Benha Journal of Applied Sciences (BJAS) **print: ISSN 2356–9751**
Vol. (10) Issue (1) (2025), (13- 15) **online: ISSN 2356–976x**
http://bjas.bu.edu.eg
Medical and Health Science

Cardiac Troponin T and Outcomes in Asphyxiated Neonates

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Abstract
Background: Worldwide, neonatal hypoxia is a major contributor to illness and death. A key result of prenatal hypoxia is hypoxic-ischemic encephalopathy (HIE), which is often associated with cardiac dysfunction.
Objective: The purpose of this research is to determine if there is a connection between cTnT levels and newborn outcomes, HIE stages, the need of inotropic support, and asphyxiated neonates.
Methods: Perinatal hypoxia was the subject of a prospective observational research including newborns. The levels of cTnT were assessed throughout the first twenty-four hours of life. By using the Sarnat staging technique, the severity of HIE was categorised. At discharge, we evaluated the neonate's neurodevelopmental state and noted their inotrope needs. In order to find connections, statistical analysis used regression.
Results: Stage III newborns with severe HIE had elevated cTnT levels compared to stage I and stage II neonates with mild or moderate HIE ($p < 0.001$). The findings showed a strong positive relationship ($r = 0.76$, $p < 0.001$) between cTnT levels and inotrope needs. There was a correlation between high cTnT levels and unfavourable outcomes, such as death and extended hospital stays (OR = 3.5, CI: 1.8-6.8, $p = 0.002$).
Conclusion: The degree of cardiac damage in newborns exposed to hypoxia may be reliably assessed by measuring cTnT levels. They have a high degree of correlation with newborn outcomes, inotropic support requirements, and HIE stages. Optimal care options for neonates with perinatal hypoxia may be achieved by early detection of high cTnT levels, which may help in risk categorisation.

Keyword : Cardiac Troponin T levels ; Inotrope requirements, HIE stages

Introduction
When a newborn suffers a severe anoxic brain damage, it causes a cascade of physiological, biochemical, and molecular alterations that are collectively known as hypoxic-ischemic encephalopathy (HIE). Brain palsy, epilepsy, intellectual disability, and behavioural disorders are among the chronic conditions that can develop as a result, and there are also acute symptoms like seizures, altered consciousness, weak breathing, poor muscle tone, or metabolic derangement (1). Premature death is another possible outcome.
As well as being the leading cause of newborn seizures, HIE is a leading cause of infant death (2). Also, the complicated pathophysiology, high mortality rate, and long-term occurrence of HIE make for a bad prognosis. According to previous studies, low- and middle-income nations have an incidence rate of 10–20 cases per 1000 live births, whereas wealthy countries have an incidence rate of 1.5 cases per 1000 live births. Neonatal intensive care unit (NICU) mortality accounts for around 24% of HIE patients. Babies that make it through the first round of medical evaluations often have motor and behavioural abnormalities like autism, epilepsy, or general developmental delay (3), as well as hearing and visual issues (10-20%) and cerebral palsy (around 40%).
The pathophysiology of foetal development has been better understood because to improved medical practices. Nevertheless, HIE is still a serious problem for full-term babies, and we need to find a way to treat it better (4).

❖ Etiology & Pathophysiology of HIE
The leading cause of HIE is perinatal hypoxia. Both prenatal and postnatal periods may cause perinatal asphyxia. Foetal causes (such as foetal bradycardia, foetal thrombosis, or foetal haemorrhage) or maternal causes (such as preeclampsia, abruptio-placentae, maternal hypotension, severe anaemia, asthma, or chronic vascular disease) or tight nuchal cord or cord prolapse can lead to inadequate placental perfusion and impaired gaseous exchange, which in turn causes intrauterine asphyxia. Severe hyaline membrane disease, pneumonia, congenital heart disease, meconium aspiration syndrome, and other causes of newborn respiratory failure may lead to postnatal asphyxia (5).
Hypoxia and brain ischaemia, or decreased cerebral blood flow and oxygen, are the fundamental mechanisms that cause HIE in both premature and full-term neonates. Acidosis, inflammatory mediator release, and free radical generation are the outcomes of a chain reaction that begins with hypoperfusion and culminates in hypoxia. Diffuse brain damage (cell death in neurones) and disruption of normal brain autoregulation are the outcomes of these pharmacological compounds. How long and how severely hypoxia lasts, as well as the brain's level of development, determine the precise kind of damage. Myelinated fibres are particularly susceptible to HIE in full-term newborns because of their increased metabolic activity (6).
Metabolism of lactate, ketone bodies, and glucose provides the foetal brain with an ongoing supply of

ATP, which in turn powers brain function. Because of its increased energy reserve capacity, the foetal brain is better able to withstand hypoxia-ischemia (HI) than the adult brain. But the foetal brain is equally vulnerable to damage in the event of a significant ATP shortage. Chronic maternal hypoxia, pre-eclampsia, umbilical cord knotting, umbilical cord prolapse, shoulder dystocia, and placental abruption are among the conditions that can cause HI, which in turn impairs the flow of oxygenated cerebral blood to the foetus and causes cellular and systemic responses, leading to this critical ATP depletion (7).

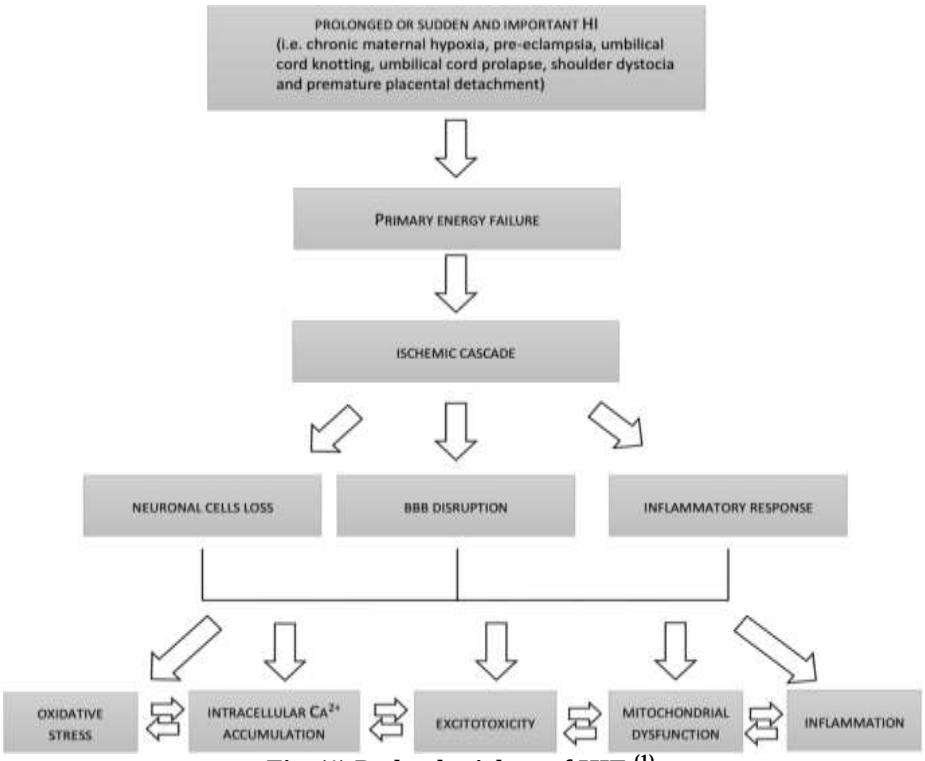

Fig. (1) Pathophysiology of HIE [(1)].

Health care providers also use the Sarnat staging criteria or an adapted version to describe the severity of encephalopathy within the first several postnatal days of life in conjunction with neuroimaging to assess the severity of the insult **[(8)],** table 1.

Table 1: Sarnet Stages of Neonatal Encephalopathy

Assessment	Stage 1	Stage 2	Stage 3
Mental status	Hyperalert	Lethargic	Stuporous
Suck reflex	Weak or absent	Weak or absent	Absent
Moro reflex	Strong	Weak	Absent
Muscle tone	Normal	Hypotonia	Flaccid
Autonomic function	Generalized sympathetic	Generalized parasympathetic	Absent
Pupils	Mydriasis	Miosis	Variable
Seizures	None	Common	Variable
EEG	Normal (awake)	Early: low-voltage theta and delta	Early: periodic pattern with isopotential phases Late: isopotential
Duration	< 24 hours	2–14 days	Hours to weeks

Conculsion

Since it may aid in the diagnosis of encephalopathy at the bedside, amplitude-integrated electroencephalography (aEEG) has seen extensive application in neonatal intensive care units (NICUs). If necessary, a paediatric neurologist may evaluate the objective record it offers. A shift from a continuous to a discontinuous background pattern may be seen in mild encephalopathy. This does not constitute a significant anomaly; it is referred to as discontinuous normal voltage. Burst suppression, characterised by periods of normal amplitude against a low-voltage background, may be seen in cases of severe damage.

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