New Zealand Paediatric Surveillance Unit*
STUDY PROTOCOL

Severe Neonatal Hyperbilirubinaemia

****Based on the Australian Paediatric Surveillance Unit Protocol***

BACKGROUND

Cases of cerebral palsy caused by preventable severe neonatal hyperbilirubinaemia have been reported with increasing frequency in Europe and North America.[1-4] There is a paucity currently of accurate severe hyperbilirubinaemia incidence data in New Zealand It is of concern that unless this information is gathered urgently, an increasing number of New Zealand children and their families could be affected unnecessarily by athetoid cerebral palsy.

Extremely high circulating levels of unbound bilirubin in the newborn period can have disastrous effects on the developing brain. Kernicterus, or bilirubin encephalopathy may result from severe neonatal hyperbilirubinaemia and cause debilitating athetoid cerebral palsy, deafness and paralysis of ocular muscles in surviving infants.[1,5,6] Timely recognition and appropriate treatment of newborn babies with hyperbilirubinaemia prevents these sequelae and thus, cases of cerebral palsy due to hyperbilirubinaemia should be avoidable.[7,8] Reasons postulated for the re-emergence of kernicterus in an age of advanced neonatal care are multifactorial and include: early hospital discharge, inadequate community newborn surveillance and lack of education about jaundice and its potential consequences among parents and care-providers.[8,9] On review of the root cause of 125 cases of kernicterus in the United States, Johnson et al found that all cases had 'multiple lapses in the care provided by multiple providers at multiple sites'.[10] Health-system failings included: failure to recognise the significance of early jaundice, failure to institute appropriate monitoring and treatment in addition to inadequate post-discharge follow-up and lactation support.[10]

We aim to establish in New Zealand the current incidence of severe neonatal hyperbilirubinaemia and its sequelae including cerebral palsy and to document the underlying causes and associated clinical risk factors. It is anticipated that these data will inform the development of important future prevention strategies such as screening initiatives and education programs for parents, care-providers and health professionals. This study will also inform future improvements to the continuity and co-ordination of newborn care, particularly after hospital discharge. Ultimately, our study strives to inform eradication of severe neonatal hyperbilirubinaemia and associated disabilities.

STUDY OBJECTIVES

The study aims to describe:

- The current incidence of severe neonatal hyperbilirubinaemia in New Zealand
- The associated diagnoses in affected infants and clinical risk factors
- The type and timing of treatment received
- The short-term* outcomes for each infant
- The proportion of infants in whom severe hyperbilirubinaemia was potentially preventable

*Long term sequelae including developmental outcome will be determined via a separate study

CASE DEFINITION

A newborn infant born **after 34 weeks** gestation and **up to 1 month post delivery** with severe hyperbilirubinaemia defined by:

- A **total** serum bilirubin ≥ 450µmol/L

OR

- clinical and/or MRI imaging evidence consistent with bilirubin encephalopathy

OR

- needing an exchange transfusion for prevention or treatment of bilirubin encephalopathy

REPORTING INSTRUCTIONS

Please report any neonate with severe hyperbilirubinaemia according to the case definition above.

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