**A series of 9 pregnancies with hyperthyroidism and Graves Disease: fetal and maternal follow up**

P. DELANNOYa, S. GRANDFILSb, M-C LEBRETHONc , F. CHANTRAINEd, C. VAN LINTHOUTb, A. BECKERSa  and H. VALDES SOCINa  
a Endocrinology, CHU Liège; b Gynecology, CHU Liège; C Pediatry, CHU Liège ; Gynecology CHR Liège.

**Introduction:**

Graves disease (GD) is an hyperthyroid auto-immune disorder. The presence of thyroid stimulating antibodies (TSAb) may lead to hyperthyroidism, thyroid orbitopathy and pretibial myxedema. GD complicates approximately 0.2% of pregnancies, carrying a risk of fetal thyroid disorders due to the transplacental transfer of thyroid-stimulating/blocking antibodies and eventually the transfer of maternal anti thyroid drugs .

**Case report**

We report a series of nine pregnancies (six patients) with Graves Disease, TSAb and fetal repercussion.

One patient had thyroidectomy, three were treated by I131 before pregnancy, and two were diagnosed during pregnancy both with cardiac failure and pulmonary hypertension due to thyrotoxicosis. Three patients presented graves ophtalmopathy.

The median age for pregnancies was 30 (28 – 33) years. The median age of gestation for the primary evaluation was 9 (7-29) weeks of gestation (WG).

The titer of TSAb are available only for five cases and during the first trimester the titer was in excess of three times the upper limit of normal. We observed a decrease of titer in three pregnancies with one normalisation

The average gestational age at delivery in our series has been 36 to 37 weeks. Two patients were managed after term and their babies presented with neonatal thyrotoxicosis. Two fetus presented a goiter that required cordocentesis, revealing hyperthyroidism. Adaptation maternal anti thyroids allowed normalize the fetal thyroid volume. No malformations was noted after birth . Six babies presented neonatal thyrotoxicosis : Five was treated by Strumazol and one by Propylthiouracil during few months.

**Discussion**

The fetal thyroid gland acquires the ability to synthesize thyroid hormones at roughly 12 weeks of gestation. Maternal TSAb may cross the placenta and stimulate the fetal thyroid gland, leading to excessive thyroid hormone secretion and goiter. The risk correlates with the TRAb titer (high if TRAb titers > 3 times the upper limit of normal).

Untreated GD hyperthyroidism is associated with maternal congestive heart failure, preeclampsia, prematurity, growth retardation, malformation and stillbirth. Umbilical blood sampling is recommended if maternal thyroid-stimulating antibody level is high, if fetal tachycardia and/or goiter to improve the control of fetal thyroid function

Antithyroid drugs (ATDs) are the mainstay of treatment for gestational hyperthyroidism due to GD. ATDs also cross the placenta and are effective on fetal thyroid hormone production.

Guidelines recommend the use of the lowest effective dose of ATDs to maintain maternal serum FT4 at or moderately above the upper limit if the reference range.

TSH and thyroid hormones should be checked every 2–4 weeks. After the completion of the first trimester, PTU must be changed to MMI or Carbimazol to reduce the risk of liver toxicity.

**Conclusion**

GD during pregnancies must be closely monitored by a multidisciplinary in order to improve maternal and fetal outcome.