Open Journal of
Clinical & Medical
Case Reports

Volume 5 (2019)
Issue 7

ISSN: 2379-1039

The case of Guillian-Barre syndrome in pregnancy

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Abstract

Miller-Fisher syndrome is a rare form of Guillian-Barre syndrome (GBS). The female, 34-years old, patient with signs of severe hyperemesis of gravidarum demonstrates the development of Miller-Fisher syndrome in the second trimester of pregnancy (the fourth parity). The morbidity in such condition is about 5-10%. It is considerable, that one third of pregnant patients needs respiratory support in the period of the disease. The paper depicts this case with description of diagnostic approach and pathogenetic treatment. Epidemiology of this nosology, clinical picture and diagnostic criteria were discussed too.

Keywords

Guillian-Barre syndrome; polyneuropathy; Miller-Fisher syndrome; complications in pregnancy

Introduction

Miller-Fisher syndrome – is A rare form of Guillian-Barre syndrome, which pertains to inflammatory polyneuropathies. In Miller-Fisher syndrome because of autoimmune aggression the peripheral nerve impairment, ataxia and oculomotor impairment occurs. In severe cases tetraparesis and paresis of respiratory muscles may accede. The causes of GBS development are often unclear, however, it was reported, that disease may occur after viral, bacterial (for example, mycoplasmatic) infections or vaccinations.

The Morbidity of Guillian-Barre syndrome vary from 0,8 to 4 percent/100 000. In Russia part of Miller-Fisher syndrome among all forms of GBS is about 2-5 percent [1,2]. In Eastern Asia the amount of GBS forms with cranial nerves involvement reaches 15-20 percent. The mortality of GBS, on average, is about 10 percent. The morbidity of GBS among pregnant women is equal to common population, but this nosology increases maternal and perinatal mortality [4].

The diagnosis is based on clinical examination, neurophysiological and laboratory data. For verification of diagnosis The WHO criteria are used routinely, but Brighton criteria of 2011are valid for use [5]. The Specific immunological marker of Miller-Fisher syndrome is autoantibody against glikolipids (GQIb). This marker may be revealed in serum of 90 percent of patients with Miller-Fisher syndrome.

Case Presentation

Patient V, 36-years-old was referred to the neurological department from infectious hospital, where she had admitted with suspicion of viral hepatitis in October of 2017. At the time of admission at neurological department the patient was in the second trimester of pregnancy (17th week). The patient suffered from hyperemesis gravidarum. This pregnancy is fourth, the previous were accompanied with hyperemesis too. During the first trimester considerable elevation of transaminases was revealed. So, she had admitted to infectious hospital with suspicion of viral hepatitis. After proper investigation HIV infection and viral hepatitis were excluded. For three days she complained of weakness of facial muscles, double vision and some problems with articulation and then she was referred to neurological department.

Common physical examination revealed mild enlargement of liver and moderate tachycardia. No heart murmur, chest rales, rash, vitiligo, skin pigmentation, or peripheral edema was detected. The patient was conscious and alert with normal mental status. Neurological examination on admission revealed bilateral prosoparesis (more significant on the right side), external ophtalmoparesis, moderate dysarthria, depression of knee reflexes and absent ankle reflexes, mild weakness in lower extremities and gait impairment with disturbance to perform the knee-heel-shin maneuver. There was loss of temperature perception, but vibratory perception was decreased in her feet. There were no meningeal signs.

Obstetric examination revealed the height of the uterine fundus corresponding to the stage of gestation with a single fetus. The liquor volume was normal. The fetal heart beat was present. Common laboratory tests were normal, but moderate elevation of transaminases and LDH was found. Several serologic studies were obtained, including a normal thyroid-stimulating hormone (TSH) level. The patient strictly refused to lumbar puncture. MRI scans of the brain were performed with no pathological signs were observed. Also EMG investigation was performed with the following result: distal motor demyelinating polyneuropathy with greater involvement of the lower extremities. Her vital capacity was normal.

Accordingly with clinical picture and investigations (EMG) the acute inflammatory polyneuropathy (Miller-isher syndrome) was diagnosed.

Treatment: The patient was treated with a course of plasmapheresis (amount of exchange is about 2500 ml for one procedure). Before this thiamine was prescribed without any improvement. During the course development of symptoms stabilized. Then regress of oculomotor impairment, improvement of dysarthria, prosoparesis was observed. The patient was charged out on 20 – week pregnancy. The full regress of ataxia was observed in one month. The clinical picture coincides with WHO characteristic of Miller-Fisher syndrome in 1990 [2,6]. The diagnosis was also supported by EMG investigation. So, it is possible to use Brighton criteria for verification [5].

Discussion

The case of demyelinating polyneuropathy was described the first time in 1834 by James Wardrope. The detailed clinical picture and hyperproteinemia in CSF was described in 1916 by Georges Guillain, Jean-

Alexandre Barre and Andre Strohl [1]. The concept of Guillain–Barré syndrome (GBS) significantly changed in the 1990s due to the recognition of acute motor axonal neuropathy (AMAN) as an axonal subtype of GBS. It is reported that acute inflammatory demyelinating polyneuropathy (AIDP) is frequent and acute motor axonal neuropathy (AMAN) is rare in European countries. In the same time, motor axonal neuropathy is common for Asia and South America [3]. The Miller-Fisher syndrome is more frequent for Eastern Asia than for European countries. The men suffer from all forms of Guillian-Barre syndrome a little more often than women (1,7:1). It is considered about age distribution, that two peaks of morbidity are exist: among 15-35 years old patients and among 50-75 years old too [2].

Guillian-Barre syndrome most commonly develops in the second part of pregnancy: 13% of cases occurs in the first trimester, 40% - in the second, and it is about 47% of GBS cases among pregnant women develops in the third trimester. Cases of development GBS syndrome during hyperemesis gravidarum with an elevation of transaminases are described [6]. However, available data are insufficient to consider this pathology as a risk factor of Guillain-Barre syndrome. It is considered, that gestosis with preeclampsia is the risk factor of Guillain-Barre syndrome development during all life time [7]. The clinical picture was notable for oculomotor impairment and ataxia coincided with one-sided prosoparesis. It is remarkable, that preeclampsia is the risk factor of Bell's palsy development in the third trimester of pregnancy and postpartum period [8].

It is not reported about any teratogenic influence of Guillian-Barre syndrome to fetum development. The way of delivery shoud be recommended in accordance with obstetric indications (there is no specific recommendations for pregnant women with Guillain-Barre syndrome).

Perinatal survival with anamnesis of GBS in time of pregnancy overlaps 96 percent [9]. It is probable, that poor outcomes of pregnancy are due to prenatal infection (for example, CMV) [10]. It is not reported about any teratogenic influence of Guillian-Barre syndrome to fetum development. The way of delivery should be recommended in accordance with obstetric indications (there is no specific recommendations for pregnant women with Guillain-Barre syndrome) [2,9]. However, in this choice clinician should pay attention to the fact that pregnant patients with Guillain-Barre syndrome need respiratory support two times more frequently than common patients with GBS.

Therapeutic approaches for pregnant patients with Guillain-Barre syndrome have no considerable differences from common recommendations: pathogenic therapy includes plasmapheresis and IVIG infusions [11]. On that point, no negative influence in these methods for fetum was described. However, IVIG transfusions are more eligible because plasmapheresis may cause some changes in volume, that especially undesirable in pregnancy. To perform plasmapheresis additional precautions are necessary: fibrinogen level before the start of the procedure and monitoring of calcium during plasma exchange [11].

It is especially important for therapeutic strategy that glucocorticoids were excluded from the all schemes of pathogenetic therapy. Besides multiple side effects it was considered that patients with GBS who received glucocorticoids get more serious resident neurologic deficit [2,12]. Adequate therapeutic strategy with IVIG or plasmapheresis perfomed well in time improves outcomes.

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Manuscript Information: Received: March 21, 2019; Accepted: May 13, 2019; Published: May 15, 2019

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Citation: Bedenko A. The case of Guillian-Barre syndrome in pregnancy. Open J Clin Med Case Rep. 2019; 1544.