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OPHTHALMOPLEGIA, DYSPHONIA AND TETRAPARESIS DUE TO GUILLAIN-BARRE'S SYNDROME IN PREGNANT AT 14 WEEKS OF GESTATION: CASE REPORT
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OPHTHALMOPLEGIA, DYSPHONIA AND TETRAPARESIS DUE TO GUILLAIN-BARRE'S SYNDROME IN PREGNANT AT 14 WEEKS OF GESTATION: CASE REPORT

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SUMMARY

Guillain-Barre's syndrome (GBS) or inflammatory/post-infectious acute polyradiculoneuropathy is due to demyelination of nerves, causing a progressive paresis or paralysis. It usually begins in the legs and sometimes goes up to the respiratory muscles and cranial nerves.
The exact mechanism of GBS occurrance is still unclear. An autoimmune disease is the assumption with the largest consensus to date. Autoantibodies related to a viral or bacterial infection in the days or weeks before the onset of symptoms damage the myelin sheaths of nerve fibers. These infections can range from a simple cold with sore throat to gastrointestinal disorders. In very rare cases, it is also recognised that the use of some drugs (streptokinase, Captopril ...) can cause GBS. The gestation is a relative immunological rest period in which autoimmune pathologies have, in their majority, a true clinical lull related to the acceptance of the embryo and the foetus by the woman's body. The onset of the GBS in the period is exceptional. We report a case of paralysis of the nerve VI accompanied by dysphonia and tetraparesis due to GBS in a pregnant of 24 year old with 14 weeks of gestation.

INTRODUCTION

GBS also called inflammatory or post-infectious acute polyradiculoneuropathy is characterized by a progressive paresis or paralysis. The first signs appear in the legs often with a gradual ascent towards the chest and cranial nerves (1-3). It affects both sexes, with a slight male predominance, children are rarely achieved (4, 5). In most cases, patient recover their physical abilities after 6 to 12 months (1, 6, 7).

In this pathology, the myelin surrounding nerve fibers is impaired (demyelination). This has the consequence slower transmission of nerve signals. The exact cause of GBS is unknown, but probably an autoimmune demyelination origin. The exact mechanism is still not known, but it is currently accepted that an overreaction of the immune defenses against infection is the cause. In fact, about two-thirds of person with GBS have suffered from a viral or bacterial infection in the days or weeks before the onset of symptoms. These infections can range from a common cold with sore throat through gastrointestinal disorders. Exceptionally, the outbreak of the syndrome may also be related to the use of some drugs (streptokinase, Captopril ...) (1, 6-9).

The gestation is a <<relative rest immunological period >> during which autoimmune pathologies have, in their majority, a real clinical lull linked to acceptance of the embryo and the fetus by the woman's body. The onset of the GBS in the period is exceptional. We report a case of paralysis of the nerve VI accompanied by dysphonia and tetraparesis due to GBS in a pregnant of 24 year old with 14 weeks of gestation..

CASE REPORT

Ms XH was 24 years old when taken into care at 14 weeks of gestation for a moderate GBS. She was treated with oxygen therapy a week, the rest of the treatment was symptomatic (analgesics, muscle massages). She made functional rehabilitation sessions over three

months. She almost fully recovered except for a few minor problems of gripping and of sensitivity of hands. Her pregnancy was completed. She delivered vaginally of a child 3,5 Kg healthy.

The Ms XH's disease story begins when she have 14 weeks of gestation by a flu syndrome. She describes the rest of the story of her illness as a period divided into five steps. Each phase is composed by signs, which complete the symptomatology of the previous, with an average setup time of about ten days, in the following order:

Step 1: a tingling sensation in the feet and hands;
Step 2: a muscle weakness she compares to a heavy legs with steppage;
Step 3: a weakness of the upper limbs associated with atrophy of Tenar eminences and Hypotenar (monkey hand sign) Figure 1
Step 4: a weakness of the sphincters responsible of a easy loss of feces and urine;
Step 5: inspiratory and expiratory moderate dyspnea associated with ophthalmoplegia (paralysis of the left VI, Figures 2 and 3) and dysphonia.

Furthermore, the review could have noted:
- an abolition of the deep tendon reflexes;
- a bradycardia (60 beats /min);
- an elevated protein level in cerebrospinal fluid;
- electromyogram (EMG) confirmed the disruption of electrical signals, muscle function was normal.

Obstetric ultrasound and brain magnetic resonance imaging (MRI) were normal.

DISCUSSION

GBS can occur during pregnancy. Even in severe cases, it does not increase the risk of miscarriage or that of fetal death. It does not affect the baby's development, and immunoglobulin treatment can be followed safely. Moreover, pregnancy does not alter the course of GBS and childbirth can be vaginally, even if the mother's paralysis (10, 11).

GBS is manifested by symptoms of severity varies from one person to another. In some case, it may even go unnoticed or be treated as an ordinary viral disease. This disease affects the sensory and motor nerves. It evolves in three phases, succeeding an infectious episode (flu, sore throat, gastroenteritis) (1-3, 6-9):

Phase 1: The first symptoms include abnormal sensations such as numbness, tingling, pins and needles, electric shock sensations and vibrations manifesting themselves in the feet and hands. Muscle weakness of varying intensity, up to paralysis. It begins in the feet, then rising to the upper limbs and head. The achievement is symmetric and progressing rapidly. Severe pain or cramps may occur especially at the back, buttocks and thighs. Various nerves may be affected at the head, responsible for various signs (ophthalmoplegia, dysphonia, swallowing disorder…). The nerves controlling the diaphragm may also be affected causing dyspnea of varying severity. Sometime the use of a respirator assistance may be necessary. This first stage lasts about three weeks or a month.

Phase 2: a phase of several days to several weeks, during which the symptoms stabilise. While paralyses are maximum, other symptoms related to the achievement of the autonomic nervous system may occur: tachycardia or bradycardia, low blood pression or high blood pression, rarely constipation. During this phase can be observed complications that occur frequently in patients hospitalised in intensive care unity (ICU) for a long time: respiratory and urinary tract infections, pressure ulcers and phlebitis.

Phase 3: symptoms regress, this phase lasts several months. Recovery can be complete, but some sequelae may persist, depending on the severity of the syndrome.

Miller-Fisher syndrome is a localised form of GBS. It combines ophthalmoplegia, ataxia and areflexia. It also follows an infectious syndrome, most often due to Campylobacter jejuni responsible for gastroenteritis (12-14).

In rare cases, death may occur from cardiac arrest, or complications observed in patients kept longer in the ICU (1, 6, 8). The risk of death increases with age. In general, the evolution is more favorable than the patient is young, the signs are moderate. In about 85% of cases, recovery is complete after six to twelve months. The myelin sheath is gradually rebuilt. In severe cases, rehabilitation can be long, and about 10% of all cases are affected negatively. Indeed, it happens that in addition to myelin, the nerve fiber itself is damaged, and it irreversibly (2, 7, 15-17). The sequelae may include decreased sensitivity to touch, persistent tingling or muscle weakness, especially in the feet or hands. Finally, relapses are possible but rare (5% of cases) (14, 18, 19).

The diagnosis of GBS is suspected based on clinical signs. Two additional tests are useful (2, 3, 7, 8):
- the lumbar puncture to detect abnormal elevation of protein in the cerebrospinal fluid
- the electromyogram which reveals a normal muscle function and disruption of the electrical activity of nerves..

Severe forms of this syndrome requires immediate hospitalization. Two main treatments of comparable effectiveness, can limit the process of nerve damage, and thereby to limit the severity of the syndrome (20-22):
- *Plasmapheresis*, which eliminates the blood of patients of autoantibodies responsible for the destruction of myelin. Several sessions are required.

Figure 1

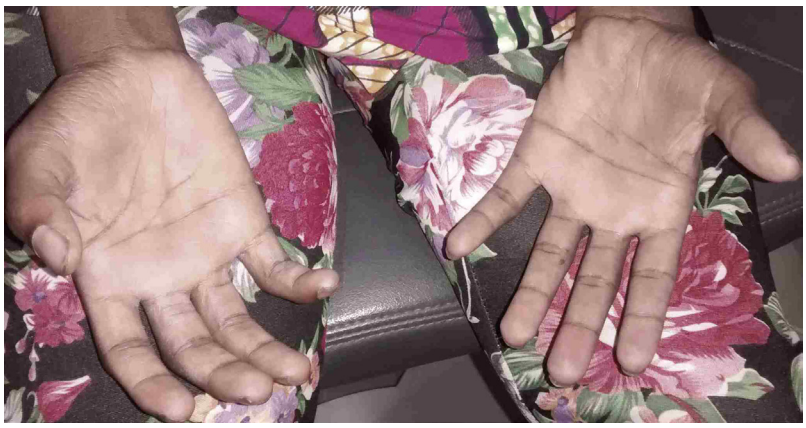


Figure 2

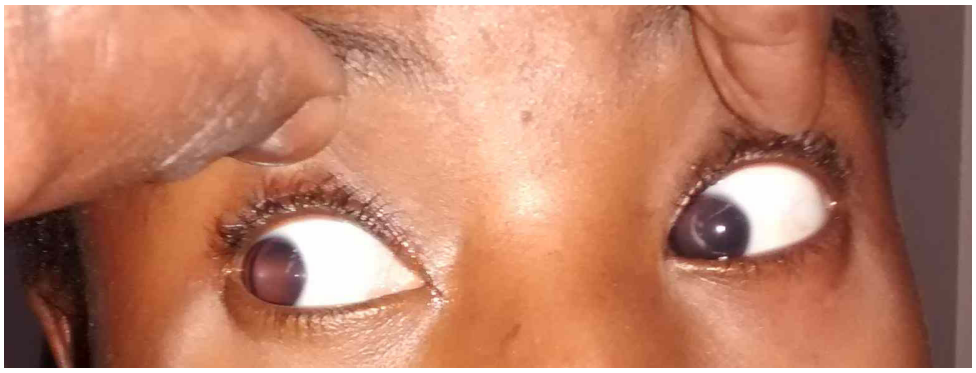
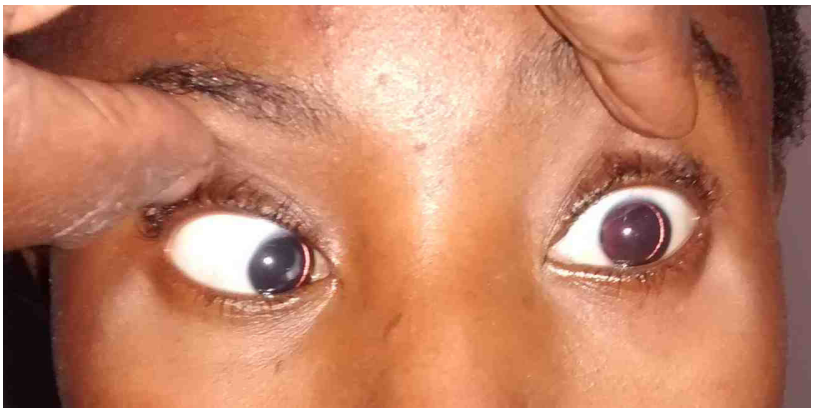


Figure 3



- *Intravenous immunoglobulin (IVIg)*, are injected to the patient (in the form of infusion) antibodies from the blood of many donors. By poorly understood mechanisms, these IVIG neutralise harmful autoantibodies. This treatment is easier to administer than plasmapheresis and its side effects (allergic reactions, muscle pain, fever, and headache) are rare.

All the symptoms associated with GBS should be treated to relieve the patient. Tracheostomy may need en prolonged assisted ventilation. En event of swallowing disorders gastric tube should be put in place. Measures against pressure ulcers should be followed by prolonged hospitalisation. Taking anticoagulant drugs reduces the risk of phlebitis. Analgesics are sometimes necessary if severe pain especially during Phase 1.

Physical therapy and physiotherapy are essential and can be put in place from the beginning of the disease (16, 17, 23).

The evolution of paralysis and progressive loss of autonomy create a state of anxiety and concern. Discouragement and waiver can be installed, especially when progress is slow. Psychological support is needed for the patient and family.

In conclusion, although exceptional, the diagnosis of Guillain-Barre's syndrome should not be disregarded during gestation. This disease can be a real problem for the obstetrician, although in most cases, it does not compromise the development of the fetus or vaginal delivery. Caesarean is sometimes the only alternative in severe forms.

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