TRANSIENT HYPOTHYROIDISM FOLLOWING I^{131} TREATMENT FOR THYROTOXICOSIS

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INTRODUCTION

The radioiodine treatment for thyrotoxicosis is still fogged with difficulties and controversies even though it has been with us for the past four decades since it was first described by Hertz and Roberts in Boston. [1] Even with the most meticulous method of estimating dosimetry, the majority of patients eventually become hypothyroid if they are followed up long enough. However, there is a group of patients who develop hypothyroidism shortly after therapy and recover, similar to those following subtotal thyroidectomy. [2] We report 8 cases out of 145 patients treated with I^{131} between October 1979 and December 1982.

METHOD

The patients comprised of seven women with a mean age of 54.4 years and a 42-year-old man. All the patients did not receive oral antithyroid drugs following the I^{131} administration. All had diffuse goitres and were not operated or given radioiodine therapy before. The doses of radioiodine ranged from 5.1 to 10.2 mCi with a mean of 8.7 mCi. Majority of the patients were followed up for over 30 months. Patients were initially followed up monthly for the first three months and subsequently every three months (Table I).

TABLE I

Patient's Serial No.	Age at Treatment (year)	Dose of I^{131} (mCi)	Duration of follow up (months)
37	57	7.10	36
58	36	10.10	36
61	48	9.99	33
65	80	5.10	33
80	42	10.00	30
81	42	10.00	30
95	70	10.20	30
124	41	7.00	18
Mean	52	8.69	30.75

The diagnosis of transient hypothyroidism was based on the drop of serum thyroxine (T4) concentration below normal levels with or without symptoms, and subsequent return to normal. Serum T4 and serum thyroid stimulating hormone (TSH) were measured by radioimmunoassay. Normal values were: T4: 64-167 nmols/l; TSH: 0-7 uIU/ml.

Table II shows the approximate time of the fall and recovery of serum T4 following the I^{131} therapy and the corresponding elevation of serum TSH.

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TABLE II

Patient's Serial No.	Biochemical Hypothyroidism: Low T4 at month of follow up	Biochemical Hypothyroidism: Raised serum TSH at month of follow up	Recovery of: Serum T4 at month of follow up	Recovery of: Serum TSH at month of follow up
37	1	1	3	3
58	2	2	5	5
61	3	4	6	6
65	3	NA*	9	NA
80	2	3	6	6
81	1	1	3	3
95	9	9	–	–
124	3	3	6	9

*NA = not affected
TSH remained suppressed

RESULTS

It was obvious that majority of the patients developed biochemical hypothyroidism within the first three months and recovered before the first anniversary of treatment.

Three patients (No.58, 95 and 124) developed symptoms of hypothyroidism within three months of therapy. Two of them were treated symptomatically. Some of the interesting first symptoms to appear were muscular pains and cramps besides the typical over-sensitivity to cold and generalised lethargy. Symptoms improved gradually and they recovered within three months.

Patient No. 95 was treated with L-Thyroxine at about the ninth month post-therapy. She developed angina pectoris and was admitted with acute pulmonary oedema about a year later. L-Thyroxine was subsequently stopped and patient has remained clinically euthyroid with the latest recorded serum T4 = 76 and serum TSH = 2.0.

Patients No. 61 and 81, after a short spell of biochemical hypothyroidism became toxic again requiring continuous carbimazole 'low dose' therapy. It is our practice to leave these patients on maintenance carbimazole 10 mg daily for six to 12 months before attempting to withdraw the drug. If on withdrawing patients are still toxic a second dose of I^{131} may be given.

DISCUSSION

Several interesting observations can be made by studying the eight patients. If the cases are followed up closely within the first few months of therapy, it is bound to notice more cases of transient biochemical hypothyroidism who are either asymptomatic or have minimal non-specific complaints. However, due to the heavy clinic follow up load, it may be impossible to do so and as such the incidence of transient hypothyroidism is much under-estimated.

It is also important to note that although patients were transiently low in their serum T4 soon after I^{131} therapy they can still be toxic later and even necessitating treatment with oral antithyroid drugs, as demonstrated by two of the patients.

The incidence of early onset of hypothyroidism is not only dependent on the dose of I^{131} given [3] but also on whether carbimazole was given pre-and post-treatment. [4] It is important to note that erroneously low serum T4 may be obtained when the blood is taken while the patient is still on carbimazole following the I^{131} therapy. Whether transient hypothyroidism is a significant pointer to prognosticate the subsequent development of permanent hypothyroid state is not certain, in view of the fact that majority of patients anyway will develop myxoedema in the second decade of post I^{131} therapy period. However, not all patients developing either biochemical or clinical hypothyroidism within the first year of treatment require replacement L-Thyroxine. A further three to six months of observation should be done. Some patients may even become toxic again as in two of the cases and the use of L-Thyroxine will be disastrous.

REFERENCES

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