Guillain-Barre Syndrome due to COVID-19 in a child with acute lymphoblastic leukemia: a case report

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INTRODUCTION

Patients with COVID-19 have a growing number of reports of neurological manifestations of COVID-19. COVID-19 stimulates the inflammatory cells and produces a large number of inflammatory cytokines, and as a result, the immune response is initiated. Guillain–Barre Syndrome (GBS) is an immune- mediated, inflammatory polyradiculoneuropathy associated with especially viral infections. Recently, numerous case reports have been published describing the relationship between COVID-19 and GBS. GBS is rarely associated with acute lymphoblastic leukemia (ALL) in children. We report a child with acute ALL who developed GBS two weeks after an acute COVID-19 infection ,during chemotherapy. We discuss the differential diagnostic problems and possible pathogenic mechanisms of GBS in children with ALL.

CASE REPORT

A 7-year-old boy was evaluated for fever, jaundice, and vomiting. Bone marrow flow cytometry was diagnostic of T-cell ALL. He was started on induction chemotherapy with vincristine, daunorubicin and L-asparaginase. In the seventh week of chemotherapy, he developed a fever and cough and his polymerase chain reaction (PCR) test for the COVID-19 result was positive. Two weeks later, he developed rapidly progressive ascending areflexic weakness of the limbs. Standard nerve conduction studies (NCS) were suggestive of sensory-motor-demyelinating neuropathy involving lower extremities. With the clinical evidence of acute onset motor sensory quadriparesis and characteristic NCS findings, a diagnosis of GBS was considered. Albuminocytological dissociation in the cerebrospinal fluid (CSF) sample also supported the diagnosis of GBS. Intravenous immunoglobulin (IVIg) at a dose of 1g/kg/day for 2 days was administered and passive physiotherapy was given as supportive therapy. The Patient showed improvement in power in both upper and lower extremities on day 4 of IVIg therapy. He became ambulatory by day 10 of the onset of quadriparesis.

DISCUSSION

We report a patient who developed GBS after COVID-19 during treatment for ALL. The clinical and electrophysiological features of our patient were consistent with acute inflammatory demyelinating polyneuropathy (AIDP). There have been scattered reports with possible GBS and concurrent evidence of COVID-19 (1-2). GBS has also been reported in association with hematologic malignancies like non-Hodgkin lymphoma, chronic lymphocytic leukemia and ALL in adults. There are only a few reports of GBS in children with ALL (3-4). The most neurological signs are dizziness and headache reported in patients with COVID-19. Other signs are less common including meningoencephalitis and GBS (5). GBS represents the most common cause of acute flaccid paralysis. It is an acute-onset, inflammatory demyelinating polyradiculoneuropathy with a wide range of clinical manifestations. Diagnosis of GBS relies on the results of clinical, electrophysiological, and CSF examinations (6). Electrophysiological studies can indicate different subtypes, including AIDP, acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN)(7). Electrophysiologically demyelinating findings and good response to IVIG treatment in our patient strongly supported AIDP. Treatment with intravenous immunoglobulin (IVIg) or plasma exchange (PLEX) is the optimal management approach (6). Although ALL and intensive chemotherapy suppressed the immune system, the development of GBS in our patient was interesting. However, autoimmune diseases can be seen in ALL. The probable mechanism of acute immune neuropathy in ALL may be the depletion of regulatory lymphocytes that suppressed autoreactive lymphocytes. GBS secondary to Covid-19 infection should be distinguished from chemotherapy-induced neurotoxicity, paraneoplastic syndrome, and the neoplastic infiltration of the spinal nerve roots. Vincristine-induced neuropathy is an electrophysiologically sensory-motor axonal neuropathy and leads to severe weakness. In our patient, both clinical and electrophysiological findings were not compatible with vincristine-induced neuropathy. Another disorder that should be considered in the differential diagnosis is critical illness neuropathy. But we did not consider critical illness neuropathy in our patient due to no intensive care or mechanical ventilation admission were required. As a cause of the patient's neuropathy, neoplastic infiltration of the nerve roots was also excluded. First, the patient did not suffer from neuropathic pain. Secondly, nerve roots involvement wasn't seen on a gadolinium-enhanced spinal cord magnetic resonance imaging (MRI). And the CSF cytology and flow cytometry results were negative.A paraneoplastic peripheral neurological syndrome was also ruled out as a cause of the patient's neuropathy due to the demyelinating involvement. In addition, ALL does not tend to cause paraneoplastic neurological syndromes. In summary,the coexistence of three different causes in our patient was interesting.

REFERENCES

1. Alberti P, Beretta S, Piatti M, et al. Guillain-Barre syndrome related to COVID-19 infection. Neurol Neuroimmunol Neuroinflamm. 2020;7(4).
2. Toscano G, Palmerini F, Ravaglia S, et al. Guillain-Barre Syndrome Associated with SARS-CoV-2. N Engl J Med. 2020.
3. Aral YZ, Gursel T, Ozturk G, Serdaroglu A. GuillainBarre´ syndrome in a child with acute lymphoblastic leukemia. Pediatr Hematol Oncol. 2001;18:343-6.
4. Brigo F, Balter R, Marradi P, Ferlisi M, Zaccaron A, Fiaschi A, et al. Vincristine-related neuropathy versus acute inflammatory demyelinating polyradiculoneuropathy in children with acute lymphoblastic leukemia. J Child Neurol. 2012;27:867-74.
5. L. Mao, H. Jin, M. Wang, Y. Hu, S. Chen, Q. He, et al., Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China, JAMA Neurol 2020
6. Leonhard SE, Mandarakas MR, Gondim FAA et al (2019) Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol 15(11):671–683.
7. Dimachkie, M.M.; Barohn, R.J. Guillain-Barré syndrome and variants. Neurol. Clin. 2013, 31, 491–510.