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Letter

Covid-19 and impairment of spermatogenesis: What if fever was the only cause?

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Sir,—We would like to congratulate you for the original research recently published in EclinicalMedicine entitled 'Impaired spermatogenesis in COVID-19 patients' [1]. The authors found an increased concentration of CD3+ and CD68+ cells and apoptotic cells in the testicles/epidymes of men who died from Covid-19. In some patients cured of COVID-19, they noted oligozoospermia, leukocytospermia and increased seminal levels of IL-6 and MCP-1. All of these signs would be, for the authors, due to an autoimmune orchitis.

First, the presence of CD3+ and CD68+ cells is physiological in epididymides [2]; they play a role in physiological sperm phagocytosis. Fever is a symptom observed in more than 80% of patients infected with COVID-19. This fever, even of limited duration, can, on its own, induce oligozoospermia and the appearance of apoptotic cells [3]. Thus, COVID-19-induced fever can alter semen parameters even in the absence of testicular immune response. The return to basal state of semen parameters may take up to three months [4].

In addition, leukocytospermia (also called pyospermia) is defined by the WHO as the presence of $>1.10^6$ granulocytes (not lymphocytes) /ml. This leukocytospermia is traditionally accompanied by an increase in IL-6 and MCP-1 [5]. Leukocytospermia can be a sign of a bacterial or viral infection, systemic inflammation or simply an infrequent ejaculation. Leukocytospermia is not evidence of inflammation of the testicles.

In conclusion, the question of SARS-CoV-2 tropism for the testicles remains unresolved. Moreover, the ability of this novel coronavirus to induce an autoimmune orchitis is not proven to date.

Declaration of Interests

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Availability of data and material (data transparency)

Not applicable.

Authors' contributions

Both authors help to write this manuscript.

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