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A clinical case

Atypical Miller Fisher Syndrome as a Mimic of Stroke: A Case Report

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Abstract

Certain neurological conditions may closely resemble acute stroke in their clinical presentation, complicating the diagnostic process and potentially delaying appropriate treatment. This report presents a clinical case that illustrates the diagnostic difficulties encountered at the onset of Miller Fisher syndrome (MFS), a rare variant of Guillain-Barré syndrome (GBS). A 42-year old male patient initially exhibited symptoms commonly associated with ischemic stroke in the posterior circulation, including unilateral ptosis, double vision, dizziness, difficulty swallowing, reduced voice strength, ataxia, and limb weakness. Despite these signs, diffusion-weighted magnetic resonance imaging (DWI-MRI) revealed no evidence of acute cerebral ischemia. The development of ophthalmoplegia and absence of reflexes raised suspicion of an alternative neurological disorder. Subsequent serological testing confirmed the presence of anti-GQ1b antibodies, and treatment with intravenous immunoglobulins was initiated, resulting in significant clinical improvement. This case highlights the importance of maintaining a broad differential diagnosis in patients with atypical neurological symptoms and emphasizes the role of comprehensive evaluation in avoiding misdiagnosis. Awareness of such presentations can aid in earlier recognition of Miller Fisher syndrome and improve outcomes for individuals with rare autoimmune neuropathies.

Key words: stroke mimics, Guillain-Barre syndrome, Miller Fisher syndrome, GQ1b antibodies.

1. Introduction

According to scientific data, stroke mimics are identified in up to 30% of patients admitted to stroke units [1]. Common stroke mimics include epilepsy, migraine, oncological conditions, functional disorders, and metabolic disturbances [2]. Between 4% and 20% of patients with stroke mimics receive thrombolytic therapy [3], and delay or error in diagnosis can lead to irreversible complications [1].

One such condition is Guillain-Barre Syndrome, an autoimmune disease in which the body's immune cells attack peripheral nerves. Current data identify eight subtypes of GBS one of them is Miller Fisher Syndrome, accounting for only 1–5% of all GBS cases globally [4]. MFS is more prevalent in Asian countries and is rare among Caucasian populations. Reports from Taiwan and Japan indicate that MFS constitutes up to 20–26% of adult GBS cases. The diagnosis of MFS is based on a clinical triad of ophthalmoplegia, ataxia, and areflexia [5]. A preceding infectious illness, such as an upper respiratory tract infection or gastroenteritis, is a common triggering factor. The most frequent pathogens include *Campylobacter jejuni* and *Haemophilus influenzae*, although others, such as *Mycoplasma pneumonia*e and *cytomegalovirus*, have also been implicated [6,7]. Unlike the classic ascending paralysis seen in typical forms of GBS, MFS often demonstrates a "top-down" progression of symptoms. Its pathogenesis involves molecular mimicry, leading to demyelination of nerve fibers and stimulation of the immune system to produce antibodies targeting gangliosides, predominantly GQ1b [8,9]. With supportive care and immunotherapy, the prognosis for MFS is generally favorable, though residual symptoms may persist in some cases [5,8].

This report describes the clinical course of a patient with MFS symptoms mimicking posterior circulation stroke. This case aimed to assess stroke mimics in a patient presenting with clinical features suggestive of stroke.

2. Case presentation

A 42-year-old man presented to the emergency department with a sudden onset of left eyelid drooping over 4 hours. He was examined by a physician, and a neurological examination showed only left-sided ptosis and low blood potassium levels and the patient explained that due to type 2 diabetes, he had been on a very strict diet for over two weeks. In the emergency department, he was given an intravenous potassium solution, his condition improved 100%, and the patient insisted on going home on his own. A day later, in the evening, the patient felt dizziness and weakness; he decided to rest and went to bed. During the night, he woke up with the following symptoms: breathing difficulties, nasal voice, unsteadiness in walking, he could not drink water, all the water poured out through his nose and mouth, and his wife called an ambulance.

In the emergency room of the stroke center of the city hospital, a neurological examination showed: dizziness, left-sided ptosis, ophthalmoplegia, diplopia on both sides, hyperesthesia of 1st and 2nd branches of the trigeminal nerve on both sides, dysphonia, dysphagia, general weakness, sensory ataxia, slightly decreased muscle tone and tendon reflexes in arms and legs bilaterally, meningeal symptoms and Babinski's symptom negative on both sides. His observation parameters on admission were stable with a heart rate of 90 beats per minute, blood pressure of 130/70 mmHg, respiratory rate of 18 breaths per minute, and temperature of 36.8 degrees Celsius.

History of life and illness: The patient reported that he had been suffering from type 2 diabetes mellitus for more than 5 years; very rarely, an increase in blood pressure to 150/90 mm Hg was observed against a background of stress. A month ago, he suffered from a cold with a temperature of up to 38.4°C for 3 days, took paracetamol, and within a week returned to work and daily routine. Works as a driver and has a family of two children.

A computed tomography (CT) scan of the brain was performed in the emergency room and showed signs

of chronic cerebral ischemia. A brain magnetic resonance imaging (MRI) could not be performed due to the patient's claustrophobia, but after persuasion, only a DWI MRI was performed, which showed no changes. Myasthenia gravis was also suspected and the patient was tested with neostigmine methylsulfate 1.0 ml subcutaneously with negative results. Changes in blood tests shows: increasing of white blood cell count (14.51*10^9/l) and glucose level (13.24 mmol/L). Blood analyses of thyroid functions and antibodies specific to HIV were normal. Lumbar puncture was not performed due to patient's phobia. The patient was admitted to the intensive care unit where treatment was initiated according to the ischemic stroke protocol.

Additional studies were conducted in the following days: neuromyography and CT scan of the chest organs - without pathologies; antibodies to acetylcholine receptor (0.141nmol/l) - negative. After some time, when the symptoms of claustrophobia were relieved by an antidepressant (amitriptyline), an MRI of the brain and cervical spine with contrast was performed, where signs of microangiopathy on the Fazekas scale 1 and a right-sided paramedian-median herniated intervertebral disc C5-C6 (3.4 mm) were found. No more pronounced lesions of the brain and spinal cord were detected. Then a blood test for diagnosis of inflammatory polyneuritis was performed (antibodies to gangliosides GM1, GM2, GM3, GM4, GD1a, GD1b, GD2, GD3, GT1a, GT1b, GQ1b), and result was positive for GQ1b+ and GT1a+ antibodies. In the neurological status, ophthalmoplegia, diplopia, dysphonia, areflexia and sensory ataxia remained, and deep sensory disturbances appeared in the form of a decrease in vibration sensitivity in the arms and legs. Superficial sensitivity is completely preserved. After that, the treatment protocol was changed to the MFS. Ophthalmoplegia, diplopia, and dysphagia persisted in the neurological status, areflexia and sensory ataxia increased, and deep sensory disturbances appeared in the form of decreased vibration sensitivity in the arms and legs.

Plasmapheresis procedures were performed in the hospital (3 times), and then patient was discharged. After 2,5 months, his condition improved; only partial internal ophthalmoplegia and diplopia in the left eye remained from the neurological deficit; all other symptoms were completely restored, however patient cannot return to work as a driver. We continue to monitor patient and hope see full recovery of all symptoms of the disease.

3. Discussion

Posterior circulation strokes, accounting for approximately 20–30% of all strokes, are more prone to misdiagnosis compared to anterior and middle circulation strokes [3]. This is largely because the posterior cerebral artery supplies critical regions such as the brainstem and occipital cortex, and its dysfunction frequently results in symptoms such as vertigo, diplopia, dysarthria, dysphagia, balance disturbances, ataxia, and visual field deficits. Similar clinical presentations can also occur with non-vascular conditions, including brain tumors, toxic or metabolic disorders, psychiatric conditions, migraines, seizures, and demyelinating diseases [10].

According to scientific data, dysphagia occurs in up to 80% of stroke cases [12]. In contrast, dysphagia and

One such condition is MFS. Diagnostic confirmation of MFS typically relies on its characteristic triad: ataxia, ophthalmoplegia, and areflexia. However, the acute onset of symptoms, atypical presentations such as dysphagia and dysphonia, along with computed tomography findings and the aforementioned triad, initially led to a misdiagnosis of stroke [5,11]. Following thorough clinical evaluation and essential tests, stroke was excluded as the diagnosis. A review of the literature revealed no previously reported cases of MFS mimicking a posterior circulation stroke in Kazakhstan.

The patient presented with dysphagia; a symptom predominantly associated with stroke.
dysphonia are observed in only about 2% of MFS patients. Studies suggest that the underlying mechanism

in such cases involves autoimmune neuropathy, specifically anti-GQ1b IgG antibodies targeting cranial nerves IX and X, leading to these rare symptoms [13].

The patient's areflexia was associated with concurrent type 2 diabetes mellitus. Elevated glucose levels of up to 13.2% indicated uncontrolled diabetes, underscoring the role of chronic hyperglycemia in weakening the peripheral nervous system and increasing susceptibility to autoimmune damage [14]. Additional symptoms of MFS, such as ataxia and ophthalmoplegia, can also mimic stroke. However, the absence of lateralization in MFS distinguishes it from posterior circulation stroke [15]. In this case, the patient initially presented with unilateral ptosis, which progressed to bilateral paralysis, a hallmark of MFS that often manifests as symmetric bilateral ophthalmoplegia [7].

The onset of the disease followed an upper respiratory tract infection one month earlier, supporting the infectious etiology commonly associated with MFS. Literature reviews indicate that over 90% of GBS patients report preceding upper respiratory or gastrointestinal symptoms. Campylobacter jejuni, the leading cause of acute bacterial gastroenteritis, is implicated in about 30% of cases [8].

The patient underwent DWI MRI, which revealed no abnormalities. This aligns with findings that neuroimaging typically does not show changes in the brain in MFS. However, in the diagnosis of acute ischemic stroke, MRI has a much higher sensitivity, ranging from 80–95% [5].

The diagnosis of MFS was ultimately confirmed through serological testing, which identified positive GQ1b+ and GT1a+ antibodies. Research highlights that GQ1b antibodies are highly specific for MFS and are detected in 90% of cases. These autoantibodies target epitopes abundantly expressed on cranial nerves III, IV, and VI, causing the characteristic ophthalmoplegia of MFS [6]. In Kazakhstan, this test is available only as a paid service, which limits its accessibility and likely leaves many patients undiagnosed due to financial constraints.

The recommended treatments for MFS include intravenous immunoglobulin and plasmapheresis. In most cases, the prognosis is favorable, though residual symptoms may persist. According to studies, complete resolution of symptoms is typically observed within six months to one year, with 96% of patients achieving full recovery [5,8].

4. Conclusions

Given the diverse presentation of stroke symptoms, it is crucial to emphasize the importance of differential diagnosis in patients presenting with stroke-like features. Awareness of rare stroke mimicking syndromes significantly reduces the risk of misdiagnosis and helps prevent irreversible complications. MFS should be strongly considered when ataxia, areflexia, and ophthalmoplegia are present. However, clinical cases exist where the disease initially manifests with unilateral ptosis, dysphonia, dysphagia, and profound sensory deficits. Accurate confirmation of MFS requires blood testing for GQ1b autoantibodies.

Conflicts of interest. The authors declare no conflicts of interest.

Author contributions: Conceptualization - Z.A. and A.J.; methodology - Z.A., A.T. and A.J.; validation - Z.A. and A.T.; formal analysis - Z.A., A.T, A.Sh. and A.J.; writing (original and draft preparation) - Z.A., A.T., and A.Sh.; writing (review and editing) - Z.A., A.T., A.Sh. and A.J.

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Инсультті еліктейтін атипиялық Миллер Фишер синдромы: Клиникалық жағдай

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Түйіндеме

Инсультті имитациялайтын неврологиялық бұзылыстар ұқсас клиникалық симптомдармен сипатталады және уақытылы диагноз қоюды қиындатып, жағымсыз клиникалық нәтиженің даму қаупін арттырады.

Ұсынылып отырған клиникалық жағдай - Гийен–Барре синдромының сирек кездесетін түрі болып табылатын Миллер–Фишер синдромының дебютінде дифференциалды диагноз қоюдағы қиындықтарды айқындайды. Біз 42 жастағы ер адамды бақылау нәтижесінде, аурудың басталуы артқы қанайналым бассейнінің ишемиялық инсультына тән келесі симптомдармен байқалатындығын анықтадық: біржақты птоз, диплопия, бас айналу, дисфагия, гипофония, атаксия және аяқ-қол әлсіздігі. Жүргізілген кешенді тексеру диффузиямен өлшенген бейнелері бар магниттік-резонанстық томографияны қамтыды, нәтижесінде жедел ишемия белгілері анықталмады. Офтальмоплегия мен арефлексия секілді қосымша симптомдардың пайда болуы өзге этиологиялы патологияға күдік тудырды. GQ1b антигеніне қарсы антиденелердің бар екенін серологиялық зерттеу растады, нәтижесінде көктамыр ішілік иммуноглобулиндермен ем басталып, оң терапиялық әсер байқалды. Бұл клиникалық жағдай атипиялық неврологиялық көріністер нәтижесінде клиникалық сергектіктің және кеңейтілген диагностикалық ізденістің маңыздылығын көрсетеді. Ұқсас бақылауларды талдау Миллер–Фишер синдромының жедел ми қанайналымының бұзылысы ретінде қате қабылдануы мүмкін екенін байқатады. Мұндай жағдайларды сипаттау диагностикалық қателіктердің жиілігін төмендетуге және сирек кездесетін аутоиммундық нейропатиялары бар науқастарға көрсетілетін медициналық көмектің сапасын арттыруға мүмкіндік береді.

Түйін сөздер: инсульт мимиктері, Гийен-Барре синдромы, Миллер Фишер синдромы, Gq1b антиденелер.

Атипичный синдром Миллера Фишера, имитирующий инсульт: Клинический случай

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Резюме

Неврологические расстройства, имитирующие инсульт, могут проявляться сходной клинической симптоматикой, затрудняя своевременную диагностику и увеличивая риск неблагоприятного исхода.

Представленный клинический случай подчеркивает сложности дифференциальной диагностики при дебюте синдрома Миллера–Фишера - редкой формы синдрома Гийена–Барре. Мы наблюдали 42-летнего мужчину, у которого заболевание началось с симптомов, типичных для ишемического инсульта в бассейне задней циркуляции: односторонний птоз, диплопия, головокружение, дисфагия, гипофония, атаксия и слабость в конечностях. Комплексное обследование включало магнитно-резонансную томографию с диффузионно-взвешенным изображением, которая не выявила признаков острой ишемии. С появлением таких симптомов как офтальмоплегия и арефлексия была заподозрена альтернативная патология. Серологическое

исследование подтвердило наличие антител к GQ1b, после чего было начато лечение внутривенными иммуноглобулинами, приведшее к положительной терапевтической динамике. Данный случай подчёркивает необходимость клинической настороженности и расширенного диагностического поиска при атипичной неврологической симптоматике. Анализ аналогичных наблюдений демонстрирует, что синдром Миллера–Фишера может маскироваться под острое нарушение мозгового кровообращения. Описание подобных случаев способствует снижению частоты диагностических ошибок и повышению качества оказания медицинской помощи пациентам с редкими аутоиммунными нейропатиями.

Ключевые слова: мимики инсульта, синдром Гийена-Барре, синдром Миллера Фишера, GQ1b антитела.