Acute Necrotising Encephalopathy of Childhood Secondary to Rotaviral Diarrhoea

A one-year-old male child presented with complaints of fever, vomiting and loose stools for 4 days. After 6 days, symptoms subsided but child developed rapid worsening sensorium and hepatomegaly. Investigations revealed marked elevation in serum transaminases (aspartate aminotransferase (AST) 10491 IU/L, alanine aminotransferase (ALT) (8990 IU/L), serum albumin was 2.4 gm/dL, prothrombin time, 32.5 seconds (International normalized ratio (INR) was 2.77, which improved to 1.53 after vitamin K supplements. Serum ammonia was initially 136.7 µg/dL which improved to 23 µg/dL on treatment. Cerebrospinal fluid analysis (CSF) was normal. Dengue NS1, IgM and IgG, HBsAg, hepatitis C IgG, hepatitis A IgM, hepatitis E IgM and ELISA for HIV were negative. Stool rotavirus antigen was positive.

Magnetic resonance imaging (MRI) of brain showed hyperintensities in the bilateral caudate nuclei, putamen, globus pallidus and restricted diffusion in the bilateral basal ganglia suggestive of acute necrotizing encephalopathy. Child was treated with intravenous methyl prednisolone (30 mg/kg/day) for 3 days, followed by oral prednisolone at 2 mg/kg/day for 15 days, which was gradual tapered over next one month. On day 6 of hospitalisation, the child's sensorium improved and he was discharged with feeding tube in situ, with residual neurological deficit, and AST of 81 IU/L and ALT of 1250 IU/L. On follow-up after 45 days of illness, his liver function tests have normalized, he can feed without the feeding tube, can speak monosyllables and can recognize parents.

Acute necrotizing encephalopathy is a para-infectious disease triggered by viral infections, most commonly by influenza and HHV 6 [1-3]. The most likely hypothesis for the pathogenesis of ANE is the exaggerated inflammatory response to viral infection leading to liver dysfunction, acute renal failure, shock, and disseminated intravascular coagulation. In nervous system, the permeability of vessels is altered without vessel wall disruption [3].

Our patient had a history of viral gastroenteritis and the stool rotavirus antigen was positive. Neurological manifestations associated with rotavirus have been described [4]. Thus, we consider rota virus as the possible etiology for ANE. Definitive treatment guidelines for ANE have not been formulated but antiviral therapy, immunomodulatory treatment, antithrombin III, therapeutic hypothermia and cyclosporin A have been variably used [1,3]. ANE is associated with a high mortality and less than 10% of patients recover completely [3]. In conclusion, a high index of suspicion for ANE is needed in a previously healthy child with sudden onset neurological symptoms following acute febrile illness.

VISHRUTHA SUJITH POOJARI, IRA SHAH AND NAMAN S SHETTY*
Pediatric Gastroenterology, Hepatology and Nutrition Department, BJ Wadia Hospital for Children, Mumbai, Maharashtra, India.
**naman.shetty@hotmail.com*

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The Neonatal Resuscitation Protocol: Keep It Simple?

Neonatal intensive care practice has its moments I would say; several actually. Sending a micropreemie home, watching a meconium aspiration pneumonia improve on high frequency ventilation and nitric oxide, managing to insert a life-saving central line catheter into a fine thread like vein, cherishing the normal neurodevelopment of a critically ill infant; all of these and more make the effort worth it. Why then, did the neonatologist in me decide to put myself at risk of criticism with this manuscript which could ripple some still waters? Albeit I am no virtuoso in the field of medical research, having faced the maelstrom of intensive care on my feet for over 10 years, I wish to comment on a guideline that makes a difference to every day practice. The neonatal resuscitation protocol (NRP) for the term newborn, can be described as "daily bread" to the genus of intensivists called neonatologists and has undergone several modifications over the last decades. Thyself followed, with great fervour, the 'reforms' made to the protocol [1]. Over the ensuing paragraphs I intend to raise my reservations on the tipping balance in NRP, between the quest for evidence based practices and pragmatism.