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ХВДП-ПОДОБНАЯ ДИЗИММУННАЯ ДЕМИЕЛИНИЗИРУЮЩАЯ ПОЛИРАДИКУЛО- НЕЙРОПАТИЯ, АССОЦИИРОВАННАЯ С ТЕРАПИЕЙ БРЕНТУКСИМАБОМ (КЛИНИЧЕСКИЙ СЛУЧАЙ)

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Brentuximab Vedotin-Induced CIDP-Like Inflammatory Demyelinating Polyradiculo- neuropathy (Clinical Case Report)

Резюме

Брентуксимаб ведотин-индуцированная дизиммунная демиелинизирующая полирадикулонейропатия является редким нежелательным побочным явлением химиотерапии, связанным с патологическим влиянием препарата на функционирование гуморальной иммунной системы. Клиническая картина заболевания характеризуется прогрессирующим развитием вялого тетрапареза и чувствительных нарушений по полиневритическому типу, нередко приводящим к выраженному нарушению качества жизни и инвалидизации пациентов. Результаты нейрофизиологического обследования представлены генерализованным поражением моторных и сенсорных волокон периферических нервов первично демиелинизирующего характера с наличием блоков проведения по моторным волокнам нервов рук в нетипичных для компрессии местах и феномена 'sural sparing pattern'. Клинико-анамнестические данные и показатели объективных методов обследования сходны с клинико-нейрофизиологическим паттерном, наблюдаемым при острой или хронической воспалительных демиелинизирующих полирадикулонейропатиях. Важной особенностью заболевания является положительный ответ на стандартную патогенетическую терапию аналогично классическим дизиммунным полирадикулонейропатиям, которая включает глюкокортикостероиды, препараты иммуноглобулинов человека для внутривенного введения и методы экстракорпоральной гемокоррекции. Обратимость неврологических нарушений на фоне патогенетической иммуносупрессивной терапии обуславливает необходимость повышения осведомленности врачей о тактике ведения данной категории пациентов. Представленный клинический случай демонстрирует пример брентуксимаб-индуцированной дизиммунной демиелинизирующей полирадикулонейропатии с прогрессирующим развитием грубых моторных нарушений, которые в течение 6 месяцев привели к полной обездвиженности пациента. Своевременное назначение патогенетической терапии в оптимальном режиме дозирования, грамотная тактика ведения, подразумевающая медленное и постепенное снижение доз глюкокортикостероидных препаратов, обеспечивает благоприятный исход при рассматриваемой курабельной лекарственно-индуцированной полирадикулонейропатии.

Ключевые слова: брентуксимаб ведотин, химиотерапия, лимфома Ходжкина, хроническая воспалительная демиелинизирующая полирадикулонейропатия, ХВДП, иммуносупрессивная терапия, глюкокортикостероидные препараты, ГКС

Конфликт интересов

Соавтор статьи Супонева Н.А. является членом редакционной коллегии журнала «Архивъ внутренней медицины». Статья прошла принятую в журнале процедуру рецензирования. Супонева Н.А. не участвовала в принятии решения о публикации этой статьи. Об иных конфликтах интересов авторы не заявляли

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Abstract

Brentuximab vedotin-induced inflammatory demyelinating polyradiculoneuropathy is a rare chemotherapy side effect. The underlying cause of disease is thought to be pathological drug effects on the humoral immune system. The typical clinical picture is characterized by progressive development of flaccid tetraparesis and severe polyneuritic manifestations, which typically leads to disability and significant impairment in quality of life. Nerve conduction study results show evidence of generalized primary demyelination, including conduction block and sural sparing pattern. Clinical and anamnestic data and objective observational findings are similar to the clinical and neurophysiological pattern observed in acute or recurrent inflammatory demyelinating polyradiculoneuropathies. It is important to emphasize that brentuximab vedotin-induced inflammatory demyelinating polyradiculoneuropathy response to accepted in guidelines pathogenetic therapy, which includes corticosteroids, intravenous human immunoglobulin therapy and extracorporeal hemocorrection methods. Clinicians should be aware of treatment possibilities due to reversibility of neurological impairment through the pathogenetic immunosuppressive therapy. This clinical case represents a case of brentuximab-induced inflammatory demyelinating polyradiculoneuropathy with progressive development of severe motor impairment, which led to complete immobilization over 6 months. This clinical case report demonstrates optimal management strategy: timely administration of pathogenetic therapy with appropriate dosing, slow and gradual reduction in corticosteroid dosage. Competent management strategy ensures favorable outcome of the curable drug-induced polyradiculoneuropathy.

Key words: *brentuximab vedotin, chemotherapy, Hodgkin disease, chronic inflammatory demyelinating polyradiculoneuropathy, CIDP, immunosuppression therapy, glucocorticoids.*

Conflict of interests

Co-author of the article Suponeva N.A. is a member of the editorial board of the journal «The Russian Archives of Internal Medicine». The article passed the journal's peer review procedure Suponeva N.A. was not involved in the decision to publish this article. The authors did not declare any other conflicts of interest

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Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article «Brentuximab Vedotin-Induced CIDP-Like Inflammatory Demyelinating Polyradiculoneuropathy (Clinical Case Report)» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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BV — brentuximab vedotin, DDP — disimmune demyelinating polyradiculoneuropathy, RCNN — The Russian Centre of Neurology and Neurosciences, CIDP — chronic inflammatory demyelinating polyradiculoneuropathy, ENMG — electroneuromyography, CTCAE — Common Terminology Criteria for Adverse Events, MRC — Medical Research Council

Introduction

Peripheral neuropathy (PN) is one of the most common adverse side effects (ASE) of chemotherapy, impairing patients' quality of life, and is a frequent cause of cancer therapy adjustments. Chemotherapy-induced polyneuropathy is frequently reported as a side effect of drugs, the mechanism of action of which involves damage to microtubules in cancer cell cytoskeleton [1, 2]. Brentuximab vedotin (BV) is a drug conjugate, comprising three components: 1) chimeric monoclonal immunoglobulin G1 anti-CD30 receptor cAC10 antibody; CD30 receptor is a tumour necrosis factor receptor; 2) active substance — microtubule-targeting agent

monomethyl auristatin E; 3) a protease-cleavable linking agent, which links both compounds together. The activity of BV is based on its ability to bind to the cells, which express CD30 receptor, and internalise into cytoplasm of the active substance, which binds to tubulin, impairs microtubule function, and breaks the cell cycle, causing target cell death [3]. In August 2011, BV was registered in the USA; in Russia, the drug was registered in 2016 [4]. Currently, BV is used to treat Hodgkin lymphoma, peripheral CD30+ T-cell lymphoma and primary skin T-cell lymphoma, usually as a third- or fourth-line therapy, where other chemotherapy options or red bone marrow stem cell autologous transplantation

were inefficient [5]. Peripheral neuropathy is the most common ASE of BV therapy. In phase II clinical trials of the drug, the rate of PNs of various degrees of severity varies between 9% and 73%: in 41–44% of cases, sensory polyneuropathy was reported; while 9–11% were motor polyneuropathy [3,6–8]. It has been noted that 11–23% of patients had to interrupt BV therapy because of severe PN [3,6,7,9]. Later it was determined that BV-induced disimmune demyelinating polyradiculoneuropathy (DDP) can account for up to 14.6% of patients with BV-induced NP and up to 7.2% among all patients treated with BV [5].

The objective of this article is to describe a clinical case of disimmune demyelinating polyradiculoneuropathy associated with brentuximab vedotin therapy, and to compare the clinical and neurophysiological pattern of brentuximab-induced demyelinating neuropathy with globally reported cases, as well as with cases of classical chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).

Clinical Case Report

Patient N., born in 1994, sought help at the Russian Centre of Neurology and Neurosciences (RCNN) for the first time in October 2024, complaining of weakness and numbness in his limbs. His medical history shows that since 2019 the patient has been followed up by an oncohematologist for Hodgkin lymphoma, type 2 nodular sclerosis, stage IVB, with involvement of neck lymph nodes, mediastinum and lungs, with penetration through pericardium and pleural membrane. After his diagnosis, the patient underwent multiagent chemotherapy: in 2019 — BEACOPP 14 (bleomycin, etoposide, doxorubicin, cyclophosphamide, vincristine, procarbazine, and prednisolone), 2022 — Nivo-BeGEV (nivolumab in a combination with bendamustine, gemcitabine, and vinorelbine); as well as radiation therapy in the mediastinum area in 2019; red bone marrow stem cell autologous transplantation in April 2022; lymph node excision in 2023. The therapy did not cause any neurological complications. From July 2023 to May 2024 the patient was treated for cancer relapse with brentuximab vedotin and showed complete reported metabolic response to the therapy in September 2023. In December 2023 (six month into his BV therapy), the patient noticed gradually progressing weakness in his limbs. His condition progressed to complete immobility by June 2024. Follow-up examinations, including positron emission tomography and computed tomography (PET/CT), showed remission of the disease. At the local outpatient clinic, the patient was diagnosed with CIDP-like neuropathy, and in July 2024 he underwent a course of IV pulse therapy with

methylprednisolone (MP) 5 g, then oral MP 64 mg daily for a week, after that the MP dose was reduced to 36 mg for four months. Thus, at the time of his visit to RCNN in October 2024, the patient's daily MP dose was 28 mg. Three weeks of high-dose immunosuppressive therapy resulted in significant improvement in limb mobility: the patient was able to walk with support, get up and sit down unaided. The rate of recovery slowed down after rapid MP dose reduction; however, his condition remained stable.

At the time of the first visit to RCNN in October 2024, the neurological status was prevailed by distal flaccid tetraparesis with diffuse areflexia, hypotonia and muscle hypotrophy: 4 points on the MRC scale in proximal limbs; up to 1–2 points in hand and finger flexors; and up to 0–1 points in hand and finger extensors, interosseal muscle of hands, and feet extensors and flexors. Superficial and deep sensibility impairment (polyneuritic form) was classified as minor. In RCNN, the patient underwent stimulation electro-neuromyography (ENMG), which showed generalised, mostly motor, neural demyelinating impairment with possible conduction in motor fibres of hand nerves in sites, which are not common for neural compression (Table 1).

Clinical and medical history data, namely the temporal association between disease onset and BV therapy initiation, positive response to the immunosuppressive therapy by the time of the visit, as well as neurophysiological examination results allowed confirming the diagnosis “CIDP-like disimmune, mostly motor, demyelinating polyradiculoneuropathy, associated with the therapy with brentuximab vedotin”.

Due to persistent significant neurologic impairment with lower rates of recovery because of the rapid reduction in the dose of MP, glucocorticosteroid therapy was corrected: it was recommended to replace MP 28 mg daily with MP 48/24 mg, alternating the high and the low doses every other day for three months, and then gradually reducing first the low dose, then the high dose of the drug. After the GCS therapy was corrected, considerable positive changes were observed, and clinical symptoms started improving more rapidly: in July 2025 (one year after GCS therapy initiation), the patient's MP dose was reduced to 20 mg once every other day. Upon examination, distal mild to moderate tetraparesis was still observed: up to 4 points (MRC) in hand and finger flexors and up to 3–4 points in finger extensors and feet flexors/extensors. The patient did not need any support while walking; he was able to get up from the chair without the help of his hands and could stand on his toes and heels. Thus, glucocorticosteroid therapy helped changing the clinical pattern from complete immobility to distal mild to moderate tetraparesis. Similar to clinical

manifestations, neurophysiological examination results showed positive changes: improved amplitude and speed parameters, improvement in possible conduction in motor fibres of hand nerves (Table 2). When this case study was reported, the patient was still consulted by RCNN specialists in order to select a maintaining dose of methylprednisolone.

Discussion and literature review

Brentuximab-induced polyneuropathy is the most common adverse effect of BV therapy [3]. In the majority of reported cases, clinical presentation comprises a classical set of symptoms: damage to thick and thin sensitive fibres with the development of predominantly superficial sensitivity impairment; deep sensitivity is reduced less frequently (polyneuritic mild to moderate impairment with signs of primary axon involvement, as demonstrated by stimulation ENMG) [8,10]. It is assumed that axon damage is associated with immediate cytotoxic effects for microtubule functioning from monomethyl auristatin E, a component of BV [11].

Nevertheless, there are global reports of isolated cases of BV-associated disimmune demyelinating poly-

radiculoneuropathy. The first case of BV-induced DDP was reported in 2013 by Pastorelli F. et al. [12]; later, individual observations were published in 2017–2022 [2, 13, 14]. The largest review by Fargeot G. et al. (2020) is a retrospective analysis of three cases (0.4 %) of Guillain-Barre syndrome and 9 cases (1.3 %) of CIDP, which were verified among 700 patients treated with BV during 2013 to 2019 [15]. Later, a single-centre study by Matthys A. et al. (2024) reported 6 clinical cases (7 %) of disimmune NP in 83 patients treated with BV [5]. It is assumed that damages to the myelin sheath may be caused by aberrated immune response as a result of the impact by chimeric monoclonal immunoglobulin G1, i.e. the first component of BV, on CD30-expressing cells, similar to the drugs, which inhibit TNF [15,16]. Histologic examination results of patients with rapidly progressing BV-induced motor neuropathy demonstrate a mixed axonal and demyelinating pattern of the damage: sural nerve biopsy specimens show reduced sheathed fibre density, signs of wallerian degeneration and sporadic accumulation of regenerating fibres. Electron microscopy results show signs of damaged axon cytoskeleton, changes in orientation and overall number of microtubules, mostly in sheathed fibres [2,17].

Table 1. Nerve conduction study results of patient N., first visit in Russian Center of Neurology and Neurosciences (03.10.2024). Electromyography machine Keypoint, Dantec (Denmark).

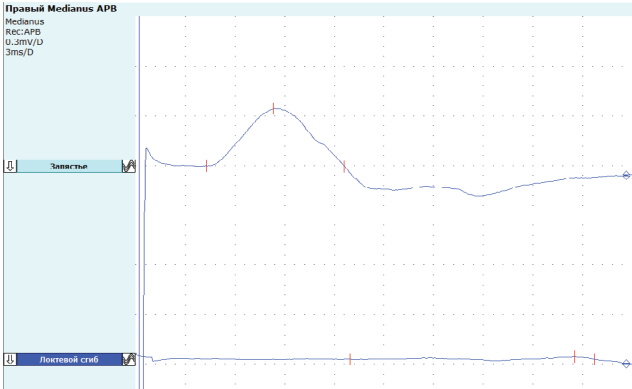
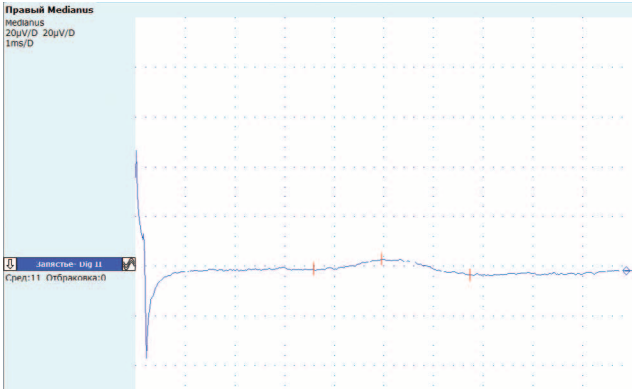
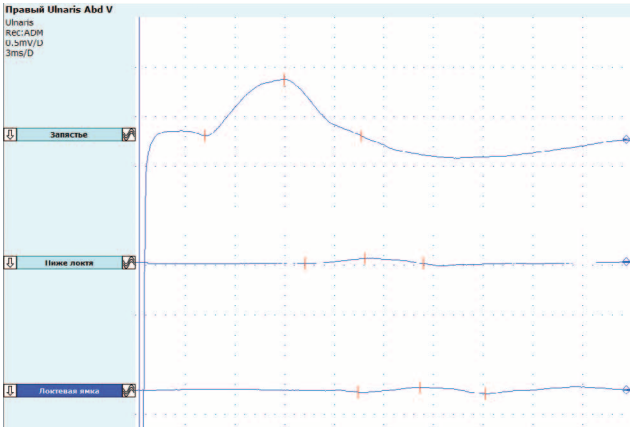
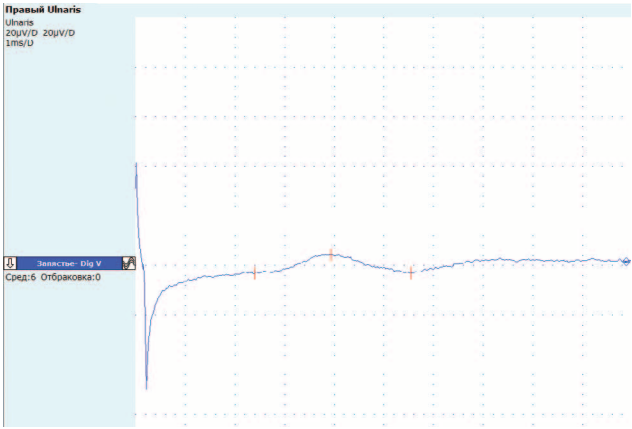
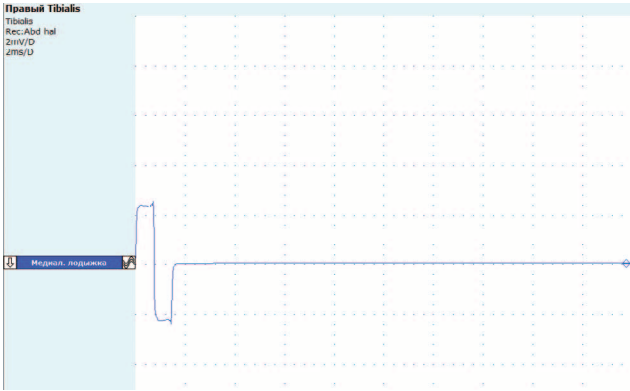
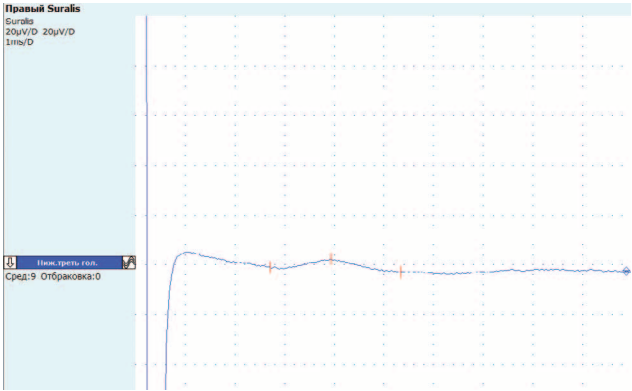
Nerve conduction study results of the right median nerve	
CMAP parameters (lead with m. APB dexter)	SNAP parameters (lead with dig. II)
	
CMAP latency, ms (N <3.5): 4.28	CNAP latency, ms (N <3.0): 3.58
CMAP amplitude, mV (N >5.0): 0.35	CNAP amplitude, µV (N >15.0): 5.0
Right median motor study shows significant temporal dispersion on 2 nd stimulation point (elbow)	Conduction velocity, m/s (N >50): 44.7
Distal CMAP duration: 8.3 (N <8.4)	
Proximal CMAP duration: 8.3 (N <8.4)	
Conduction velocity, m/s (N >50) (elbow-wrist segment): 25.5	—

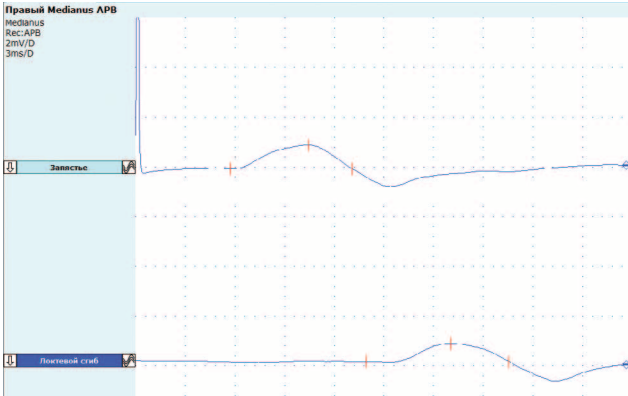
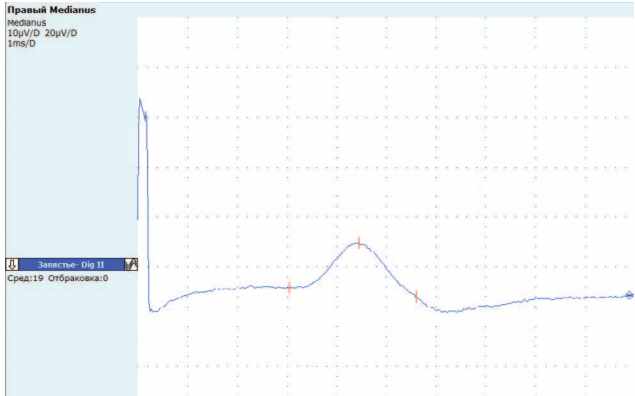
Table 1. (The End).

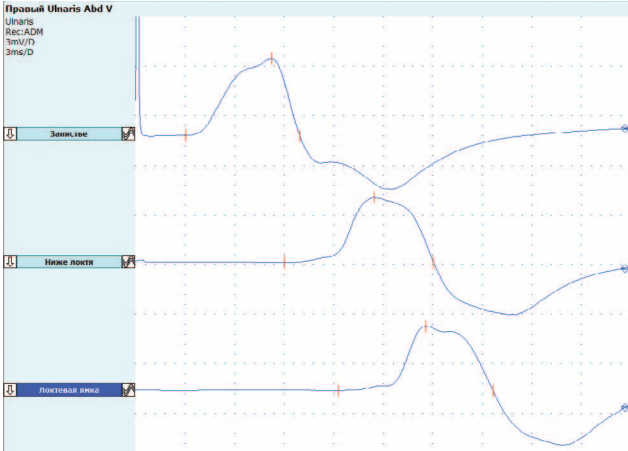
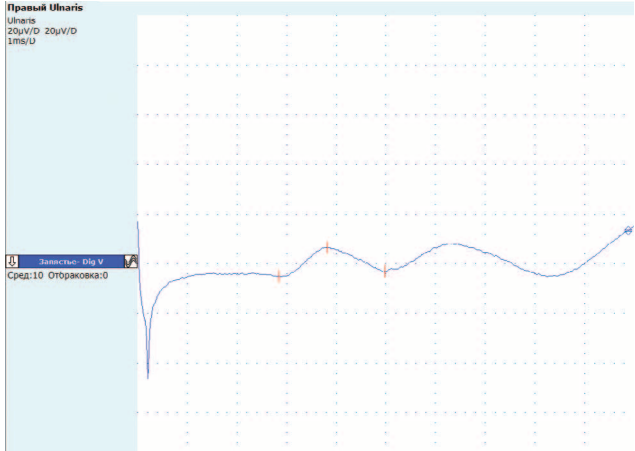
Nerve conduction study results of the right ulnar nerve	
CMAP parameters (lead with m. ADM dexter)	SNAP parameters (lead with dig. V)
	
CMAP latency, ms (N <3.0): 4.28	CNAP latency, ms (N <3.0): 3.58
CMAP amplitude, mV (N>6.0): 0.57	CNAP amplitude, µV (N>15.0): 7.6
Right ulnar motor study shows probable conduction block below elbow: 91 %	—
Conduction velocity, m/s (N>50) (below elbow-wrist and above elbow to below elbow segments): 33.2 — 40.6	Conduction velocity, m/s (N>50): 60.4

Nerve conduction study results of the right tibial nerve	Nerve conduction study results of the right sural nerve
CMAP parameters (lead with m. AH dexter)	SNAP parameters
	
Right tibial motor study showed no responses	SNAP latency, ms (N <4.0): 2.71
—	SNAP amplitude, µV (N>6.0): 4.2
—	Conduction velocity, m/s (N>40): 44.3

Note: ADM — abductor digiti minimi manus muscle; APB — abductor policis brevis muscle; AH — adductor hallucis; CMAP — compound muscle action potential; dig. — digitus; N — norm; SNAP — sensory nerve action potential

Table 2. Nerve conduction study results of patient N., dynamics in 8 months on steroid therapy (03.07.2024). Electromyography machine Keypoint, Dantec (Denmark)

Nerve conduction study results of the right median nerve	
CMAP parameters (lead with m. APB dexter)	SNAP parameters (lead with dig. II)
	
CMAP latency, ms (N <3.5): 5.72	SNAP latency, ms (N <3.0): 3.04
CMAP amplitude, mV (N >5.0): 0.96	SNAP amplitude, μV (N >15.0): 10.0
Conduction velocity, m/s (N >50) (elbow-wrist segment): 26.9	Conduction velocity, m/s (N >50): 47.7

Nerve conduction study results of the right ulnar nerve	
CMAP parameters (lead with m. ADM dexter)	SNAP parameters (lead with dig. V)
	
CMAP latency, ms (N <3.0): 3.05	SNAP latency, ms (N <3.0): 2.84
CMAP amplitude, mV (N >6.0): 4.6	SNAP amplitude, μV (N >15.0): 10.7
Conduction velocity, m/s (N >50) (below elbow-wrist and above elbow to below elbow segments): 36.9 — 42.7	Conduction velocity, m/s (N >50): 44.0

Note: ADM — abductor digiti minimi manus muscle; APB — abductor policis brevis muscle; CMAP — compound muscle action potential; dig. — digitus; N — norm; SNAP — sensory nerve action potential.

According to case studies and individual cases reported in global publications, there is a common clinical and neurophysiological pattern of BV-induced DDP [5,12–15]. The majority of publications report that disimmune BV-induced neuropathy is usually abrupt or subacute in onset; it starts from isolated sensory disorders, mostly paresthesias and neuropathic pain syndrome; then severe ataxia and motor deficit of various degrees of severity develop, which are more noticeable in distal muscles of the limbs. According to Fargeot G. et al. (2020), 73 % of patients required extra support for walking [15]; among the patients described by Matthys A. et al. (2024), complete immobility developed in one out of six patients [5]. Other reported symptoms are diffuse areflexia and impaired vibration sensitivity [5,12,13]. A chronic condition in the publication by Fargeot G. et al. (2020) was reported in 36 % of cases [15]. The studies do not indicate any clear predisposing factors of demyelinating polyradiculoneuropathy development in patients treated with BV. However, the publication by Matthys A. et al. (2024) reported potential impact of a higher cumulative BV dose on the development of disimmune NP [5]. It has been established that disimmune NP appears on the average 10–14 weeks after BV therapy initiation. Polyneuritic disorders start 2 to 36 weeks after BV therapy initiation [5, 15].

This case study is yet another example of BV-induced DDP, which manifested 24 weeks after BV therapy initiation. The condition was chronic; motor disorders progressed and were more prominent distally. In six months, the patient was completely immobile; it corresponds to grade 4 under the Common Terminology Criteria for Adverse Events (CTCAE). The distinctive characteristic of this case is prevailing motor disorders both at the onset and at the peak of clinical symptoms, as well as more prominent muscle involvement (hand and feet extensors).

According to study results, the data obtained during stimulation ENMG in patients with BV-induced DDP correspond to EDX criteria of CIDP and acute inflammatory demyelinating polyradiculoneuropathy (AIDP), i.e. primary demyelinating damage to peripheral nerves are present [5,15]. In 78 % of cases, the sural sparing pattern was observed (reduced amplitude of sensory potential of the sensory section of the median or ulnar nerve with preserved amplitude of sensory potential of the sural nerve) [15], which is a highly specific early marker of AIDP and is observed in CIDP [18]. Of note, in a later observational study by Pezullo L. et al (2025), patients treated with BV also had the sural sparing pattern, despite primary axial changes seen during stimulation ENMG [10]. The common characteristic of BV-induced DDP, noted by researchers, was failure to register any F-waves from hand and feet muscles, as well as conduction in motor

fibres of hand nerves in sites, which are not common for neural compression [5,12–14]. ENMG results in this case study correspond to published data: predominant involvement of motor fibres of peripheral nerves; demyelinating damage; sural sparing pattern.

The differential diagnosis of BV-induced NP includes chemotherapy-induced toxic axonal NPs, paraproteinemic demyelinating NP, as well as paraneoplastic NP and neurolymphomatosis, given that this patient category is treated with BV for lymphatic cancers [5]. Thus, various studies may be required, including anti-neuronal antibody screening, nerve ultrasound under the Ultrasound Pattern Sum Score (UPSS) protocol [19] or brachial plexus magnetic resonance imaging, PET/CT, serum protein electrophoresis, and 24-hour urine test with immunofixation [5].

The severity of BV-induced polyneuropathy is dose-dependant, therefore, recovery depends on BV therapy duration and mode [5]. According to the guidelines on the management of patients treated with BV, who develop CTCAE grade 2–3 NP, BV therapy should be interrupted until clinical symptoms improve to CTCAE grade 1 or resolve completely; then the therapy should be resumed at a lower dose [9, 20]. CTCAE grade 4 NP requires BV therapy discontinuation [9,20]. Based on the therapy results reported in case studies, it can be concluded that BV-induced disimmune NP responds to the standard pathogenetic therapy, similar to classical CIDP [5,12–15]. The sample data by Fargeot G. et al. (2020) demonstrated that in 82 % of cases, the neurological status improved after the IV human immunoglobulin (IVHI) therapy (2 g/kg) together with BV therapy discontinuation; however, complete recovery has not been observed in any of the cases [15]. In the publication by Matthys A. et al. (2024), IVHI helped achieve significant improvement in two cases; in two cases, positive changes were observed with the use of glucocorticosteroid therapy; also, there was one case of spontaneous recovery once BV dose had been reduced [5]. Apparently, pathogenetic therapy duration is determined on a case-by-case basis and depends on the clinical effect, similar to classical variants of disimmune neuropathy. The question of repeated initiation of BV after disimmune NP is resolved remains unanswered. In addition to clinical improvements after pathogenetic therapy, neurophysiological characteristics (as shown by ENMG) also improve: in some clinical cases, motor and sensory responses reverted to their normal levels [12,14]. This case study confirms the overall trend of the positive impact of the standard pathogenetic therapy on BV-induced DDP: pulse therapy with methylprednisolone, then high-dose oral MP therapy with gradual controlled MP dose reduction helped achieving positive changes — from complete immobility to mild distal tetraparesis within a year. It is

worth noting that gradual, slow reduction of glucocorticosteroid therapy ensures higher rates of regression of neurological deficit and optimal return of the quality of life [21].

Conclusion

Brentuximab vedotin-induced disimmune demyelinating polyradiculoneuropathy is a severe debilitating complication of chemotherapy, the course and clinical characteristics of which can resemble acute or chronic inflammatory demyelinating polyradiculoneuropathy. Both acute and chronic muscle weakness, ambulation disorders and sensory ataxia are clinical red flags of possible disimmune origin of impairment and the indication for stimulation electroneuromyography in order to identify the pattern of peripheral nerve damage. Medical professionals following up patients with haematological and neurological conditions should be aware of this complication of brentuximab vedotin therapy, because prompt discontinuation of the drug and initiation of immunosuppressive pathogenetic therapy are the optimal management approach for such patients, ensuring favourable outcome and recovery.

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