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Neonatal Suffocation And Forensic Medicine Review

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ABSTRACT

The scientific literature on neonatal encephalopathy has expanded exponentially over the past few decades, and malpractice lawsuits in obstetrics and neonatology have grown to represent a significant danger to the healthcare system. Currently, scientific evidence is insufficient to definitively determine in each individual case whether the hypoxic insult formed during labour, in the early hours after birth, or otherwise whether the damage has to recognise a distant and pervasive source acting during pregnancy. In order to clearly identify all the data in favour of or against the assumption that there is a causal correlation between neonatal encephalopathy and medical misbehaviour, a more in-depth analysis of all these cases is desired, according to some authors, who believe that this scientific uncertainty leads to a higher percentage of civil suit decisions that are prone to recognising a guilty medical behaviour. This article will concentrate on the medico-legal approach to a prenatal hypoxic-ischemic incident, addressing the pertinent information that must be gathered in order to determine the medical and legal aetiology of the neonatal harm.

Keywords: Neonatal asphyxia, forensic medicine, malpractice, diagnostic criteria, consensus statement.

INTRODUCTION

Birth or intra-partum asphyxia, with an incidence ranging from 1 to 6 neonates per 1000 live fullterm births, continues to be a significant concern in perinatal medicine despite the frequent use of more reliable techniques throughout labour to check the foetal well-being [1]. According to the Fenichel and Sarnat classifications, [2,3] approximately 50% of these newborns will develop hypoxic-ischemic encephalopathy (HIE), and those with moderate (stage 2) to severe (stage 3) HIE are at a significant risk of developing cerebral palsy (CP; [4]).

Additionally, according to recent data, preterm birth (28%) and infections (36%) are the two other leading causes of newborn deaths, with intrapartum hypoxia or asphyxia coming in third. [5]. The mechanisms of peripartum asphyxia have been the subject of various articles in recent years in an effort to draw a distinct line between prenatal and postnatal causes and to establish a set of criteria for CP instances. Despite all of the efforts, the primary diagnostic criteria still place a greater emphasis on subjective problems than on signs and symptoms.

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The Scores Score and the presence of multi organ dysfunction (MODs) signs, which were once thought to be necessary for the diagnosis, have been dropped from the list of essential criteria for the diagnosis of CP in favour of a stricter clinical manifestation of the movement disability (only the spastic quadriplegia and the dyskinetic type) and the unmistakable exclusion of other pathologies. A prenatal blood pH of 57.0 and a base deficit of 41.412 mmol/L in a newborn displaying premature symptoms of moderate to severe encephalopathy have been the cornerstones of all classifications that have been taken into consideration thus far.

In the human term baby with encephalopathy, MRI may be able to identify two primary patterns of brain injury [13]. The watershed prominent pattern, which affects the white matter and may extend toward the grey matter if the injury is severe enough, appears to be connected to a prolonged suffocation.

Acute deep hypoxia with a predominance of grey matter is shown by the basal nuclei dominating pattern.The investigation of biomarkers connected to the hypoxia insult may yield different results [14].Although a number of intriguing molecules have been identified (including S100b, serum/CSF interleukin-1b, serum interleukin-6, and CSF NSE), there is yet to be any widespread usage of such indicators in a clinical environment.

The lack of a clear definition for each term used when referring to the conditions of perinatal asphyxia, hypoxic-ischemic encephalopathy, neonatal encephalopathy, and cerebral palsy can make clinicians and forensic experts testifying as expert witnesses in court uncomfortable at times [8]. This topic has been referred to as the 'Bermuda Triangle' of neonatology. Its timing, duration, and results are also poorly defined.[9-10]

Hypoxemia, hypercapnia, and the emergence of metabolic acidosis are all symptoms of intrapartum asphyxia, which develops when blood gas exchange is impaired [10]. Neonatal encephalopathy is a clinically recognised syndrome of abnormal neurologic function that appears in the term infant in the first few days of life and is characterised by difficulties with breathing induction and maintenance, depression of tone and reflexes, subnormal levels of consciousness, and frequently seizures [11].

A maternal blood pH of 57.0 and a base deficit of 41.412 mmol/L in a newborn displaying premature symptoms of moderate to severe encephalopathy have been the cornerstones of all classifications that have been taken into consideration thus far.

These consensus principles suggest that in addition to casting doubt on the presence of an intrapartum injury, the purported causal relationship between severe metabolic acidosis and cerebral injury has also been called into question. When using cord blood pH at the time of birth, the incidence of having an umbilical arterial pH57 was 3.7 per 1000 term live births, but only 23.1% of them had neonatal neurologic morbidity or mortality, according to Graham et al. [12] who reviewed several studies correlating an umbilical arterial pH57 to neonatal neurologic morbidity and mortality.

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Additionally, the promising field of magnetic resonance imaging (MRI) techniques, including proton magnetic resonance spectroscopy and diffusion tensor imaging (DTI), and the examination of brain injury biomarkers are not yet definitive.

The investigation of biomarkers connected to the hypoxia insult may yield different results [12]. Although a number of intriguing molecules have been identified (including S100b, serum/CSF interleukin-1b, serum interleukin-6, and CSF NSE), there is yet to be any widespread usage of such indicators in a clinical environment.

In the clinical environment, uncertainty appears to be the only data that can be shared. The presence of a pH47.0 with a base deficit of 41–412 mmol/L, however, can be used as solid evidence to demonstrate that the origin of the cerebral damage cannot be attributed to a peripartum hypoxic event and must instead be attributed to a different origin in a clinical period during which neither the gynaecologist nor the neonatologist could take any steps to prevent such damage.

For this reason, a CP claim necessitates that the doctor supply all information that is at their disposal, particularly information that relates to other potential causes of the brain damage or that rules out the possibility that the hypoxic-ischemic event occurred during pregnancy.

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