Thus overall, 11 total patients (25%) from the patient population of patients who recovered from COVID-19 had some semen count pathology [5]. The severity of illness from COVID-19 was also recorded: 5 patients (11.6%) were admitted to the intensive care unit, 26

patients (60.5%) were hospitalized in the internal medicine unit, and 12 patients (27.9%) were not hospitalized. There data showed that azoospermia has a strong relation to the severity of COVID-19 illness: azoospermia was found in 3 of the 26 patients hospitalized in the internal medicine unit and 4 out of 5 patients admitted to the intensive care unit, while only one of the 12 patients not hospitalized during the span of their COVID-19 illness were found to have azoospermia ( $P < 0.001$ ) [5]. In another study by Guo et al. [6], the authors reported transient abnormalities, in particular a decrease in sperm motility, in semen parameters in 22 patients affected by COVID-19. Similar to Gacci et al. [5], they did not, however, have baseline semen parameters before the infection.

At the cellular level, SARS-CoV-2 (virus causing COVID-19 disease) is a single-stranded, non-segmented, positive sense RNA virus belonging to the *Coronaviridae* family. Mechanistically, SARS-CoV-2 has been shown to enter cells via binding to angiotensin-converting enzyme 2 (ACE2) receptor cells via its S glycoprotein [8]. Further, recent studies have shown that TMPRSS2, a host cell protease, plays an important role in COVID-19 pathogenicity, as it cleaves the viral S glycoprotein, facilitating viral activation [8]. Studies have shown that ACE2 can be found in all organs to some degree, including the testes, suggesting that COVID-19 could affect spermatogenesis.

There have been studies that analyzed ACE2 expression in the reproductive system. Results indicate that ACE2 is heavily found in Leydig cells, which function in producing testosterone, along with Sertoli cells and seminiferous ducts cells in the testis [9]. Further, studies have shown that spermatogonia and spermatids have a high concentration of TMPRSS2 [9]. Thus, given the expression of ACE2 in Leydig cells, Sertoli cells, seminiferous duct cells, and spermatogonia, and given the expression of TMPRSS2 in spermatogonia and spermatids, we hypothesize that SARS-Cov-2 could alter the function of the human testis, with potential for a negative impact on fertility. Indeed, studies have shown that there is a positive correlation between ACE2 expression in the testis and symptoms where the testicular tissue with high ACE2 expression was more likely to have reproductive dysfunction due to COVID-19 [10].

The results in this case presented here cannot be generalized; however, it might indicate a likely correlation between COVID-19 and asthenozoospermia, which needs to be further investigated. The strength in this case study is the fact that we had the semen parameters before and after COVID-19 disease, thus providing a clearer comparison and possible indicator of the reason for asthenozoospermia. Whether Remdesivir administration was the reason for the asthenozoospermia is unlikely due to the lack of evidence.

It is unknown whether the asthenozoospermia would be transient in all cases. There is also a lack of recommendation on how frequent a semen analysis should be performed to test for the recovery of the sperm. It would be worthwhile investigating this phenomenon to better counsel patients,

especially those going through infertility treatments. We do not know how long this abnormality might last and time is of the essence with infertility patients. One suggestion might be to freeze sperm across the board to avoid waiting for recovery and wasting time. Further studies are needed to confirm this.

## Conclusion

Severe COVID-19 has been shown to cause alteration in semen parameters which could be temporary. Whether this is due to the severe illness itself or due to the type of virus acting on the ACE2 receptors remains to be determined. With more variants of the virus emerging, it might be prudent, though unclear, for males who have borderline or low sperm counts, to freeze their sperm as a precautionary measure.

**Author Contributions** All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by Serin Seckin, Hadi Ramadan, and Zaher Merhi. The first draft of the manuscript was written by Serin Seckin, Hadi Ramadan, Zaher Merhi, Marco Mouanness, Melvin Thornton, and Ariel Gidon commented on previous versions of the manuscript. The authors read and approved the final manuscript.

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## Declarations

**Consent for Publication** Informed consent was obtained from all individual participants included in the study.

**Conflict of Interest** The authors have no relevant financial or non-financial interests to disclose.

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