

Case Series

Spectrum of Post-COVID-19 Complications in Pediatric Patients: A Case Series

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A B S T R A C T

The COVID-19 epidemic posed significant challenges to global health systems, highlighting the need for research into the virus's complex pathology and the development of targeted interventions. Despite existing management options, post-COVID complications in paediatric patients with underlying haematological and immunological vulnerabilities present a pressing concern. This case series presents four paediatric patients with complex post-COVID complications and underlying disorders, including chronic myeloid leukaemia with disseminated intravascular coagulation, Evans syndrome with coeliac disease, Guillain-Barré syndrome, and multisystem inflammatory syndrome in children (MIS-C). These cases underscore the importance of early recognition and multidisciplinary management in achieving optimal outcomes.

Keywords: CML, DIC, COVID-19, MIS-C.

Introduction

Coronavirus disease 2019 (COVID-19) is a communicable disease caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Despite the fact that the global COVID-19 pandemic has officially ended, we continue to feel its effects. It has been shown that the disease can be associated with a variety of complications in various disorders. The dysregulated immune pathways in severe COVID-19 shape the disease course and reflect the functional diversity of the paediatric immune system and the role of various treatment modalities.¹ We report a case series of four children presented to our tertiary care centre in North India with various underlying diseases and varied covid-related complications. First we present a case of chronic myeloid leukaemia (CML) complicated by

disseminated intravascular coagulation (DIC); second, a case of Evans syndrome later diagnosed with coeliac disease and immune thrombocytopenia; third, a case presenting with Guillain-Barré syndrome (GBS); and fourth, a case with necrotising pneumonia and post-COVID multisystem inflammatory syndrome (MIS-C) with an underlying disorder under evaluation associated with clubbing.

Case - I

An 8-year-old boy was presented in July 2023 with complaints of abdominal distention and intermittent fever for 1 month with a recent escalation of fever to continuous high-grade for a week. He also had multiple bluish swellings on the left arm, face, and thigh for 2 weeks. There was no past history of any bleeding or blood transfusion, no history of trauma, chronic fever, jaundice, rashes, intake

of alternative medicine or family history of bleeding. On examination, he was sick-looking, in respiratory distress with pain and tenderness at the sites of bleeding. He required oxygen with continuous positive airway pressure support, which was gradually weaned off. He had massive hepatosplenomegaly, with a liver span of 16 cm and spleen size of 15 cm, along with multiple ecchymoses and swellings on the left arm, face, and thigh. The swelling in the left thigh was soft and fluctuant. Initial investigations revealed a haemoglobin level of 6.0 g/dL, a white blood cell count of 570,000/mm³ and platelets of 480,000/mm³ (Table 1). The peripheral smear showed features of hyperleukocytosis with 5% myeloblasts, 8% promyelocytes, 25% myelocytes, 8% metamyelocytes, 6% bands, 20% neutrophils, 4% lymphocytes, 1% monocytes, 15% eosinophils, and 8% basophils.

The Ultrasound of the thigh lesion showed altered echogenicity suggestive of an abscess. However, only unclothed blood was noted in the aspirate from this lesion, leading to the diagnosis of haematoma and underlying coagulopathy likely due to disseminated intravascular coagulation (DIC) based on the clinical presentation. Although the screening coagulation profile, including prothrombin time (PT) and activated partial thromboplastin time (APTT), was within normal limits. However, the D-dimer levels were elevated (Table 1). COVID neutralising antibodies were positive (2517 AU/mL). Besides DIC, the differential diagnosis was disease progression to blast crisis. The peripheral blood was sent for flow cytometry. The report showed 1% CD45 dim blasts, ruling out accelerated phase or blast crisis. Another differential diagnosis was acquired coagulopathy due to malignancy. But his coagulation profile was normal even before administering FFP. Factor XIII deficiency is associated with severe bleeding and normal PT and APTT, as in our case. Subsequent workup, performed after FFP replacement due to logistic issues, showed that Factor XIII activity was normal. The next differential was malignancy-associated platelet dysfunction as a paraneoplastic syndrome. However, the patient presented with multiple subcutaneous and intramuscular haematomas rather than petechiae. Fluorescent in situ hybridisation (FISH) studies done from peripheral blood confirmed the presence of the Philadelphia chromosome (Fig. 3). Additionally, reverse transcriptase polymerase chain reaction (RT-PCR) detected the p210 transcript of the BCR-ABL1 gene (Fig. 4), further confirming the diagnosis. He was started on imatinib based on clinical and peripheral smear findings in view of his critical condition. He required multiple FFP transfusions over one week, besides starting

imatinib on an emergency basis, after which there was noticeable improvement in the haematomas. After a month of inpatient care, he was discharged in stable condition. Since discharge, he has been on regular follow-up with serial quantitative RT-PCR for BCR-ABL1 transcript monitoring to assess treatment response and disease progression. At the three-month follow-up, quantitative RT-PCR for BCR-ABL1 showed transcript levels below 10%, indicating a good early molecular response (Fig. 4). However, at 1.5 years of treatment, his quantitative RT-PCR BCR-ABL1 titers started rising despite adequate dose and compliance with imatinib. Imatinib resistance mutation analysis (IRMA) was negative. The patient was switched to dasatinib. Repeat analysis at three months of dasatinib is pending. Currently, the patient has completed two years of treatment. The spleen is no longer palpable.

Case-2

A 5-year-old female child presented in August 2022 with symptoms of loose stools, fever and swelling of feet for one week. She had also developed pallor, shortness of breath, and fatigue for 5 days. The patient was born to a non-consanguineous marriage and had no history of bleeding or blood transfusions in the past. There was no family history of bleeding or haematological disorders. Her immunisation status was up-to-date and development appropriate. On examination, the patient was underweight, had severe pallor and had signs of congestive cardiac failure.

The patient received packed red blood cells and platelet transfusions. She had persistent fever and thrombocytopenia, leading to a suspicion of MIS-C. COVID-neutralising antibodies were also positive (371 AU/ml). She received a course of intravenous immunoglobulin, and her fever improved. Based on the clinical picture and investigations, a diagnosis of Post-COVID Evans Syndrome (ES) was made initially. ES is a rare condition characterised by immune thrombocytopenia (ITP) and autoimmune haemolytic anaemia (AIHA). However, the patient had persistent thrombocytopenia on follow-up visits. The workup done for secondary ITP is shown in Table 2.

The final diagnosis of the patient was post-COVID ES, coeliac disease and secondary ITP. The patient was started on a strict gluten-restricted diet, and her parents were counselled on alternative dietary options. With dietary management, her haemoglobin and platelet counts gradually improved over the next three months. The patient showed significant clinical improvement, and laboratory results remained normal on follow-up.

Table 1. The laboratory investigations of case 1

Hemoglobin(g/dl)	6
Platelet/mm ³	480,000
WBC count/mm ³	570,000

Peripheral smear	5% myeloblasts, 8% promyelocytes, 25% myelocytes, 8% metamyelocytes, 6% bands, 20% neutrophils, 4% lymphocytes, 1% monocytes, 15% eosinophils, and 8% basophils consistent with CML..
prothrombin time and activated partial thromboplastin time	Normal
D Dimer	2.41 mcg/ ml (<0.5mcg/ ml)
COVID neutralising antibodies (AU/mL)	2517
Flow cytometry	1% CD45dim blasts, ruling out accelerated phase or blast crisis
Fluorescent in situ hybridization (FISH)	Philadelphia chromosome present. RTPCR BCR ABL1 confirmed p210 transcript.

Table 2.The Laboratory Investigations of case 2

Hemoglobin(g/dl)	3.8
Platelet/mm ³	24,000
WBC count/mm ³	9,200
Peripheral smear	Microcytic, hypochromic RBCs, No malarial parasites seen.
LDH (U/dL)	655
Direct Coombs test (DCT)	Negative
Reticulocyte count(%)	8.6
HIV, HBsAg, Anti HCV	Non- Reactive
Antinuclear Antibody(ANA)	Weakly positive
Prothrombin time and activated partial thromboplastin time	Normal
Bone marrow examination	Erythroid hyperplasia with megakaryocytic proliferation
Tissue transglutaminase level	297 U/mL
Endoscopy and duodenal biopsy	Suggestive of celiac disease
Thyroid profile	Normal

Case-3

An 8-year-old female patient presented in December 2023 with a one-day history of tingling sensation and numbness in her lower limbs, followed by weakness in her lower limbs for four hours. She had a history of fever for one day a week prior to presentation. On physical examination she had tachypnea. However. There were no signs of pneumonia or congestive cardiac failure. Neurological examination showed decreased tone in all limbs, absent deep tendon reflexes and decreased power in both upper limbs (3/5) and lower limbs (2/5), with intact sensory examination. Given the clinical suspicion of GBS, treatment was initiated with intravenous immunoglobulin (IVIG). Table-3 shows laboratory investigations. The patient was intubated due to worsening respiratory distress and impending respiratory failure. The diagnosis of post-COVID GBS was confirmed by

positive COVID neutralising antibodies (4000 AU/mL) and nerve conduction studies revealing acute motor axonal neuropathy. Autonomic dysfunction manifested on day 5 in the form of hypertension, for which initially amlodipine and later clonidine were added. Due to the lack of significant improvement after two doses of IVIG, rituximab was administered on day 24. On day 42, the first session of plasmapheresis was initiated due to slight improvement following rituximab and two cycles of IVIG, with a total of seven cycles given. The patient was gradually weaned off the respiratory support. The patient was successfully weaned off oxygen support by day 76. At discharge (day 114), the patient demonstrated significant improvement, with the ability to sit independently, although she was unable to bear weight. The patient was discharged with an ankle-foot orthosis and continues to follow up in the paediatric neurology clinic and has had no relapses.

CASE -4

A 4-year-old male child was referred in April 2023 with bilateral ear discharge and intermittent cough for a year that had worsened for the last month and shortness of breath for 2-3 days prior to presentation. On physical examination he had tachypnea and tachycardia with hypoxia. The oxygen saturation was 90% on room air. He had clubbing, probably due to chronic hypoxaemia and central cyanosis. Chest examination revealed bilateral crepitations with intercostal and subcostal retractions and hepatosplenomegaly.

The chest X-ray showed right-sided bronchopneumonia with pulmonary oedema and cardiomegaly (cardiothoracic ratio 0.6). The child was put on oxygen by high-flow nasal cannula and managed with injection of hydrocortisone, nebulisation with bronchodilators and injection of ceftriaxone and vancomycin. Dobutamine, furosemide and enalapril were also added along with blood transfusion in view of possible congestive cardiac failure secondary to low haemoglobin (5.5 g/dl). On day 4 of admission, the fever worsened, and facial congestion, palmar erythema, and a maculopapular rash appeared.

Echocardiography suggested mild left atrial and ventricular dilatation and LVEF of 50-55%. CECT chest indicated left lobe consolidation along with evidence of pulmonary arterial hypertension. On the 20th day of admission, again the fever worsened, palmar erythema and a maculopapular rash appeared all over the body, and an eye examination suggested bilateral conjunctivitis. COVID antibody test results were 11,000 AU/mL, LDH was 2263 U/dL, and ferritin was 327 ng/mL. The tropical infections were ruled out. The clinical picture was suggestive of post-COVID MIS-C. The child was managed with IVIG and methylprednisolone pulse therapy. Inotropes were added in view of shock. Antibiotics were further upgraded, and amphotericin B was started. The child was subsequently required for mechanical ventilation and respiratory failure. Unfortunately, the child did not respond to therapy and succumbed to the illness in May 2023. Although the CECT chest didn't show any evidence of bronchiectasis and the echocardiogram didn't show any underlying cyanotic heart disease, the workup for the underlying disorder for clubbing was planned after recovering from the PICU.

Table 3. The laboratory investigations of case 3

Date	31/12/23	8/1/24	17/1/24	23/1/24	7/2/24	12/2/24	20/2/24	5/3/25	12/3/24	9/4/24	13/4/24
Hb (g/dl)	11.3	9.4	15.8	-	8.7	11.8	-	-	-	-	12.7
TLC /mm ³	9400	19800	11600	-	9400	12500	-	-	-	-	7600
DLC(%)	N(87%) L(13%)	-	-	-	-	-	-	-	-	-	-
Plate let /mm ³	1.26	2.57	-	-	2.39	2.84	-	-	-	-	2.9
Urea/creat(mg/dl)	16/0.4	22/0.7	20/0.3	21/0.4	16/0.5	-	-	-	-	-	12/0.5
Na / k (meq/l)	134/3.8	130/3.8	141/4.9	-	-	-	-	-	-	-	134/4.4
SGOT/SGPT (U/L)	33/34	31/29		-	-	-	-	-	-	-	-
Urine r/m & culture	NAD Sterile	NAD Sterile	NAD Sterile	NAD Sterile	E.coli	Klebsillela	-	-	-	-	NAD Sterile
Blood culture & sensitvity	-	Sterile	Sterile	-	-	-	-	-	-	-	-
CRP (mg/L)	-	-	-	-	-	-	-	47	98	48	24
Procalcitonin (ng/mL)	-	0.695	-	-	-	-	0.52	0.274	0.695	-	-
Viral markers (HIV,HbsAg,Anti-HCV)		-	NR	-	-	-	-	-	-	-	-

CSF (cyto,bio,culture)	No cells , protein-61.4,Sugar-71mg/dl,Sterile		-	-	-	-	-	-	-	-
Anti MOG	-	-	Neg	-	-	-	-	-	-	-
MRI brain & spine	-	Normal	-	-	-	-	-	-	-	-

Table 4.Laboratory investigations of case 4

Date	25/4/23	2/5/23	8/5/23	12/5/23	13/5/23	17/5/23
Hb (g/dl)	10.8	-	-	-	5.5	10.5
TLC /mm ³	5900	-	-	-	1.2	12800
DLC(%)	N47/L43	N48/L42	-	-	N27/L67	-
Platelet /mm ³	2.48	-	-	-	30000	4.26
Urea/creat/uric acid (mg/dl)	17/0.9/6.6	40/0.9/10.4	32/0.9/5.3	-	29/1.1/5.3	63/0.9/7.9
Na / K(meq/l)	139/4.3	-	-	-	-	133/4.9
SGOT/SGPT (U/L)	77/29	-	-	-	-	70/24
CPK MB (IU/L)	25	-	-	-	-	-
Urine r/m& culture	NAD	-	-	-	-	NAD
Blood culture & sensitivity	-	-	sterile	Candida albicans	sterile	CoNS
CRP/ Procalcitonin (mg/L)/(ng/mL)	-	Negative	12/0.83	-	6/0.4	-
LDH (U/dL)	540	-	2263	1445	-	-
Covid antibody(AU/mL)	-	-	-	-	11000	-
2D echocardiography	-	Mild dilatation of left atrium & left ventricle, EF-50-55%	-	-	-	-

Discussion

The challenges that we face in maintaining health are malnutrition, the escalation of antimicrobial resistance, outbreaks of infections and pandemics. The Covid-19 pandemic had devastating effects on our health system. While there are options available for management, such as monoclonal antibodies, antivirals, and novel immune therapies,

continued research in this field is essential to understand the complexities of COVID-19 and develop targeted interventions to alleviate the symptoms.¹ This case series highlights the diverse and complex manifestations of COVID-19 in children, particularly in the context of underlying haematological and chronic disorders. We report four cases that demonstrate the varied presentations and outcomes of post-COVID complications in paediatric patients.

The first case, a newly diagnosed case of CML with DIC, illustrates the challenges in managing a rare but life-threatening condition. Haemorrhage in chronic phase chronic myeloid leukaemia (CML-CP) is an uncommon occurrence, as platelet counts and coagulation parameters are typically preserved, allowing for adequate haemostasis. Clinically, bleeding is more often considered a marker of disease progression rather than an initial presentation. Additionally, initiation of treatment with tyrosine kinase inhibitors and interferon-gamma therapy has been associated with platelet dysfunction and bleeding.^{2,3} In the present case, the child exhibited significant bleeding even before the initiation of any treatment, despite having normal routine coagulation parameters. Factor XIII deficiency, characterised by normal PT, INR, and APTT, typically presents with severe, prolonged deep-tissue bleeding,⁴ but Factor XIII activity was normal even though the patient continued to have bleeding. Generally, superficial and mucosal bleeding are indicative of platelet disorders, while deep-seated bleeding involving muscles, joints or internal organs is more characteristic of coagulation defects. In our patient he had deep-seated bleeding suggesting coagulation abnormality. An extensive review of the literature revealed that the development of DIC in a paediatric cancer patient with COVID-19 is a severe, life-threatening condition associated with poor prognosis and high mortality.⁵

SARS-CoV-2 activates the immune system with an excessive release of pro-inflammatory cytokines and increased activation of the coagulation system, leading to excessive activation of the fibrinolytic system,⁶ creating an imbalance in the coagulation and fibrinolytic systems. Also, it infects platelets and epithelial cells of blood vessels, further contributing to the activation of fibrinolysis.⁷ During hyperfibrinolysis, the activating factors of the fibrinolysis system (tPA, plasminogen) increase, while the inhibitory factors (PAI-1, TAFI, α 2AP) decrease, leading to an increase in bleeding complications.⁸ The result is increase D-dimer and decreased fibrinogen.

The second case highlights the rare association between Covid-19 infection and ES and coeliac disease in a paediatric patient. Coeliac disease is an autoimmune disorder triggered by gluten and environmental factors, leading to small intestine inflammation and villous atrophy. It can manifest with classic symptoms like diarrhoea and abdominal pain or non-classic symptoms such as iron deficiency anaemia and osteoporosis. The gold standard test for diagnosis is duodenal biopsy, but serological tests like anti-tissue transglutaminase antibodies (TTGA) are highly sensitive and specific.⁹ In our case, coeliac disease was diagnosed based on elevated TTGA levels and duodenal biopsy findings. Notably, coeliac disease has been associated with various autoimmune disorders.¹⁰ Miller's study reported two adult

cases of coeliac disease and AIHA; one also had psoriasis and the other hypothyroidism.¹¹ Another case report by Uddin et al. reported a case of coeliac disease and ES.¹² Our patient developed ES, likely triggered by COVID-19, as evidenced by positive COVID-19 antibodies, a negative systemic lupus erythematosus workup, and non-reactive HIV status. The patient responded well to a strict gluten-restricted diet, with significant clinical improvement and normalisation of platelets on follow-up on a gluten-free diet.

The third case demonstrates the severe neurological complications that can arise in the context of COVID-19, as seen in GBS. The patient's presentation with rapid progression to respiratory failure necessitating mechanical ventilation highlights the need for early recognition and aggressive management of GBS. The typical GBS presents with distal paraesthesia, rapidly progressive ascending bilateral weakness of the extremities, and diminished or abolished tendon reflexes. GBS is frequently associated with dysautonomia and pain. *Campylobacter jejuni* is the primary pathogen associated with GBS. Others are *M. pneumoniae*, *influenza* and *Zika* viruses. The estimated global incidence of GBS is between 0.8 and 1.9 cases per 100,000 inhabitants/year.¹³ The first GBS case after COVID-19 was reported in January 2020.¹⁴ Since then, there are several published reports. Palaiodimou et al. analysed 18 observational studies, with a total of 136,746 COVID-19 patients, estimating an incidence of 15 GBS cases per 100,000 SARS-CoV-2 infections.¹⁵ Comparing this relative incidence with other viruses, GBS related to SARS-CoV-2 infection was higher than influenza (7.3 cases per 100,000 infected).¹⁶ and lower than Zika virus (20 cases per 100,000 infected).¹⁷ COVID-19 patients had an increased risk of demyelinating GBS, the most common form, and the clinical outcomes were comparable to historical GBS controls. In our case, the patient presented with an axonal form of GBS, highlighting the variability in clinical manifestations. Despite the complexity and severity of the case, the patient showed significant improvement, emphasising the importance of prolonged and intensive care in achieving a favourable outcome. The available literature shows that the symptoms of GBS are more severe in patients who have previously suffered from COVID-19.^{18,19} Studies have shown that GBS is a rare but serious complication of COVID-19, and early recognition and treatment are critical to preventing long-term sequelae.

The fourth case illustrates the potentially fatal nature of MIS-C, a rare but severe complication of COVID-19 in children.²⁰ The patient's presentation with fever, rash, and cardiac involvement, followed by rapid deterioration, underscores the need for early recognition and aggressive management of MIS-C.²¹ Despite intensive care and multimodal therapy, the patient succumbed to the illness, as seen in the present case, highlighting the importance

of continued research into the pathophysiology and management of MIS-C. In our case, the diagnosis of MIS-C was established based on the World Health Organization criteria, which include age of presentation <19 years, fever and elevated inflammatory markers persisting for more than 3 days, presence of palmar erythema and maculopapular rash, accompanied by bilateral non-purulent conjunctivitis, history of shock/myocardial infarction and exclusion of other microbial causes of inflammation. This case highlights the importance of recognising and promptly managing MIS-C, a serious condition that can have severe consequences if left untreated.

In conclusion, these cases demonstrate the varied and complex manifestations of COVID-19 in children, particularly in the context of underlying haematological and immunological disorders. They emphasise the need for a high index of suspicion, early recognition, and aggressive management to achieve optimal outcomes. Further research is necessary to understand the long-term implications of COVID-19 in children and to develop effective strategies for prevention and treatment of post-COVID complications. By combining clinical experience with research findings, we can improve our understanding of COVID-19 in children and develop better approaches to managing this complex disease.

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References

1. Rotulo GA, Palma P. Understanding COVID-19 in children: immune determinants and post-infection conditions. *Pediatric research*. 2023 Aug;94(2):434-42. [Google Scholar]
2. Kostos L, Burbury K, Srivastava G, Prince HM. Gastrointestinal bleeding in a chronic myeloid leukaemia patient precipitated by dasatinib-induced platelet dysfunction: case report. *Platelets*. 2015 Nov 17;26(8):809-11. [Google Scholar]
3. Zheng WL, Zhang GS, Shen JK, Pei MF, Peng HL, Xu M. Acquired factor VIII inhibitor as presentation of chronic myelogenous leukemia during interferon-alpha therapy. *Thrombosis research*. 2011 Aug 1;128(2):202-3. [Google Scholar]
4. Kruse-Jarres R, Singleton TC, Leissinger CA. Identification and basic management of bleeding disorders in adults. *The Journal of the American Board of Family Medicine*. 2014 Jul;27(4):549-64. [Google Scholar]
5. Soehalino S, Setyoningrum RA, Cahyadi A. Coagulopathy and clinical outcomes of pediatric cancer patients with COVID-19 infection in Surabaya. *Journal of Medicinal and Pharmaceutical Chemistry Research*. 2025 Jun;7(6):1119-29. [Google Scholar]
6. Abudouleh EA, Alhamlan F, Al-Qahtani AA, Bohol MF, Al Hazzani A, Khorfan K, Alkaff M, Owaidah T, Al-Qahtani AA. Changes in the fibrinolytic system of patients infected with severe acute respiratory syndrome coronavirus 2. *Journal of Clinical Medicine*. 2023 Aug 10;12(16):5223. [Google Scholar]
7. Ahmad F, Kannan M, Ansari AW. Role of SARS-CoV-2-induced cytokines and growth factors in coagulopathy and thromboembolism. *Cytokine & Growth Factor Reviews*. 2022 Feb 1;63:58-68. [Google Scholar]
8. Saes JL, Schols SE, Van Heerde WL, Nijziel MR. Hemorrhagic disorders of fibrinolysis: a clinical review. *Journal of Thrombosis and Haemostasis*. 2018 Aug;16(8):1498-509. [Google Scholar]
9. Tarar ZI, Zafar MU, Farooq U, Basar O, Tahan V, Daglilar E. The progression of celiac disease, diagnostic modalities, and treatment options. *Journal of investigative medicine high impact case reports*. 2021 Oct;9:23247096211053702. [Google Scholar]
10. Cooper BT, Holmes GK, Cooke WT. Coeliac disease and immunological disorders. *British medical journal*. 1978 Mar 4;1(6112):537-9. [Google Scholar]
11. Butt N. A case of polyautoimmunity: celiac hepatitis, Grave's disease and autoimmune hemolytic anemia. *Journal of the College of Physicians and Surgeons Pakistan*. 2019 Jan 1. [Google Scholar]
12. Uddin SM, Haq A, Haq Z, Yaqoob U. Case Report: Rare comorbidity of celiac disease and Evans syndrome: The rare correlation of Celiac and Evans syndrome. *F1000Research*. 2019 Feb 14;8:181. [Google Scholar]
13. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-barré syndrome. *The lancet*. 2021 Mar 27;397(10280):1214-28. [Google Scholar]
14. Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barré syndrome associated with SARS-CoV-2 infection: causality or coincidence?. *The Lancet. Neurology*. 2020 Apr 1;19(5):383. [Google Scholar]
15. Palaodimou L, Stefanou MI, Katsanos AH, Fragkou PC, Papadopoulou M, Moschovos C, Michopoulos I, Kokotis P, Bakirtzis C, Naska A, Vassilakopoulos TI. Prevalence, clinical characteristics and outcomes of Guillain-Barré syndrome spectrum associated with COVID-19: a systematic review and meta-analysis. *European journal of neurology*. 2021 Oct;28(10):3517-29. [Google Scholar]
16. Stowe J, Andrews N, Wise L, Miller E. Investigation of the temporal association of Guillain-Barre syndrome with influenza vaccine and influenzalike illness using the United Kingdom General Practice Research Database. *American journal of epidemiology*. 2009 Feb 1;169(3):382-8. [Google Scholar]

17. Mier-y-Teran-Romero L, Delorey MJ, Sejvar JJ, Johansson MA. Guillain–Barré syndrome risk among individuals infected with Zika virus: a multi-country assessment. *BMC medicine*. 2018 May 15;16(1):67. [Google Scholar]
18. Yusari IG, Sudira PG, Samatra DP. Clinical characteristics of Guillain–Barre syndrome in COVID-19: a systematic review and meta-analysis of observational studies. *The Egyptian Journal of Neurology, Psychiatry and Neurosurgery*. 2023 Mar 27;59(1):40. [Google Scholar]
19. Filosto M, Cotti Piccinelli S, Gazzina S, Foresti C, Frigeni B, Servalli MC, Sessa M, Cosentino G, Marchioni E, Ravaglia S, Briani C. Guillain-Barré syndrome and COVID-19: an observational multicentre study from two Italian hotspot regions. *Journal of Neurology, Neurosurgery & Psychiatry*. 2021 Jul;92(7):751-6. [Google Scholar]
20. Prashanth B, et al. *Int J Res Med Sci*. 2023;11(9):3409-13. [Google Scholar]
21. Jain S, Kashyap A, Singh A. Spectrum of Multisystem Inflammatory Syndrome in Children (MIS-C)—a Report of Three Cases. *SN Comprehensive Clinical Medicine*. 2021 Dec;3(12):2635-9. [Google Scholar]