

Infant Feeding in HIV and Gastroesophageal Reflux

We read with interest the IYCF Guidelines 2016 [1] published recently in Indian Pediatrics. We would like to bring to the attention of the readers and policy makers the following issues:

1. Regarding "HIV and infant feeding", point "h" needs attention in light of WHO recommendations and recent literature [2,3]. In its guiding practice statement, WHO clearly states that in mothers living with HIV, mixed feeding is better than "no breastfeeding at all."
2. Mothers living with HIV who plan to return to work/school (increasing trend seen in young Indians), a shorter duration of breastfeeding is better than not initiating breastfeeding at all [2].
3. Regarding "HIV and infant feeding", point "m" dealing with exclusive replacement feeding (ERF), WHO is more emphatic regarding avoidance of animal milk in first six months of life. However, animal milk is a valid option for ERF in children above six months [2].
4. Regarding "Infant feeding in various conditions related to the infant", point (iv) deserves attention as recent literature shows that "upright positioning for 30 minutes after feeds" does not hold true in reducing gastroesophageal reflux (GER) [4]. The prone

position was superior to the supine or upright positions while patients were awake or asleep. However, prone position cannot be recommended due to its association with sudden infant death syndrome (SIDS). The policy statement on task force on SIDS recommends supine position for infants including infants with GER [4].

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Editor's note: *The corresponding author of the guidelines in question did not provide a point-by-point response to these queries, and stated that these issues will be addressed at the time of next revision of guidelines.*

Need for Revision of Guidelines for Management of DR-TB in Children

Overall prevalence of drug resistant tuberculosis (DR-TB) in pediatric patient is increasing [1]. The Directly Observed Treatment, Short Course (DOTS) strategy has emerged as a possible solution to the rising number of tuberculosis (TB) cases and has been incorporated in India's Revised National Tuberculosis Control Programme (RNTCP) as well. RNTCP multidrug resistant (MDR) TB treatment regimen consist of 6 drugs [kanamycin (km), levofloxacin (lvx), ethionamide (eto), pyrazinamide (Z), ethambutol (E) and cycloserine (cs) during 6-9 months of an intensive phase and 4 drugs (lvx, eto, E and cs) during the 18 months of the continuation

phase [2]. In recent studies it has been found that there is increasing fluoroquinolone and ethionamide resistance [1].

At our institute, we had a 10-year-old boy who was diagnosed with rifampicin-resistant (RR) miliary tuberculosis with pneumothorax by GenXpert on sputum sample. We started the child on second-line anti-tubercular therapy (ATT) consisting of moxifloxacin, amikacin, PAS, cs and clofazimine along with prednisolone, based on our previous experience with prevailing DR-TB in Mumbai and the sensitivity pattern [1]. Child improved on above regimen and was discharged with advice to follow-up with drug sensitivity testing (DST) report. After 15 days of above regimen, during a visit to a DOTS center for the medicines, drug regimen was changed to lvx, eto, Z, E, cs and km. After one week of above regime, child's condition deteriorated, and he

developed fever, headache and altered sensorium with signs of raised intracranial pressure. On neuroimaging, he had multiple tuberculomas with communicating hydrocephalus. Child was started on previous ATT regimen that we had put him on initially, and he was treated with dexamethasone along with 3% sodium chloride for his raised intracranial tension. Subsequently, his DST report arrived which showed resistance to rifampicin, isoniazid, km, ofloxacin, E and eto with sensitivity to moxifloxacin, Z, linezolid, PAS, amikacin, clofazimine and capreomycin. Thus as per the medicines that he was receiving from DOTS, the child would be receiving only two drugs (Z and km) to which the DST showed sensitivity with all the other drugs being resistant. This could have led to worsening of his clinical condition. Though it may be argued that appearance of tuberculomas may suggest a paradoxical reaction, the child even after one month of hospitalization was bed ridden, and needed a ventriculoperitoneal shunt for his

hydrocephalus suggesting that he developed CNS TB as part of his worsening of TB.

Thus, there is a need for revision of national guidelines for management of DR-TB patients to avoid worsening of the disease condition.

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Bartter Syndrome with Nephrogenic Diabetes Insipidus and Vitamin D Resistant Rickets

A 1-year-old boy presented with polyuria, polydipsia and poor weight gain noticed since three months of age. He weighed 5 kg (-3SD), Height 64 cm (-3 SD), and had features of some dehydration. Hyponatremia (165 mEq/L) and metabolic acidosis (pH 7.27, bicarbonate levels 17 mEq/L) were detected. Serum creatinine, potassium, calcium, magnesium and chloride levels were within normal reference ranges. Serum osmolality and urine osmolality were 355 mOsm/L and 145 mOsm/L, respectively. He was diagnosed as nephrogenic diabetes insipidus after a vasopressin challenge test failed to increase the urine osmolality levels. Renal ultrasonography was normal. He was treated with spironolactone.

At the age of 3 years, he presented with rickets and hypocalcemic tetany (ionized calcium 2.2 mg/dL) in association with hypophosphatemia (2.2 mg/dL) and secondary hyperparathyroidism (PTH levels 180.2 pg/mL). The rickets was refractory to therapy with Vitamin D; and the child developed fractures of bilateral ulnae and femur requiring hip spica and plaster casts. He was still showing poor weight gain (weight 7.9 kg, -3SD).

Triangular facies, prominent eyes and forehead, and large ears were appreciated. Blood pressure was normal. At this juncture, he was found to have hypokalemia (2.5 mEq/L), metabolic alkalosis (pH 7.52, bicarbonate levels 35.2 mEq/L) and hypercalciuria (spot calcium: creatinine ratio 1.4). Serum magnesium and creatinine levels were normal; urine chloride was >20 mEq/L. Plasma renin activity was high (38.9 ng/mL/h), confirming Bartter syndrome. Wrist X-ray showed metaphyseal cupping and splaying. Serum 25 hydroxycholecalciferol levels were 31.4 ng/mL. He is currently on potassium chloride (8 mEq/kg/day), indomethacin (2 mg/kg/day), enalapril (0.5 mEq/kg/day), and calcium supplements. At the last follow up at age of 4 years, his serum potassium, sodium, creatinine, calcium and phosphate levels are normal, and he is showing satisfactory weight gain.

The presentation of this child with Bartter syndrome is unusual for two reasons. The first being the initial paradoxical presentation with hypernatremic dehydration and metabolic acidosis; the second being the association with vitamin D resistant rickets (leading to secondary hyperparathyroidism). The former presentation has been anecdotally reported in the literature [1,2]. Bettinelli, *et al.* [1] reported a child who presented with severe hypernatremia, who was initially diagnosed as nephrogenic diabetes insipidus, but on follow up was diagnosed as Bartter syndrome. They concluded that in a few cases of Bartter syndrome, hypokalemia and/or metabolic alkalosis may be absent during the initial few