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Clinical short communication

Persistent parvovirus B19 DNAemia in B-cell depletion: Implications for family planning in multiple sclerosis and related disorders

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ABSTRACT

Background: B-cell depletion has transformed the management of multiple sclerosis (MS) and can be a treatment option in other neuroimmunological disorders, such as neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein-associated disease (MOGAD). However, its immunosuppressive effects increase susceptibility to viral and bacterial infections. During a recent parvovirus B19 (B19V) outbreak across Europe, we noted cases of persistent B19V infections in B-cell depleted patients.
Aim: To determine the clinical implications of persistent B19V for the management of B-cell-depleted individuals with MS and related neuroimmunological disorders.
Methods: In this retrospective observational study, patients with neuroimmunological disorders followed between 2013 and 2023 and during a B19V outbreak in 2024 undergoing B-cell depletion and diagnosed with B19V infection were included. Clinical presentation, laboratory findings and treatment response were reviewed.
Results: We identified three B-cell depleted individuals with confirmed B19V infection (two with relapse-remitting MS (RRMS) on ocrelizumab, one with MOGAD on rituximab), all female and of childbearing age. Two patients presented with symptomatic acute viral infection consistent with Fifth Disease, including neutropenia, anemia, and elevated liver enzymes. The third patient contracted B19V during early pregnancy, presented with mild flu-like symptoms not specific to B19V, however, her fetus developed signs of fetal anemia. Intravenous immunoglobulin treatment led to rapid clinical and laboratory improvement, yet all patients showed persistent B19V DNAemia.
Conclusions: Given the demographic characteristics of people with MS and related disorders, exposure to communicable childhood diseases may represent a relevant risk factor for potentially severe complications, including adverse pregnancy outcomes, during B-cell depletion therapy.

1. Introduction

Multiple sclerosis (MS) is a chronic autoimmune disorder of the central nervous system and the leading cause of non-traumatic disability in young adults worldwide [1]. Neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein-associated disease (MOGAD) are rare differential diagnoses of MS exhibiting distinct autoantibody-associated pathophysiological mechanisms, patterns of CNS involvement and clinical courses [2,3]. Current treatment strategies aim to manage acute relapses and prevent disease progression through immunosuppressive, disease-modifying therapies (DMT) [4]. Given the typical age at diagnosis, treatment decisions must include family planning considerations, including pregnancy and early parenthood. Therefore, increasing attention has been directed toward assessing the reproductive safety profiles of DMT, and recently updated consensus recommendations guide individualized risk-benefit assessments [5,6].

Among the most effective therapeutic options, B-cell-depleting agents, such as rituximab, ocrelizumab and ofatumumab, significantly

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reduce relapse rates and delay disease progression [7–9]. However, their immunosuppressive effects increase the susceptibility to infections [10,11], an important consideration in the context of parenthood, particularly in households with young children, where communicable childhood diseases are highly prevalent [12].

Here, we report three cases of parvovirus B19 (B19V) infections with sustained viral PCR positivity following a recent pan-European outbreak [13]: two in people with MS (pwMS) treated with ocrelizumab and one in a patient with MOGAD receiving rituximab. We discuss the clinical implications of persistent B19V DNAemia for the management of B-cell-depleted pwMS and related neuroimmunological disorders.

2. Methods

2.1. Study design and participants

In this monocentric observational retrospective study, individuals with neuroimmunological disorders receiving B-cell-depleting treatment and diagnosed with B19V infection were included during a B19V outbreak in 2024 and by systematically searching the clinical records of all patients treated at the neuroimmunology outpatient clinic of the University Hospital Zurich, Switzerland, between 2013 and 2023. We obtained demographic and clinical information through review of regular medical records. Inclusion and exclusion criteria are detailed in Supplementary material 1.

2.2. Standard protocol approvals, registrations, and patient consents

The three patients included in the study provided written informed consent for data collection. The retrospective analysis was approved by the Ethics Board of the Canton of Zurich (KEK-ZH Nr. 2024–00239). This study was approved by the data governance board of the University Hospital Zurich (No. DGR0000815).

3. Results

3.1. Demographics and clinical presentation

For this retrospective observational study, we identified three patients from our neuroimmunology outpatient clinic undergoing B-cell depletion, who presented with prolonged infectious symptoms consistent with B19V infection during a pan-European outbreak in March to October 2024. Demographic and clinical characteristics are summarized in Table 1. A systematic search of clinical records from 2013 to 2023 did not identify additional people with B19V infections under B-cell depletion (Supplementary material 1).

Table 1
Demographics and clinical characteristics.

	Patient 1	Patient 2	Patient 3
Age, *y*	38	45	39
Sex	female	female	female
Diagnosis	RRMS	RRMS	MOGAD
Disease duration at B19V infection, *y*	7	19	2
EDSS at B19V infection, absolute	0.0	3.5	0.0
DMT at B19V infection	ocrelizumab	ocrelizumab	rituximab
duration of therapy, m	31	24	13
number of infusions (n)	4	4	3
total dose (mg)	2400	2400	1710 (3x375mg/m^2)
previous DMT	dimethyl fumarate 09/2018–05/2021 glatiramer acetate 09/2017–09/2018	dimethyl fumarate 01/2022–03/2022 glatiramer acetate 2005–2013 and 10/2017–03/2018	none
Clinical features of B19V infection	rash, fever, arthralgia	rash, fever, arthralgia	flu-like symptoms
duration of symptoms, weeks	*4*	*2*	*2*
treatment, dose	*IVIg 1 g/kgBW (90 mg total)*	*IVIg 0.4 g/kgBW every 4 weeks (110 mg total)*	*IVIg 2 g/kgBW (120 mg total)*
Laboratory parameters at B19V diagnosis			
*Initial B19 viral load [copies*ml^{-1}]*	441′344′467	33′081	1′400′000
B19V IgM seroconversion	at diagnosis	nd	nd
B19V IgG seroconversion, weeks after diagnosis	3	16	8
CD19$^+$20$^+$ B cells [μL^{-1}] (% leukocytes)	6 (<1)	0 (0)	0 (0)
*Serum-IgG [g*L^{-1}]*	10.5	6.2	6.1
RBC [nadir, μL^{-1}] [nadir, μL^{-1}] (time of occurrence in reference to day of diagnosis (d))	3.07 (+6)	3.62 (−32)	4.06*
Neutrophils [nadir, μL^{-1}] [nadir, μL^{-1}] (time of occurrence in reference to day of diagnosis (d))	0.54 (+6)	1.08 (−32)	4.3*
*Serum ALT [peak, IU*ml^{-1}] (time of occurrence in reference to day of diagnosis (d))*	283 (0)	157 (−4)	14*

ALT alanine-aminotransferase, *BW body weight, DMT* disease modifying treatment, *EDSS* expanded disability status scale, *G/l* gram per liter, *Hb* hemoglobin, *Ig* immunoglobulin, *m* months, MOGAD myelin oligodendrocyte glycoprotein-associated disease, *n* number, *nd* not detected, *RBC* red blood cell, *RRMS* relapsing-remitting multiple sclerosis, *y* years. *Laboratory parameters of patient 3 remained normal at all times.

3.2. Case descriptions

3.2.1. Case 1

A 38-year-old female with relapse-remitting multiple sclerosis (RRMS), receiving ocrelizumab every 9 months (Table 1), presented to her general practitioner in March 2024 with persistent arthralgia, intermittent fever up to 40 °C, and worsening general condition. She was referred to our outpatient clinic for further evaluation. Laboratory investigations revealed pure red cell aplasia (PRCA), moderate neutropenia, and elevated liver enzymes. A suspected B19V infection was confirmed by PCR (Table 1), while initial serology showed B19V IgM, but not B19V IgG-seroconversion (Fig. 1 A). Ocrelizumab treatment was paused and the patient received intravenous immunoglobulins (IVIg, 1 g/kg body weight (BW)), which led to rapid clinical and laboratory improvement. Follow-up testing three weeks later demonstrated presence of B19V-IgG, which remained detectable twelve months later, suggesting seroconversion and not passive transfer of IVIg-derived B19V-IgG. However, a low-grade B19V DNAemia persists up to the last follow-up at 15 months after first symptoms, despite B-cell reconstitution and additional two cycles of IVIg in May and June 2025 (Fig. 1 A). Ocrelizumab was switched to glatiramer acetate due to a stable MS disease course.

Posthoc epidemiological history indicated likely transmission from the patient's school-aged child, who had experienced a brief febrile illness with nonspecific symptoms two weeks prior. A B19V outbreak had been reported at the child's school during the same period.

3.2.2. Case 2

A 45-year-old female with RRMS, receiving ocrelizumab every 6 months (Table 1), presented to her general practitioner (GP) in March 2024 with fever up to 40 °C, arthralgia, a skin rash, and worsening general condition (Fig. 1B). Laboratory work-up revealed PRCA, mild neutropenia, and elevated liver enzymes. Due to diagnostic uncertainty, she was referred to our dermatology department for evaluation of the rash. Skin biopsy showed nonspecific dermatitis consistent with a

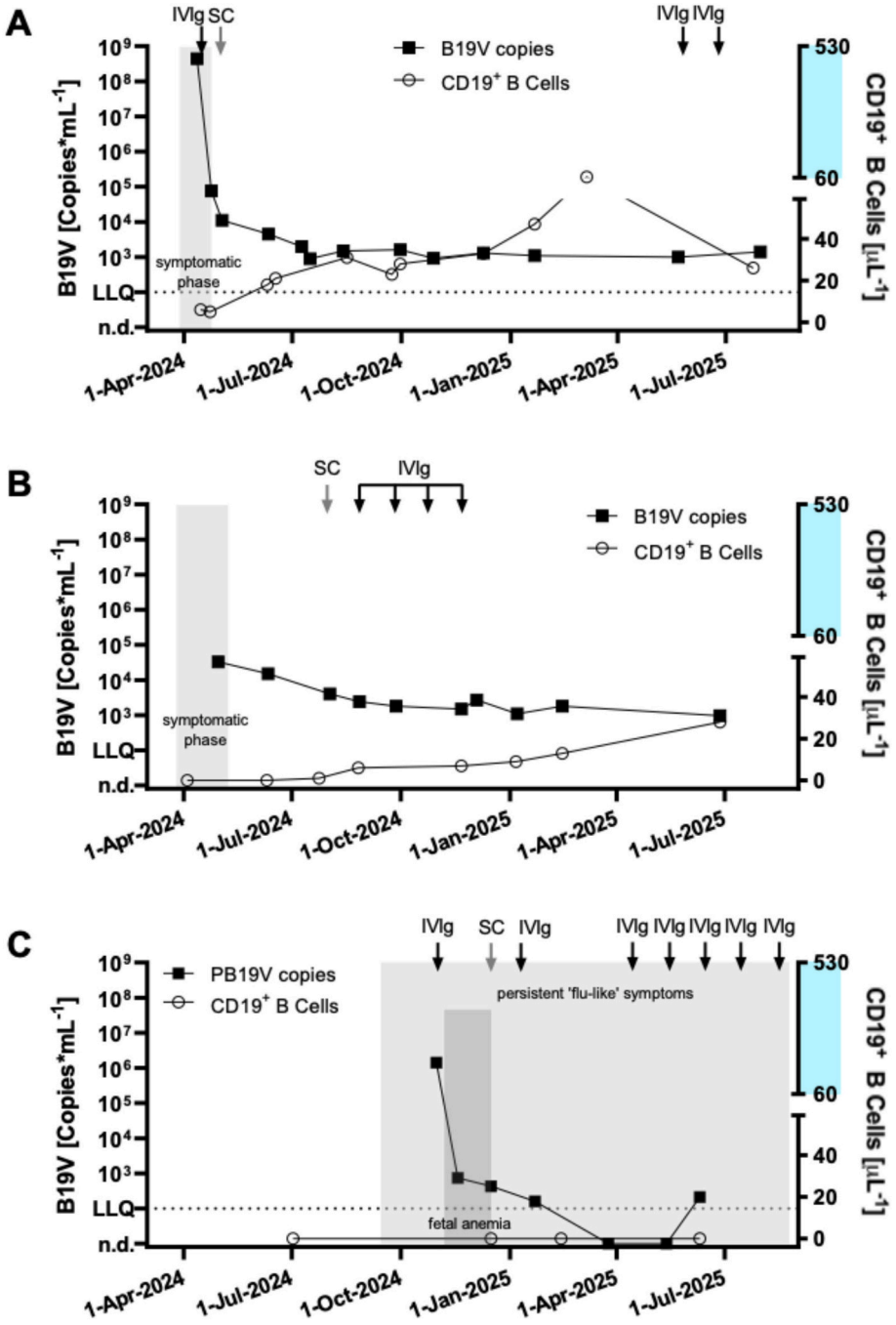

Fig. 1. Persistent B19V DNAemia under B-cell depletion despite IVIg treatment.
(A) patient 1, (B) patient 2, and (C) patient 3. Overview of clinical course (grey), B19V DNAemia (square), B-cell count (circle), IVIg treatment (arrow) and timepoint of IgG seroconversion (SC, arrow) as shown.
Physiological range of $CD19^+$ B cell count: 60–530*µL^{-1} (light blue). *IVIg* intravenous immunoglobulin, *B19V* Parvovirus B19 virus, *Lower Limit of Quantification* (LLQ) defined as 10^2 genome copies*ml^{-1}, *SC* seroconversion.

paraviral exanthema. One week after referral to our outpatient clinic, and 32 days after initial presentation to her GP, PCR confirmed B19V infection. By this time, the patient's symptoms and laboratory results had already normalized (Table 1). Despite clinical resolution, follow-up PCR testing revealed a persistent low-grade B19V DNAemia despite B19V-IgG seroconversion twelve weeks later. To support viral clearance during persistent B-cell depletion, we serially administered IVIg (1 g/kg BW) every 4 weeks (Fig. 1B) starting sixteen weeks after diagnosis, however, B19V DNA remained detectable. In January 2025, we discontinued IVIg treatment after four cycles because of rising B cell counts (Fig. 1B) and continued to monitor the patient clinically. The patient has remained clinically stable up to the latest follow-up at 15 months after the first symptoms with respect to both B19V infection and RRMS. Ocrelizumab treatment is currently paused and a switch to an oral DMT is planned.

Epidemiological history revealed that the patient's children had experienced a rash with mild constitutional symptoms a few days prior to the patient's first symptoms. No formal B19V outbreak was reported at their school.

3.2.3. Case 3

A 39-year-old female patient with relapsing MOGAD treated with rituximab (Table 1) reported her second pregnancy 6 months after her second rituximab infusion. In October 2024, at gestational week 14, she presented to her gynecologist with persistent coughing. Her gynecologist suspected B19V infection, which was confirmed by PCR. Initial serological testing showed neither B19V-IgM nor IgG seroconversion and B cells were fully depleted. Following consultation with the gynecology department, rituximab was paused, and the patient was hospitalized to receive IVIg (1 g/kg BW). This led to rapid DNAemia regression and B19V-IgG seroconversion eventually was confirmed eight weeks later (Fig. 1C). However, prenatal ultrasound revealed increasingly elevated peak systolic velocity in the fetal middle cerebral artery in November 2024, consistent with B19V-associated fetal anemia. As no hydrops fetalis developed, intrauterine transfusions were not required. However, the decision was made to repeat the IVIg treatment in January 2025. Serial follow-up monitoring of the pregnancy thereafter demonstrated a stable fetal condition and the delivery occurred at 39 + 1 weeks of uncomplicated gestation with no perinatal complications. B19V DNAemia in the patient, however, remained detectable over 10 months up to the latest follow-up (Fig. 1C). We therefore decided not to restart rituximab and instead continued IVIg, serving both as treatment for the B19V infection and as maintenance therapy for the underlying MOGAD. The patient experienced persistent flu-like symptoms including cough and rhinorrhea (Fig. 1C).

Detailed history revealed that the patient's older daughter likely contracted B19V at her daycare, where an outbreak had been reported days earlier. The child remained asymptomatic.

4. Discussion

Persistent or unexpectedly severe B19V infections have been reported in patients treated with B-cell-depleting agents such as rituximab and ocrelizumab, including in the context of MS [14–16]. Here, we report three cases of autoimmune demyelinating diseases treated with B-cell depleting drugs affected by a prolonged B19V infection during a recent post-pandemic upsurge of B19V infections in Europe [17–20].

It is hypothesized that, despite normal immunoglobulin levels, patients undergoing B-cell depletion fail to mount an adequate immune response to B19V infection due to the absence of pre-existing B19V-specific immunoglobulins and the inability to generate new B19V-specific plasma cells. In B-cell-depleted patients, persisting viral replication may result in severe complications, including agranulocytosis and anemia due to lytic bone marrow suppression, as well as B19V-associated hepatitis [21–23]. Consequently, as illustrated in cases 1 and 3, hospitalization may become necessary. Treatment with IVIg is thought to compensate for the deficient antibody-mediated immunity by providing exogenous B19V-specific antibodies [24]. In our cases, this led to a rapid decline in B19V DNAemia in both patients 1 and 3 (Fig. 1). However, complete viral clearance was not achieved in any patient, and persistent low plasma B19V DNA could be detected by PCR more than fifteen months after infection. Furthermore, low-level B19V DNAemia persisted despite B-cell reconstitution in patient 1 and repetitive IVIg therapy in patient 2. B-cell depletion can lead to a prolonged suppression of memory B-cells [25]. While beneficial in controlling autoimmunity [26], sustained memory B–cell suppression may therefore crucially hamper B19V clearance [27]. Our cases therefore add important real-world data to the safety profile of long-term ocrelizumab treatment [28], suggesting that B-cell depletion may result in a prolonged and potentially underrecognized impairment of B-cell-dependent antiviral immunity.

Notably, apart from patient 3 who continued to experience flu-like symptoms during the entire follow-up period, persistent B19V DNAemia was not accompanied by clinical relapse of B19V-associated symptoms. This disease course despite persistent B19V DNAemia is consistent with other reports of exacerbated B19V infections in the context of B-cell depletion [14] and has also been observed in adult recipients of solid organ transplants [29,30]. Because we considered the DNAemia as indicator of persisting viral replication in these immunosuppressed patients, we treated all 3 cases with repeated IVIg infusions. However, it is unclear whether the B19V DNA detected in plasma reflects viral replication or rather a shedding of non-functional, attenuated viral remnants [31]. This consideration is supported by longitudinal blood donor screening studies, which have observed persistent B19V DNAemia in asymptomatic, immunocompetent donors, while virtually no cases of transfusion-transmitted B19V infection have been reported. The authors argue, that this was either due to the absent clinical relevance of such infection, or because the presence of this DNAemia does not translate into infectious potential [31,32].

In all three cases, transmission likely occurred through exposure to symptomatic children or confirmed B19V outbreaks in school or daycare settings. Although the children were not tested and a definitive link cannot be established, the timing and symptomatology support this route of infection. This highlights an often-overlooked risk for patients receiving B-cell-depleting therapies. Given the typical age distribution of people with MS and related disorders, many patients are parents of young children, who commonly transmit benign, but potentially severe infections in immunosuppressed adults, such as B19V. Clinicians should consider this exposure risk in differential diagnoses and patient counseling.

Furthermore, MS, NMOSD and MOGAD often affect people of childbearing potential, making family planning counseling essential. While recent consensus recommendations address the teratogenic risks of DMTs [5,6], the impact of maternal immunosuppression in infection risk during pregnancy remains underrecognized. In case 3, fetal anemia appears to have resulted from acute infection representing a rare but serious complication of B19V infection during pregnancy despite prior IVIg treatment [33,34]. This case highlights that B-cell depletion may increase maternal and fetal vulnerability to infections.

The key limitation of our study is the small number of eligible individuals diagnosed with B19V under B-cell depletion. In our review of patient data from 2013 to 2023 we did not find further documented cases of B19V infection under B-cell depletion. This most likely limits the occurrence to the recent B19V outbreak across Europe in 2023/2024 [17–20], however, we cannot rule out that more B-cell depleted patients were affected by B19V infection yet did not experience typical symptoms or complications such as neutropenia, thereby remaining undiagnosed.

In conclusion, given the prevalence of common childhood illnesses [12] and the real-world infection burden in young families of pwMS and related disorders, our observations underscore the need for vigilance and strategies for infection prevention in pwMS and related disorders on B-cell-depleting therapies.

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CRediT authorship contribution statement

Lorenz Karnbrock: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation. **Marina Herwerth:** Writing – review & editing. **Guido Vincent Bloemberg:** Writing – review & editing, Investigation. **Michael Huber:** Writing – review & editing, Investigation. **Patrick Roth:** Writing – review & editing. **Irene Alma Abela:** Writing – review & editing. **Veronika Kana:** Writing – review & editing, Supervision, Conceptualization.

Ethics statement

Written informed consent was obtained from the individuals for the publication of any potentially identifiable data included in this article. The retrospective analysis was approved by the Ethics Board of the

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Declaration of competing interest

L. Karnbrock, G.V. Bloemberg and M. Huber report no conflicts of interest.

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