Propylthiouracil-induced hepatotoxicity in Graves’ disease: A case report.

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INTRODUCTION

Propylthiouracil-induced severe hepatotoxicity is a relatively rare occurrence, with very few cases reported in the literature. The management of this complication in pregnancy can be a challenge because of the effects of the various treatment options on the fetus.

CASE REPORT

We report a case of hyperthyroidism in a 28-year-old woman that occurred at 20 weeks gestation.

This situation was due to Graves’ disease. Propylthiouracil (PTU) treatment was used.

The following of the pregnancy doesn’t show any side effects.

15 days after delivery, she has a vomiting and fatigue.

Check list show elevated ALAT = 1000U/l.

Table 1 : Biological Characteristics

<table>
<thead>
<tr>
<th>With PTU</th>
<th>After stopping PTU</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALAT</td>
<td>1000 Ui/l</td>
</tr>
<tr>
<td></td>
<td>21 Ui/l</td>
</tr>
</tbody>
</table>

This liver toxicity has been managed by stopping this medication. After 4 days, ALAT remain to normal rate (Table 1).

DISCUSSION

Although asymptomatic liver dysfunction is observed in up to one-third of patients who received propylthiouracil, severe hepatotoxicity is rarely seen.

Propylthiouracil-induced severe hepatotoxicity is a relatively rare occurrence, with very few cases reported in the literature. Despite observed transient increases in liver function test values at the initiation of treatment. A one in 10,000 incidence of liver failure following PTU treatment has been reported in the United States of America.

The diagnosis of propylthiouracil-induced toxic hepatitis was established by the temporal relation between the drug initiation and hepatic dysfunction, and exclusion of other causes of liver damage including viral hepatitis, alcoholic liver disease, autoimmune hepatitis, hereditary disorders, and other hepatotoxins.

The clinical manifestations are usually nonspecific except abnormal liver function tests. Discontinuation of propylthiouracil is warranted if hepatic function deteriorated during the use of the drug. The pathophysiology of propylthiouracil-induced hepatotoxicity is still unclear. Some evidence implied that autoimmune disorder may play an important role. It is believed that the use of propy-lthiouracil may cause ANCA-positive vasculitis.

In one case report of hepatitis after propylthiouracil administration, positive ANA and slightly positive anti-smooth muscle antibodies had been found too.

The clinical presentation of propylthiouracil-induced hepatotoxicity includes jaundice, right upper quadrant abdominal pain, nausea, vomiting, malaise and abnormal liver function tests.

CONCLUSION

This case illustrates a rare complication of treatment with a presumed safe drug during pregnancy followed by adverse maternal outcomes due to the hepatotoxicity treatment.

References: