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Case Report

Beyond Erythema Infectiosum: A Rare Case of Parvovirus B19 with Neurological, Renal, Hematological & Cardiac Manifestations

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Abstract

Acute Parvovirus B19 infection can lead to severe neurological and systemic complications. This case describes a rare presentation of Parvovirus B19 encephalitis with posterior reversible encephalopathy syndrome (PRES) with symptomatic seizure accompanied by dilated cardiomyopathy (DCM) with atrial septal defect-secundum, acute kidney injury (AKI), sepsis and pure red cell aplasia (PRCA). The complex interplay of these conditions posed significant diagnostic and management challenges. Neurological involvement manifested as encephalitis with PRES, contributing to symptomatic seizures and progressing to status epilepticus. The concurrent development of DCM and AKI suggested a profound systemic inflammatory response, exacerbated by sepsis. PRCA further complicated the clinical course by inducing severe anemia. This case highlights the need for early recognition, comprehensive monitoring, and multidisciplinary intervention to improve patient outcomes in severe Parvovirus B19 infections with multi-organ involvement.

Keywords: *Parvo B19, Erythema Infectiosum, Case Report*

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Introduction

There are 5 different types of parvoviruses that affect human of which Parvovirus B19 has gained the status of an expanding and emerging virus. In 1975, Yvonne Cossart first identified parvovirus particles by electron microscopy while screening for Hepatitis B virus.[1] Parvovirus B19 infection is common in childhood and by the age of 15 years half of them have specific anti parvovirus B19 antibodies.[2]

Parvo B19 primarily spreads through respiratory droplets but can also be transmitted through blood transfusion and from infected mother to fetus, potentially causing serious condition like hydrops fetalis or fetal death during pregnancy.[3] Secondary transmission to seronegative individual is frequent, with a secondary attack rate of 20-30% among susceptible adults in environments like school or households during erythema infectiosum outbreaks.[4] Parvovirus B19 is a small, single stranded DNA virus that binds to P antigen receptor on erythrocyte.[5] Its genome encodes three known functional proteins: nonstructural protein (NS1) and two capsid proteins, viral protein 1(VP1) and viral protein 2(VP2)[6]. NS1 promotes various replication processes and is cytotoxic to host cell.[6] Parvo B19 has been associated with wide range of clinical manifestations from asymptomatic to multisystem involvement like erythema infectiosum,[7] arthropathy, thrombocytopenia, hepatitis, acute kidney injury with proteinuria, nephritic presentation,[8] myocarditis, cardiomyopathy,[9] and neurological disease in healthy host, pure red cell aplasia in immunocompromised one, transient aplastic crisis in patients with increases red cell turnover.[10] Of these the neurological involvement in children has been rising[11] that may even culminate in sinister consequences. The neurological manifestations commonly include acute encephalitis, encephalopathy with posterior reversible encephalopathy syndrome,[12] meningitis, cerebellar ataxia, transverse myelitis, stroke and peripheral neuropathy.[13]

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Case Presentation

A 13-year-old boy, previously healthy, hailing from Barishal presented to Dhaka Medical College Hospital with generalized swelling and scanty micturition for four days and occasional headache and low-grade fever over the preceding 3 days. Two weeks prior, he had history of sore throat and fever which resolved spontaneously. The patient developed facial swelling that progressed to involve his limbs and abdomen, accompanied by decreased urine output with high colored urine. From the third day of admission, he developed headache, nausea, occasional breathlessness and clouding of consciousness (E3V3M4). On the fourth day of admission, he developed 2-3 episodes of generalized tonic-clonic seizure lasting for 3–4 minutes, associated with tongue biting, frothing and urinary incontinence. Post-ictal symptoms included dizziness, headache and mild visual disturbance. The seizure was initially controlled by anticonvulsant. Five days later, he again developed recurrent episodes of seizures accompanied by fever (102°F). This seizure recurrence following the febrile episode required an increase in anticonvulsant dosage. There was no history of rash, oral ulcer, joint pain, intake of date juice or nephrotoxic drug use. His past medical history was unremarkable, and his immunizations were up to date according to the Expanded Program on Immunization (EPI) schedule.

On examination, the patient appeared ill-looking and severely anemic. His blood pressure was 110/70 mmHg, pulse was 92 beats per minute, and temperature was 101°F. Generalized edema was present. A firm, non-tender right posterior cervical lymph node (2× 1.5 cm) was palpable which gradually increased in size from the 7th day of admission to onwards. Neurological examination revealed normal higher mental functions, motor strength of 4/5 in all extremities, normal reflexes and intact sensory functions. On fundoscopic examination, optic disc was pale with clear margin. Abdominal examination revealed mild hepatosplenomegaly and shifting dullness, suggesting ascites. Cardiovascular and respiratory examinations were unremarkable.

Laboratory findings including full blood count revealed normocytic normochromic anemia-5.1g/dl (After getting few units of blood transfusion hemoglobin was corrected to 9.2 g/dl and later after 3 weeks of admission hemoglobin again dropped to 6.6g/dl), neutrophilic leukocytosis(WBC count-16800/cumm, Neutrophil 78%), thrombocytosis, raised ESR (64mm) and elevated D-dimer levels (2.42 µg/ml). Serum complement levels (C3 and C4) were reduced and CRP was mildly elevated (4.66 mg/L). Reticulocyte count was in normal range. Renal function tests indicated acute kidney injury evidenced by rapidly rising creatinine (5.33 mg/dl, later normalized to 0.99 g/dl after 3 sessions of hemodialysis) and urea (118mg/dl) with the urine analysis showing 40–50 pus cells, 1-2 red blood cells per high-power field (No dysmorphic RBC) and protein was nil. Reduced complements levels suggested immune complex-mediated injury.

The cerebrospinal fluid (CSF) analysis using the VIASURE multiplex Real Time PCR Kit detected the presence of Human Parvovirus B19. CSF study showed an elevated white blood cell count of 35 cells/cumm, predominantly neutrophils (60%) with 40% lymphocytes. Both Gram stain and AFB stain were negative for organisms. CSF protein was slightly increased at 51.80 mg/dL, while glucose levels were within the normal range at 57.06 mg/dL.

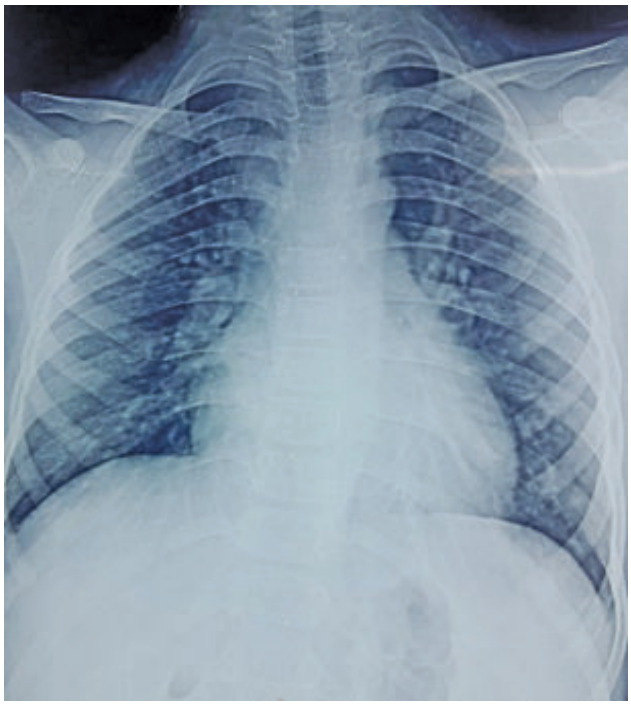

Fig.-1: *Chest X-ray P/A view showing cardiomegaly with pulmonary oedema*

Imaging studies revealed cardiomegaly and pulmonary oedema on chest X-ray, mild hepatosplenomegaly with passive hepatic congestion and bilateral swollen echogenic kidney on abdominal ultrasonography, and mild cerebral edema on CT of brain. MRI of Brain demonstrated bilateral symmetrical hyperintense (bright) signals in the fronto-parietal and occipital areas in T2 weighted and FLAIR predominantly affecting subcortical white matter, indicating vasogenic edema findings consistent with PRES[14]. Echocardiography showed global hypokinesia, mild left ventricular systolic dysfunction (ejection fraction, 48%), and mild pulmonary hypertension and ASD secundum (6×8×9mm). Hemoglobin electrophoresis, osmotic fragility test, ANA, *p*-ANCA, *c*-ANCA, Coombs test, PT, APTT and thrombophilia panel were normal.

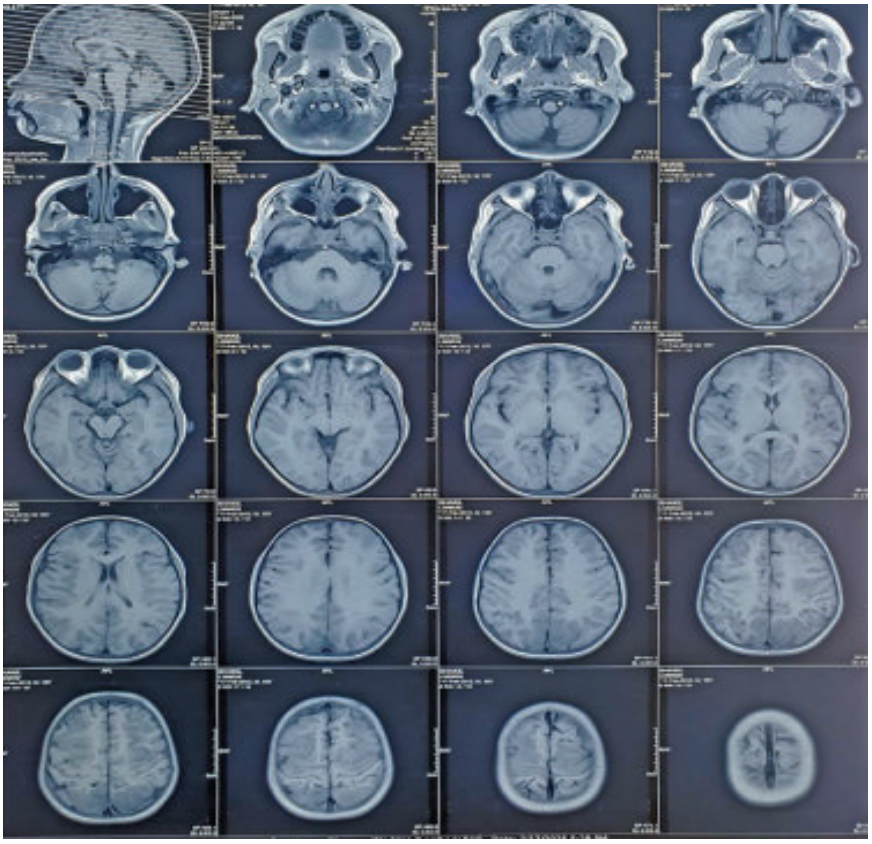

Fig.-2: T1-weighted MRI shows bilateral symmetrical hypointense area in fronto-parietal and occipital area

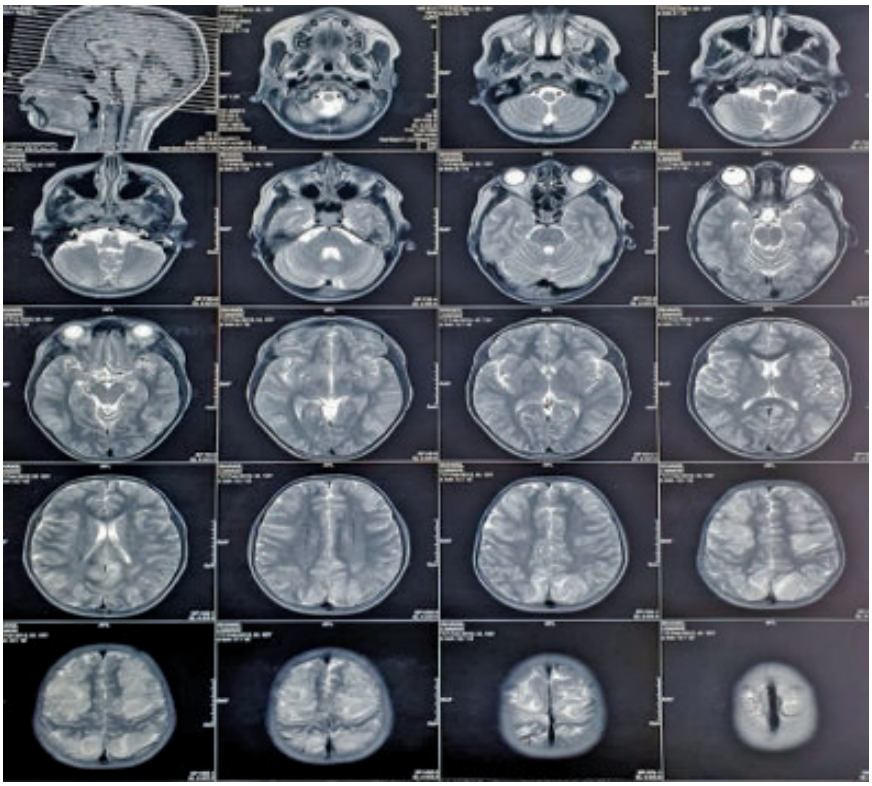

Fig.-3: Bilateral symmetrical hyperintensity in fronto-parietal and occipital area in T2 weighted MRI

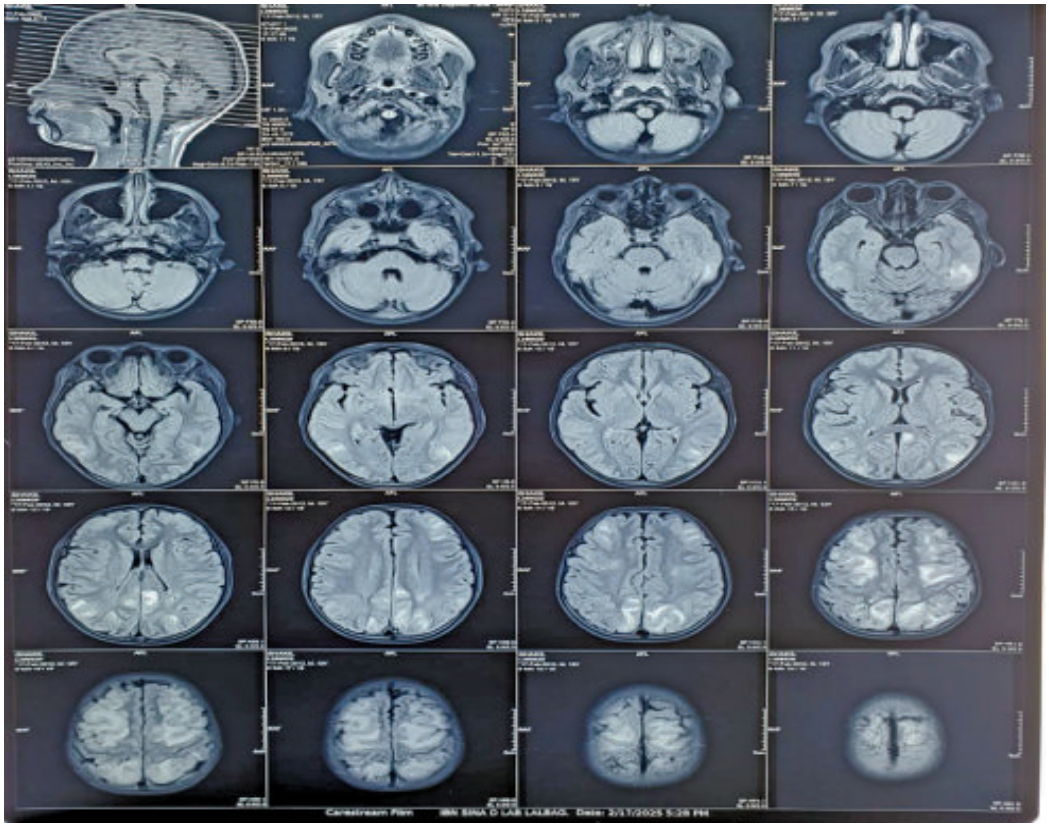

Fig.-4: Bilateral symmetrical hyperintensity noted in fronto-parietal and occipital area in T2 FLAIR sequences.

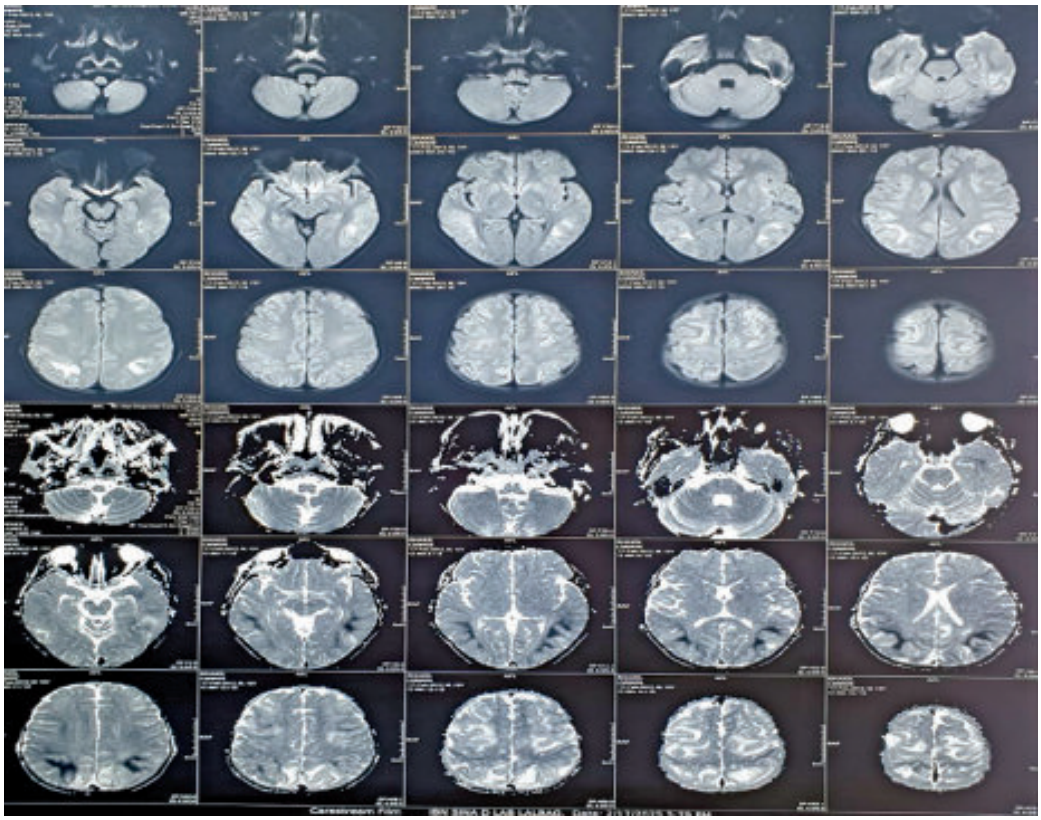

Fig.-5: Bilateral hyperintensity involving parieto-occipital area in DWI which shows low signal in ADC

A diagnosis of acute viral encephalitis due to Parvovirus B19 was made, complicated with Posterior Reversible Encephalopathy Syndrome (PRES) with symptomatic seizure, Acute Kidney Injury (AKI) due to sepsis (resolved), DCM with ASD Secundum and Pure Red Cell Aplasia (PRCA). The patient showed significant clinical improvement, with resolution of AKI and no further seizures after anticonvulsant adjustment. His renal function normalized with three sessions of hemodialysis and his cardiac function required ongoing monitoring. After two months on a follow up visit, his anemia was fully resolved with a normal hemoglobin level and renal function tests within normal limits. The symptoms associated with PRES (Posterior Reversible Encephalopathy Syndrome) were completely reversible with no residual neurological deficits.

Discussion

Parvovirus B19 has been linked to a spectrum of neurological, cardiac, renal and hematological disorders including encephalitis, PRES, myocarditis resulting in cardiomyopathy, acute kidney injury and pure red cell aplasia. PRES is a rare complication characterized by reversible vasogenic edema, typically associated with hypertension, renal dysfunction or sepsis.[14] In this case, AKI and sepsis likely contributed to the development of PRES. In encephalitis, Parvovirus B19 (PVB19) is hypothesized to cause direct neuronal injury or trigger an autoimmune response leading to inflammation of the central nervous system (CNS). The presence of PRES in this case suggests endothelial dysfunction, which may be exacerbated by inflammatory cytokines and immune activation. PRES is thought to occur due to disruption of the blood-brain barrier, leading to vasogenic edema which can be reversible with early intervention. PVB19 primarily infects erythroid progenitor cells via the P antigen receptor.[14] This leads to cytopathic effects and immune-mediated destruction of infected cells, resulting in hematologic abnormalities such as transient aplastic crisis or pure red cell aplasia (PRCA).[15] In case of our patient the fully reversible anemia with normal reticulocyte count supported the diagnosis of PRCA. Hemoglobin electrophoresis and osmotic fragility test were normal ruling out hemoglobinopathies and red cell membrane disorders. Normal upper GI Endoscopy with negative stool OBT ruled out any occult blood loss. Bone marrow examination could not be done due to unstable physical condition. Renal involvement, such as AKI, may result from immune complex deposition in the glomeruli, leading to glomerulonephritis.[8] Reduced complement levels with elevated D-dimer levels in our patient suggest an immune-mediated injury mechanism.[16]The presence of global hypokinesia and mild left ventricular systolic dysfunction elevated levels of troponin I + CK-MB raised concerns about Parvovirus B19-associated myocarditis,[17] potentially progressing to DCM. Cardiac manifestations, including DCM, are believed to arise from direct myocardial infection or an exaggerated immune response leading to myocardial inflammation and fibrosis. Studies have documented the presence of PVB19 DNA in cardiac tissue of affected patients,[18] supporting viral myocarditis as a contributing factor. In this patient ASD Secundum along with viral myocarditis complicated the clinical picture by developing DCM.

Diagnosing PVB19-associated encephalitis is difficult due to its atypical symptoms, overlap with other conditions, complex multi-organ involvement, limited availability of specific tests and the potential for misdiagnosing PRES without early imaging. Previous studies have documented the neurological manifestations of PVB19 but comprehensive reports including simultaneous encephalitis, PRES, AKI, DCM and PRCA in a patient are limited. Kerr et al.[19] reported a case of PVB19-associated encephalitis, emphasizing the role of immune-mediated mechanisms in CNS involvement. Similarly, Bock et al.[20] described PVB19-induced myocarditis, attributing myocardial dysfunction to viral invasion and inflammatory response. Our case aligns with these findings but extends the discussion by illustrating concurrent renal and neurological complications, highlighting the multisystemic impact of PVB19. Regarding PRES, Hinchey et al.[21] first identified the condition as a reversible neurological syndrome related to endothelial dysfunction. While PRES is frequently associated with hypertensive crises, its occurrence in viral encephalitis with AKI and Sepsis, as observed in our patient remains relatively rare. Levine et al.[22] documented PVB19-induced nephropathy, supporting our hypothesis that immune complex deposition plays a role in AKI pathogenesis. The overlapping findings in these studies reinforce the necessity of a multidisciplinary approach to manage such complex cases. Our case also adds to the evidence supporting immunomodulatory therapy as using steroid initially improved the outcome. Heegaard and Brown[10] reviewed the role of IVIG and corticosteroids in severe PVB19 infections, addressing the need for early intervention to mitigate immune-mediated damage. The favorable outcome in our patient, achieved through multidisciplinary management, aligns with their recommendations and highlights the importance of tailored therapeutic strategies.

Conclusion

While PVB19 is generally considered a benign pathogen, its potential for causing severe systemic disease should not

be underestimated. Early recognition with advanced diagnostic technique, aggressive supportive therapy, a multidisciplinary approach and long term follow up are essential for optimizing outcomes in patients with severe complications of Parvovirus B19. Clinicians should maintain a high index of suspicion in patients presenting with unexplained cytopenias, recurrent seizures and fluid overload or other presentations of multi system dysfunction. This case contributes to the growing body of evidence on PVB19-related encephalitis and highlights the need for increased clinical awareness, particularly in pediatric patients with multi-organ dysfunction.

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