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Group B Streptococcal Chorioamnionitis with Intact Amniotic Membranes. A Rare?

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Abstract

Group B streptococcus infection (GBS) is an important cause of perinatal infection. Maternal colonization by GBS can cause amniotic infection and thus fetal infection, sometimes causing fetal death. This infection generally occurs in association with premature rupture of membranes. We present here an infrequent case of GBS chorioamnionitis in full-term gestation with intact amniotic membranes. The curiosity of the case lies in the integrity of the amniotic membranes, the asymptomatic clinical presentation, and the adverse result (term fetal death).

Keywords

Chorioamnionitis, Group B Streptococcus, Intact Amniotic Membranes

1. Introduction

Streptococcus agalactie is a gram positive, catalase and oxidase negative and optional anaerobic coconut belonging to group B of the Lancefield classification, which is why it is also called group B Streptococcus or GBS. The gastrointestinal tract is the natural human reservoir for GBS and therefore the probable source of vaginal colonization. The vaginorectal colonization rate for GBS is highly variable. In Europe, rates between 6.5% and 36% have been reported, with figures close to 20% [1]. In Spain [2] colonization rates have been published in pregnant women from 11% to 18%.

The newborn is colonized by GBS as it passes through the colonized birth canal, intrauterine after rupture of the membranes. Although amniotic fluid has antibodies, complement levels, and phagocytes, it is a good culture medium for GBS, and even more so once the membranes have ruptured. Approximately 50% of the newborns of carrier mothers are colonized by GBS [3].

In the absence of preventive measures, between 1% and 2% of the newborns colonized during childbirth develop an early infection (in the first 7 days of life) [4]. This condition occurs as sepsis, pneumonia or meningitis, and in the vast majority of cases it occurs during the first 24 hours of life [5] [6] [7]. Mortality, which in the years 1970-1980 reached 50%, has now been reduced to 4% - 5% as a result of advances in neonatal care. However, as a consequence of the infection, 25% - 30% of the affected newborns suffer important neurological sequelae [8].

In addition to GBS maternal colonization, different factors increase the risk of early neonatal infection. Risk factors include: prematurity, prolonged rupture of the membranes (more than 18 h), intrapartum fever (≥38˚C), GBS bacteriuria during pregnancy and a previous newborn affected by GBS infection. However, 50% of infections occur in newborns without risk factors [9]. Fortunately, the incidence of early neonatal GBS infection in Spain has decreased by 73% after the implementation of prevention measures, standing at a rate of approximately 0.33% to 0.18% [10].

GBS is also an important cause of infection during pregnancy (chorioamnionitis) and in the postpartum period (endometritis), and although the data is not conclusive, an association has also been reported between vaginal colonization by GBS and premature delivery, premature rupture of membranes (PROM), low weight at birth and intrauterine death [11] [12].

As for chorioamnionitis, it is known to originate from the passage of microorganisms into the amniotic cavity and the fetus. This access occurs mainly via the ascending route. It is generally seen in the context of PROM.

Here we present a case of chorioamnionitis with the result of intrauterine fetal death in full-term gestation in which the pregnant woman had integrity of the amniotic membranes.

2. Clinical Case

The case refers to a healthy 36-year-old [gravida 2, para 1 (cesarean section)]. It is hypothyroid, without presenting any other personal history or risk factors of interest. Current pregnancy is proceeding normally. A urine culture performed at week 28 of gestation was considered negative, *with less than* 10,000 *CFU/mL*; *however, the GBS screening carried out in the urine culture was clearly positive*. At 34 weeks gestation, another obstetric revision was performed, being completely normal. She is scheduled for the Fetal Well-being Consultation at the week 39 of gestation, indicating that she should only undergo coagulation tests for epidural anesthesia to be administered, but that vaginal-rectal GBS check-up, between 35 - 37 weeks, was not required, since she was considered a carrier of GBS at detection of the presence of this bacterium in the urine culture previously performed.

At 38 weeks gestation, he went to the emergency room due to decreased fetal movements of one day of evolution, without other associated signs or symptoms. Report not having expelled liquid. When performing a fetal ultrasound, intra-

uterine fetal death was verified, being the amount of normal amniotic fluid. Induction of labor with misoprostol was performed after informing the patient and her family. 24 hours after initiation of induction, a 3410-gram male fetus was born, Apgar 0/0, with a loose twist of the neck cord and without macroscopic characteristics to suggest the cause of death.

The fetal autopsy subsequently revealed a degree of maceration corresponding to a date of death greater than one week, absence of malformations, bacillary proliferation in the pulmonary bronchi and severe acute hypoxia within the uterus. The placental pathology study corroborates marked histological signs indicative of infection (chorioamnionitis) associated with bacillary proliferation and postmortem thrombotic changes. The cultures taken at birth, both from the fetus (nasal, buccal and rectal) and placental, all demonstrate development of GBS. In conclusion, both postmortem cultures and the pathological anatomy of the placenta suggest chorioamnionitis with group B strep fetal sepsis as a probable cause of fetal death, in a patient with a prior positive urine culture for this microorganism. The curiosity of the case lies in the integrity of the amniotic membranes, the asymptomatic clinical presentation and the adverse result given in term gestation.

3. Discussions

Introducing phrases such as "Group B streptococcus chorioamnionitis with intact membranes", "Chorioamnionitis caused by Streptococcus group B with intact membranes" and "Intrauterine fetal death in a pregnant woman carrying group B streptococcus" in PubMed, are only two jobs:

Katz and Bowes [13], in 1988, reviewed perinatal mortality due to GBS infection over a three-year period at a tertiary center. In 6 of the 16 perinatal deaths due to GBS, infection occurred with the membranes intact. A review of the obstetric and pediatric literature reports they conducted that 10% - 50% of GBS infection occurred in this manner, ascendingly.

Neri *et al.* [14] reported a case of GBS chorioamnionitis with intact membranes in a quintuple preterm delivery, unusual for three reasons: premature delivery was associated with intact membranes and GBS-infected amniotic fluid; the five newly born were contaminated with GBS and all the newly born survived.

In term pregnancies, chorioamnionitis occurs in the presence of a PROM and complicates approximately 2% - 4% of full-term births. In the case provided here, chorioamnionitis presented in a pregnant woman at term (38 weeks) and in the absence of PROM.

The case presented here, of course, falls within this third type of chorioamnionitis, due to the absence of clinical signs of infection, without analytical repercussion, but with marked histological signs in the placenta suggestive of infection (chorioamnionitis) associated with bacillary proliferation. Ascending infection from the genitourinary tract to the amniotic fluid/fetus was considered to

be a culprit of the chorioamnionitis.

It is generally observed in the context of a PROM, although it can occur with intact membranes, as in the case provided here. One of the main risk factors for chorioamnionitis is prolonged rupture of the membranes. In the case provided here, the only existing risk factor was the presence of GBS in the urine culture performed at week 28 of gestation.

Chorioamnionitis is associated with increased maternal and perinatal morbidity and mortality [15]. Neonatal morbidity and mortality related to chorioamnionitis is inversely proportional to gestational age at birth and includes: neonatal sepsis, pneumonia, intraventricular hemorrhage, and cerebral white matter damage with possible short and long-term sequelae, such as cerebral palsy [16]. PROM, at term or preterm, is associated with an increase in neonatal sepsis [17]. Prior to the widespread use of maternal intrapartum chemoprophylaxis, maternal colonization with GBS conferred an increased risk of intraamniotic infection and infection in the early postpartum period. It is not clear whether there is an association between maternal GBS colonization during pregnancy and preterm delivery, but it is known that GBS causes fetal deaths in the third trimester [18], as occurred in the case reported here.

Vaginal-rectal colonization of GBS causes fetal/neonatal infection during labor, which is considered due to direct contact between the GBS flora and the fetus. Intrapartum antibiotic prophylaxis, therefore, is recommended to prevent neonatal GBS infection. Also, GBS, after PROM, can cause fetal/neonatal infection; this is also due to direct contact with GBS (in the amniotic fluid) and the fetus. Here, the current fetus was considered infected through intact membranes. In this sense, this case highlights that fetal GBS infection can be caused even without PROM or vaginal delivery.

Due to this positive screening for urine GBS carried out in week 28, this pregnant woman was already considered to be a carrier of GBS, and, as indicated in the Consensus Document [2] prepared in 2012 by different Spanish scientific societies, the taking of the vaginal-rectal culture was not necessary. Specifically, said Document indicates: "All pregnant women with the presence of GBS in urine during the pregnancy require the administration of intrapartum antibiotic prophylaxis, without it being necessary perform vaginal-rectal culture at weeks 35 - 37".

4. Conclusions

- As can be seen, it is complex to find publications, clinical cases that specifically refer to the coexistence of chorioamnionitis caused by GBS, which causes intrauterine fetal death in a full-term pregnancy and that infection takes place with the entire membranes, such as in the case provided here.
- Although one of the main risk factors for chorioamnionitis is prolonged rupture of the membranes, it must always be borne in mind that it can also occur with intact membranes.

- There may be symptoms of chorioamnionitis without any clinical evidence and without analytical repercussions.
- The main pathogenic mechanism of chorioamnionitis is the ascending pathway, even in the presence of intact membranes.
- Although the data are not conclusive, an association has also been reported between vaginal colonization by GBS and intrauterine death.
- One of these germs implicated in the etiology of amniotic infection is, without a doubt, GBS. Therefore, it is mandatory to identify pregnant women who are carriers of this microorganism, to take adequate prophylactic measures [2] [19] [20].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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