Etiology and clinical profile of childhood optic nerve atrophy at a tertiary eye care center in South India

Sir,

I have two comments on the interesting study by Chinta *et al*.[1]

First, Chinta *et al*.[1] addressed that hypoxic ischemic encephalopathy (HIE) seen in 41% of patients was the most frequent cause of childhood optic nerve atrophy (ONA), followed by idiopathic (30%), hydrocephalus (7%), compressive etiology (5%), infective (6%), congenital (2%), inflammatory (2%), respectively. I presume that the actual prevalence of HIE in the studied cohort is higher than 41% already reported. This is based on the following two points. (1) It is obvious that HIE is a common and serious complication of prematurity. The increasing rates of preterm births coupled with better survival of these infants have resulted in the higher prevalence of systemic and ocular complications associated with prematurity. In addition to retinopathy of prematurity, infants who are born preterm may suffer from severe visual impairment as a result of HIE, hypoglycemia, and other metabolic imbalances. The effect of these processes on the anterior visual pathway may result in optic atrophy, optic nerve hypoplasia or optic disc cupping and affection of the posterior visual pathway leads to cortical visual impairment. Other ocular associations include strabismus, nystagmus, and ocular motor abnormalities such as tonic down gaze and defective saccades and pursuits.[2] In the last decade, improved survival of infants with low birth weight (LBW) has been achieved in many developed countries. However, Indian subcontinent has still the highest child mortality rates along with a very high frequency of prematurity and LBW. Maternal education, exposure to passive smoking, age at first pregnancy ≥25 years, birth interval <2 years, previous history of LBW baby, weight gain ≤4 kg during pregnancy, maternal weight at last week of gestation ≤45 kg, pregnancy induced hypertension, high risk pregnancy, and late antenatal registration emerged as significant risk factors contributing to prematurity and LBW.[3] Apart from the aforementioned risk factors and constrained socioeconomic standards, consanguinity, as a common means of family property retention, has significantly contributed to the increased pregnancy loss, prematurity, and birth weight <2500 g.[4] (2) Prematurity and LBW are generally associated with short- and long-term sequelae. Hence, they could operate leading to ONA through various contributions, notably traumatic, congenital, hydrocephalus, vascular, compressive, and infectious.

Second, it is obvious that ONA is among ocular manifestations of human immunodeficiency virus (HIV) infection. In India, HIV infection is on the rise among children. The estimated number of people living with HIV in India has been reported to be 2.4 million (1.93–3.04 million) in 2009 and children under 15 years of age accounted for 4.4% of all infections.[5] Surprisingly, no case of HIV-associated ONA was reported among Chinta *et al*.'s.[1] studied cohort. I presume that limited awareness on pediatric HIV infection and constrained financial resources were the main factors that hampered arranging for CD4 (+) T-lymphocyte counts and viral overload estimation among Chinta *et al*.[1] studied population.

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