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Reply to Letter to the Editor: "The Increase in Pediatric Postural Orthostatic Tachycardia Syndrome During the Pandemic May be due to Autonomic Neuropathy as a Complication of SARS-CoV-2 Infection"

LETTER TO THE EDITOR
REPLY

To the Editor,

We would like to thank the author(s)[1] for their interest in and valuable comments on our study entitled "Increased Diagnosis Rates and Clinical Characteristics of Pediatric Postural Orthostatic Tachycardia Syndrome During the COVID-19 Pandemic", published in *The Anatolian Journal of Cardiology*.[2] We greatly appreciate their contribution, which has allowed us to further clarify the quality, comprehensiveness, and limitations of our work.

1. Study design

It is true that retrospective studies have certain limitations such as missing data and recall bias. We explicitly addressed this limitation in the discussion section of our article. We applied the most objective methods possible during patient selection. By implementing well-defined inclusion and exclusion criteria, we ensured the most reliable data collection possible. Under the constraints of the pandemic, the retrospective design enabled the evaluation of a relatively large patient group over an extended time period.

2. Population representativeness

Our study included all consecutive patients presenting with syncope or orthostatic intolerance symptoms to a tertiary pediatric cardiology center. Therefore, while it may not fully represent the general pediatric population, it reflects real-world experience and referral patterns at a major reference center. Accordingly, the observed prevalence rates may be higher than expected, a point we explicitly acknowledged as a study limitation.

3. Small fiber neuropathy (SFN)

We agree that SFN may contribute to the pathophysiology of postural orthostatic tachycardia syndrome (POTS). However, advanced diagnostic tools such as skin biopsy, corneal confocal microscopy, or Sudoscan are not routinely available at our center. Therefore, SFN assessment could not be incorporated into our study. Nonetheless, in both the introduction and discussion sections, we emphasized that viral infections such as SARS-CoV-2 may trigger autoimmune and autonomic dysfunction mechanisms.

4. History of COVID-19 infection

During the pandemic, it was not possible to access patient SARS-CoV-2 PCR records due to the confidentiality policies of the Ministry of Health. Therefore, a direct assessment of prior infection in patients diagnosed with POTS could not be performed. However, in our discussion, we specifically addressed the potential role of viral infections and autoimmune mechanisms in the development of POTS. While knowledge of patients' PCR results would undoubtedly have provided more reliable data to associate our findings with the pandemic, this was not legally permissible within the regulations governing our institution.

Mustafa Mertkan Bilen
Gamze Vuran
Murat Muhtar Yılmazer
Timur Meşe
Cem Doğan

Department of Pediatric Cardiology, University of Health Sciences, Dr. Behçet Uz Pediatric Diseases and Surgery Training and Research Hospital, İzmir, Türkiye

Corresponding author:
Mustafa Mertkan Bilen
✉ bilen.uygar@gmail.com

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5. Evaluation of other autonomic disorders

As the primary aim of our study was to compare the frequency and clinical characteristics of POTS, it was not feasible to comprehensively evaluate all autonomic system disorders. Nevertheless, we acknowledge that future prospective studies should also investigate multisystemic autonomic involvement. We consider research into all potential contributing etiological factors highly valuable.

6. Assessment of anxiety

As correctly noted, due to the retrospective design, we could not employ standardized questionnaires or psychiatric evaluations. Anxiety-related findings were recorded based on patient and family reports. This limitation was explicitly mentioned in the discussion section of our study.

In conclusion, our study demonstrated a significant increase in pediatric POTS diagnoses during the pandemic. While lifestyle changes and psychosocial stress were among the primary contributing factors, we also believe the possible impact of viral infections should be considered. We once again thank the authors for their insightful contributions.

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