UNRAVELING THE CONNECTION BETWEEN CORTISOL AND PEDIATRIC IDIOPATHIC INTRACRANIAL HYPERTENSION

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Background: Idiopathic intracranial hypertension (IIH) is a condition of elevated intracranial pressure without identifiable secondary causes. The childhood incidence is 0.7/100,000 and increases with age, obesity, and female gender. Few case reports in the literature, and our own experience, suggest there may be an association between IIH and adrenal insufficiency (AI) but the real extent is unknown.

Materials/Methods: Retrospective chart review identified all children who presented with IHH and had cortisol measured between January 2010 and September 2019. Based on morning, random or 1 mcg ACTH stimulated cortisol levels, adrenal functioning was classified as: (1) deficient (peak cortisol <16 µg/dl, AM cortisol <5 µg/dl), (2) at risk (peak cortisol 16 - 20 µg/dl, AM cortisol 5 - 13 µg/dl or random < 13 µg/dl), or (3) sufficient (peak cortisol >20 µg/dl, AM or random cortisol >13 µg/dl). Descriptive data present mean (SD), and $\chi 2$ tests of differences are used to examine differences between groups.

Results: Participants (N=64) were 40.6% male, of mixed ancestry, with a mean age of 10.8 (4.8) years. Cortisol levels were obtained at an average of 0.6 (1.9) years after diagnosis of IIH; 23% and 52% of patients had insufficient or at risk cortisol levels. The majority of those in the insufficient (70%) or at risk (80%) groups were exposed to topical, nasal or inhaled corticosteroids, but not systemic. Only 60% and 12 % of those with IIH and insufficient or at risk cortisol testing, respectively, underwent definitive testing with a stimulation test. Adrenal function did not differ by age, race/ethnicity, zBMI, nor prolong exposure to steroids (> 2 weeks), time between IIH diagnosis and cortisol testing. Those in the deficient group were less likely to be female (33%) than those in the at risk (61%; P=.001) or sufficient (81%; P<.001) groups. Those with AI were more likely to have history of asthma (53%; vs: 18% at risk and 12% normal; both P<.05)

Conclusions: Steroid use and AI are common in IIH and need consideration as a cause of IIH. Appropriate diagnosis and treatment of AI in children who present with IIH may lead to its resolution, significantly impacting clinical outcomes of these children. In our cohort, the majority had AI or at risk cortisol levels, and many did not undergo further testing. All young children who present with IIH should be evaluated for steroid exposure, including non-systemic steroids, and undergo evaluation for AI. Caution should be utilized by providers prescribing these drugs.