

IODINE INDUCED THYROTOXICOSIS-CASE REPORTS AND REVIEW OF LITERATURE

Pages with reference to book, From 122 To 124

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Abstract

Iodine in the form of Lugol's iodine is commonly prescribed for the treatment of goitre. Eighteen patients, who had positive history of ingestion of iodine are described. Some of the patients had hyperthyroidism when seen while others had thyrotoxicosis like symptoms after taking iodine. Hyperthyroidism was confirmed clinically, radio-isotopically and by response to treatment. Iodine was incriminated after exclusion of other causes of hyperthyroidism.

Introduction

Iodine in the form of cough mixtures, topical ointment, multivitamin mineral tablets, anti-diarrhoeal agents and lugol's iodine is commonly used. There are lack of facilities for investigation of goitre in Pakistan and lugol's iodine is commonly prescribed for patients having goitre. The following patients had developed thyrotoxicosis or thyrotoxicosis like symptoms following ingestion of iodine containing drugs.

Patients and Methods

There were 8 women and ten men. Their ages ranged from 20-71 years. All patients were referred to the thyroid clinic and they were examined before investigations. Thyroid uptake and P.B.1-131 were measured as previously reported (Khan et al., 1979). Thyroxine and triiodothyronine were assayed using commercial kits (Radiochemical Centre, Amersham, U.K.).

Results

The clinical features, history of iodine ingestion and the results of investigations are given in the accompanying table.

Table: Iodine Induced Thyrotoxicosis

S.No.	Name	Age	Sex	History of iodine ingestion	Thyroid uptake 4 hrs	Thyroid uptake 24 hrs	PBI-131 %AD/L	T3 ng/ml	T4 ug/100 ml	Comments
1	S.B	54	F	Lugol's Iodine	41%	67%	0.25	Not done	Not done	Thyrotoxicosis
2	A.K	35	M	Lugol's Iodine (180 ml)	40%	55%	0.4	4.15	13.1	Thyrotoxicosis (T3 Toxicosis)
3	V.J	25	M	Lugol's Iodine (30 ml)	89%	88%	0.67	4.5	24	Thyrotoxicosis
4	A.S	28	F	Lugol's Iodine (120 ml)	92%	97%	0.19	3.35	8.6	Thyrotoxicosis (T3 Toxicosis)
5	H.R	71	M	Lugol's Iodine	15%	38%	1.3	3.7	16.3	Thyrotoxicosis
6	S.W.K	20	M	Lugol's Iodine (600 ml)	37%	52%	0.08	Not done	Not done	Developed Thyrotoxicosis like symptoms on ingestion of Lugol's Iodine.
7	C	25	F	Lugol's Iodine for two weeks	27%	57%	1.7	1.95	9.9	Developed palpitation, choking sensation, weakness of oligomenorrhoea following ingestion of Lugol's Iodine.
8	A.K	54	M	8 hydroxyquinoline derivatives for six years as prophylaxis against amoebiasis	51%	76%	0.34	4.35	21	Thyrotoxicosis
9	—	32	M	Lugol's Iodine (60 ml)	31%	53%	0.3	4.5	22	Thyrotoxicosis
10	A	45	F	Lugol's Iodine (90 ml)	23 %	52%	0.8	Not done	Not done	Thyrotoxicosis (ingestion of Lugol's Iodine lead to an increase in the symptoms and signs of thyrotoxicosis).
11	I	30	F	Lugol's Iodine (90 ml)	9%	13%	—	4.5	21	Thyrotoxicosis
12	S	40	F	Lugol's Iodine (60 ml) for two months	61%	65%	0.5	Not done	Not done	Thyrotoxicosis
13	H	20	F	Lugol's Iodine (180 ml)	26%	42%	0.72	3.25	12.8	T3 Toxicosis
14	M.Z	43	M	Lugol's Iodine (15 ml)	60%	52%	0.19	2.15	6.6	Developed headache, insomnia, nervousness, tremors and falling of hair on ingestion of Lugol's Iodine
15	G.B	35	F	Lugol's Iodine (120 ml) for one month	74%	8%	—	—	—	Symptoms and signs of thyrotoxicosis were intensified on ingestion of iodine
16	M.S	22	M	Lugol's Iodine (360 ml) for six months	56%	73%	0.1	2.65	6.2	Developed thyrotoxicosis like symptoms
17	B.M	30	F	Lugol's Iodine (90 ml)	—	—	0.1	2.86	9.5	Developed thyrotoxicosis like symptoms
18	A.W	34	M	8 hydroxyquinoline derivatives (frequent use)	72%	74%	—	—	—	Developed Thyrotoxicosis

Discussion

The role of iodine in the aetiology of hyperthyroidism has been well established since 1821 when

Coindet described six goitrous patients who developed clinical features of thyrotoxicosis following treatment with iodine. Kocher (1910) drew attention to this phenomenon and called it Jod-Basedow. In 1920's, there were series of reports from the United States regarding increased incidence of thyrotoxicosis after iodisation of salt (Kimball, 1925; McClure, 1927). Connolly et al (1970) reported that the incidence of thyrotoxicosis on the island of Tasmania had more than doubled following the iodisation of bread. Vagenakis et al (1972) discussed the possible mechanisms for the iodide induced thyrotoxicosis. Iodine induced thyrotoxicosis may also occur in apparently normal persons (Savoie et al., 1975). Thyroid storm has also been reported after cardiac angiography (Blum et al., 1976) Livadas et al (1977) gave potassium iodide to sixteen cases of toxic adenoma and noted progressive increase in the serum T4 level. All patients in the present series had history of ingestion of commercially available Lugol's iodine except patients A.K. and A.W. (No. 8 and 18) who had 8 hydroxyquinoline derivatives. Diagnosis was confirmed by clinical feature radioisotope studies and by response to treatment and by exclusion of other thyrotoxic states (Graves, Disease, thyrotoxicosis factitia, thyrotoxicosis associated with hydatidiform mole and choriocarcinoma and thyrotoxicosis caused by ectopic thyroid tissue). There are no reports of 8 hydroxyquinoline induced thyrotoxicosis, but the iodine content in these drugs varies from 40 to 63.9 percent and thyroid enlargement has also been noted (Goodman and Gillman, 1975). The two patients (A.K. and A.W.) who self administered these drugs, used the drugs, for a long time (six and 3 years) as prophylaxis against amoebiasis. Prolonged use of these drugs not only induced hyperthyroidism as in the above two patients but can cause neurotoxicity (Oakley 1973). The experience elsewhere in the world and the experience in Peshawar indicates that iodine may induce persisting hyperthyroidism in subjects with goitre. These studies also show that iodine should not be used in patients with goitre except in iodine deficiency goitre or in preoperative preparation of preexisting thyrotoxicosis.

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