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PERIPHERAL NERVOUS SYSTEM LESSON IN CORONAVIRUS INFECTION COVID-19

Ya.N. Majidova,
Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan
N.M. Azimova,
Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan
M.K. Temirova,
Tashkent Pediatric Medical Institute, Tashkent, Uzbekistan

ABSTRACT: Infection caused by the SARS-CoV-2 (COVID-19) coronavirus, accompanied by various lesions of various organs and body systems, is associated with a certain level of mortality, especially in the presence of risk factors [1]. The interest of neurologists in this disease is due to the frequent defeat of various parts of the central and peripheral nervous system (PNS), reports of which appeared in the medical literature already in the first half of 2020.

KEYWORDS: Infection, coronavirus, PNS, tremor, myelitis, encephalopathy, anosmia, dysgeusia, Hyena-Barre cider.

INTRODUCTION

The literature review provides information on peripheral nervous system damage in COVID-19 coronavirus infection. One of the most severe forms of PNS lesion is Guillain—Barre syndrome (GBS), which creates a real threat to the patient's life and often leads to persistent neurological deficit. As a rule, GBS occurs against the background of various infectious diseases, including viral ones. An increased risk of developing GBS was noted in patients with various variants of coronavirus infection, in particular, MERS-CoV and currently, SARS-CoV-2 [2, 3]. When monitoring a cohort of 71,904 patients with COVID-19 hospitalized in 61 emergency departments in Barcelona (Spain), GBS was diagnosed in 11 of them [4]. The prevalence of GBS was significantly higher among patients with COVID-19 than among patients without COVID-19 (0.15% and 0.02%, OR 6.30, 95% CI 3.18–12.5), which after standardization amounted to 9.44 and 0.69 cases per 100,000 person-years, respectively (OR 13.5, 95% CI 9.87-18.4). Patients with GBS on the background of COVID-19 were more likely to have taste and smell disorders than patients

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with GBS developed on the background of another, non-sars-cov-2 infection (27.59 OR, 95% CI 1,296–587.0).

THE MAIN FINDINGS AND RESULTS

The authors noted that although patients with COVID-19 and GBS more often required treatment in intensive care units, the combination of these diseases was not accompanied by a significant increase in mortality. Analysis of available data on the association of ATYPICAL pneumonia-2 with the development of GBS allowed us to establish that the presence of coronavirus infection is associated with the development of one additional case of GBS for 63,762 cases of SARS-2 infection [5]. This is significantly less compared to the frequency of GBS, which develops against the background of other infectious agents, in particular Campylobacter jejuni (~1 case per 1000 cases) and Zika virus (~1 case per 4000 cases). According to a number of authors, the high frequency of GBS in patients with sars-CoV-2, noted in early studies (March-April 2020), was to a certain extent due to the use of broad and heterogeneous diagnostic criteria, as a result of which patients with GBS were included in the studies, the etiological affiliation of which was confirmed by various diagnostic tests (serological, polymerase chain reaction) or only clinically. Subsequently, when the diagnostic criteria became more stringent, the number of cases of GBS caused by SARS-2 decreased. According to the results of two systematic reviews, clinical manifestations of GBS in patients with COVID-19 occur on the 14th day (interquartile range of 7-20 days) and after 11.5 days (7.7-16.0 days) [5, 6]. It is likely that immunomodulatory therapy and glucocorticosteroids used to treat patients with COVID-19 can change the timing of the development of GBS, positively affecting its course and consequences [7]. It was also noted that patients with COVID-19 and GBS have relatively infrequent myalgia and radiculopathy (14.2%), which are registered in about 2/3 of patients with GBS without COVID-19 [6, 8]. Cases of isolated lesions and cranial nerves (oculomotor, abductor, etc.) in patients with COVID-19 have been described [9, 10]. As a rule, cranial neuropathy is combined with an impaired sense of smell and taste sensitivity. Due to the relatively low frequency of cranial nerve lesions, it is not possible to assess their diagnostic and prognostic significance. Of exceptional interest are cases of delayed development of neurological deficits in patients who have undergone COVID-19. Thus, the development of GBS is described in a 46-year-old patient who suffered a confirmed coronavirus infection with pneumonia, with full recovery, who on the 54th day from the appearance of the first symptoms of COVID-19 acutely developed tetraparesis, paresis of the respiratory muscles (artificial ventilation of the lungs was required),

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weakness of facial muscles and neuropathic pain syndrome [11]. The diagnosis was confirmed by the results of a study of cerebrospinal fluid (protein content 2.8 g / l), a decrease in the speed of the pulse along the peripheral motor nerves (according to electroneuromyography).

CONCLUSION

The specific nature of GBS in this patient was excluded according to the serological study, the authors did not provide information about the possible re-infection of Atypical pneumonia-2 and the content of antibodies. Full recovery was noted at the 4th week from the moment of the development of neurological deficit.

CONCLUSION

To date, there is no convincing evidence that all cases of delayed development of PNS lesions in patients who have undergone COVID-19 are caused by the ATYPICAL pneumonia-2 virus. Further observation of patients who have undergone COVID-19 will answer the question about the possibility and specificity of this kind of delayed lesion.

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