Graves’ Disease and Pregnancy
Summarized by Susan E. Calico

A woman with Graves’ disease faces difficult choices when she considers pregnancy. Though the fetus is at far more risk than the mother, both may be temporarily or permanently affected by her condition and its treatments.

Unfortunately, there is no way to monitor or predict the effect on the fetus of any treatment given to the mother. The few studies on Graves’ pregnancies are so few and small that the results are inconclusive. Even after birth, neither infant appearance nor blood tests are conclusive predictors of the child’s future.

Risks of Pregnancy

The first precaution is to normalize the mother’s condition before conception. Women using anti-thyroid drugs should establish a stable maintenance dose of PTU. For women treated with surgery or radioactive iodine, replacement thyroid hormone dose should be stabilized. Waiting six months after radioactive iodine treatment to conceive minimizes the effects of the radiation on developing eggs.

But the child can be affected even when the mother’s Graves’ is well under control. No matter how long ago they were diagnosed and treated, most women with a history of Graves’ have measurable levels of thyroid-stimulating antibodies (TSAb, TBII and LATS) present in their blood. These antibodies can cross over the placental barrier and cause Graves’ symptoms in the fetus. The symptoms – hyperthyroid, goiter, bulging eyes – usually disappear within a few months after birth, when the baby is no longer exposed to the antibodies. But in some, the symptoms linger for years or reoccur later in childhood. In these cases, the mother’s antibodies may have triggered Graves’ disease in the child.

Poorly Monitored Graves’

Because pregnancy “stirs up” the thyroid, even women who presumably have little of their thyroid left after radioactive iodine or surgery can’t rule out a hyperthyroid episode. The normal elevation of thyroid hormones in pregnancy can mask a recurrence for women with a history of Graves’. For this reason, close monitoring of fetal signs and maternal blood levels (every 2 to 4 weeks) is important.

Though neither synthetic replacement hormone nor the mother’s natural thyroid cross the placental barrier, too much or too little can have serious indirect affects on the fetus. Hypothyroidism (or undertreatment with replacement hormone) causes prolonged gestation and underdevelopment of the fetus. Hyperthyroidism (or overmedication with replacement hormone)
imperils both the mother and the fetus. These infants are more likely to die shortly before and after birth. Premature delivery is likely, though the infant will have fewer “preemie” problems because its development is accelerated by the excess thyroid hormones. And later on, the child may have shorter stature and subtle nervous-system defects. The mother risks the life-threatening thyroid storm, especially during labor and delivery.

**Controlling Graves’ During Pregnancy**

If Graves’ reoccurs or first occurs during pregnancy, the options are limited by the direct and indirect effects of many drugs on the fetus. Tapazole™ should be avoided during pregnancy and breastfeeding because it can suppress the infant’s thyroid, but PTU in similar doses has more moderate and less permanent effects. Dosage of PTU should be limited to less than 300 mg during the first and second trimester, PTU can usually be stopped during the third trimester as the natural suppression of the immune system during pregnancy causes a gradual remission of Graves’ symptoms. PTU is not concentrated in breast milk, so moderate doses of 200 mg or less are thought to have little effect on nursing infants.

Thyroid surgery is another option for controlling Graves’ during pregnancy. The surgery is usually performed in the second trimester to maximize fetal survival.

Management of Graves’ during pregnancy is complicated by the crossing-over of some thyroid-related medications to the fetus. Iodine treatment is the most dangerous of these, whether radioactive iodine or iodine drops (used to temporarily suppress the thyroid). After 10 weeks gestation, iodine passes easily to the fetus, with high rates of death; radioactive iodine results in destruction of the fetus’ thyroid and parathyroid, and sometimes chromosomal damage. Therefore, any treatment or testing with iodine during pregnancy should be strictly avoided.

Propranolol (beta blocker) can also affect the fetus and infant, suppressing heart rate, respiration and growth, and affecting the liver and glucose metabolism. Its use is avoided during pregnancy and breastfeeding.

**Source:**
“Hyperthyroidism is Pregnancy” by Dorothy R. Hollingsworth, published in Werner’s *The Thyroid* ed. Sidney H. Ingmar and Lewis E. Braverman, Summarized by Susan Elisabeth Calico

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