

Causality Association Between COVID-19 Infection and Aortic Dissection

To the Editor,

The cardiovascular complications of coronavirus disease 2019 (COVID-19) disease are not hidden from anyone. These side effects were exacerbated when the role of angiotensin-converting enzyme-II (ACE-II) receptors as the gateway to the virus was confirmed. Recently, some case reports and case series studies have presented some evidences on acute aortic dissection as an adverse sequel of COVID-19 disease, and accordingly, COVID-19 has been mentioned as one of the serious and potential causes of this complication. In some cases described, even, the diagnosis of infectious disease was made at random and on the basis of evidence of general and mild manifestations of the disease even without evidence of previous cardiovascular involvement. In a case described by Fukuhara et al¹ a 52-year-old COVID-19 patient with a low-grade fever without cough or dyspnea was described with acute type A (DeBakey type I) aortic dissection in computed tomography (CT) angiography assessment with a primary intimal tear at the level of the sinotubular junction, and a markedly compressed true lumen in the descending thoracoabdominal aorta was detected. In another case described by Tabaghi and Akbarzadeh² a 47-year-old woman presented with a history of dyspnea, shakes, dry coughs, and bloody diarrhea from 2 weeks before admission but without a history of cardiovascular events. On the eighth day of admission, she developed chest pain, loss of consciousness, and bradycardia along with a dilated ascending aorta of about 50 mm in chest CT and with severe aortic insufficiency, dilated aortic root and ascending aorta, a double lumen with an intimal flap of the ascending aorta in trans-thoracic echocardiography suspecting acute type A aortic dissection. Shahzad et al³ also described their experience on a 35-year-old male with initial suspicion of Marfan syndrome and a definitive diagnosis of COVID-19. He denied any chest, back, or abdominal pain; however, CT pulmonary angiogram (CTPA) showed an aortic dissection involving the root of the aorta extending to the arch of the aorta and right common carotid artery. In a case presented by Akgul et al⁴ a 68-year-old woman was described with a pulseless, right femoral artery, systolic hypertension, and an aortic diastolic murmur in examination, in their further assessment by CT, a type A aortic dissection flap extending through the right common iliac artery was revealed. In other studies such as case series or short communications, with the report of experiences related to the occurrence of aortic dissection due to COVID-19 disease, the importance of paying more attention to this causal relationship was raised. As reported by Murana et al⁵ an upward trend of patients with type A acute aortic dissection in parallel to the outbreak of COVID-19 was described. Increasingly, they showed a dramatical increase in the time between the onset of symptoms and the hospital admission leading to an increase in aortic dissection-related death before arriving in the operating room. In a large, multicenter study by Fukuhara et al⁶ in the United States and China, 52 consecutive patients with type A aortic dissection during COVID-19 pandemic were described, leading to an overall prevalence of aortic

LETTER TO THE EDITOR

Mehmet Altuğ Tuncer ¹

Hakimeh Sadeghian ²

Mehrdad Sheikhvatan ^{3,4}

Mahtab Toulany ⁵

¹Department of Cardiovascular Surgery, Okan University Hospital, Istanbul, Turkey

²Department of Echocardiography, Shariati Hospital, Tehran University of Medical Science, Tehran, Iran

³Tehran Heart Center, Tehran University of Medical Science, Tehran, Iran

⁴Department of Cardiology, Heidelberg University Hospital, Heidelberg, Germany

⁵Tehran University of Medical Science, Tehran, Iran

Corresponding author:

Mehrdad Sheikhvatan

✉ med.msv@gmail.com

Cite this article as: Altuğ Tuncer M, Sadeghian H, Sheikhvatan M, Toulany M. Causality association between COVID-19 infection and aortic dissection. *Anatol J Cardiol* 2022;26(4):338-339.



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dissection in the background of COVID-19 as 5.7% and the time interval between COVID-19 infection and occurring dissection as preoperative to 6 weeks after surgery but with 33.3% postoperative death rate. Overall, the causal relationship between the COVID-19 disease and the occurrence of type A aortic dissection is quite expected, and in this regard, the poorer prognosis of treatment protocols is quite evident in these patients.⁷ Following the outbreak of COVID-19, efforts to discover the destructive effects of this infection on vascular components also intensified. In this regard, it has been well shown that the increasing likelihood of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-mediated endothelial injury potentially by entering through ACE-II receptors on vascular target cells and activating inflammatory cascade ultimately leads to endothelial defects.⁸ But how this infection directly predisposes people to aortic dissection is shrouded in obscurity and requires further clinical and histopathological evaluation. Various environmental factors can change the behaviors of genes that involve arterial dissection.⁹ In fact, some non-genomic factors (such as infections) can induce upregulation or downregulation of the genes and thus can trigger or inhibit the gene-related processes related to arterial dissection. Such infectious factors have been well identified in carotid or craniocervical artery dissections.¹⁰ Some evidence is available in the association between SARS-CoV-2 infection and aortic, cervical artery, carotid, vertebral, and even coronary artery dissection.^{11,12} It seems that SARS-CoV-2 can potentially induce pathophysiological processes related to dissection; however, these processes remain uncertain.

Acknowledgments: The authors thank all the colleagues and university officials for their sincere cooperation.

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