

# TRIAC TREATMENT RESPONSE TO THYROID HORMONE RESISTANCE

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## INTRODUCTION

Resistance to thyroid hormone (RTH) is a disease characterized by decreased sensitivity to thyroid hormone in the peripheral tissues such as cell membrane, metabolism, or nuclear receptor. In THR $\beta$  gene mutation induced RTH, the effect of T<sub>3</sub> on TR $\beta$  mutant receptors in the liver and the pituitary decreased, on TR $\alpha$  receptors located in the brain and heart increased. Triac's activity is similar

## CASE

A 1-month-old girl patient was admitted because of detection of elevated TSH level in the national screening program. With regards to her family history there was no thyroid disease in the family and in the mother of the patient during pregnancy. The patient's physical examination revealed a live gaze, anterior fontanel 3x3 cm, body weight of 3500g (10-25 p), height 52 cm (25-50 p), pulse: 190/min, blood pressure : 90/pulse, thyroid stage 0, and puberty tanner stage 1. In his laboratory, sT<sub>3</sub>: 8.62 pg/ml (2.76-4.38), sT<sub>4</sub>: 3.31ng/dl (0.75-1.49), TSH: 7.86  $\mu$ IU/ml (0.77-5.64), thyroglobulin: 105 ng/dl(1.6-59.9), Iodine(urine): 12.7 ug/dl, TSH receptor antibody (TRAB): 8.36 U/L (0-14), thyroid autoantibodies were negative. Her mother tested normal for thyroid functioning. In the genetic analysis of the patient suspected of thyroid hormone resistance, the P453H c.1358C>A mutation was detected heterozygously at the 10th exon of the THR $\beta$  gene. Triac therapy began at 0.5 mg/kg/ day (1750 mcg/ day) In the follow up examination, the patient's pulse rate decreased to normal and sT<sub>3</sub>: 6.09 pg/ml (2.76-4.38), sT<sub>4</sub>: 1.41 ng/dl (0.75-1.49),TSH: 7.44  $\mu$ IU/ml (0.77-5.64).

## CONCLUSION

In RTH due to THR $\beta$  gene mutations, Triac binds to mutant TR $\beta$  receptors, reducing the effect of TR $\alpha$  receptors on the decrease in thyroid function tests and the decrease in fT<sub>3</sub> levels. Like in our case, cases of high levels of fT<sub>3</sub> have been reported in the literature when clinical recovery was obtained with Triac therapy. This effect is thought to be secondary to the cross-reaction. In view of this, or this reason, triac dose titration should take into account clinical improvement as well as thyroid function tests.

