high-blood-glucose-levels/-ketonuria-in all but(n=2) where ketonuria was negligible.Interventions-were-directed,-/-established-guidelines-for-DKA-HHS-/relevant associations/-emerging-complications-driven. Comparatively, Intractability, brittleness,-evanescence--Somogyi-Dawns-Phenomena/-poor outcome were more common in the cases presenting unusually/atypically/or with rare associations.

Conclusions-importance: This study reiterates the need for an early-structured-approach to the evaluations,-management of-DKA-HHS presenting-atypically,-or-with an uncommon/-rare-remarkable associations.Identifying these unusual/-striking associations are crucial because specific adjunctive-interventions-differ.An obligatory base-line-interval-investigations is recommended for all cases with unusual features-associations,-/-tardively-resolving comatose-states,-labile blood-glucose-profiles,-lactic-acidosis,-shock, /cases with the need for overenthusiastic insulin,-fluid, -electrolyte,-applications,-MRDM,-/-Mariucs syndrome,and the other therapeutically-difficult cases with severe abdominal pains. In such challenging cases minimum-investigations including relevant,-imaging,-coagulations-serological,-complete-haematological-studies,-in addition to others which will be emerging- complications-driven, is proposed for children living between the-Tropics-of-Cancer-Capricorn, also a mandatory- Pancreatic-function /abdominal-Ultrasonographic evaluations to exclude-disseminated-FCPD,and LFT-tests to rule out hepatic-cirrhosis,-CLL,-hepatic-infiltrations-steatosis will be worthwhile. In all concerned cases,-investigations/ interventions-should be more aggressive/-comprehensive.

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BILIRUBIN UPREGULATES GENE EXPRESSION OF SLC7A11 AND INCREASES CYSTINE UPTAKE MEDIATED BY SYSTEM X_c⁻ IN SH-SY5Y NEUROBLASTOMA CELLS

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Background and aims: Severe neonatal hyperbilirubinemia can produce encephalopathy. We have previously demonstrated that exposure of SH-SY5Y cells to unconjugated free bilirubin (Bf) for 24 h induces several genes as the both components of

a glutamate-cystine antiporter (System X_c⁻). Cystine is implicated in glutathione (GSH) formation.

Present study was performed to analyze if the upregulation of System X_c⁻ induced by Bf offers protection from oxidative stress.

Methods: Cell viability was assessed by MTT test in SH-SY5Y cells exposed to 140 nM Bf for 24 h (T0). [¹⁴C]-cystine uptake was measured with and without l-quisqualate (an specific System X_c⁻ inhibitor). Cells were then growth in medium for 48h (T48) and 156h (T156) in the absence of Bf. GSH level and viability were measured after H₂O₂ exposure.

Results: Cell viability was reduced 30-40% after 24 h of Bf exposure. Cystine uptake was increased in surviving cells after 24 h Bf exposure. Further incubation with l-quisqualate for 4 h reduced cystine uptake to normal levels. GSH levels were 2.5 times that in control cell both at T0 and T48 but returned to normal levels at T156. Cells were protected from H₂O₂ exposure when GSH levels were high, but cells death was identical to controls at T156 when GSH levels returned to basal levels.

Conclusions: SH-SY5Y cells respond to Bf exposure by an increased gene expression and activity of the System X_c⁻. This was associated with a higher incorporation of cystine and GSH content which protected the cell from oxidative stress H₂O₂ induced.

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HYPOTHERMIA TREATMENT PROLONGS THE POSSIBLE TIME TO RECOVERY OF AEEG TO PREDICT NORMAL COGNITIVE AND MOTOR OUTCOME

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BACKGROUND AND Aims: This study aims to investigate the correlation between amplitude-integrated EEG (aEEG) and outcome at 24 months in full-term infants treated with systemic hypothermia after perinatal asphyxia and moderate/severe hypoxic-ischemic encephalopathy (HIE)

Methods: In 2007, 21 infants with HIE were treated with systemic hypothermia (body temperature 33-34°C for 72 hours) using a Thecoterm® device.