Polyradiculoneuropathy of Guillain-Barré during pregnancy: a case report of a successful maternal and fetal outcome

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Guillain-Barré syndrome (GBS) is an acute autoimmune, inflammatory, demyelinating disease affecting the peripheral nervous system, responsible for a typical ascending flaccid paralysis with the classic albuminocytological dissociation in the cerebrospinal fluid. Its occurrence during pregnancy is a rare eventuality. Through the clinical observation of a 33-year-old pregnant woman who had GBS during third trimester and a literature review, we will study the clinical, diagnostic, therapeutic and maternal-fetal prognostic aspects of this pathology during pregnancy.

Keywords: pregnancy; polyradiculoneuropathy; Guillain-Barré syndrome.

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For reference: Benjilany A, Bouhtouri Y, Kouach J. Polyradiculoneuropathy of Guillain-Barré during pregnancy: a case report of a successful maternal and fetal outcome. Nevrologiya, neiropsikhiatriya, psikhosomatika = Neurology, Neuropsychiatry, Psychosomatics. 2022:14(1):97–98. DOI: 10.14412/2074-2711-2022-1-97-98

Guillain-Barré syndrome (GBS) is an acute autoimmune inflammatory polyradiculoneuritis of the peripheral nervous system [1]. The typical form was described by Georges Guillain and Jean Alexandre Barré, but many variants have been identified more recently. The incidence is 1/100,000, with no clear gender predominance. There is habitually a recent history of respiratory or digestive viral infection (70% of cases). It occurs very rarely during pregnancy.

Observation

Mrs. F.Z., 33 years old, with no medical or surgical history. The current pregnancy is estimated at 37 amenorrhea weeks (AW) according to an early ultrasonography, having benefited from 3 prenatal consultations and 3 obstetrical ultrasounds, with a prenatal check-up objectifying a gestational diabetes, unrecognized with no maternal-fetal consequences.

At 32 AW, she presented a rapidly progressive worsening heaviness and fatigability of both lower limbs, followed two days later by heaviness of both upper limbs, without subjective sensory or sphincter disorders, evolving in a context of apyrexia and conservation of general state. It's undeniably an ascending bilateral and symmetrical tetraparesis, with an acute installation. The extension phase of symptoms lasted 2 weeks before a plateau is reached. Neurological examination showed a motor deficit in the four limbs, predominantly distal and in the lower limbs, associated with osteotendinous areflexia in all four limbs. There were neither superficial or deep sensory disorders, nor damages of cranial pairs. The electroneuromyogram showed a mixed mechanism motor neurogenic disorder with demyelination predominance of the four limbs, mainly in the lower limbs; indeed, there was an abolition of the H reflex, alterations of F waves' conduction speeds, a decreased motor nerve conduction velocity with prolonged distal motor conduction latencies. The cytological and biochemical study of cerebrospinal fluid (CSF) showed no cytology with an albuminorachy at 0.46 g/l, then an albuminocytological dissociation. Viral (HIV, HBV, HCV, CMV, EBV, HSV) and bacterial (Syphilis, Lyme disease, brucellosis, Campylobacter jejuni)

serologies were negative. Immunological analysis (anti-DNA, antinuclear, antiphospholipid antibodies, cryoglobulinemia) was normal.

Diagnosis of Guillain-Barré syndrome was therefore retained on the basis of clinical criteria, electroneuromyogram and biological data. She was hospitalized in Neurology Department. Therapeutic management included a cure of polyvalent immunoglobulin (TEGELINE®) at a dose of 400 mg/kg/day for five days with daily motor rehabilitation. Evolution was marked by a clear downward recovery of the motor deficit after one week.

Patient consulted in our formation at 35 AW, for assessment of pregnancy. Vaginal birth was accepted by multidisciplinary staff subject to the absence of complications. She presented at 37 SA and 3 days, for management of her delivery, in which clinical examination found a hemodynamically and neurologically stable patient, in the early stages of labor with a uterine height greater than the gestational age (37 cm with a fatty pannicle of 2 cm), obstetrical ultrasound showed an evolving monofetal pregnancy in summit presentation, biometries corresponded to gestational age with an estimation of fetal weight at 3600 g, the amniotic fluid quantity was normal. Active labor management (artificial rupture of membranes + oxvtocin) was instituted, allowing a vaginal delivery of a female newborn, APGAR score 10/10 at 1, 5 and 10 minutes of life, birth weight of 3750 g, received and examined by the pediatrician, judged in good health and then given to the mother for early breastfeeding. Immediate postpartum was without particularities, patient did not present any neurological complications.

Patient was seen again in the Department of Neurology after a month of postpartum, neurological examination was strictly normal except for a weakness of osteotendinous reflexes in the left lower limb.

Discussion

Less than 2% of pregnants have a neurological pathology [1]. Acute polyradiculoneuritis is involved in 1.5% of cases [1]. This incidence does not seem to be different from that observed in the general population. It occur very rarely during pregnancy.

Neurological damage does not seem to arise at a specific term of pregnancy, although in the majority of cases, symptoms are encountered in last trimester, and more particularly in early postpartum period. Indeed, the retrospective study of Cheng et al. on a group of pregnant women with acute polyradiculoneuritis concluded that the risk of occurrence was mostly increased during two first weeks of postpartum [2].

Pathophysiological mechanism remains the same and is immunological. humoral, cellular or mixed [3]. From cases described in literature, it is difficult to conclude whether the immunological state generated by pregnancy favors or protects against acute polyradiculoneuritis. Moreover, the existence of recurrent forms in pregnant women and the aggravation of symptoms during peripartum period could suggest a hormonal role [1, 4]. Evolution and prognosis of GBS is not impacted by pregnancy. In our patient, motor and especially respiratory evolution was uneventful.

GBS in pregnancy does not affect fetal development and does not increase abortion or fetal death risk, suggesting that the agent causing GBS does not cross the placental barrier or affect the fetus [5, 6]. Nelson et al. reported a fetal survival rate of 96% in 29 patients with GBS during pregnancy, which is not different from that in unaffected pregnant women [7]. However, severe forms of acute polyradiculoneuritis appears to carry an increased risk of preterm delivery [5].

Respiratory distress represents the major complication in pregnant women, caused by increased ventilatory requirements and decreased residual capacity during pregnancy [5]. However, severe cases of GBS complicated by maternal respiratory distress have been reported with a satisfying fetal development and an ordinary delivery, when early respiratory support, minimal benzodiazepine sedation and early caesarean section are performed [5].

Therapeutic management of pregnant women with GBS has no particularities [5, 8]. Recently, it has been shown that immunoglobulins alone at an effective dose are sufficient to reach a clinical improvement [3]. Indeed, in our patient, a patent clinical improvement was noted in the first week after a five-day cure of immunoglobulin. Yik-Si Chan et al. have shown that there is no maternal-fetal risk and that immunoglobulins should be considered as the treatment of choice for GBS during pregnancy [9]. Other authors consider this treatment to be more effective than plasmapheresis because of less blood spoliation and infectious risk [9, 10].

Prevention of hemodynamic, respiratory, and infectious complications must be rigorous. Besides, clinical and ultrasound monitoring of fetal development must be part of therapeutic management [11].

Delivery is achieved in the majority of cases by vaginal delivery, without particular difficulties in parturients with GBS [11]. However, it has been noted that in GBS, induction of labor and/or cesarean section seem to cause a deterioration of delivery conditions and recovery delay [7, 10]. No fetal malformations have been described at birth, even when GBS occurs at first trimester [1].

Conclusion

Guillain-Barré syndrome remains a rare pathology during pregnancy, requiring close collaboration between obstetrician and neurologist once the diagnosis is confirmed. Its occurrence and evolution do not seem to be impacted by pregnancy. Similarly, except in severe cases, GBS does not seem to affect pregnancy and fetal development. However, prevention of respiratory, hemodynamic and infectious complications must be rigorous and regular clinical and ultrasound monitoring of fetal development must be an integral part of therapeutic management.

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Received/Reviewed/Accepted 24.09.2021/01.11.2021/08.11.2021

Conflict of Interest Statement

The investigation has not been sponsored. There are no conflicts of interest. The authors are solely responsible for submitting the final version of the manuscript for publication. All the authors have participated in developing the concept of the article and in writing the manuscript. The final version of the manuscript has been approved by all the authors.