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GUILLAIN-BARRÉ SYNDROME IN A FEMALE PATIENT WITH MULTIPLE SCLEROSIS IN THE SETTING OF PROLONGED EXPOSURE TO ANTI-CD20 THERAPY: A CLINICAL EXPERIENCE

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СИНДРОМ ГІЄНА — БАРРЕ У ХВОРОЇ НА РОЗСІЯНИЙ СКЛЕРОЗ НА ТЛІ ТРИВАЛОЇ АНТИ-CD20 ТЕРАПІЇ: КЛІНІЧНИЙ ДОСВІД

Keywords: *multiple sclerosis; Guillain-Barré syndrome; demyelination; peripheral nervous system; autoimmune diseases*

Background. Multiple sclerosis (MS) and Guillain-Barré syndrome (GBS) are autoimmune demyelinating disorders affecting different parts of the nervous system: the central nervous system in MS and the peripheral nervous system in GBS. Despite partially overlapping immunological and genetic mechanisms, the coexistence of these two conditions in a single patient is extremely rare.

Objective. To analyse a case of Guillain-Barré syndrome developing in a patient with multiple sclerosis during long-term anti-CD20 therapy.

Materials and Methods. A retrospective analysis of clinical data, laboratory findings, neuroimaging results and neurophysiological studies was performed in a patient with multiple sclerosis who developed Guillain-Barré syndrome during prolonged anti-CD20 treatment.

Results. The patient had a five-year history of multiple sclerosis and had been receiving anti-CD20 therapy due to high disease activity. Acute deterioration was characterised by fever, bulbar dysfunction and progressive limb weakness resulting in peripheral tetraparesis. Initial cerebrospinal fluid examination revealed neutrophilic pleocytosis suggesting infection; subsequent analysis demonstrated lymphocytic predominance and infectious aetiology was not confirmed. Magnetic resonance imaging showed no evidence of active demyelinating lesions. Electroneuromyography demonstrated axonal involvement of peripheral nerves supporting the diagnosis of Guillain-Barré syndrome.

Conclusions. The coexistence of multiple sclerosis and Guillain-Barré syndrome is rare but should be considered in clinical practice. Patients receiving long-term anti-CD20 therapy require careful monitoring for potential infectious and autoimmune complications.

Ключові слова: *розсіяний склероз; синдром Гієна — Барре; демієлінізація; периферична нервова система; аутоімунні захворювання*

Актуальність. Розсіяний склероз (РС) та синдром Гієна — Барре (СГБ) є аутоімунними демієлінізуючими захворюваннями, що уражають різні відділи нервової системи: при РС — центральну, при СГБ — периферичну. Попри частково спільні імунологічні та генетичні механізми патогенезу, поєднання цих патологій в одного пацієнта трапляється вкрай рідко, а в науковій літературі описано лише поодинокі випадки.

Мета роботи. Проаналізувати клінічний випадок розвитку синдрому Гієна — Барре у пацієнтки з розсіяним склерозом на тлі тривалої терапії анти-CD20 препаратами.

Матеріали та методи. Проведено ретроспективний аналіз клінічних даних, результатів лабораторних, нейровізуалізаційних та нейрофізіологічних досліджень пацієнтки з РС, у якої під час тривалої терапії анти-CD20 препаратами розвинувся синдром Гієна — Барре.

Результати. Пацієнтка з п'ятирічним анамнезом РС отримувала терапію анти-CD20 препаратами у зв'язку з високою активністю захворювання. Гостре погіршення стану супроводжувалося гарячкою, бульбарними розладами та прогресивним зниженням м'язової сили в кінцівках із формуванням периферичного тетрапарезу. У спинномозковій рідині первинно виявлено нейтрофільний плеоцитоз, що зумовило підозру на інфекційний процес; надалі визначено лімфоцитарний характер ліквору, інфекційну етіологію не підтверджено. За даними МРТ-ознак активності демієлінізуючого процесу не виявлено. Електронейроміографія виявила аксональне ураження периферичних нервів, що дало змогу встановити діагноз синдром Гієна — Барре.

Висновки. Поєднання розсіяного склерозу та синдрому Гієна — Барре є рідкісним, проте можливим клінічним феноменом. Пацієнти з РС, які тривало отримують терапію анти-CD20 препаратами, потребують ретельного клінічного спостереження щодо можливого розвитку інфекційних та аутоімунних ускладнень.

Guillain-Barré syndrome is an acute autoimmune demyelinating polyneuropathy that affects the peripheral nervous system and, in most cases, develops after a prior infection [1]. The most common clinical form of the disease is acute inflammatory demyelinating polyradiculoneuropathy [2]. Multiple sclerosis, in turn, is a chronic immune-mediated inflammatory demyelinating disease of the central nervous system that develops as a result of a complex interaction between genetic and environmental factors [3].

Although these conditions affect different parts of the nervous system, they share similar underlying immunopathological mechanisms that lead to demyelination and axonal damage. Activation of nonspecific inflammatory cascades and autoantibody-mediated responses is believed to contribute to the development of demyelinating processes in both the central and peripheral nervous systems [1; 4]. The shared individual biomarkers and metabolic alterations observed in multiple sclerosis and Guillain-Barré syndrome further support the possible uniformity of pathogenetic mechanisms underlying these disorders [2; 3; 6].

The coexistence of multiple sclerosis and Guillain-Barré syndrome in a single patient is extremely rare and has been reported in contemporary scientific literature only as isolated clinical observations [7; 8]. It is believed that such a combination may suggest a generalized demyelinating process involving different levels of the nervous system, likely sharing common immunopathogenetic mechanisms [9;10].

Objective: to review a clinical case of Guillain-Barré syndrome developing in a female patient with multiple sclerosis in the setting of prolonged exposure to anti-CD20 therapy.

A retrospective analysis was performed, involving clinical findings, laboratory results, and neuroimaging and neurophysiological investigations in a female patient with a confirmed diagnosis of multiple sclerosis who developed Guillain-Barré syndrome in the setting of prolonged anti-CD20 therapy.

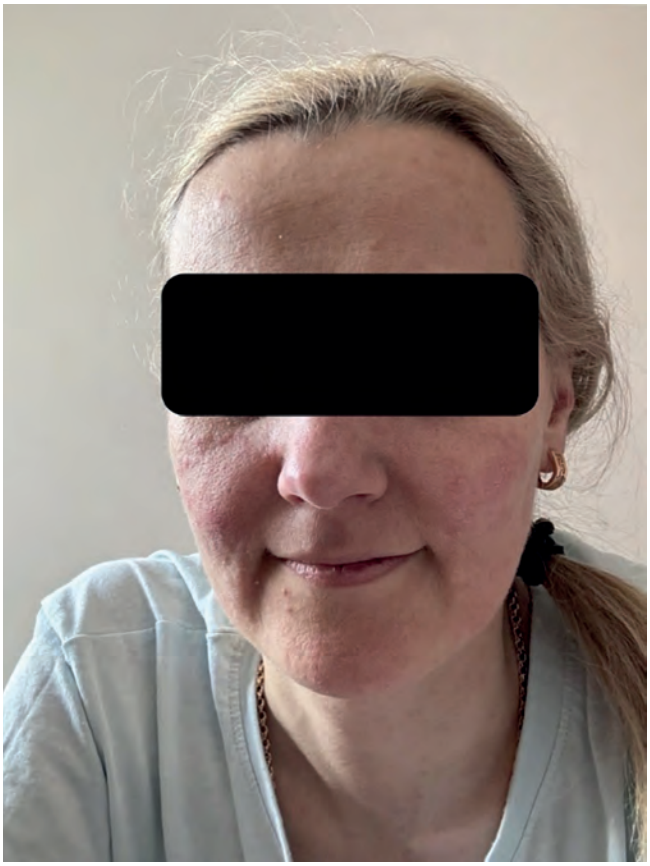
In 2016, patient O. (born in 1981) was diagnosed with multiple sclerosis, which was subsequently characterized by escalating clinical and radiological activity of the disease with development of a persistent neurological deficit.

The onset of the disease manifested as weakness in the upper limb. At that time, brain magnetic resonance imaging (MRI) revealed focal demyelinating lesions. The patient received a course of intravenous pulse therapy with methylprednisolone, which resulted in clinical improvement.

Two years after disease onset (2018), the patient's condition deteriorated and was considered an exacerbation: gait impairment worsened, the patient developed weakness in her lower limbs with subsequent formation of lower spastic paraparesis, and cerebellar disorders became apparent.

In 2020, two exacerbations of the disease were documented, which triggered initiation of high-efficacy anti-CD20 therapy, the last dose of which was administered in early June, 2024.

An acute deterioration of the general condition developed after visiting a swimming pool on August 22, 2024, when the patient experienced an increase in body temperature up to 38 °C accompanied by chills. On August 23, 2024, general weakness, psychomotor slowing, generalized tremor, headache, fever, and skin rash (Figure) appeared.



Skin rash observed in the patient at the onset of the acute phase of the disease

On August 24, 2024 the patient was hospitalized with suspected pneumonia. Rapid antigen test for SARS-CoV-2 was negative. No signs of an acute cerebrovascular event were detected on a brain CT scan dated August 24, 2024. Chest X-ray dated August 24, 2024 showed no focal infiltrative changes. Brain MRI showed focal lesions without signs of activity.

Neurological examination demonstrated positive meningeal signs, and cerebrospinal fluid test showed marked neutrophilic pleocytosis, with a cell count of 310 cells/μL, predominantly consisting of neutrophils. With a preliminary diagnosis of meningitis, the patient was transferred for further intensive treatment to Lviv Regional Clinical Infectious Diseases Hospital, where a diagnosis of meningoencephalitis was established and treatment in an intensive care unit was initiated. The patient was hospitalized in the above institution from August 24, 2024 to September 4, 2024.

The neurological status in the intensive care unit was characterized by bulbar dysfunction and signs of cranial nerve involvement, including weakened convergence, convergent strabismus of the left eye, limited abduction of the left eyeball, flattening of the right nasolabial fold, and deviation of the tongue to the right; horizontal nystagmus was later observed. Reflexes were heterogeneous: tendon reflexes in the upper extremities were decreased, whereas those in the lower extremities were increased with enlargement of reflexogenic fields;

abdominal reflexes were absent. Meningeal signs were strongly positive, and nuchal rigidity was subsequently noted; the Babinski sign was described as inconsistent on both sides. According to the evolution of neurological findings as of August 26, 2024, the working diagnoses included acute meningoencephalitis and cerebral edema. On repeat examination on August 28, 2024, meningitis in the setting of multiple sclerosis with lower spastic paraparesis was considered, accompanied by progression of lower spastic paraparesis and the development of hypertonia in the muscles of the upper extremities.

Laboratory findings during the acute phase reflected an inflammatory response, signs of anemia, and transient changes in biochemical parameters.

Cerebrospinal fluid findings demonstrated a biphasic pattern. Initial assessment performed at the First Territorial Medical Association on August 24, 2024 revealed neutrophilic pleocytosis, with a total cell count of 310 cells/μL and neutrophil predominance. In contrast, cerebrospinal fluid analysis performed at the Lviv Regional Clinical Infectious Diseases Hospital on August 26, 2024 showed clear, colorless fluid with lymphocytic pleocytosis of 15 cells/μL, protein level of 0.33 g/L, and glucose concentration of 4.2 mmol/L. A repeated examination on August 30, 2024 demonstrated an increase in cell count to 18 cells/μL (lymphocytic predominance) along with a decrease in glucose concentration to 3.1 mmol/L, while the protein level remained unchanged at 0.33 g/L.

Polymerase chain reaction test of the cerebrospinal fluid performed on September 04, 2024 did not detect Epstein-Barr virus DNA, cytomegalovirus DNA, herpes simplex virus types 1, 2, or 6 DNA, or *Toxoplasma gondii* DNA; serum immunoglobulin M antibodies to *Toxoplasma gondii* were negative.

Brain MRI on August 27, 2024 revealed findings consistent with a demyelinating disease of MS type in remission, with no evidence of disease activity; no other structural brain abnormalities were identified. Magnetic resonance imaging of the cervical and thoracic spine with contrast enhancement performed on September 03, 2024 also did not demonstrate any active demyelinating lesions; however, magnetic resonance signs of a demyelinating process in the spinal cord at the levels of C_1—C_7 and Th_3—Th_{12} without signs of activity were noted, along with degenerative changes of the cervical and thoracic spine and a protrusion of the intervertebral disc at the Th_7—Th_8 level.

Electroneuromyography performed on September 2, 2024 demonstrated axonal involvement of the left peroneal nerve with a decrease in distal function to 47 % of the lower limit of normal, while function at the level of the upper third of the lower leg remained preserved; distal parameters on the right corresponded to the lower limit of normal. No signs of involvement of other examined peripheral nerves of the upper and lower extremities were detected.

In the setting of acute disease, the patient developed a pronounced motor deficit described in her clinical records as peripheral tetraparesis with predominant involvement of the right extremities: active movements in the right upper limb were absent, movements in the lower extremities were markedly limited, and movements in the left upper limb were partially preserved. In parallel with the neurological deficits, dysphagia requiring nasogastric tube feeding was observed.

On admission, the patient complained of weakness in the extremities and difficulty speaking. Objectively, the patient was conscious and oriented; the general condition was assessed as severe; no skin rash was observed at the time of examination. Vesicular breath sounds were present without wheezing; respiratory rate was 17 per minute and oxygen saturation was 98 %. Hemodynamic findings were stable: blood pressure 100/70 mmHg, pulse rate 60 per minute, heart sounds were clear and rhythmic, with no murmurs detected. The abdomen was soft and non-tender; the liver and spleen were not enlarged; Murphy's punch sign (Pasternatsky's sign) was negative; no peripheral edema was present; urination was spontaneous; the thyroid gland was not enlarged.

Neurological examination revealed limited contact, slowed and quiet speech, and a weakened voice (dysphonia). Pupils were equal with preserved light reflex; convergence was weakened; ocular movements were full; a fine horizontal nystagmus was noted on lateral gaze. The face was symmetrical and the tongue was midline. Swallowing remained impaired, and feeding was continued via a nasogastric tube. Motor deficit was pronounced: active movements in the right lower limb were absent; movements in the left lower limb and right upper limb were markedly limited (only finger gestures were available); movements in the left upper limb were preserved but accompanied by impaired coordination. Tendon reflexes were diminished and abdominal reflexes were absent; no abnormal plantar responses or meningeal signs were detected at the time of examination.

Based on the results of the conducted examinations, the patient was diagnosed with multiple sclerosis, relapsing-remitting course, remission phase during anti-CD20 therapy; sequelae of a previously developed infectious-allergic polyradiculoneuropathy (Guillain-Barré syndrome) with pronounced tetraparesis and loss of limb function.

The patient had laboratory evidence of persistent anemia with microcytic features and fluctuations in inflammatory activity, as well as changes characteristic of a urological complication and pronounced leukopenia and lymphocytopenia (Grade 3 toxicity).

In-patient treatment for the acute phase of Guillain-Barré syndrome in this patient lasted four months. After that, she had several courses of rehabilitation therapy and pharmacological restorative therapy, which continued for an additional 6 months.

At present, the patient is experiencing a clinical improvement, with progressively less intense pain and partial recovery of motor function: the patient is able to sit independently and take up to 5–6 steps. However, dysuric symptoms persist in the form of urinary frequency.

The coexistence of multiple sclerosis and Guillain-Barré syndrome in clinical practice is very rare. However, current evidence suggests that simultaneous demyelinating involvement of both the central and peripheral nervous systems is not always a coincidental finding. In some cases, these conditions may share common underlying immunopathogenetic mechanisms [4; 8].

Both disorders are autoimmune demyelinating processes where activation of the inflammatory immune response plays a central role in development, involving T- and B-lymphocytes, macrophages, and the production of autoantibodies. This, in turn, leads to myelin sheath damage, axonal injury, and the development of persistent neurological deficits [1]. Data from experimental models, particularly autoimmune encephalomyelitis and autoimmune optic neuritis, have demonstrated the possibility of cross-reactive immune responses between myelin antigens of the central and peripheral nervous systems. Such mechanisms support the hypothesis of so-called immune "epitope spreading", where an abnormal immune response may extend between different parts of the nervous system [5].

Although central and peripheral myelin differ in their protein composition, they may share common antigenic determinants capable of inducing a systemic autoimmune response. This potentially creates conditions for the spread of the pathological process from the central nervous system to the peripheral nervous system, or vice versa, which may clinically manifest as the development of peripheral demyelinating neuropathy in patients with an already established diagnosis of multiple sclerosis [11].

In recent years, particular attention has been paid to the role of the B-cell response in the pathogenesis of demyelinating diseases. Current therapeutic approaches for multiple sclerosis, including the use of monoclonal antibodies targeting CD20, are aimed at the depletion of B-lymphocytes as key effectors of autoimmune inflammation [12—14]. At the same time, prolonged immunomodulatory therapy in the setting of B-cell depletion may influence the balance between regulatory and effector immune response mechanisms, thereby modifying the course of concomitant autoimmune processes, including demyelinating neuropathies [15; 16]. Reported cases of anti-CD20 therapy use in refractory forms of chronic inflammatory demyelinating polyneuropathy further highlight the complex immunoregulatory effects of anti-CD20 therapy on the peripheral nervous system [17].

At the same time, studies of demyelination biomarkers have demonstrated similarities in the metabolic and immune profiles of cerebrospinal fluid in patients with multiple sclerosis and Guillain-Barré syndrome, which may reflect shared pathophysiological mechanisms of myelin damage [6]. Genetic and immunological factors may also play a role in the development of both conditions, particularly through mechanisms involving Fc receptor regulation and macrophage activation [9].

Thus, although multiple sclerosis is traditionally regarded as a demyelinating disorder of the central nervous system and Guillain-Barré syndrome as a disorder of the peripheral nervous system, accumulated clinical and experimental evidence suggests a potential immunopathogenetic link between these two diseases.

In the presented clinical case, the development of Guillain-Barré syndrome in a female patient with a long-standing course of multiple sclerosis receiving pathogenetic immunomodulatory therapy is described. Initially, the clinical presentation was interpreted as a possible infectious involvement of the central nervous system. However, subsequent instrumental and neurophysiological examinations confirmed a combined demyelinating lesion of both the central and peripheral nervous systems.

The presented case demonstrates that the development of peripheral demyelinating neuropathy in patients with multiple sclerosis may clinically mimic both an exacerbation of the underlying disease and an acute infectious process, thereby significantly complicating timely diagnosis. In such situations, the use of additional diagnostic methods, particularly electroneuromyography, is essential to clarify the level of nervous system involvement.

The obtained clinical findings emphasize the importance of careful differential diagnosis between exacerbation of multiple sclerosis, infectious involvement of the nervous system, and the development of autoimmune polyneuropathy, especially in patients receiving modern immunomodulatory therapy.

Therefore, the coexistence of multiple sclerosis and Guillain-Barré syndrome, despite its rarity, should be considered in clinical practice when new neurological symptoms appear in patients with demyelinating diseases of the nervous system, particularly in the setting of prolonged administration of disease-modifying therapy with a pronounced immunosuppressive effect.

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Limitations of the study

It should be noted that the presented clinical case has several limitations, primarily related to the retrospective nature of the analysis and the description of the disease course in a single patient. Such a design does not allow for definitive conclusions regarding causal relationships between the development of peripheral demyelinating neuropathy and the course of multiple sclerosis or the administered immunomodulatory therapy.

In addition, the initial clinical presentation was considered suggestive of a possible infectious involvement of the central nervous system, which could have influenced the timing of the final diagnosis and the sequence of therapeutic interventions. The absence of a control group also limits the ability to assess the impact of individual factors on the disease course and treatment outcomes.

Given the rarity of the coexistence of multiple sclerosis and Guillain-Barré syndrome, the obtained data are descriptive in nature and require further confirmation in larger prospective studies. Generalization of the results to a broader patient population should therefore be undertaken with caution.

Prospects for further research

The obtained clinical data indicate the need for further investigation of the mechanisms underlying combined demyelinating involvement of the central and peripheral nervous systems in patients with multiple sclerosis. Considering the complexity of differential diagnosis between exacerbation of the underlying disease, infectious processes, and the development of autoimmune polyneuropathy, the identification of clinical, laboratory, and neurophysiological markers that could facilitate earlier recognition of Guillain-Barré syndrome in this patient population appears to be a promising direction.

Particular attention should be paid to assessing the impact of long-term immunomodulatory therapy on the course of concomitant autoimmune processes, as well as identifying potential risk factors for the development of peripheral demyelinating lesions during treatment with monoclonal antibodies. Prospective multicenter studies with extended follow-up periods may help to clarify the frequency of such combined conditions, their clinical characteristics, and possible pathogenetic associations, which could have practical implications for timely diagnosis and optimal treatment strategies.

Conflict of interest

The authors have submitted the completed International Committee of Medical Journal Editors (ICMJE) Conflict of Interest Disclosure Form, available at: http://www.icmje.org/conflicts-of-interest/. The authors declare the absence of any actual or potential conflict of interest related to the results of this work with pharmaceutical companies, manufacturers of biomedical devices, or other organizations whose products, services, or financial support may be associated with the subject of the submitted materials or that may have sponsored the conducted research.

Ethics statement

The authors confirm that the preparation of this article was carried out using data from the patient's primary medical records. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki of the World Medical Association on medical research involving human subjects, Directive 86/609 of the European Community on the participation of humans in biomedical research, as well as the requirements of the Order of the Ministry of Health of Ukraine No. 690 dated September 23, 2009.

Use of Generative Artificial Intelligence

The authors acknowledge the use of generative artificial intelligence tools during the preparation of this manuscript. In accordance with the Generative Artificial Intelligence Delegation Taxonomy (GAIDeT, 2025), tasks related to proofreading, editing, and translation were delegated under full human supervision. ChatGPT-5 (OpenAI, version released in June 2025) was used for this purpose. All outputs generated by this tool were carefully reviewed, edited, and approved by the author, who assumes full responsibility for the content and conclusions of the publication. Generative artificial intelligence tools are not listed as authors and bear no responsibility for the final results. This declaration does not apply to the use of basic tools for grammar, spelling, or reference formatting.

Data availability statement

The authors confirm that the study is based on the results of original clinical observations that were systematized and analyzed by the author. The primary data include summarized clinical indicators of the patient, laboratory test results, examination protocols, and obtained quantitative characteristics. All materials are archived by the researchers and may be provided upon reasonable request to the corresponding author, in compliance with confidentiality requirements and ethical standards.

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