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Hyperthyroid in Pregnancy: A Case Report

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ABSTRACT
Hyperthyroidism in pregnancy is generally caused by Grave's disease which can cause thyroid crises, premature, abortion and fetal death. Graves disease often becomes more severe in the first trimester of pregnancy and will experience an exacerbation in the postpartum period. A 35-year-old woman with a third pregnancy 23-24 weeks with fetal death in the womb. The patient has been suffering from Grave's disease for 10 years but has not taken the drug for the past 1 year. No history of trauma and bleeding. FT4 levels 75.62 pmol / L and TSHs levels 0.005 µIU / mL. Wayne's index score is 23 and the Burch Wartofsky scale is 45 or impending thyroid storm. Management of hyperthyroidism is given before termination of pregnancy. Pulmonary breath shortness at two hours after delivery and given a diuretic and vasodilator. Supervision is carried out until the patient's condition improves. The function of the thyroid gland in pregnancy is increased which is influenced by increased levels of thyroxin binding globulin (TBG), thyroid-stimulating factors (TSF) from the placenta and decreased iodine supply in the thyroid gland. These complaints are more severe in the first trimester of pregnancy and will experience an exacerbation in the postpartum period. In inadequate treatment, hyperthyroidism complicated to maternal heart failure and fetal death. Hyperthyroidism can cause complications in the mother and fetus and increase morbidity and mortality. Proper initial treatment will improve outcomes.

Keywords: Pregnancy, hyperthyroidism, complications.

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INTRODUCTION
Hyperthyroidism is hyperfunction of the thyroid gland characterized by a 15-20% increase in basal metabolism, sometimes accompanied by a mild enlargement of the thyroid gland. About 90% of hyperthyroidism is caused by Grave's disease, both solitary and multiple toxic nodules and toxic adenomas. Grave's disease is generally found at a young age that is between 20 to 40 years and more often found in women than men with a ratio of 5: 1.[1]
Hyperthyroidism is estimated at 2: 1000 of all pregnancies, but if it is not controlled it can cause thyroid crisis, premature labour abortion and fetal death. Graves disease often becomes more severe in the first trimester of pregnancy and will experience an exacerbation in the postpartum period. [2] In this case, pregnant women with fetal death and postnatal complications caused by hyperthyroidism.

CASE REPORT
A 35-year-old pregnant woman came to the emergency department with complaints of fetal movements not felt since 1 day ago. At present the patient is the third pregnant at the age of 23-24 weeks, so far the regular control to the midwife and specialist doctors. The patient has never experienced bleeding or trauma before. A history of previous labour was normal with a healthy baby. The patient has been suffering from Grave's disease for the past 10 years and has not been treated for one year. During pregnancy, the patient feels palpitations and tightness during activity. The patient also complaint about weight loss and fatigue.
On physical examination, the patient's general condition appeared weak with a blood pressure of 197/87 mmHg and pulse 148 times per minute regular. The exophthalmos and the thyroid gland feel soft in the neck without pain. Laboratory tests found an increase in FT4 levels of 75.62 pmol / L and low TSHs levels of 0.005 µIU / mL. Wayne index with a value of 23 or found signs of toxicity and the Burch Wartofsky scale with a value of 45 or impending thyroid storm
Patients diagnosed with Grave disease in pregnancy with impending thyroid storm and IUFD at 23-24 weeks gestation. The patient was taken to the endocrine section and given methimazole 30 mg twice daily, propranolol 30 mg twice daily, and Lugol 5 drops per 6 hours. The termination of pregnancy is carried out by vaginal delivery and administration of oxytocin postpartum.
The patient complained of shortness of breath and anxiety after two hours postpartum. Patients were consulted in the Cardiology division with pulmonary oedema and advised giving diuretics and vasodilators. Patients were given nitroglycerin at a dose of 5 meq per hour and furosemide 30 mg per hour intravenously to improve the condition of pulmonary oedema Close monitoring is carried out on the patient for several days until the patient's condition is stable. Patients are planned to go home for outpatient care after monitoring side effects and postpartum complications. Hemodynamic condition is stable with blood pressure 110/70 mmHg and pulse 92 times per minute. Patients were given anti-thyroid therapy and beta-blockers on discharge. During treatment at home, patients are advised to monitor thyroid function to the clinic every once a month.

DISCUSSION
During pregnancy, the thyroid gland's physiology increases and in many ways the activity of the thyroid gland resembles that of hyperthyroidism. Before the development of chemical measurement techniques for the physiology of the thyroid gland, people assumed that the occurrence of goitre and an increase in basal metabolism in pregnant women were due to hyperactive thyroid gland. This assumption is based on histological features in the form of hypertrophy and hyperplasia of the thyroid gland follicles in pregnant women. From various studies show that the

prevalence of goitre during pregnancy varies geographically.[1,2]

In a Scottish study, 70% of pregnant women developed goitre, more than non-pregnant women (38%). In contrast to studies in Iceland, where there was no increase in the incidence of goitre during pregnancy. Also, a study in the United States showed no increase in the incidence of goitre in pregnant women. From the results of the study note that this is due to the iodine content in Iceland and the United States is higher than in Scotland.[3]

According to Glamour, pregnancy is a unique condition, in which the thyroid gland's physiology is affected by 3 changes, 1) a change in thyroid size due to increased levels of thyroxin binding globulin (TBG) in response to increased estrogen levels and increased levels of iodine binding protein starting weeks 12th which reaches 2 times normal levels which will increase T4 and T3 levels in serum; 2) an increase in the secretion of thyroid-stimulating factors (TSF) from the placenta especially human chorionic gonadotropin (HCG); and 3) pregnancy is accompanied by a decrease in iodine supply in the thyroid gland due to an increase in renal clearance of iodine and iodine loss through the photo-placental complex at the end of pregnancy so that it will cause a relative iodine deficiency state. [4]

A person with known Grave hyperthyroidism, a classic clinical characteristic can be used as a guide for diagnosis. Clinical signs that can be used as a guide for diagnosis are the presence of tremors, non-infiltrative or infiltrative eye disorders, weight loss without knowing why, local myxedema, myopathy and onycholysis. All of these conditions never occur in a normal pregnancy. If the resting pulse exceeds 100 times per minute and does not slow down with Valsalva manoeuvring, this gives a strong possibility of hyperthyroidism. Pregnant hyperthyroidism patients can experience hyperemesis gravidarum which can only be treated with anti-thyroid medications.[5]

Grave's disease often becomes more severe in the first trimester of pregnancy, so the highest incidence of hyperthyroidism in pregnancy will be found especially in the first trimester of pregnancy. Until now the cause is not known with certainty. At an older gestational age, Grave's disease has a tendency to remission and will experience an exacerbation in the postpartum period.[4]

In pregnancy, there will be a decrease in the maternal immune response which is thought to be caused by increased fetal suppressor T cell activity that exerts suppressor factors. These suppressor factors cross the placental barrier thereby suppressing the mother's immune system. After the placenta is released, these suppressor factors will disappear. This can explain why the exacerbation of hyperthyroidism occurs in the postpartum period. After delivery, there is an increase in TSAb levels which peak 3 to 4 months postpartum. This increase can also occur after abortion. A survey conducted by Amino et al. (1979-1980) showed that 5.5% of Japanese women suffer from postpartum thyroiditis. The clinical features of postpartum thyroiditis are often unclear and difficult to detect. Postpartum thyroiditis usually occurs 3-6 months after delivery with clinical manifestations of transient hyperthyroidism followed by hypothyroidism and then spontaneous recovery.[6]

Hyperthyroidism will cause various complications both for the mother and fetus and the baby to be born. The state of hyperthyroidism in pregnancy can increase serious maternal morbidity, especially heart trouble. The exact mechanism of hemodynamic changes in hyperthyroidism is still confusing. There is plenty of evidence that the long-term effects of increasing levels of thyroid hormone can cause myocardial damage, cardiomegaly and ventricular dysfunction. Thyroid hormones can affect the myocardium directly or indirectly.[7]

Clinically there will be an increase in the ejection fraction at rest, where this can also be caused by the pregnancy itself. Ventricular dysfunction will get worse if accompanied by anaemia, preeclampsia or infection. These risk factors often occur together in pregnant women. Davis et al. mentioned that heart failure is more common in pregnant women with uncontrolled hyperthyroidism especially in the last trimester.[4]

One serious complication that can occur in pregnant women with hyperthyroidism is a thyroid crisis. This can occur due to precipitating factors including labour, operative measures including cesarean section, trauma and infection. In addition thyroid crisis can also occur in pregnant hyperthyroidism patients who are not diagnosed or receive inadequate treatment.[5]

According to Davis LE et al's report, of 342 pregnant hyperthyroidism sufferers, thyroid crisis occurred in 5 patients who had received anti-thyroid treatment, 1 patient who received operative therapy, 7 patients who were undiagnosed and not treated. Thyroid crises are characterized by severe manifestations of hyperthyroidism and hyperpyrexia. Body temperature can increase to 41oC accompanied by anxiety, agitation, tachycardia, heart trouble, nausea, vomiting, diarrhoea, delirium, psychosis, jaundice and dehydration.[5]

Antithyroid drugs that are widely used are the group of thioamides which work to inhibit thyroid hormone synthesis through the blockade of the tyrosine molecule iodination process. Anti-thyroid drugs are also immunosuppressive by suppressing the production of TSAb through their work affecting the activity of thyroid gland lymphocyte T cells. Because this drug does not affect the release of thyroid hormones, the clinical response only occurs after thyroid hormones stored in colloids are used up. So the time needed to reach the euthyroid state depends on the number of colloids contained in the thyroid gland. In general, clinical improvement can be seen in the first week and a new euthyroid state is reached after 4-6 weeks of treatment.[8]

Propylthiouracil (PTU) and methimazole have been widely used in pregnant women with hyperthyroidism. However, PTU has many advantages compared to methimazole, including PTU can inhibit the change of T4 to T3 besides inhibiting thyroid hormone synthesis and crossing the placenta less than methimazole because PTU has strong protein binds and is difficult to dissolve in water. In early pregnancy before the formation of the placenta, a dose of PTU can be given as in a nonpregnant state, starting from a

dose of 100 to 150 mg every 8 hours. After the controlled situation is indicated by clinical improvement and a decrease in serum T4 levels, the dose should be reduced to 50 mg 4 times a day. When euthyroid conditions have been reached, the dose of PTU is given 150 mg per day and after 3 weeks 50 mg is given 2 times a day. Examination of serum T4 levels should be done every month to monitor the course of the disease and treatment response. In the second and third trimesters, the dose of PTU should be reduced as low as possible even in most patients remission can occur during the third trimester, so that sometimes no administration of anti-thyroid medications is needed.[9]

Gladstone reports that the use of propranolol can cause small placenta, fetal growth restriction, impaired response to anoxia, postnatal bradycardia and hypoglycemia in neonates. Therefore propranolol is not recommended as a long-term first-choice drug against hyperthyroidism in pregnant women. However, quite a lot of researchers report that giving beta-blockers to pregnant women is quite safe. Beta-blockers can accelerate the control of thyrotoxicosis when combined with iodide. The combination of propranolol 40 mg every 6 hours with iodide usually results in clinical improvement within 2 to 7 days. Iodide rapidly inhibits the binding of iodide in the thyroglobulin molecule (Wolff-Chaikoff effect) and blocks the secretion of thyroid hormone. However, long-term treatment of iodine can be bad because it causes goitre and hypothyroidism in the fetus. As a substitute, Lugol solution can be given 5 drops 2 times a day, but should not be more than 1 week.[10]

Complications include heart trouble. The state of hyperthyroidism in pregnancy can increase serious maternal morbidity, especially heart trouble. The exact mechanism of hemodynamic changes in hyperthyroidism is debated. There is plenty of evidence of the long-term effect of increasing levels of thyroid hormone that can cause myocardial damage, cardiomegaly and ventricular dysfunction. Thyroid hormones can affect the myocardium directly or indirectly.[10]

CONCLUSIONS

Hyperthyroidism in pregnancy is hyperfunction of the thyroid gland accompanied by a mild enlargement of the thyroid gland which usually experiences menstrual disorders or infertility which is generally caused by Grave's disease. Grave's disease is more severe in the first trimester of pregnancy, the highest hyperthyroidism in the first trimester of pregnancy.

Hyperthyroidism is a disorder that occurs when the thyroid gland produces excessive thyroid hormone from the body's needs. Hyperthyroidism may be due to overall over the function of the gland or a less common condition. During pregnancy, the thyroid gland's physiology increases and in many ways the activity of the thyroid gland resembles that of hyperthyroidism.

Hyperthyroidism will cause various complications both for the mother and fetus and the baby to be born. The state of hyperthyroidism in pregnancy can increase serious maternal morbidity, especially heart trouble. Ventricular dysfunction will get worse if accompanied by anaemia, preeclampsia or infection. One serious complication that can occur in pregnant women with hyperthyroidism is a thyroid crisis.

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