

Refractory Polyradiculoneuritis Responsive to Cyclophosphamide: A Case Report of a Patient with a History of Diffuse Large B Cell Lymphoma and CAR-T Cell Therapy

Luisa Kreß MD^{1*}, Nurcan Üçeyler MD¹, Magnus Schindehütte MD², Christian Geis MD³, Hayrettin Tumani MD⁴, Claudia Sommer MD¹, Karl Georg Haeusler MD⁴

Abstract

Background: Polyradiculoneuritis is a rare but serious neurological condition with diverse etiologies, including autoimmune, paraneoplastic, and therapy-induced mechanisms. In oncologic patients, particularly those with hematologic malignancies and prior immune-directed therapies, diagnosis and treatment can be especially challenging.

Case presentation: We report on a 69-year-old woman with acute onset and rapid progression of a polyradiculoneuritis with a history of B-cell lymphoma and chimeric antigen receptor (CAR)-T cell therapy. While response to standard glucocorticoid, intravenous immunoglobulin, or plasma exchange was insufficient, the patient was successfully treated with cyclophosphamide (CYC).

Conclusions: This case highlights the importance of considering autoimmune polyradiculoneuritis in immunotherapy-exposed patients with rapidly progressive neuropathy. When first-line treatments fail, and histopathology shows T-cell predominance, CYC may be an effective therapeutic option.

Keywords: Polyradiculoneuritis; Inflammatory axonal radiculitis; Cyclophosphamide; Immunotherapy; Peripheral neuropathy.

Introduction

Polyradiculoneuritis in oncological patients is a rare but potentially life-threatening neurological condition, with autoimmune, infectious, paraneoplastic, and therapy-related etiologies. Patients present with rapidly progressive sensorimotor deficits, often affecting spinal nerve roots and in some cases also the cranial nerves [1-5]. The standard therapies for polyradiculoneuritis are high-dose intravenous (i.v.) glucocorticoids or i.v. immunoglobulins (IVIg) and plasma exchange (PLEX) in patients with acute or rapidly progressing clinical symptoms and in chronic conditions upon an inadequate response to glucocorticoids [6, 7]. Refractory cases may require immunosuppressive agents like rituximab (RTX) or cyclophosphamide (CYC) [8, 9], as RTX targets B lymphocytes, while CYC inhibits abnormal cell proliferation, B and T cells [8, 9]. In oncologic patients, especially in those with hematologic malignancies and a history of intensive immune-directed therapies, the pathogenesis of polyradiculoneuritis requires careful etiological distinction [10-13]. Autoimmune processes must be differentiated from paraneoplastic mechanisms or therapy-associated immune dysregulation [12]. Chimeric antigen receptor (CAR)-T cell therapy

Affiliation:

¹Department of Neurology, University Hospital Würzburg, Würzburg, Germany

²Department of Neuroradiology, University Hospital Würzburg, Würzburg, Germany

³Department of Neurology, University Hospital Jena, Jena, Germany

⁴Department of Neurology, University Hospital Ulm, Ulm, Germany

*Corresponding Author

Dr. Luisa Kreß, MD: Department of Neurology, University Hospital Würzburg, Josef-Schneider-Str. 11, 97080 Würzburg, Germany.

Citation: Dr. Luisa Kreß, Nurcan Üçeyler, Magnus Schindehütte, Christian Geis, Hayrettin Tumani, Claudia Sommer, Karl Georg Haeusler. Refractory Polyradiculoneuritis Responsive to Cyclophosphamide: A Case Report of a Patient with a History of Diffuse Large B Cell Lymphoma and CAR-T Cell Therapy. Archives of Clinical and Medical Case Reports. 10 (2026): 29-36

Received: December 09, 2025

Accepted: January 19, 2026

Published: February 26, 2026

is known to induce acute immune-mediated toxicities such as cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS), typically occurring within days to weeks after infusion [12, 14-16]. Polyradiculoneuritis in oncologic patients may also represent a primary autoimmune process unrelated to malignancy or its treatment [4]. Due to the altered immune milieu in heavily pretreated and systemically compromised individuals, the clinical presentation and progression can be unusually severe and accompanied by a notably immune-reactive profile [17]. Here, we report the case of a 69-year-old woman with a history of diffuse large B-cell lymphoma (DLBCL), previously treated with CAR-T cell therapy and multiple chemotherapy regimens, who developed a severe, therapy-refractory, progressive autoimmune polyradiculoneuritis with cranial nerve involvement.

Case Presentation

A 69-year-old woman with a history of DLBCL first diagnosed four years ago, presented in our emergency room (day 0) with double vision, tactile hypoesthesia, and weakness of the left lower extremity, along with back pain. DLBCL had been treated with CAR-Tcells (10 months prior to the current presentation) for a relapse. The patient had been in remission from DLBCL upon presentation. Previous therapies included RTX, CYC, doxorubicin, vincristine (R-Chop), followed by RTX, gemcitabine, cisplatin, and dexamethasone (R-GDP). Neurological symptoms had started twelve days before admission to the emergency department (day-12) and worsened progressively. Neurological examination revealed a complex oculomotor dysfunction involving the left oculomotor and trochlear nerves, predominantly proximal paresis of the left lower extremity, and mild distal weakness

Table 1: Overview of the pathological signs in neurological examination during the first hospital stay and their changes in follow-up examinations.

	1 st hospital stays: Day 0 – day 10	2 nd hospital stays: Day 14 – day 84	During repetitive CYC applications
Cranial nerves	Anisocoria (right > left), diplopia ptosis (left), gaze evoked nystagmus	<i>After PLEX:</i> Oculomotor paresis (right > left) <i>After IVIg:</i> Ptosis (right) improved, gaze holding weakness right eye <i>After CYC:</i> Anisocoria improved, no ptosis, no gaze holding weakness	3 rd stay: No diplopia 4 th stay: NP 5 th stay: NP 6 th stay: NP
Motor system	Mild paresis of the proximal left LE, reflexes of LE bilaterally NE	<i>Admission:</i> Severe paresis of the left LE, mild paresis of the right LE <i>After PLEX:</i> Increase of the right-sided paresis <i>After IVIg:</i> Paraplegia LE bilaterally <i>After CYC:</i> Paraplegia distal LE, severe paresis of the proximal LE bilaterally 2 nd stay: No changes	3 rd stay: Moderate paresis of LE bilaterally, slightly more pronounced distally 4 th stay: Mild paresis of LE, slightly more pronounced distally bilaterally 5 th and 6 th stay: Mild toe paresis bilaterally 5 th and 6 th stay: PTR bilaterally NE, ATR bilaterally weakly
Sensory system			
- Tactile hypoesthesia (residual sensation)	Left leg (not quantified)	<i>Admission:</i> Left LE (proximal > distal) <i>After PLEX/IVIg:</i> No changes <i>After CYC:</i> No hypoesthesias, prickling paresthesias LE with an increasing distal gradient bilaterally	3 rd : Prickling paresthesias of the lower legs with an increasing distal gradient 4 th stay – 6 th stay: Prickling paresthesias of soles of the feet
- Vibration		<i>After IVIg:</i> Bilaterally pallhypesthesia of LE	4 th stay: Bilateral pallhypesthesia (improved in comparison to 2 nd hospital)
- Propriozeption		<i>After IVIg:</i> Position sense LE absent up to upper ankle joint <i>After CYC:</i> Position sense LE intact in metatarsophalangeal joints, <i>After IVIg:</i> Absent up to knee joint on the right, intact on the left	4 th -6 th stay: N. A.
Coordination		2 nd stay: Lack of trunk control, severe ataxia	3 rd stay: Trunk control
Gait	<i>Admission:</i> Without pathology <i>Discharge:</i> Inability to walk independently	2 nd stay: Inability to walk	3 rd stay: Stand with assistance 4 th stay: Walk a few hundred meters with a rollator 5 th stay: Salk with a rollator, small steps, unsteady, toe walking and tandem gait with support, heel standing impossible 6 th stay: Unsteady gait without assistive devices
Pain	<i>Admission:</i> Back pain <i>Discharge:</i> Severe radicular pain	2 nd stay: Stabbing pain LE from proximal to distal	3 rd – 6 th stay: No pain

Abbreviations: ATR = Achilles tendon reflex; CYC = Cyclophosphamide; IVIg = intravenous immunoglobulin; LE = lower extremity; N.A. = not applicable; NE = not elicitable; NP = no pathology; PLEX = plasma exchange; PTR = patellar tendon reflex.

Citation: Dr. Luisa Krefß, Nurcan Üçeyler, Magnus Schindehütte, Christian Geis, Hayrettin Tumani, Claudia Sommer, Karl Georg Haeusler. Refractory Polyradiculoneuritis Responsive to Cyclophosphamide: A Case Report of a Patient with a History of Diffuse Large B Cell Lymphoma and CAR-T Cell Therapy. Archives of Clinical and Medical Case Reports. 10 (2026): 29-36.

in the right lower extremity, compromising her ability to walk (see Table 1 for details).

Cerebrospinal fluid (CSF) analysis thirteen days after symptom onset (day 1) showed pleocytosis (31 [normal: 0-4] cells/mm³) and a mildly elevated protein level 73.8 [normal: 0-50] mg/dl. Viral and bacterial tests were without pathological findings, while Fluorescence-Activated Cell Sorting (FACS) revealed T-cell proliferation. Further laboratory evaluation was unremarkable (Table 2).

Detailed representation of CSF findings, excluding individual parameters measured in blood, which are documented accordingly. Laboratory-specific reference ranges are indicated in parentheses. * T-cell differentiation without any pathological findings.

Somatosensory evoked potentials (SSEP) of the tibial nerve showed prolonged P-40 latencies, and motor evoked potentials of the tibialis anterior muscle (MEPs) revealed prolonged total and “central motor” latencies (Table 3). Nerve conduction studies were essentially normal (Table 3).

Table 2: Immunological CSF and blood profile over time

Test (ref. range)	Day 1	Day 6	Day 14	Day 49
CSF leukocyte count (0-4) n/µl	31	11	21	83
Glucose (CSF/serum ratio) (>0.5)	0.26	0.49	0.45	0.51
CSF total protein (0-50) mg/dl	74	47	56	490
CSF lactate (1.2-2.1) mmol/l	2.1	2.8	3.5	4.4
Q-Albumin (CSF/serum ratio)	15.1	8.42	10.7	91.9
IgG Index (<0.7)	N.A.	N.A.	5.13	53.3
IgG-synthesis (%; Reiber formula)	N.A.	N.A.	10%	60%
β2-microglobulin (0-2) mg/dl	4.4	2.6	3.1	8.1
Oligoclonal IgG bands (CSF/serum type 1-5)	N.A.	N.A.	Type 2	Type 2
FACS-analysis T-cells (CD3+) B-cells (CD19+) CD4/CD8 ratio	72%* 0.02% 0.79	N.A.	93%* 0.05% 1.04	93%* 0% 0.96
CSF cytopathology (IHC)	N.A.	Predominant CD3+ T cells, no malignancy	T cells, monocytes, no malignancy	T cells, monocytes, no malignancy
Autoantibody screening (e.g., ANA, ANCA) in blood	N.A.	Negative	N.A.	Negative
Neuronal/ ganglioside/ paraneoplastic autoantibodies in CSF and blood	N.A.	Negative	Negative	Negative

Detailed representation of CSF findings, excluding individual parameters measured in blood, which are documented accordingly. Laboratory-specific reference ranges are indicated in parentheses. * T-cell differentiation without any pathological findings.

Abbreviations: CSF = cerebrospinal fluid; FACS = Fluorescence-Activated Cell Sorting; IHC = immunohistochemistry; N.A. = not applicable.

Table 3: Overview of the results of the electrophysiological examinations.

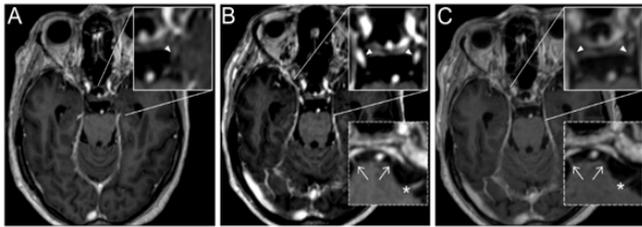
Date	NCS	EMG	Tibial nerve SSEP	MEPs of ant. muscle
Day 1 and Day 6	LE normal	Chronic neurogenic in LE muscle	Prolonged P40 latencies	Prolonged total and “central” motor latencies to the left
Day 14	Motor nerves LE: Axonal damage pattern	N.A.	Left: no response; Right: prolonged P40 latency	Cortical responses not reproducible, prolonged total and “central” motor latencies to the left
Between day 29 and day 205	Motor nerves LE: Axonal damage pattern progressively worsening	N.A.	N.A.	N.A.
Day 330	Predominantly axonal damage pattern of the motor nerves of LE (idem to previous)	Essentially normal in LE	Left: no response; Right: prolonged P40 latency (idem to previous)	Prolonged total and “central” motor latencies to the left (improved to previous)

Abbreviations: ant = anterior; EMG = electromyography; LE = lower extremity; MEPs = motor evoked potentials; N.A. = not applicable; NCS = nerve conduction studies; SSEP = somatosensory evoked potentials.

Citation: Dr. Luisa Krefß, Nurcan Üçeyler, Magnus Schindehütte, Christian Geis, Hayrettin Tumani, Claudia Sommer, Karl Georg Haeusler. Refractory Polyradiculoneuritis Responsive to Cyclophosphamide: A Case Report of a Patient with a History of Diffuse Large B Cell Lymphoma and CAR-T Cell Therapy. Archives of Clinical and Medical Case Reports. 10 (2026): 29-36.

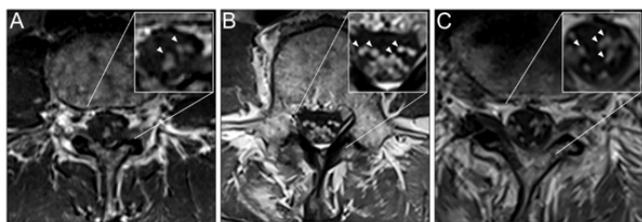
In magnetic resonance imaging (MRI) the left oculomotor nerve (Figure 1a) and fibers of the cauda equina (Figure 2a) showed contrast enhancement.

Figure 1: T1-weighted gadolinium-enhanced cranial MRI.



Day 2 (A) shows a radiocontrast enhancement of the left oculomotor n. (arrowhead). By day 48 (B) inflammation progresses with radiocontrast enhancement of oculomotor nerve bilaterally (arrowhead), abducens nerves bilaterally (arrows), and the left facial nerve (asterisk). On day 99 (C) radiocontrast enhancement of oculomotor nerve (arrowhead) and left facial nerve (asterisk) was regredient. Abbreviations: MRI = magnetic resonance imaging; n = nerve.

Figure 2: T1-weighted gadolinium-enhanced axial MRI at L4-5 level.



Gadolinium enhancement of the cauda equina fibers is visible on day 2 (A), with extensive involvement of the entire lumbosacral nerve roots on day 48 (B), followed by marked regression on day 99 (C). Abbreviations: MRI = magnetic resonance imaging.

Suspecting an inflammatory granulomatous disease, high dose i.v. glucocorticoids (1g methylprednisolone/day, days 1-3) were administered. Subsequent CSF findings (day 6: persistently elevated cell count [11/mm³]) and neurological symptoms did not improve. The patient was discharged home on day 10 after i.v. glucocorticoid treatment. An oral glucocorticoid tapering regimen was provided, followed by a recommendation for re-evaluation using MRI and lumbar puncture in four weeks.

Fourteen days after symptom onset, the patient returned to our emergency room with progressive radicular pain, rapidly progressing paraparesis, and sensory disturbances in the lower extremities, resulting in immobility. Neurological examination at this point in time is summarized in Table 1. Within days, the paraparesis proceeded to distal paraplegia and a proximal high-grade paresis, accompanied by severe ataxia with a lack of trunk control (Table 1). Radicular pain worsened despite hydromorphone, pregabalin, and amitriptyline treatment.

Repeated CSF analysis showed signs of cellular and humoral inflammatory activity combined with blood-CSF barrier dysfunction with increasing pathological values over disease course summarized in Table 3. Electrophysiological examination indicated predominantly axonal damage of the motor nerves of the lower extremities and prolonged latencies in SSEP and MEPs (detailed in Table 3). Brain MRI on day 48 revealed contrast enhancement in both oculomotor nerves, both abducens nerves, and left facial nerve (Figure 1b). Spinal MRI showed intense and widespread contrast enhancement of the cauda equina fibers (Figure 2b). Due to the predominant involvement of proximal nerve roots, a fascicular biopsy of the L5/S1 spinal root was performed. The procedure was conducted microsurgically under intraoperative monitoring, and notably, no new neurological deficits occurred postoperatively. This approach yielded decisive diagnostic information, revealing high-grade axonal loss with massive T-cell infiltration and no B-cell infiltration (Figure 3).

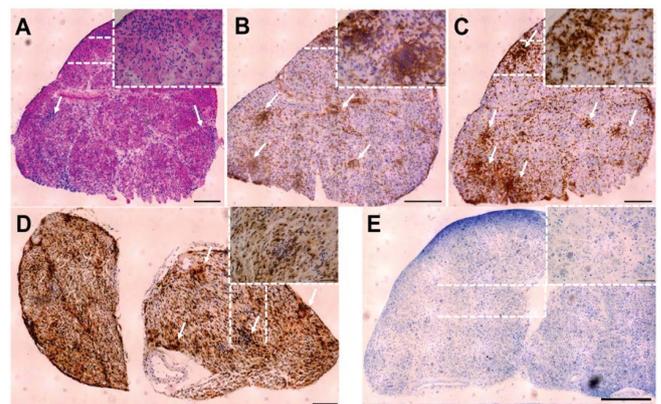


Figure 3: Fascicular biopsy L5/S1 on the left body side (day 57).

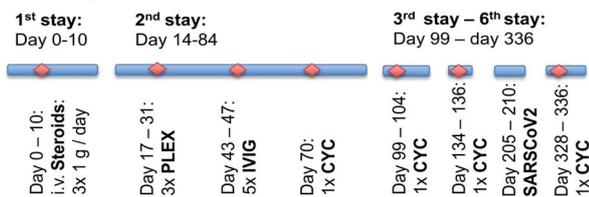
Representative photomicrographs illustrating inflammation in labeling CD3-immunoreactive T cells (using Leu4 as pan T-cell marker), cytotoxic T-cells (CD8), and macrophages (CD68). Fiber morphology was assessed using semithin sections, and cellular infiltration was visualized with H&E staining. H&E staining demonstrated a cell-rich fascicle with focal, perivascular, and diffuse infiltrates (A). At a dilution of 1:1000 (regular dilution not assessable due to high cellularity), Leu4 staining revealed massive T-cell infiltration (B), including prominent perivascular clusters. Most of these T cells were CD8-positive cytotoxic T cells (C). Macrophages (D) densely infiltrated the entire fascicle, particularly concentrated around perivascular T-cell clusters. The semithin section showed severe, slightly inhomogeneous fiber loss with signs of active axonal degeneration but no regeneration clusters or onion bulbs (E). Thickly myelinated fibers were predominantly affected, whereas thinly myelinated fibers were largely preserved. Thick arrows indicate areas of cellular infiltration (A), T cells (B–C), or macrophages (D). Asterisks denote examples of acute axonal degeneration; hash marks indicate preserved thickly myelinated fibers. Magnification: A–F ×40. Scale bars: Detailed sections = 50 µm, overview = 300 µm. Abbreviations: H&E = hematoxylin and eosin; L5/S1 = fifth lumbar vertebra/first sacral vertebra.

Seven cycles of PLEX stopped progression but did not improve clinical symptoms. A subsequent IVIg therapy (0.4 g/kg bodyweight/day for 5 days) slightly improved the oculomotor impairment and radicular pain (see Table 1 for details). The patient experienced complications while in hospital, including respiratory syncytial virus infection with bacterial superinfection, acute SARS-CoV-2 infection, port infection, and deep vein thrombosis of the left posterior tibial vein.

Based on personal experience with similar cases and the massive T-cell infiltration in fascicle biopsy, CYC administration (600 mg/m² body surface) was started on day 70, resulting in improvement of the paraparesis and further recovery from cranial nerve impairment from day 73 on (Table 1). CYC was well-tolerated, apart from temporary leukopenia. Following the first maintenance treatment with CYC (four weeks after the induction cycle) and a stay in a rehabilitation clinic, significant improvement was documented on day 99. Cranial nerve functionality had nearly recovered apart from mild anisocoria, lumbar pain was well controlled by using pregabalin and amitriptyline. Moreover, the patient was now able to stand independently, and sensory disturbances had regressed. Nerve conduction studies revealed isolated axonal damage in the tibial nerve. MRI scans showed lesser contrast media enhancement of cranial nerves (Figure 1c) and cauda fibers (Figure 2c) compared to the prior images. Outcome and follow-up: On day 134, cranial nerve functions were normal, the patient was pain free and able to walk several hundred meters without help, despite remaining paresthesias at the soles.

On day 328, slight gain instability and mild distal paresis of the left lower extremity were still present (Table 1) before the fourth and final administration of CYC. Four months post treatment, clinical status, evoked potentials, and MR imaging showed continuous improvement. The residual mild paresthesias did not interfere with activities of daily life. Our final disease classification was autoimmune, inflammatory polyradiculoneuritis with cranial nerve involvement. Intensive

Figure 4: Overview of hospital admissions and timing of major pharmacological interventions.



High-dose intravenous steroids were administered during the first stay (Day 0–10), followed by plasmapheresis (PLEX), intravenous immunoglobulins (IVIg), and multiple cycles of cyclophosphamide (CYC) during subsequent stays. A SARS-CoV-2 infection occurred between Day 205 and 210. Abbreviations: BW = body weight; CYC = Cyclophosphamide; i.v. intravenous; IVIg = intravenous immunoglobulin; PLEX = plasma exchange.

physical and occupational therapy continued throughout the treatment. A timeline of the hospital stays and administered pharmacological therapies is given in Figure 4.

During regular follow-up visits up to one year after discharge, no evidence of relapse of the B-cell neoplasia was found in laboratory tests, CSF analysis, or imaging. However, fourteen months after the onset of neurological symptoms, the patient developed a new recurrence of aggressive retroorbital DLBCL.

Discussion

This case report underscores potential challenges in managing polyradiculoneuritis in patients with hematologic malignancy and extensive prior immuno-chemotherapy. Our patient presented with a rapidly progressive, painful polyradiculoneuropathy with cranial nerve involvement and marked T-cell infiltration in fascicular nerve root biopsy, reflecting a profoundly immune-reactive state and suggesting secondary axonal pathology associated with inflammation. Of note, fascicular biopsy of a spinal nerve root is rarely performed in clinical practice but proved to be essential in this case. In contrast to a standard sural nerve biopsy, this approach enabled direct assessment of the affected proximal nerve tissue and guided diagnosis and treatment decisions.

Considering the medical history of our patient, a link between the polyradiculoneuritis and prior CAR-Tcell infusion needs to be discussed. Most immune effector cell-associated neurotoxicities, including ICANS, typically occur within days to weeks after infusion [15, 16, 18]. Commonly reported adverse effects are B cell depletion (aplasia), hypogammaglobulinemia, cytopenia, and infections [18, 19]. Information on long-term adverse effects of CAR-T cell infusion is sparse so far, and severe late-onset CAR-T cell related neurotoxicity has rarely been reported [18]. In the presented case with a CAR-T cell infusion 10 months prior to symptom onset, a direct causal relationship between polyradiculoneuritis and CAR-T cell therapy seems unlikely; thus, a coincidental autoimmune activation remains a plausible alternative. However, this group of patients is particularly at risk due to the repeated, aggressive immunotherapy and may be particularly susceptible to subsequent autoimmune reactions.

We further considered a paraneoplastic etiology as differential diagnosis [11, 20, 21]. However, no clinical evidence of malignancy was found during oncologic work-up until month 14, and the analyzed CSF as well as nerve root fascicle biopsy showed no malignant cells. Moreover, the marked and sustained clinical improvement following CYC argues against a paraneoplastic mechanism, as such responses are typically absent in cases with untreated or occult malignancy. SARS-CoV-2 and bacterial infections occurred later during the clinical course and could, in theory,

have modulated immune responses or mimicked some of the observed aspects. However, the onset of neurological symptoms clearly preceded these infections, and there was no evidence of direct infectious involvement of the nervous system (e.g., normal CSF microbiology, absence of systemic inflammation at onset). Moreover, the robust and sustained response to immunosuppressive therapy (CYC) argues for a primarily immune-mediated pathogenesis rather than a post-infectious or parainfectious mechanism. While a contributory role of these infections in shaping the broader immune milieu cannot be fully excluded, they are unlikely to be the primary trigger or driver of the polyradiculoneuritis in this case.

Given the presumed immune-mediated pathogenesis, the lack of improvement under initial therapies (glucocorticoids, PLEX, IVIg) necessitated escalation. The decision to administer CYC was based on the severe clinical course, strong T-cell infiltration in the fascicle biopsy, our prior clinical experience with similar immune-mediated neuropathies, and massive T-cell infiltration in fascicle biopsy. CYC exerts its immunosuppressive effect primarily by inducing apoptosis in activated, proliferating T cells and by modulating the balance between effector and regulatory T-cell populations [8]. This mechanism is particularly relevant in our case, given the marked T-cell infiltration observed in the fascicular biopsy. CYC therapy was followed by sustained clinical improvement and reduction of inflammatory activity on imaging and in CSF as also seen in other case series in different types of polyradiculoneuropathies [22, 23]. Although no randomized trials exist for CYC in this setting, its efficacy in chronic inflammatory demyelinating polyneuropathy and Guillain-Barré syndrome-like neuropathies has been demonstrated in case reports [24-27] and animal studies. Our findings support considering CYC in selected patients with therapy-refractory immune neuropathies with prominent T-cell involvement.

Our case underlines the need for heightened awareness of autoimmune neurotoxicity in patients with hematologic malignancies, particularly those exposed to multiple immune-directed therapies. The therapeutic approach should be guided by clinical severity, immune profiles, and histopathology when available.

Conclusion

The increasing use of immunotherapies in oncology introduces new challenges in managing neurological complications. In case of severe, therapy-refractory polyradiculoneuritis with signs of immune activation, CYC may represent a valuable treatment option, as it selectively targets activated T cells and suppresses pathogenic immune responses, particularly in T cell-driven inflammatory processes. Multidisciplinary collaboration and long-time surveillance of this vulnerable patient cohorts are essential. Further research is needed to elucidate pathophysiological

mechanisms and establish evidence-based treatment strategies.

List of Abbreviations

CAR = Chimeric antigen receptor; CRS = cytokine release syndrome; CSF = cerebrospinal fluid; CYC = cyclophosphamide; DLBCL = diffuse large B-cell lymphoma; FACS = Fluorescence-Activated Cell Sorting; ICANS = immune effector cell-associated neurotoxicity syndrome; i.v. intravenous; IVIg = intravenous immunoglobulin; MEPs = motor evoked potentials; MRI = magnetic resonance imaging; PLEX = plasma exchange; RTX = Rituximab; SSEP = somatosensory evoked potentials.

Consent for Publication

Written informed consent of publishing the clinical routinely collected data was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

Availability of Data and Material

All data generated or relevant for this case are presented within this published article. For any additional inquiries, please contact the corresponding author.

Competing Interests

The authors declare that they have no competing interests.

Funding

LK was funded by the Interdisciplinary Center for Clinical Research (project numbers: Z-2/CSP_22; Z-3 BC-18).

Authors Contributions

LK and KGH contributed to the conception of the case report, conducted the literature review, and drafted the manuscript. LK, KGH, NÜ were responsible for clinical management of the patient and supported data collection. All authors participated in the analysis and interpretation of the findings. MS interpreted imaging findings and provided radiological expertise for the case description. All authors reviewed, critically revised the case report, and approved the final version for submission.

Acknowledgements

We sincerely thank Prof. Guido Stoll for his expert consultation and treatment advice based on experience from comparable cases. Our special thanks go to the entire care team for their exceptional management of the patient, with special recognition to the nursing staff and physiotherapists. We also acknowledge the Department of Neurosurgery,

especially José Perez, for performing the fascicular biopsy, as key diagnostic element and the Department of Hematology and Oncology for their valuable support and co-assessment. Finally, we thank the patient for providing consent to publish this case.

References

1. Apiraksattayakul N, Jitprapaikulsan J, Sanpakit K, et al. Potential neurotoxicity associated with methotrexate. *Sci Rep* 14 (2024): 18548.
2. Brittain G, Roldan E, Alexander T, et al. The Role of Chimeric Antigen Receptor T-Cell Therapy in Immune-Mediated Neurological Diseases. *Ann Neurol* 96 (2024): 441-452.
3. Rałowska-Gmoch W, Koszewicz M, Łabuz-Roszak B, et al. Diagnostic criteria and therapeutic implications of rapid-onset demyelinating polyneuropathies. *Exp Mol Pathol* 140 (2024): 104942.
4. Shastri A, Al Aiyan A, Kishore U, et al. Immune-Mediated Neuropathies: Pathophysiology and Management. *Int J Mol Sci* 24 (2023).
5. Perrinjaquet C, Desbaillets N, Hottinger AF. Neurotoxicity associated with cancer immunotherapy: immune checkpoint inhibitors and chimeric antigen receptor T-cell therapy. *Curr Opin Neurol* 32 (2019): 500-510.
6. Doneddu PE, Nobile-Orazio E. Management of chronic inflammatory demyelinating polyradiculopathy. *Curr Opin Neurol* 31 (2018): 511-516.
7. Singhal S, Khanna R, Surendranath A, et al. Chronic Immune Sensory Polyradiculopathy (CISP): A Systematic Review of the Literature. *Neurol Int* 16 (2024): 1214-1222.
8. Xiang Q, Cao Y, Song Z, et al. Cyclophosphamide for Treatment of Refractory Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Systematic Review and Meta-analysis. *Clin Ther* 44 (2022): 1058-1070.
9. Dalakas MC, Rakocevic G, Salajegheh M, et al. Placebo-controlled trial of rituximab in IgM anti-myelin-associated glycoprotein antibody demyelinating neuropathy. *Ann Neurol* 65 (2009): 286-293.
10. Delios AM, Rosenblum M, Jakubowski AA, et al. Central and peripheral nervous system immune mediated demyelinating disease after allogeneic hemopoietic stem cell transplantation for hematologic disease. *J Neurooncol* 110 (2012): 251-256.
11. Ingber S, Buckstein R. Paraneoplastic lumbosacral axonal polyradiculopathy preceding the diagnosis of nodular lymphocyte predominant Hodgkin lymphoma: a case report. *Leuk Lymphoma* 49 (2008): 2009-2011.
12. Bompaire F, Birzu C, Bihan K, et al. Advances in treatments of patients with classical and emergent neurological toxicities of anticancer agents. *Rev Neurol (Paris)* 179 (2023): 405-416.
13. Okada K, Seki M, Yaguchi H, et al. Polyradiculoneuropathy induced by immune checkpoint inhibitors: a case series and review of the literature. *Journal of Neurology* 268 (2021): 680-688.
14. Jung S, Greiner J, von Harsdorf S, et al. Fatal late-onset CAR T-cell-mediated encephalitis after axicabtagene-ciclioleucel in a patient with large B-cell lymphoma. *Blood Adv* 5 (2021): 3789-3793.
15. Brudno JN, Kochenderfer JN. Recent advances in CAR T-cell toxicity: Mechanisms, manifestations and management. *Blood Rev* 34 (2019): 45-55.
16. Brudno JN, Kochenderfer JN. Toxicities of chimeric antigen receptor T cells: recognition and management. *Blood* 127 (2016): 3321-3330.
17. Abu-Shakra M, Buskila D, Ehrenfeld M, et al. Cancer and autoimmunity: autoimmune and rheumatic features in patients with malignancies. *Ann Rheum Dis* 60 (2001): 433-441.
18. Cappell KM, Kochenderfer JN. Long-term outcomes following CAR T cell therapy: what we know so far. *Nat Rev Clin Oncol* 20 (2023): 359-371.
19. Cordeiro A, Bezerra ED, Hirayama AV, et al. Late Events after Treatment with CD19-Targeted Chimeric Antigen Receptor Modified T Cells. *Biol Blood Marrow Transplant* 26 (2020): 26-33.
20. Koike H, Tanaka F, Sobue G. Paraneoplastic neuropathy: wide-ranging clinicopathological manifestations. *Curr Opin Neurol* 24 (2011): 504-510.
21. Pelosof LC, Gerber DE. Paraneoplastic syndromes: an approach to diagnosis and treatment. *Mayo Clin Proc* 85 (2010): 838-854.
22. Jarius S, Bräuninger S, Chung HY, et al. Inositol 1,4,5-trisphosphate receptor type 1 autoantibody (ITPR1-IgG/anti-Sj)-associated autoimmune cerebellar ataxia, encephalitis and peripheral neuropathy: review of the literature. *J Neuroinflammation* 19 (2022): 196.
23. Rajabally YA. Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Current Therapeutic Approaches and Future Outlooks. *Immunotargets Ther* 13 (2024): 99-110.
24. Howell NA, Arya S, Tai PC, et al. Guillain-Barré syndrome

as an early manifestation of angioimmunoblastic T-cell lymphoma. *BMJ Case Rep* 15(2022).

25. Mangano K, Dati G, Quattrocchi C, et al. Preventive and curative effects of cyclophosphamide in an animal model of Guillain Barré syndrome. *J Neuroimmunol* 196 (2008): 107-115.

26. Calixto OJ, Poveda S, Meneses-Toro MA, et al. Chronic

inflammatory demyelinating polyradiculoneuropathy in a patient with systemic lupus erythematosus without systemic activity. *Lupus* 32 (2023): 1123-1125.

27. van Laarhoven HW, Rooyer FA, van Engelen BG, et al. Guillain-Barré syndrome as presenting feature in a patient with lupus nephritis, with complete resolution after cyclophosphamide treatment. *Nephrol Dial Transplant* 16 (2001): 840-842.



This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC-BY\) license 4.0](https://creativecommons.org/licenses/by/4.0/)