Common pathological mechanisms for cardiac complications following SARS-CoV-2 infection or COVID-19 vaccination

Dear Editor,

I read the article by Çekmen *et al*., which was an elegant elucidation of the comorbidities impact on coronavirus disease of 2019 (COVID-19) mortality.[1] They found that hypertension and coronary artery disease are common comorbid conditions among COVID-19 patients. Nonetheless, they did not offer any explanation for that. This issue should be looked at in more detail because current evidence suggests that COVID-19 infection and vaccines can trigger cardiovascular complications. According to existing literature, the rate of cardiac complications is higher with the COVID-19 infection than with the COVID-19 vaccine. Nonetheless, it is hypothesized that there is an overlap between the underlying pathophysiological mechanisms. This overlap may be a consequence of structural and biochemical similarities between the virus spike (S) protein and vaccine-derived S protein. The S protein is an important structural protein that mediates virus entry into host cells via interaction with angiotensin-converting enzyme 2 (ACE2). Concurrently, ACE2 is a key component of the renin–angiotensin system (RAS), which is critically involved in cardiovascular function.

COVID-19 pathophysiology is driven by different mechanisms, including the disruption of the RAS balance. RAS dysregulation may arise upon natural infection or vaccination.[2] This triggers a cascade of adverse events that may contribute to endothelial dysfunction, high blood pressure, and cardiovascular complications.

Another possible mechanism is molecular mimicry. Induction or development of cardiovascular diseases may be the outcome of high structural similarity between spike protein and human self-antigens, including α-myosin. Cross-reactivity between the S and host protein can trigger autoimmune disease and hence cause damage to the host.[3]

Both COVID-19 and COVID-19 vaccines may trigger a variety of thrombotic events, which can increase the risk of cardiovascular problems. Emerging evidence suggests that platelets play an essential role in thrombus formation. The interaction between the S protein and the ACE2 receptor on platelets increases platelet aggregation, activation, release of various proinflammatory mediators, and thrombosis.[4]

Hyperinflammation is another suggested mechanism for cardiovascular adverse effects in vaccinated and unvaccinated individuals. Some people could develop uncontrolled inflammation after COVID-19 infection or vaccination. Multisystem inflammatory syndrome (MIS) is a good example of how a dysregulated inflammatory response can become harmful. This serious and even deadly threatening complication affects multiple body systems, including the cardiovascular system, and is characterized by exaggerated innate and adaptive immune responses. Super antigenic properties of the S protein may provide new insight into the pathogenesis of MIS.

The massive T cell activation and cytokine release induced by superantigen may play a key role in the pathogenesis of MIS and cardiac failure.[5]

In addition to the above-mentioned mechanisms, there are many other extrinsic or intrinsic factors (including sex, age, the general health status of the person, and different vaccine manufacturing technologies) that can influence the individual response to vaccines or infection. Therefore, further investigations should be performed to further reveal fundamental underlying mechanisms.

Overall, COVID-19 infection or immunization can result in several cardiovascular events. So, understanding the pathophysiologic mechanism is crucial to be able to promote the prevention, diagnosis, and treatment of cardiovascular diseases.

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Conflicts of interest

There are no conflicts of interest.

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