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Case Report

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Analysis of risk factors for sepsis caused by streptococcus agalactiae in a newborn

Abstract

Despite the advances in neonatal care and the adequate use of antibiotics, the influence of early sepsis on neonatal morbidity and mortality remains significant. Its most frequent etiology is bacterial, and within them Group B Streptococcus plays an important role. We analyze the history of a normopeso newborn, PEG, a product of pregnancy, which highlights the lack of GBS, and a pregnancy and childbirth without complications that at 16hours of life presents symptomatology compatible with early neonatal sepsis. Early diagnosis is difficult to perform in newborns because of the proteic nature of their clinical manifestations as well as their easy confusion with other conditions. Therefore, identifying risk factors for infection raises clinical suspicion and enables early interventions in asynchronous patients. Clinical guidelines have been developed at the national level and hospitalizations in which risk factors are identified that allow taking behaviors before and after birth. Several reports indicate that this entity can occur in children without these classic risk factors, since although the guides are very useful, they are imperfect when used in each particular patient. This paper aims to describe the risk factors for early sepsis of GBS and to analyze the usefulness of complementary examinations to sensitize health professionals about early diagnosis and appropriate and timely treatment.

Keywords: *Streptococcus agalactiae*, newborn, risk factor's

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Introduction

Sepsis is the invasion and proliferation of bacteria, fungi or viruses in the bloodstream.[1] In the newborn, it is classified as early or late according to the time of clinical manifestation. Early sepsis is the one that occurs in the first seven days of life and represents the most frequent form (85%); Most cases occur within the first 48hours.[1,2]

Despite advances in neonatal care and use of antibiotics, the influence of early sepsis on neonatal morbidity and mortality remains significant.[3] Its most frequent etiology is bacterial, only 1% is of fungal or viral cause. The most frequent bacteria are *S. Agalactiae* or *Streptococcus* group B (GBS) and *E. Coli*. The etiology varies according to the local epidemiology and the child's birth weight.[4] Prior to the antibiotic era, Gram-positive cocci predominated, mainly GBS. With the implementation of prophylaxis strategies, there has been a significant decrease in the incidence of this disease reaching 90% in some series.[2]

In Uruguay, strategies for the management of SBG infection were incorporated into the national protocols for the diagnosis and treatment of newborns.[1] However, the suspicion and timely diagnosis of this infection continues to be a problem in part related to the lack of individualization of the application of these general recommendations. There are currently three challenges to early neonatal sepsis: identifying children with suspected symptoms to begin antibiotic treatment in a timely manner; distinguish children at high risk for infection and discontinue antibiotic treatment once the diagnosis was discarded.

This paper aims to describe the risk factors for early sepsis of GBS and to analyze the usefulness of complementary tests to sensitize health professionals about early diagnosis and appropriate and timely treatment.

Clinical case

Newborn, male, 16 hours of life

Mother 20years old, healthy, group O Rh^+. First gestation, precocious pregnancy, poorly controlled, low genital infection during the first and second trimester, received adequate treatment. Uroculture in the third trimester without development. Serology for Hepatitis B, Lus and HIV negative. Toxoplasmosis immunized. No GBS search was performed. Vaginal birth, spontaneous rupture of membranes of 3hours, fluid meconial amniotic fluid. She did not receive antibiotics during labor. Mature term, 39weeks of Gestational Age (EG) by date of last menstruation, birth weight 2560g, small for EG, harmonic, Apgar 9/10. The physical exam highlights epispadia. Rest without alterations.

In joint mother-child accommodation begins with difficulty in suction. Capillary glycemia with limit values. At 13hours of age he added respiratory distress syndrome with intermittent moaning, flutter and polypnea. Hiperreactivity, hypotonia, discreet skin paleness, prolonged recoloration time. Moves to CTI. In evolution, respiratory difficulty is on the increase, requiring orotracheal intubation and mechanical ventilatory assistance for 5days; Hemodynamic instability that is managed with volume and inotropes. Increased markers of inflammatory response in the first 72hours: white blood cells 12,000cells/mm^3, 89% neutrophils at 32000cells/mm^3, 76% neutrophils; C reactive protein (CRP) of 12 to 94mg/dL, Procalcitonin (PCT) 80 to 100ng/L. It is not possible to obtain cerebrospinal fluid for cytochemical analysis. Cultivation of cerebrospinal fluid without development. GBS is isolated in the blood culture. Empiric treatment with ampicillin plus gentamicin i/v is initiated and maintained for 14days. It shows good evolution, without complications.

Discussion

The early diagnosis of this pathology is difficult due to the proteiformity of its clinical manifestations and the similarity with other diseases in this stage of life. Identifying risk factors for infection raises clinical suspicion and allows early interventions in asymptomatic patients. For this purpose, international clinical recommendations and guidelines have been elaborated and, at the national level, allow the adoption of pre-natal and post-natal behaviors. Risk factors included in these recommendations are: premature and prolonged rupture of membranes, maternal colonization by GBS, prematurity, and coriomanionitis.[1,5]

Neonatal sepsis to GBS is usually caused by the rise of germs from the female genital tract, with infection of the amniotic fluid and the fetus before or during labor. Maternal colonization by GBS occurs during pregnancy and can be constant or intermittent.[6] Bacteriuria by GBS in the last trimester of pregnancy and the history of neonatal sepsis by GBS are indicators of colonization.[2,3] International data report that the prevalence of colonization varies between 10 and 30%.[4] National guidelines for the care of pregnant women recommend the systematic search of this microorganism at 35-36weeks of gestation through recto-vaginal exudate and the performance of uroculture in the last trimester. In this case the state of maternal colonization is unknown since this study was not performed.[7]

Premature and prolonged rupture of membranes beyond 18hours and chorioamnionitis have been associated with an increase in the incidence of neonatal sepsis in GBS.[1] In this case, these risk factors were not present. The mother was asymptomatic with normal physical examination. No fever, uterine tenderness, tachycardia; No fetal tachycardia was recorded and amniotic fluid was clear.[2]

Neonatal risk factors include prematurity and low birth weight, especially weight less than 1500grams. The latter is inversely related to the risk of sepsis.[5,8,9] These factors were also not present in this clinical case.

However, several reports indicate that GBS sepsis can occur in children without the risk factors classically described, hence the importance of individualizing clinical assessment and decision making.[2,9] Other elements that may suggest an increased risk of maternal colonization have been reported. Examples are maternal malnutrition, sexually transmitted infections, maternal age under 20years[2] and prolonged labor.[10] In the presented clinical situation, the following risk factors can be identified in this patient: absence of GBS search, maternal age under 20years, presence of low genital infections, small male infant for gestational age, weight at Be born 2540g.

This should alert the health team to the active search and suspicion of SBG infection: The clinical manifestations of the infection are varied and nonspecific and appear within the first 48hours of life. In early neonatal infection the most common form of presentation is pneumonia or sepsis, with less frequent meningitis.[1,2] Among the most frequent manifestations are respiratory distress, digestive intolerance, alterations in thermoregulation, apneas, convulsions, poor peripheral perfusion, cyanosis without other cause, metabolic acidosis, hypo or hyperglycemia, general malaise, lethargic or irritability. However, these symptoms are non-specific and may correspond to other non-infectious clinical syndromes that simulate neonatal sepsis.[5] This patient had difficulty in suctioning and hypoglycemia in the first hours of life, which, together with the previous risk analysis, should have led to an earlier suspicion of the diagnosis.[5,7]

Acute phase reactants are useful adjuncts in asymptomatic infants with risk factors.[2] it is necessary to remember that the inflammatory response to the infection requires at least six hours, so it is necessary to request blood withdrawal between the 6-12hours of life.[5] The interpretation of the hemogram should be correlated with the gestational age and the hours of life. The most specific value is neutropenia.[1,5] Acute phase reactants: PCR They ascend at 6-8hours after infection, and peak at 24hours. Procalcitonin begins to rise at two hours after infection, peaking at 12o'clock. PCT has higher sensitivity, but less specificity.

Although in this case the acute phase reactants were very high, their usefulness was lower since the patient's clinic was sufficient to make an adequate diagnosis.

The etiological diagnosis requires the carrying out of blood culture and/or culture of liquids and/or samples of normally sterile sites.[8] In this clinical situation, we obtained a resection of positive hemovulatory to the mentioned germ. It is noteworthy that the treatment of this germ does not offer greater complications since it is sensitive to penicillin, so a treatment instituted early has a good prognosis.

Conclusion

The current guidelines and/or recommendations for the prevention of diagnosis and treatment of neonatal GBS infection do not include all clinical situations. As with other recommendations, individualization is required when making decisions in clinical practice it is necessary to consider all possible factors of reisgo maintain high clinical suspicion to avoid delays in diagnosis and better results.

They pose as challenges in this pathology:

A. Identify neonates with clinical signs of early neonatal sepsis, through high suspicion, bearing in mind that patients present symptoms within the first 24hours

B. Identify patients without symptoms, at high risk and requiring antibiotic treatment.

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Conflict of interest

Author declares that there is no conflict of interest.

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