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Ischemic Stroke in Multisystem Inflammatory Syndrome in Children

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Letter to the Editor | The interesting article by Chang *et al*. describing two adolescent females (patient-1: 15-year-old; patient-2: 16-year-old) who developed acute ischemic stroke shortly after mild COVID-19 infection provides important insight into the neurological complications associated with multisystem inflammatory syndrome in children (MIS-C) [1]. Patient-1 presented with aphasia and right hemiparesis 10 days after COVID-19 onset, whereas patient-2 developed indifference and mutism 3 days after infection [1]. The authors attributed the cerebrovascular events to MIS-C and treated both patients with intravenous immunoglobulins and corticosteroids, while patient-2 additionally received anticoagulation because of an apical ventricular thrombus [1]. Although the report is clinically valuable, several issues require further clarification. A major concern is the unclear etiology of the ventricular thrombus in patient-2. Since the patient exhibited systolic dysfunction with a reduced ejection fraction (45%), thrombus formation may have resulted from impaired ventricular contractility and blood stasis. However, because the patient also developed deep venous thrombosis (DVT), paradoxical embolization through a patent foramen ovale (PFO) should also be considered [2]. Therefore, it would be important to know whether a PFO was excluded through transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), or cardiac magnetic resonance imaging (cMRI). Another possible explanation is COVID-19-associated myocarditis or endocarditis, both previously reported cardiac complications of SARS-CoV-2 infection that may predispose to intracardiac thrombus formation [3,4]. Additionally, ventricular thrombus formation has already been described in MIS-C patients and may represent a direct complication of the inflammatory syndrome itself [5]. MIS-C-associated vasculitis and aortitis with secondary thromboembolic events have also been reported [6]. Therefore, clarification is needed regarding whether aortitis, aortic thrombus, or aortogenic embolism were adequately excluded. Furthermore, the possibility that the supposed ventricular thrombus represented left ventricular hypertrabeculation/noncompaction (LVHT/LVNC) should also be considered, particularly if advanced cardiac imaging was limited [7]. Another important limitation is the lack of follow-up data concerning the ventricular thrombus in patient-2. The authors should specify whether the thrombus completely resolved after anticoagulation therapy and whether repeat TTE, TEE, or cMRI demonstrated normalization. It also remains unclear why the thrombus was not initially visualized on TTE despite being identified later, raising questions regarding imaging sensitivity and diagnostic timing. The mechanism underlying systolic dysfunction in patient-1 also remains unexplained. Myocarditis secondary to COVID-19 or MIS-C should be considered, especially because myocardial inflammation is increasingly recognized in pediatric COVID-19 patients [3,4]. Takotsubo syndrome (TTS), which has also been associated with SARS-CoV-2 infection, represents another differential diagnosis. Moreover, myocardial infarction due to coronary involvement or hypercoagulability should have been excluded in patient-2 as a potential contributor to reduced ventricular function. It would also be valuable to know whether ventricular thrombus was systematically excluded in patient-1. Overall, this interesting report raises several unanswered questions regarding the pathophysiology of stroke, systolic dysfunction, and ventricular thrombus formation in pediatric COVID-19 and MIS-C. Further clarification of the cardiac findings, advanced imaging results, and long-term follow-up would substantially strengthen the interpretation and clinical significance of the study.

Key Words SARS-CoV-2, COVID-19, ischemic stroke, multisystem inflammatory syndrome, neurological

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