Transient congenital hypothyroidism: About 6 cases

IINTRODUCTION

Transient neonatal hypothyroidism (T N HT) is a rare entity important to recognize. It is due to placental transfer of antibodies antirécepteurs of pituitary TSH.
It is distinguished from permanent congenital forms of hypothyroidism, because it requires only limited substitution treatment in time.
OBJECTIVE : /Report the observations of 6 children who presented T N HT

Population, methodology

This is a retrospective study of six children(with two brothers) hospitalized for suspiscion of hypothyroidism. All children underwent a complete clinical examination with questioning of the parents, hormonal exploration (FT4, TSH, AC Anti-TPO), and cervical ultrasound .Once the diagnosis established, treatment with levothyroxine is undertaken. Regular reassessments were performed.

RESULTS

The average age at diagnosis is 3 months (20 days-5 months). Sex ratio F / G is 4/2. Hypothyroidism was diagnosed in front of clinical signs in 4 cases, persisting neonatal jaundice in 2 cases. Biological assessment confirmed hypothyroidism: FT4 average 6,5 pmol/L. , mean TSH 35 mUI/L. TPO antibodies (TPO) were found in all cases (mean 30 ng / ml). Questioning the parents revealed an autoimmune thyroiditis in mothers . Clinical and radiological examinations did not show any malformation. LT4 replacement therapy undertaken was screeching halt after 4,85 months (3- 6). Hormonal reassessments showed no abnormalities and anti TPO were négatives. Growth stature and psychomotor development were normal

DISCUSSION

The transient congenital hypothyroidism is a form of congenital hypothyroidism due to a non-permanent deficit in thyroid hormones. It is more common in iodine deficiency areas. The symptoms may be those of the permanent HC, but are usually less marked and transient; transient HC can also be asymptomatic.

Possible causes transient HC are multiple. Placental transfer of maternal anti-TSH receptor antibodies (thyroid-stimulating hormone) can cause blockage of thyroid receptors in the newborn, lasting up to 3 to 6 months after birth, duration necessary to the fall of maternal antibodies. Maternal treatment with antithyroid drugs can decrease neonatal thyroid hormonosynthèse for a few days to two weeks after birth. Mutations in the gene DUOXA2 (15q15) are responsible for iodine organification abnormality; a partial organification defect may cause a transient HC, but these mutations may be responsible for a form of permanent thyroid dyshormonosynthèse by HC. Neonatal exposure to iodine (especially premature infants) can also develop a transient HC. The HC insufficiency / excess iodine intake (occurs in fetuses or newborns whose mothers were exposed to inappropriate amounts of iodine during pregnancy can manifest as a thyroid deficiency and goiter. An overproduction of enzyme iodothyronine deiodinase type 3 can be observed in case of congenital hepatic hemangioma, originally a consumer hypothyroidism, characterized by low levels of thyroxine (T4), a high TSH and an increase in reverse triiodothyronine (rT3)

Regular monitoring of serum TSH during treatment with L-thyroxine helps differentiate HC transitional forms of permanent forms and adapt the treatment. In the absence of proof of permanent HC at age 2 or 3 years (elevated serum TSH greater than 20 mU / L sub therapeutic dosing), a therapeutic window of 30 days is recommended. If serum T4 or free T4 remain normal, the HC is supposed transient and treatment is no longer necessary. The prognosis of children treated early is excellent

CONCLUSION

GHD rarely is rarely revealed to the neonatal period. The existence of signs of other hormone deficiencies and M A should evoke it precociously

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