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REVIEW

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Relationship between COVID-19 infection and erectile dysfunction; a literature review examining the link and proposed mechanisms behind this phenomenon

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Abstract

It is now only in the wake of coronavirus disease 2019 (COVID-19) that we are beginning to understand many of the extra-respiratory manifestations of the condition. There is now growing evidence that erectile dysfunction (ED) is closely linked with the disease. We carry out one of the first literature reviews to consolidate the current evidence of the causal link between COVID-19 and ED and explore the proposed mechanisms that underpin this phenomenon. We carried out a literature search of the databases; PubMed (MEDLINE), Scopus, Web of Science and the Cochrane library. Search time frame was between December 2019 and March 2022. Only studies deemed of acceptable quality were included. Five studies were found highlighting the link between COVID-19 and ED. A further Nineteen studies were utilized to illustrate the proposed biological mechanisms underpinning COVID-19 related ED. Clear evidence has been documented through multiple studies internationally recognizing reduction in erectile scores and reduced sexual activity. It appears there is likely indirect and direct cytopathic effects on endothelial cells, in addition to hormonal and psychosocial factors. The associated ED is likely a result of a multitude of mechanisms including direct and indirect endothelial dysfunction, vasoactive cytokines, endocrine dysregulation, and psychosocial factors. This is the first literature review to delve into the likely underpinning mechanisms of the virus that drive ED.

Keywords

Erectile dysfunction; COVID-19; Virus

1. Introduction

The emergence of the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that first emerged in December 2019 has been responsible for over six million deaths world-wide. The condition was named coronavirus disease 2019 (COVID-19) by the World Health Organisation and only in the wake of its destructive path have we begun to identify a multitude of non-respiratory related sequalae [1]. These include an array or cardiac, neurological and haematological complications however, there is now growing evidence that COVID-19 has deleterious effects on male sexual function, namely erectile function [2]. Practitioners world-wide may see a growing influx of this presentation to their respective outpatient departments and therefore a comprehensive review of the evidence and mechanisms underpinning this phenomenon is required.

The American Urological Association (AUA) defines erectile dysfunction (ED) as the recurrent inability to attain and/or maintain an erection sufficiently to permit satisfactory sexual intercourse. ED is common, and not limited to elderly men with an overall prevalence of roughly 60% in men aged 45 and older [3]. Previously felt to be a purely psychogenic disorder, it is now well understood that 80% of cases involve an organic cause [4]. These are broadly divided into endocrine and non-endocrine aetiologies with vasculogenic dysfunction accounting for the majority of non-endocrine cases. COVID-19 may be able to affect both of these domains leading to ED in men who contract the disease.

In this literature review, we investigate the current evidence linking COVID-19 with ED and further clarify the mechanisms underpinning this phenomenon. An extensive literature review was carried out to answer the key questions; firstly, does COVID-19 cause erectile dysfunction in men and secondly, what are the biological mechanisms that likely underpin this?

2. Methods

A summary of methodology for this non-systematic literature review can be found in Table 1. We carried out a search of the databases PubMed (MEDLINE), Scopus, Web of Science and the Cochrane library. The search was carried out by two authors who collaborated the most relevant articles to be included in the review. Dates of search included all articles

TABLE 1. Summary of methods utilised by literature review.

Items	Specification
Date of Search (specified to date, month and year)	Dec 2019–Apr 2022
Databases and other sources searched	MEDLINE (through PubMed), Scopus, Web of Science
Search terms used (including MeSH and free text search terms and filters)	COVID-19, SARS-CoV-2, corona virus and Erectile dysfunction, impotence, sexual dysfunction
Timeframe	2 years, 4 months
Inclusion and exclusion criteria (study type, language restrictions *etc.*)	Any study type, excluding articles in language other than English
Selection process (who conducted the selection, whether it was conducted independently, how consensus was obtained, *etc.*)	GB conducted literature review and writing of paper. VG assisted with literature review and edits to the final study.

COVID-19: coronavirus disease 2019; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2.

published between December 2019 to March 2022.

2.1 Search strategy

Search terms utilised are found in Table 1. Any article type was included which analysed erectile dysfunction during or following COVID-19 infection. Articles published in a language other than English were excluded. Search strategy for databases included the following key words arranged in variable combinations; COVID-19 or SARS-CoV-2 or corona virus and erectile dysfunction or impotence or sexual dysfunction. Summary of study selection can be found in Fig. 1.

2.2 Quality assessment

We utilized the risk of bias in non-randomized studies-of interventions (ROBINS-I) checklist quality assessment tool for non-randomized studies. Studies were included if they had a risk of bias score of low or moderate. Studies with a mean risk of bias score of serious or critical were not included.

3. Results

Five studies were included highlighting the link between COVID-19 and ED (Table 2). A further Nineteen studies were utilised to illustrate the proposed biological mechanisms underpinning COVID-19 related ED. Only studies that adhered to ethical guidelines were included in this review.

3.1 COVID-19 linked with ED

A prospective case-control study carried out in Egypt screened healthy individuals who contracted COVID-19 for ED. Utilising the validated Arabic version of the international index of erectile function (IIEF-5), 197 participants were scored during Sars-CoV-2 infection. They found a significant reduction in erectile function scores between the infected and control groups (22.6 *vs.* 23.5, $p < 0.041$). Interestingly, they also found a significant reduction in testosterone levels between infected and control participants (3.9, 5.0, $p < 0.001$), concluding this could be an indirect mechanism for ED. Multiple logistic regression models revealed COVID-19 severity, baseline erectile function scores and smoking to be important predictive factors of ED for these patients. While this was a quality, adequately sized prospective study, there were several limitations. Firstly, the study only assessed participants for the preceding 4 weeks before joining the study for both erectile scores and serum testosterone levels. Therefore, the study did have incomplete data sets of each participants' sexual function before contracting COVID-19. It is also to note that a drop in one point of the IIEF-5 score although found to be statistically significant, does not likely translate to clinically significant ED [5].

The study by Duran *et al.* [6] (2021), retrospectively reviewed 12 centres in Turkey and identified ED amongst other andrological diagnosis rates to be significantly increase during the COVID-19 pandemic when compared to pre-pandemic rates. Rates of ED during COVID were found to approach 8.7% (n = 150) compared to pre-COVID rates or 6.6% (n = 214), $p = 0.008$. Limitations of the study include the non-randomized, retrospective design creating confounding variables. Due to the restrictions imposed during the pandemic, there were unequal numbers in patients in the pre-COVID-19 group and COVID-19 periods thus creating another limitation to the study. In reality, there is likely a range of biopsychosocial elements that play a role influencing these results. The concluded ED is likely also influenced by financial stressors, change in lifestyle (working at home) and overall anxiety related to the pandemic.

Harirugsakul *et al.* [7] (2021) helped highlight the link between COVID-19 and ED in this study based in Bangkok. They excluded participants suffering from severe mental health or medical conditions to assess erectile function (IIEF-5 score) in a total of 153 patients with COVID-19. They found amongst this COVID-19 cohort; ED prevalence was 64.7% which was much higher than the normal Thai population prevalence of 37.5–42.2%. Limitations include the cross-sectional methodology without a control group, making it difficult to explore causal relationships between ED and COVID-19. Also, these patients were comprised of inpatients which limits the generalizability of the results to the population with mild COVID-19. Recall bias is again noted to be a confounder with questionnaire studies [7].

Sansone *et al.* [8] (2021) carried out an Italian online survey for the effects of COVID-19 on sexually active males. Of the 100 patients included, the prevalence of ED was significantly higher in the COVID-19 positive group compared to those unaffected (28% *vs.* 9.33%; $p = 0.027$). Logistics regression

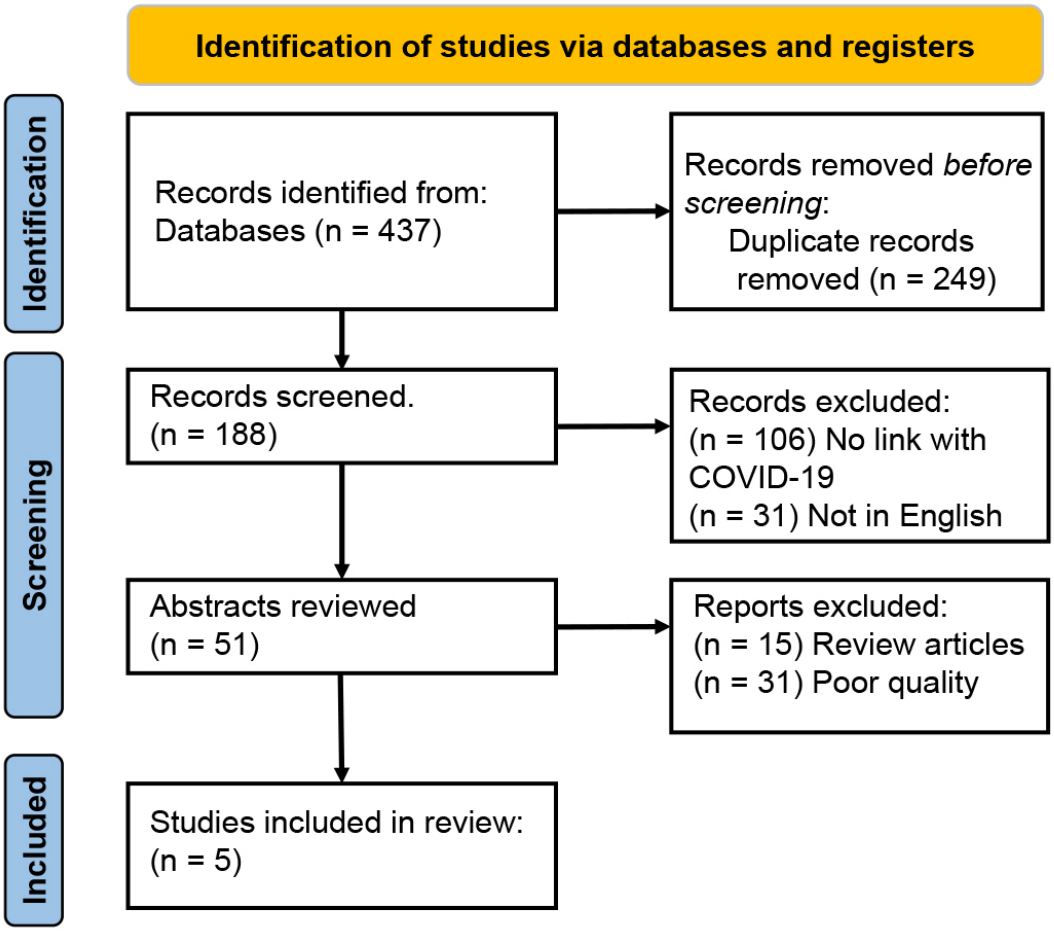

FIGURE 1. Flow diagram of studies included in review. COVID-19: coronavirus disease 2019.

TABLE 2. Summary of study characteristics and the effects of COVID-19 on ED.

Study	Study type	Sample size	COVID-19 effects on ED
Saad *et al.* [5] 2022	Prospective case control study	n = 197	IIEF-5 score reduced significantly in COVID-19 infected participants compared to control (22.6 *vs.* 23.5).
Duran *et al.* [6] 2021	Non-randomized retrospective study	n = 4955	Rates of ED during the COVID-19 pandemic were significantly higher than pre-COVID-19 era (8.7% *vs.* 6.6%).
Harirugsakul *et al.* [7] 2021	Cross-sectional survey study	n = 153	ED rates in patients with COVID-19 were significantly higher than general local population (64.7% *vs.* 37.5%).
Sansone *et al.* [8] 2021	Cross-sectional survey study	n = 100	A significant increase in ED was self-reported in patients with COVID-19 than those without (28% *vs.* 9.33%).
Karsiyakali *et al.* [9] 2021	Cross-sectional survey study	n = 1356	Significant decline in erectile function and sexual behaviours.

COVID-19: coronavirus disease 2019; ED: erectile dysfunction; IIEF-5: international index of erectile function.

models confirmed this effect to be independent from other variables such as age, body mass index (BMI), or psychological status. Strengths of this paper include the propensity score matching utilized to remove possible bias resulting from age and BMI. Removing other psychosocial confounders to these results highlights that COVID-19 infection may directly affect erectile function and be an independent risk factor for ED in males [8].

An evaluation of sexual functioning carried out in Turkey observed a reduction in sexual functioning during COVID-19. Over one thousand participants were included to complete an internet-based survey which revealed a statistically significant

drop in sexual intercourse as well as masturbation rates during this period. Limitations included not evaluating mental health statuses of participants at the time of survey such as anxiety or depression which may skew data. The nature of the study using self-reporting internet surveys also predisposes to recall bias, as well as not objectively confirming COVID-19 infection with polymerase chain reaction tests [9].

3.2 Proposed biological mechanisms underpinning COVID-19 related ED

3.2.1 Endothelial dysfunction

Endothelial function has clearly been described as having a crucial role in maintaining erectile function. The corpora cavernosa requires dynamic, responsive, and healthy vasculature for the erectile process. After initiation, nitric oxide, prostaglandin E1 and acetylcholine are released which signal the smooth muscles of the penile arterioles to relax. This vasodilatation increases blood flow to the corporal body sinusoids which fill with blood and compress the sub-tunical venous plexus between the tunica albuginea and the peripheral sinusoids, thus reducing venous outflow. The stretching of the tunica occludes emissary veins to further decrease venous outflow to a minimum to allow for corporal engorgement and the complete erection [10].

SARS-CoV-2 has extensive evidence for its deleterious effects on blood vessels. In the wake of the disease, scientists have now found that that the virus affects the vascular system as much as it does the respiratory system. Most well-known are the endothelial dysfunction that leads to deep vein thrombosis and subsequent life-threatening pulmonary emboli. The corporal bodies require patent and healthy blood vessels for the dynamic erectile process as described. It has been uncovered that endothelial cells in the corpus cavernosa contain the protein angiotensin-converting enzyme 2 (ACE2) receptor. This receptor is also found in the lungs and is key to the pathological mechanism of COVID-19 causing respiratory distress [11]. SARS-CoV-2 uses the ACE2 receptor to facilitate entry into these target endothelial cells. This is mediated by transmembrane serine protease 2 and cathepsin L which helps cleave the viral particle permitting engagement with the receptor. Studies examining vascular beds of respiratory tissue in COVID-19 patients reveals widespread endothelial activation and associated microangiopathy and thrombosis with angiogenesis. Comparing these findings with lung samples of influenza virus which are absent of these vascular changes confirms this process to be specifically a SARS-CoV-2 sequalae and not merely a viral effect [12, 13].

The endothelium possesses an extremely intricate and complex set of properties to sustain an immaculate homeostasis. Unlike other tissues, endothelium has the unique quality of maintaining blood in the liquid form and prevents the formation of clotting and coagulation cascades to sustain life. These endothelial cells are lined with heparin sulfate proteoglycans, thrombomodulin and prostacyclins promoting anticoagulant properties at the lumen wall. In conjunction with these factors, the endothelial cells also produce tissue-type plasminogen activator and uro-kinase plasminogen activator forming profibrinolytic properties to inhibit clot formation and platelet aggregation [14]. Other essential components required for erection are dynamic vasodilation for crucial corpus cavernosa filling. This is facilitated by nitric oxide, endothelial-derived hyperpolarizing factor and prostacyclin.

SARS-CoV-2 effectively counteracts many of these protective mechanisms. The virus acts directly on these endothelial cells by binding and entering triggering widespread cytopathic effects by reducing VE-Cadherin, degrading basement membranes resulting in endothelial sloughing and death. It also acts indirectly on the cells by triggering a pro-inflammatory cytokine storm which leads to excessive production of the ligands interleukin (IL)-1α, IL-6 and tumour necrosis factor (TNF)-α [15]. Tissue factor, thromboxane, cluster of differentiation (CD)40 ligand are all expressed in response to these stressors triggering pathological pro-coagulant pathways leading to microthrombi of the vessels [16]. There is evidence these microthrombi can infarct capillary beds and end arterioles leading to resistance in blood flow to the corpora. Much like the heart in pro-inflammatory, pro-coagulant conditions; blood supply to the end organ is restricted resulting in ED. The vasodilatory mechanisms are also affected as the inflammatory cytokines and cellular damage causes a shift in the arachidonic acid pathway, decreasing prostacyclin production and increasing production of thromboxane A2 [14]. The high circulating levels of thromboxane have strong vasoconstrictive properties which lead to insufficient filling of the corporal bodies to impede formation of a full erection.

These mechanisms which have been studied extensively in other areas of the body are likely how SARS-CoV-2 causes ED. This local effect on blood vessels however is likely not the sole organic mechanism through which the virus affects erectile function.

3.2.2 COVID-19 related Hypogonadism

Growing evidence supports an endocrine dysfunction model as a secondary causative agent for ED in COVID-19 infection. Studies have emerged showing an acquired hypogonadism is present in up to 90% in males admitted to hospital with COVID-19 [17]. The ACE2 receptor is demonstrated to be readily expressed on cells of the testes including spermatogonia, Sertoli cells and Leydig cells which are crucial in testosterone production [18–20]. The ACE2 gene is essential in steroidogenesis as ACE2 knockout mice studies exhibit spermatogenesis and hormonal dysregulation [20]. The proposed mechanism COVID-19 related hypogonadism results from destruction and damage of Leydig cells *via* direct and indirect viral mechanisms. Leydig cells are the major site for testosterone production. The release of luteinizing hormone (LH) from the pituitary gland stimulated G protein-coupled receptors causing increases in cyclic adenosine 3′, 5′-cyclic monophosphate (cAMP) formation which leads to cholesterol conversion to testosterone which releases the steroid hormone to circulate. Damage and destruction to these cells causes reduced synthesis of testosterone.

The effects of testosterone on erectile function are broad and likely act in a multitude of processes. Firstly, central effects of testosterone are important in initiating the erectile process. Several areas in the brain including the hypothalamus, medial preoptic area and most importantly the amygdala all express

androgen receptors. These areas facilitate sexual behaviours in presence of appropriate stimuli. This is demonstrated clinically in studies of patients with low testosterone correlating with significant reductions in sexual desire and libido [21]. Conversely, testosterone therapy can significantly improve ED [22]. In fact, self-defined unfaithful men were found to have significantly higher androgen levels than their faithful counterparts [23].

Low testosterone is also closely linked with metabolic syndrome as well. This syndrome constitutes a combination of known ED risk factors including dyslipidaemia, hypertension, hyperglycaemia and obesity. All these conditions are risk factors for developing ED. Lastly, although considered modest, testosterone also has a positive impact on the enzymatic steps necessary for the initiation of the erectile process. Testosterone is shown to have positive effects on nitric oxide which is essential for vasodilation of arterioles and filling of the corporal bodies [24]. Immunohistochemical analyses have revealed reduced nitric oxide expression in the corpora cavernosum of COVID-19 infected men compared to non-infected men [25]. Testosterone is also important for mediating inhibition of RhoA-Rho-kinase located in the corporal bodies which leads to vasodilatation of arterioles further facilitating erectile function. Low testosterone levels are associated with significantly raised levels of RhoA and Rho-kinase in the corpora cavernosa. These increased levels prevent the relaxation of penile smooth muscle and vasodilatation to initiate a complete erection [26].

3.2.3 Other factors

The influence of COVID-19 on erectile function should not solely be put down to its biological effects on the erectile process. As many are aware, the pandemic has led to a multitude of psychosocial factors that indirectly influence not only erectile function but sexual activities. In the wake of a natural disaster such as earthquake and flooding, there is well documented evidence that sexual function in males and females drops markedly [27, 28]. COVID-19 is no different as we see anxiety related to the pandemic and certain lifestyle and financial stressors plays a large role on sexual function. Isolating at home also changes the relationship dynamics where intimacy can be affected with the "over-exposure" to each other. Other factors to consider are deterioration in general health and wellbeing from the virus impacting cardiovascular health and thus erectile function. Furthermore, mental health deterioration also heavily contributes to the reduction in erectile function and sexual activities in the population.

Future research should create large scale prospective trials to assist with further correlating the link between COVID-19 and ED. Moreover, further studies utilising electron microscopy to characterize the SARS-CoV-2 replication cycle and pathological mechanisms within penile tissues would be beneficial in uncovering more about pathogenesis behind COVID-19 related ED.

4. Conclusions

There is now an abundance of growing data illustrating the link between COVID-19 and ED. There is a broad range of mechanisms involved. Local direct and indirect effects on endothelial cells, secondary hypogonadism and endocrine dysfunction as well as psychosocial factors all play an important role in the pathophysiology. These mechanisms underpinning ED in COVID-19 may serve as potential targets in the future for developing strategies to minimize its severity. The current literature lacks large scale prospective trials further examining the effects of COVID-19 on ED.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

GB—was involved with the conception, data collection and writing of the article. VG, JKu and JKi—were involved in editing and reformatting of article. AM—was involved in assistance and oversight of the article.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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