ENDOCRINOLOGY E09 LOW/MEDIUM DOSES OF INHALED CORTICOSTEROIDS CAN CAUSE ADRENAL INSUFFICIENCY.

J. FUDVOYE [1], M. LEBRETHON [1], A. PARENT [1].

[1] CHU Liege, Liège, Belgium, Pediatric endocrinology/diabetology

Results: We report the cases of three children treated with inhaled corticosteroids (ICS) for asthma presenting adrenal suppression (AS) associated with features of cortisol deficiency, including non-specific symptoms (fatigue, growth retardation,...) but also life-threatening conditions (hypoglycemia, coma). ICS are commonly prescribed to asthmatic children. Until recently, a dose lower than 500 µg/day of fluticasone proprionate was considered to be safe regarding the risk of AS. However, recent literature reported children who presented adrenal insufficiency when using 500 µg or less/day of Fluticasone Proprionate. Case 1: A five year-old boy was referred to the endocrine clinic for growth evaluation. He presented recurrent respiratory infections treated by Fluticasone, 500 µg daily. Patient's growth parameters demonstrated short stature (-2.7 SD for height) and decreased growth velocity. Glucagon stimulation testing showed undetectable cortisol (<0.1 µg/dl) with morning ACTH very low (3.1 pg/mL). Because of the ACTH deficiency, hydrocortisone therapy was initiated. Brain nuclear magnetic resonance was normal. After reducing the dose of ICS, the dose of hydrocortisone was slowly decreased with progressive elevation of cortisol and ACTH on blood samples collected at 8 am (ACTH: 33.8 pg/mL et Cortisol à 8 µg/dl). An ACTH stimulation test showed a good response in cortisol (16.5 µg/dL after one hour). Case 2: A five year-old girl presented growth retardation. She was receiving 1 inhalation, twice daily, of a fixed combination of Salmeterol 25 µg/fluticasone 250 µg for persistent asthma. Patient's growth parameters demonstrated relatively short stature (-1.6 SD) and a decreased growth velocity (-2.9 SD). Glucagon stimulation test showed a very low basal cortisol at 5.2 µg/l with ACTH < 3.8 ng/l with cortisol reaching a maximum of 107.3 µg/l. Treatment with Seretide was then discontinued and clinical examination 6 months later revealed a significant increase in growth velocity (9.2 cm/year). Case 3: A 4.5 year-old boy presented severe hypoglycemia (10 mg/dl) with coma. He had been found unconscious in his bed in the morning. He had fever and low appetite the previous day. Glucose infusion was given with rapid clinical improvement. Cortisol level collected during hypoglycemia was 13,8 µg/dl. Insuline was undetectable. Clinical examination was without particularity except palor. Glucagon stimulation test showed low basal cortisol at 2 µg/L and low ACTH at 4.2 ng/L without elevation of cortisol (13.2 µg/L). Hydrocortisone therapy was started with an initial dose of 10 mg/m2 of body surface area. Brain nuclear magnetic resonance was normal. The boy was treated for persistent asthma with 2 inhalations, twice daily, of a fixed combination of Salmeterol 25µg/fluticasone 125 µg. Doses of Seretide are currently being decreased and adrenal function is recovering. Sensitivity to AS associated with ICS is difficult to predict in children. This sensitivity might involve genetic factors which are still unknown. Millions of children receive long term ICS to control asthma, however, clinically symptomatic AS is uncommon. This condition can lead to potential serious complications, especially in stress conditions. In order to prevent adrenal insufficiency, the minimal required dose of ICS should be defined and at risk children should be identified.