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Hypotensive Brain Stem Necrosis in a Stillborn

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Summary. Hypotensive brain stem necrosis is reported in a stillborn. Additional postmortem findings included evidence of intrauterine distress, shock, and a pure blood culture of group B β-hemolytic streptococci. These findings suggest group B β-hemolytic streptococcal sepsis in utero, with a subsequent episode of transitory circulatory failure prior to intrauterine demise.

Key words: Hypotensive brain stem necrosis – Cardiac arrest encephalopathy – Intrauterine, group B β-hemolytic streptococci – Still birth

Introduction

Hypotensive brain stem necrosis [5], also known as cardiac arrest encephalopathy [8], is a well recognized pattern of ischemic brain damage seen at autopsy in cases who survived an episode of acute, transitory circulatory failure [1, 3, 5–9, 11]. As first described by Gilles [5], these changes are characterized by bilateral, symmetrical necrosis of brain stem nuclei. Because of the frequent history of a cardiac arrest, Janzer and Friede [8] have proposed the term "cardiac arrest encephalopathy".

The lesion probably occurs more frequently in infants than in adults [8, 9], but it has not yet been reported in a stillborn. We present the clinical history and postmortem findings in a term stillborn whose brain exhibited typical hypotensive brain stem necrosis. The implication is that this fetus suffered an acute cardiac event in utero and survived long enough to develop the histopathologic changes.

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Case Report

Clinical History

The black female infant, product of the third, uncomplicated pregnancy in a 30-year-old mother, was delivered stillborn at term. The mother detected fetal movements immediately prior to the onset of labor, but no fetal heart tones were heard on admission to the hospital several hours later. The stillborn was delivered vaginally after labor augmentation and was noted to be markedly meconium-stained.

Postmortem Findings

Size and weight were adequate for gestational age of 40 weeks. The skin, nails, and chorioamnionotic membranes were meconium-stained. No other gross abnormalities were identified.

Microscopic examination of the viscera revealed histological changes indicative of intrauterine stress and shock. There was meconium deposition with the Hofbauer cells of the chorioamnionotic membranes and stress involution of the thymus and fetal adrenal cortex. The liver and spleen were markedly congested, and juxtamedullary hyperemia of the kidneys was prominent. The other organs revealed slight autolysis. Cultures of the lungs and spleen showed no growth; the blood culture, however, grew innumerable colonies of group B β-hemolytic streptococci.

The appearance of the brain was unremarkable, and the weight was appropriate for the age. On microscopic examination, bilateral, symmetrical necrosis of the pontine tegmental nuclei was identified. The neurons of the reticular formation, the seventh and eighth cranial nerve nuclei, the superior olivary nuclei, and the vestibular nuclei demonstrated eosinophilic degeneration. The remainder of the brain and spinal cord revealed no abnormality.

Discussion

Ischemic brain stem lesions occurring as a result of perinatal asphyxia [4] or prenatally without clear evidence of an asphyxial episode [10] have been reported; however, isolated finding of hypotensive brain stem necrosis in a stillborn is not recorded. Of perhaps greater significance is the implication of this finding in conjunction with the visceral findings at autopsy.

The histological changes indicate that the fetus sustained a severe, acute event in utero and survived it for a time.

The sequence of events that lead to the intrauterine demise in our case cannot be determined with certainty; however, the positive and pure postmortem blood culture suggests the possibility of group B β-hemolytic streptococcal sepsis as the acute insult. Group B β-hemolytic streptococcus is a particularly virulent pathogen in the neonatal period and has been reported to cause intrauterine death and spontaneous late abortions in the absence of inflammatory manifestations in either the fetal lung or in placenta, membranes, and cord [2]. It may be that this fetus developed group B β-hemolytic streptococcal sepsis in utero with subsequent septic shock resulting in cardiac arrest. This sequence of events, although speculative, would explain the pathologic changes observed at autopsy.

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