METHIMAZOLE INDUCED AGRANULOCYTOSIS AND HEPATITIS IN A PATIENT WITH GRAVES' DISEASE APPEARED AFTER RADIOIODINE TREATMENT

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INTRODUCTION: Toxic nodular goiter is a common cause of hyperthyroidism. It develops after the mutation of gene for TSH receptor, which is followed by enhanced synthesis of thyroid hormones. Radioiodine (RAI) is the therapy of choice for toxic nodular goiter. Methimazole is commonly used for the treatment of Graves' disease. Agranulocytosis and toxic hepatitis are most severe side effects of this treatment.We present a case of patient with toxic nodular goiter in whom RAI triggered development of Graves' disease. Treatment of hyperthyroidism with methimazole led to agranulocytosis and hepatotoxicity.

A CASE REPORT: A 75–year old woman was treated with RAI because of toxic nodular goitre. Before radioiodine therapy, serum Thyroid Stimulating Immunoglobulin (TSI), thyroid peroxidase antibodies (antiTPO) and thyroglobulin antibodies (antiTg) were negative. Three months after treatment the patient developed severe hyperthyroidism with appearance of TSI. Methimazole therapy was started. Four weeks later agranulocytosis and mild liver failure were found. Methimazole was stopped and the granulocyte colony stimulating factor and antibiotic treatment were initiated. After 4 days of therapy the leucocyte count and liver tests became normal. The second administration of RAI because of Graves' disease was found successful. At the follow up she developed hypothyroidism. The l-thyroxin therapy was introduced.

CONCLUSIONS: Toxic nodular goiter does not exclude predisposition for autoimmune thyroid disease. In predisposed patients with toxic nodular goiter the Graves' disease can be triggered by RAI treatment. Disadvantages of methimazole treatment are side effects such as agranulocytosis and hepatotoxicity. RAI therapy is the most convenient and safe in treatment of Graves' disease.