Oral Presentation – 10

Blood Lead Level Association with Neurological Features in 972 Children Affected By an Acute Severe Lead Poisoning Outbreak in Northern Nigeria

Paul Dargan[1, 2], **Jane Greig**[3], **Natalie Thurtle**[1], **Lauren Cooney**[1], **Cono Ariti**[4], **Leslie Shanks**[1]
[1]*Médecins Sans Frontières (MSF), Amsterdam, The Netherlands*
[2]*Guys and St. Thomas' NHS Foundation Trust and King's College London, London, UK*
[3]*Médecins Sans Frontières (MSF), London, UK, London, UK*
[4]*London School of Hygiene and Tropical Medicine, London, UK*

Abstract

Objectives: In 2010, *Médecins Sans Frontières* (MSF) investigated reports of high mortality in young children in Zamfara State, Nigeria, leading to confirmation of widespread acute severe lead poisoning in at least seven villages in the region. The aim of this study was to conduct a retrospective analysis to determine venous blood lead level (VBLL) thresholds and risk factors for encephalopathy using MSF programmatic data from the first year of the outbreak response.

Method: For the period June 2010 to the end of June 2011, we included all children aged ≤5 years with a first-ever VBLL ≥45µg/dL recorded before chelation therapy and whose neurological status was recorded within 7 days of this VBLL. Neurological status was described by a composite measure of neurological signs or symptoms and AVPU; severe neurological features were defined as seizures witnessed by medical staff; and/or altered consciousness (an AVPU or V or P or U). Odds ratios (OR) for neurological features were estimated; the final model was adjusted for age and baseline VBLL, using random effects for village of residence. The role of malaria was assessed in patients who had been tested for malaria with a malaria rapid diagnostic test (RDT). Data were analysed using STATA 10.1 (StataCorp, Texas, USA).

Results: 972 children met inclusion criteria: 885 (91%) had no neurological features; 34 (4%) had severe features; 47 (5%) had reported recent seizures; and 6 (1%) had other neurological abnormalities. The geometric mean VBLLs for all groups with vs those without neurological features were >100µg/dL vs 66µg/dL. The adjusted OR for neurological features increased with increasing VBLL: from 2.75, 95%CI 1.27-5.98 (80-99.9µg/dL) to 22.95, 95%CI 10.54-49.96 (≥120µg/dL). Neurological features were more likely in those aged 1-<3 years rather than 3-≤5 years (peak for 1-<2 years OR 4.77, 95% CI 2.50-9.11). Severe neurological features were only seen at VBLL <105µg/dL in those with malaria.

Conclusions: VBLL of ≥80µg/dL, age 1-<3 years and a positive malaria RDT result were strongly associated neurological features. Severe neurological abnormalities were not seen in children with VBLL <105µg/dL in the absence of malaria. These factors will help clinicians managing children with lead poisoning to determine which individuals are likely to be at greatest risk of developing neurological features and therefore potentially life-threatening encephalopathy. This is particularly important when managing large outbreaks for determining urgency of therapy and to help guide chelation protocols.