



## Incidence of Hypothyroidism in Patients with Diffuse and Toxic Nodular Goiter Treated with 131I

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### Abstract

The aim of this study is to evaluate the outcome of 131I treatment in these two variants of hyperthyroidism, respectively, to assess the incidence of hypothyroidism in these groups of patients compared to groups of patients with diffusely enlarged thyroid.

**Material and Methods:** We observed 226 patients treated with 131I during the five-year period, from 2002 to 2007, 174 (76.9%) with Graves' disease and diffuse toxic goiter. 25 (11%) with multi-nodular and 27 (11.95%) with toxic nodule.

There were 42 (20.80%) males and 184 (81.24%) female patients, aged 22 to 70 years.

The treatment dose of 131-I was based on the size of the nodule or thyroid gland, respectively, and radioiodine uptake.

Patients are followed for three to five years after they received I-131.

**Results:** The incidence of hypothyroidism was greater in Graves' disease than in multi-nodular toxic and toxic nodular (Toxic Adenoma).

Most of the patients (96%) with the solitary toxic nodule remained euthyroid up to five years after treatment with 131-I.

**Keywords:** 131-I; Graves' Disease; Multi-Nodular Toxic Goiter and Toxic Adenoma

**Introduction**

Graves’ disease (GD) is the most common cause of hyperthyroidism in iodine-sufficient areas. The global prevalence of GD is approximately 0.5% [1,2], and its incidence is approximately 0.5-1.0 per 1000 person-years [2,3].

The forms of primary therapy for Hyperthyroidism are available

- Destruction of the thyroid by 131I
- Partial of total surgical ablation of the thyroid, and
- Blocking of hormone synthesis by anti thyroid drugs.

Selection of therapy depends on a multiplicity of considerations [4-8].

In United States, 131I therapy is the initial modality of therapy selected by members of the American Thyroid Association for management of uncomplicated Graves’ disease and toxic nodular goiter. It is used most patients who have prior thyroid surgery, because the incidence of complications, such as hypoparathyroidism and recurrent nerve palsy, is especially high in this group if a second thyroidectomy is preformed [9].

Likewise, is the therapy of choice for any patient who is a poor risk for surgery because of complications.

In the early 1960s, it was recognized that a complication of RAI therapy was a high incidence of hypothyroidism.

This reached 20-40% in the first year after therapy and increased about 2.5% per year, so that by 10 years 50-80% of patients had low function [10,11].

This outcome is supposed to be better in patients with toxic nodular goiter or toxic multi nodular goiter.

**Material and Methods**

Patients can be treated directly after diagnosis, without prior therapy with anti thyroid drugs. This is safe and common in patients with middle hyperthyroidism and especially those without eye problems. But all our patients have undergone pretreatment with antithyroid drugs. The medicine is stopped 2-3 days before therapy if the patients were treated with PTU and 3-5 days before therapy if patients was treated with methimazole.

The treatment dose of 131-I was based on size of the nodule or thyroid gland, respectively, and radioiodine uptake.

Disease	Graves’s disease and diffuse toxic goiter	Multi nodular toxic goiter	Toxic nodule	Total
Patients	174 (76.9%)	25 (11%)	27 (11.5%)	226
Male	38 (16.8%)	2 (0.88%)	2 (0.88%)	42 (20.80%)
Female	136 (60.18%)	23 (10.18%)	25 (11.06%)	184 (81.42%)

**Table 1:** There were 42(20.80%) males and 184(81.24%) female patients, aged 22 to 70 year. Period from 2002 to 2007.

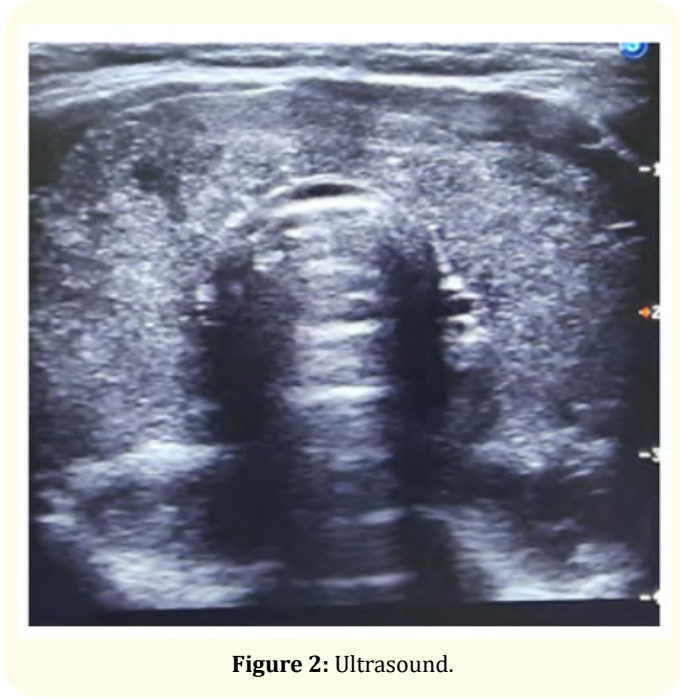
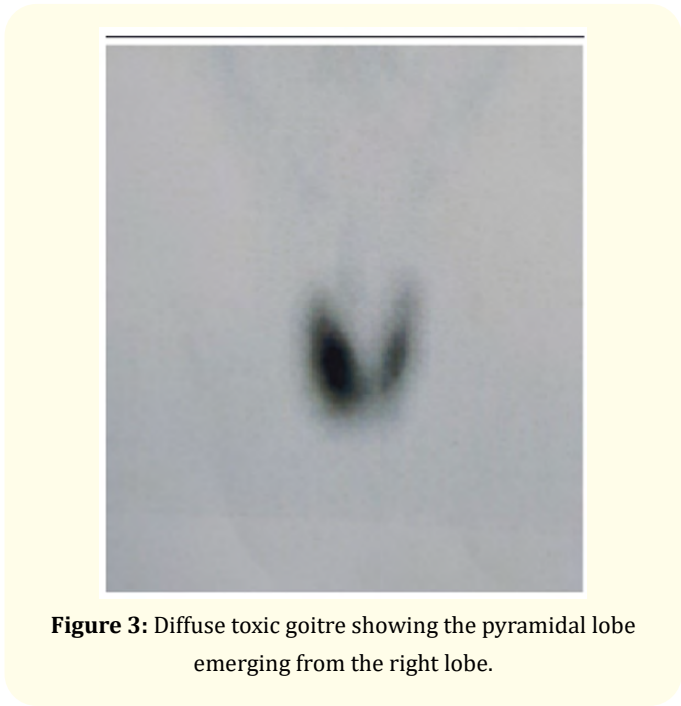
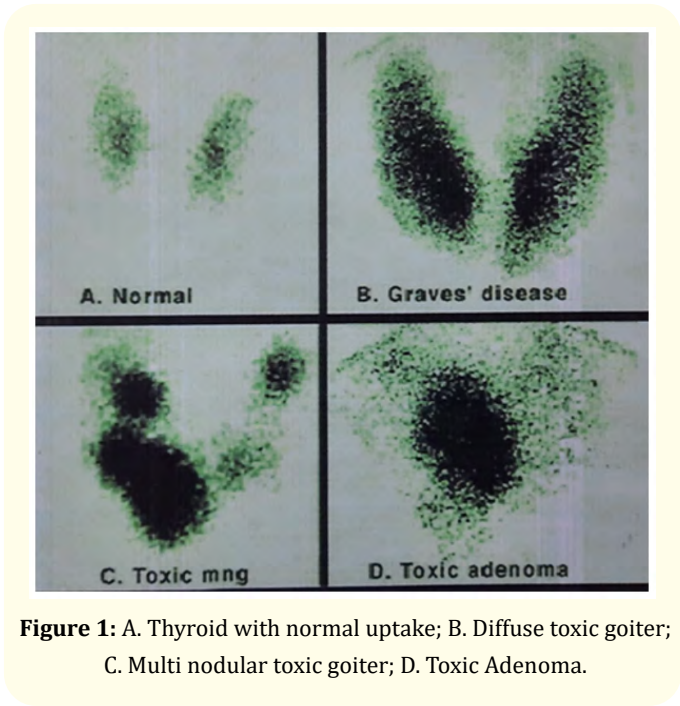
Dose of radioiodine varied from 10-17 mCi (370-594MBq) in patients with Graves’s disease and diffuse toxic goiter, and 14-22 mCi (518-814MBq) in patients with toxic multi nodular goiter and solitary toxic nodule.

**Results**

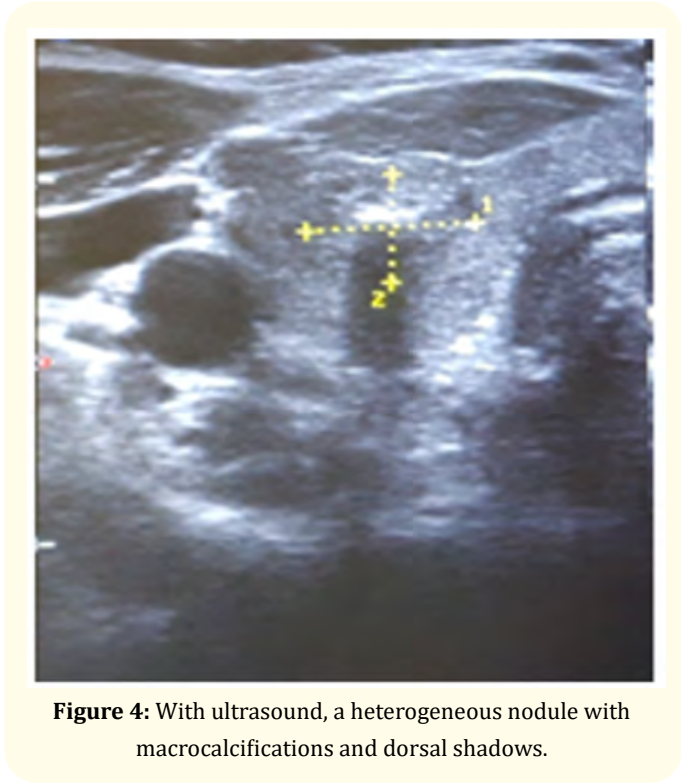
Incidence of Hypothyroidism in the group of patients with enlarged thyroid gland was 130 out 174 patients (74.7%) or 57.52

all 226 patients included in the study. In the group of patients with single toxic nodule there were only one out of 27 patients or 3.7% (or 0.44%) from all 226 patients), whereas in patients with toxic multi nodular there were 2 patients of 25 or 8% (or 0.88%) of all 226 patients included in the study) table 2.

Most of patients (96%) with the solitary toxic nodule remained euthyroid up to five years after treated with 131-I.



**Figure 2:** Ultrasound.



**Figure 4:** With ultrasound, a heterogeneous nodule with macrocalcifications and dorsal shadows.

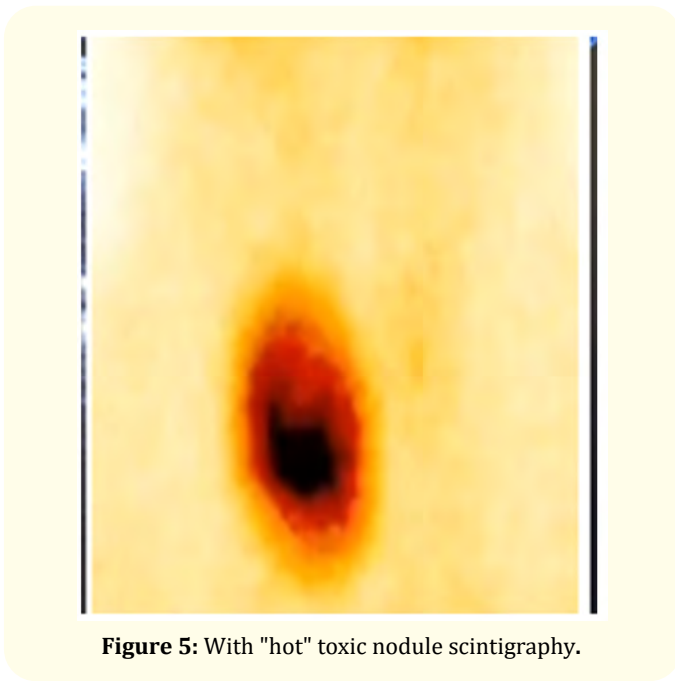


Figure 5: With "hot" toxic nodule scintigraphy.

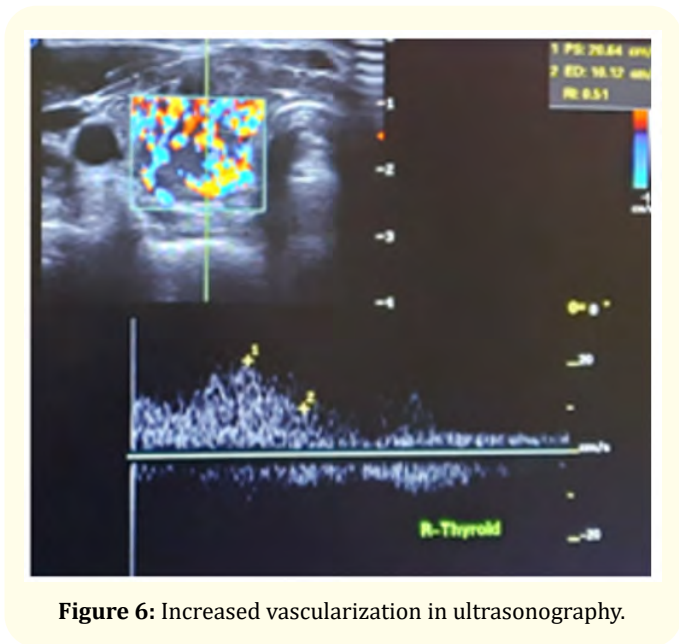


Figure 6: Increased vascularization in ultrasonography.

Disease	Incidence of Hypothyroidism	Total number of patients = 226	Number of separate disease
Graves' disease and diffuse toxic goiter	130 (57.52%)	174	130 (74.7%) of 174
Multi nodular toxic goiter	2 (0.88)	25	2 (8%) of 25
Toxic nodule	1 (0.44%)	27	1 (3.7%) of 27

Table 2: Incidence of Hypothyroidism followed for three to five years after they received 131-J.

**Discussion**

Jodine-131J therapy is quick, easy, moderately expensive, avoid surgery, and is without significant risk in adults and probably late teenagers. The larger doses required to give prompt and certain control generally induce hypothyroidism, and low doses are associated with a frequent requirement for retreatment or ancillary medical management over one to two years.

Surgery was selected as primary therapy for patients with large goiters. Most Therapists attempted to restore euthyroidism by use of 131J or surgery

Toxic adenoma and toxic multi nodular goiter (MNG) are common causes of hyperthyroidism, second in prevalence only to Graves' disease. The prevalence of toxic nodular goiter increases with age and in the presence of iodine deficiency and may therefore

be more common than Graves' disease in older populations in regions of iodine deficiency. Toxic adenoma and MNG are the result of focal and/or diffuse hyperplasia of thyroid follicular cells whose functional capacity is independent of regulation by thyroid-stimulating hormone (TSH). Twenty to 80 percent of toxic adenomas and some nodules of MNGs have somatic mutations of the TSH receptor gene that confers autonomous hyperactivity [12,13].

Although any surgery is risky, major complications of thyroid surgery occur rarely in patients operated on by an experienced thyroid surgeon. These complications include damage to the parathyroid glands that are next to the thyroid and control your body's calcium levels (causing problems with low calcium levels) and damage to the nerves that control your vocal cords (causing you to have a hoarse voice).

After your thyroid gland is removed, the source of your hyperthyroidism is gone and you will become hypothyroid. As with hypothyroidism that develops after radioiodine treatment, your thyroid hormone levels can be restored to normal by treatment once a day with a thyroid hormone supplement [14].

We concluded that treatment with toxic nodular goiter and toxic multi nodular goiter is especially convenient for patients because of very low incidence of hypothyroidism.

Prospective randomized, controlled trials 24,25 [16,17] and detailed retrospective analyses 26,27 [18,19] have shown that precision dosimetry does not improve outcome, while the use of a small range of fixed doses, which can take into account clinically established variables, is safe, simple and cost-effective.

## Conclusion

Radioactive iodine has been used to treat patients for hyperthyroidism for over 60 years and has been shown to be generally safe. Importantly, there has been no clear increase in cancer in hyperthyroid patients that have been treated with radioactive iodine. As a result, in the United States more than 70% of adults who develop hyperthyroidism are treated with radioactive iodine. More and more children over the age of 5 are also being safely treated with radioiodine [10].

We concluded that treatment with toxic nodular goiter and toxic multi nodular goiter is especially convenient for patients because of very low incidence of hypothyroidism.

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