COVID-19, Immune System and Sex Hormones

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INTRODUCTION

The very rapid spread of COVID-19 and the inability of some people's immune systems to detect and destroy it may indicate that people with severe forms of the disease do not have opportunity to activate the immune system and to fight the virus. The extent of the disease and the rate of spread appear to be due to several factors. That is, the rate at which the virus spreads and propagates in the body is so fast and progressive that some people's immune systems are unable to cope with the virus, so they appear to have little clinical symptoms but die suddenly, this may be the reason why people with immunodeficiency (cancer patients, etc.) have a severe illness. On the other hand, vitamin D receptors have a positive role in activating the immune system, therefore, a sufficient amount of it acts as a control agent for the disease, for this reason, older people who are naturally deficient in vitamin D may be more likely to die from the virus. But the important question is for patients who are young and have no history of illness but suddenly die of exposure to the virus, what I think is important about these patients, the speed of the virus will kill the immune response time.

Mortality from COVID-19 was higher in several groups of patients:

Men [1]

Black communities [2]

Hispanic [3]

Older people (especially men) [4]

In young men, blacks, and Hispanics, a common factor can be found that is inversely related to the immune system. In all three groups, testosterone levels are higher than their counterparts, and it has been shown that testosterone has an adverse effect on the immune system, and that increasing testosterone levels reduces immune function. Testosterone is higher in men than women and women have stronger immune systems than men [5]. Testosterone levels in blacks are higher than in Hispanics and whites [6, 7]. In the elderly, in addition to vitamin D deficiency, signs of testosterone and related factors can be found. The role of androgen receptors may be even greater than that of testosterone. This means that testosterone levels decrease in the elderly, but androgen receptors do not decrease as the hormone decreases and probably provides a good breeding ground for the virus and its effect on the immune system.

Therefore, for prevention in this phase of the disease and given our limited information, it may be best to synthesize inflammation in healthy, young, non-viral individuals to effect this inflammation and immune activation as a vaccine and in people with high levels of testosterone or androgen receptors, the use of antagonists such as flutamide may be effective.

REFERENCES


