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LIPID PROFILE IN PATIENTS WITH HYPERTHYROIDISM AND ITS RESPONSE TO ANTITHYROID DRUG THERAPY: A HOSPITAL-BASED COMPARATIVE STUDY FROM A TERTIARY CARE CENTRE IN NORTH-WESTERN INDIA

Dr Vijay Pal Bhamu*	Senior Resident, Department Of General Medicine, S.m.s. Medical College And Attached Hospitals, Jaipur, Rajasthan, India*Corresponding Author
Dr Ajeet Singh Gurjar	Senior Resident, Department Of General Medicine, Sjp Medical College, Bharatpur, Rajasthan India
Dr Kanika	Assistant Professor, Department Of General Medicine, S.m.s. Medical College And Attached Hospitals, Jaipur, Rajasthan, India

ABSTRACT

Background: Thyroid hormones are powerful regulators of lipoprotein synthesis, transport and catabolism, and thyrotoxicosis is characteristically accompanied by hypocholesterolaemia. Whether the favourable lipid profile of untreated hyperthyroidism reverses fully once euthyroidism is restored with antithyroid drugs has been incompletely characterised in Indian patients. This study compared lipid parameters between hyperthyroid patients and euthyroid controls and prospectively assessed their change after antithyroid drug therapy. **Methods:** A hospital-based comparative study with a prospective pre–post treatment component was conducted in the Department of General Medicine, S.M.S. Medical College and attached hospitals, Jaipur, from July 2023 to December 2024. Sixty-one adults with biochemically confirmed hyperthyroidism and 61 age- and sex-matched euthyroid controls were enrolled. Patients with diabetes mellitus, renal, hepatic or coronary artery disease, pregnancy, genetic dyslipidaemia or use of lipid-altering drugs were excluded. Thyroid function and fasting lipid profile were measured at baseline in both groups, and repeated in the hyperthyroid group after 3–12 months of antithyroid drug therapy. Data were analysed using the chi-square test, unpaired and paired t tests, analysis of variance and Pearson correlation; $p < 0.05$ was considered significant. **Results:** The groups were comparable for age (34.5 ± 8.2 vs 35.1 ± 7.9 years, $p = 0.78$) and sex distribution, with a female preponderance (68.9%) among cases. At baseline, hyperthyroid patients had significantly lower total cholesterol (152 ± 25 vs 185 ± 30 mg/dL, $p < 0.001$), LDL cholesterol (85 ± 20 vs 115 ± 25 mg/dL, $p < 0.001$) and HDL cholesterol (45 ± 10 vs 50 ± 12 mg/dL, $p = 0.032$); triglycerides did not differ significantly (110 ± 30 vs 120 ± 35 mg/dL, $p = 0.154$). The differences persisted across age, sex and body mass index strata. After antithyroid drug therapy, thyroid function normalised and total cholesterol rose by 26 mg/dL (+17.1%), LDL cholesterol by 23 mg/dL (+27.1%) and HDL cholesterol by 3 mg/dL (+6.7%) ($p < 0.001$, < 0.001 and 0.041, respectively), the increment being greater with longer treatment duration. Therapy was well tolerated, with 83.6% of patients reporting no adverse effect. **Conclusions:** Untreated hyperthyroidism is associated with a substantial reduction in total, LDL and HDL cholesterol. Restoration of euthyroidism with antithyroid drugs reverses this pattern, with LDL cholesterol showing the largest proportionate rise. Lipid profile should therefore be interpreted in the light of thyroid status, and re-assessed once the patient is euthyroid rather than during thyrotoxicosis.

KEYWORDS : Antithyroid agents; Cholesterol, LDL; Dyslipidaemias; Hyperthyroidism; Lipoproteins; Thyrotoxicosis

INTRODUCTION

Thyroid disorders are among the commonest endocrine conditions encountered in clinical practice, second only to diabetes mellitus in India, where thyroid abnormalities are estimated to affect several tens of millions of people.[1,2] In iodine-sufficient populations the prevalence of overt hyperthyroidism is approximately 0.2–1.4%, with a further 0.7–1.4% having subclinical disease, and women are affected considerably more often than men.[3] Graves' disease and toxic nodular goitre account for the large majority of cases, and antithyroid drugs remain the most widely used first-line therapy in the Indian setting.[4]

Thyroid hormones influence virtually every step of lipoprotein metabolism. Triiodothyronine up-regulates hepatic low-density lipoprotein (LDL) receptor expression through a thyroid hormone response element in the receptor gene promoter, thereby accelerating the clearance of LDL particles from plasma.[5,6] It also modulates cholesterol biosynthesis through sterol regulatory element-binding protein 2 and 3-hydroxy-3-methylglutaryl coenzyme A reductase, enhances cholesteryl ester transfer protein and hepatic lipase activity, and stimulates adipose tissue lipolysis with an increased turnover of free fatty acids and glycerol.[7,9] The net effect in thyrotoxicosis is a fall in total and LDL cholesterol, a variable fall in high-density lipoprotein (HDL) cholesterol, and relatively preserved triglyceride concentrations.[10,11]

This apparently favourable lipid pattern is a consequence of the hypermetabolic state rather than a marker of cardiovascular protection, and it is reversed once euthyroidism is achieved. A systematic review and meta-analysis of 166 studies including 12,855 patients reported that treatment of overt hyperthyroidism was associated with a rise in total cholesterol of approximately 44 mg/dL and in LDL cholesterol of approximately 31 mg/dL, whereas no significant lipid change followed treatment of subclinical hyperthyroidism.[12] Older prospective series had already documented the same directional change after radioiodine and after antithyroid drugs.[13,14] The clinical implication is twofold: a lipid profile measured during thyrotoxicosis will underestimate a patient's true atherogenic burden, and a rise in cholesterol after treatment may represent unmasking of pre-existing dyslipidaemia rather than a drug effect.

Most of the available evidence comes from Western and East Asian populations. This matters, because South Asians have a higher percentage of body fat and a more adverse lipid phenotype at any given body mass index, and because dietary patterns differ substantially. Indian data describing lipid alterations in hyperthyroidism are relatively few, and prospective data documenting the change in lipid parameters in the same patients before and after antithyroid drug therapy are fewer still.[15,17] The present study was therefore undertaken to compare the lipid profile of patients with hyperthyroidism with that of age- and sex-matched euthyroid controls, and to assess the change in lipid parameters in the same patients following antithyroid drug treatment at a tertiary care

teaching hospital in north-western India.

MATERIALS AND METHODS

Study design, setting and duration

This was a hospital-based comparative study with a prospective pre–post treatment component, carried out in the Department of General Medicine, S.M.S. Medical College and attached group of hospitals, Jaipur, Rajasthan, between July 2023 and December 2024. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from every participant before enrolment. The study was conducted in accordance with the Declaration of Helsinki and the National Ethical Guidelines for Biomedical and Health Research involving Human Participants (ICMR, 2017).

Participants

Consecutive adults older than 18 years attending the medicine outpatient department or admitted to the medical wards with biochemically confirmed hyperthyroidism — suppressed serum thyroid stimulating hormone (TSH) with raised free thyroxine (FT4) and/or free triiodothyronine (FT3) — were enrolled as cases. Euthyroid adults with normal thyroid function tests, matched to the cases for age and sex, were enrolled as controls.

Participants in either group were excluded if they had diabetes mellitus, chronic kidney disease, chronic liver disease or established coronary artery disease; were pregnant or lactating; had a known genetic lipid disorder such as familial hypercholesterolaemia; or were receiving lipid-lowering therapy or any other drug known to alter thyroid function or lipid metabolism. Cases were additionally excluded if they had hypothyroidism, prior thyroidectomy or radioiodine therapy, thyrotoxicosis due to thyroiditis or excessive iodine intake, or thyroid storm.

The sample size was calculated on the basis of an expected mean HDL cholesterol of 37.8 mg/dL in the hyperthyroid group and 41.35 mg/dL in the healthy group, with a common standard deviation of 7.87 mg/dL. To detect a mean difference of at least 4 mg/dL at 95% confidence ($\alpha = 0.05$) and 80% power, 61 participants were required in each group. The same sample size provided adequate precision for the other lipid parameters studied.

Treatment and follow-up

Patients in the hyperthyroid group were started on antithyroid drug therapy as per departmental protocol, with dose titration guided by clinical response and serial thyroid function tests.[19] Thyroid function and fasting lipid profile were repeated after 3 to 12 months of therapy. Patients in whom euthyroidism was biochemically restored at the time of reassessment were classified as responders, and those in whom it was not as non-responders. Adverse effects attributable to antithyroid drugs — cutaneous rash, gastrointestinal intolerance and derangement of liver enzymes — were recorded at each visit. Controls were assessed once, at baseline.

Statistical analysis

Data were entered in a spreadsheet and analysed using standard statistical software SPSS 26.

RESULTS

Baseline characteristics

One hundred and twenty-two participants were studied, 61 in each group. The two groups were well matched: mean age was 34.5 ± 8.2 years in the hyperthyroid group and 35.1 ± 7.9 years in controls ($p = 0.78$), and the largest single stratum in both groups was 18–30 years. Women predominated among cases (42 women, 19 men; 68.9% female), a distribution that did not differ significantly from controls (40 women, 21 men). Body mass index and height were comparable between the groups. Hyperthyroid patients weighed significantly less than controls (57.1 ± 8.95 vs 62.8 ± 7.01 kg, $p < 0.001$) and had higher random blood glucose values (105.4 ± 11.14 vs 86.2 ± 11.37 mg/dL, $p = 0.001$), consistent with the catabolic and insulin-antagonistic effects of thyroid hormone excess (Table 1).

Table 1. Baseline demographic and clinical characteristics of the study population

Parameter	Hyperthyroid group (n = 61)	Control group (n = 61)	p value
Age, years (mean ± SD)	34.5 ± 8.2	35.1 ± 7.9	0.78
18–30 years, n	24	26	0.78
31–40 years, n	16	15	
41–50 years, n	13	11	
51–60 years, n	8	9	
Sex, male / female, n	19 / 42	21 / 40	0.78
Weight, kg	57.1 ± 8.95	62.8 ± 7.01	<0.001
Height, m	1.61 ± 0.05	1.62 ± 0.17	0.06
Body mass index, kg/m²	22.3 ± 3.1	23.0 ± 2.8	0.20
Blood glucose, mg/dL	105.4 ± 11.14	86.2 ± 11.37	0.001

SD, standard deviation. Age and sex distribution compared by chi-square test; continuous variables by unpaired t test.

Thyroid function and lipid profile at baseline

Baseline thyroid function confirmed the diagnostic separation of the two groups, with markedly suppressed TSH and elevated FT4 and FT3 in the hyperthyroid group (all $p < 0.001$). The hyperthyroid group had significantly lower total cholesterol (152 ± 25 vs 185 ± 30 mg/dL, $p < 0.001$), LDL cholesterol (85 ± 20 vs 115 ± 25 mg/dL, $p < 0.001$) and HDL cholesterol (45 ± 10 vs 50 ± 12 mg/dL, $p = 0.032$) than controls. The difference in triglycerides was small and not statistically significant (110 ± 30 vs 120 ± 35 mg/dL, $p = 0.154$), indicating that the effect of thyroid hormone excess in this cohort was largely confined to cholesterol-carrying lipoproteins (Table 2, Figure 1).

Table 2. Baseline thyroid function tests and fasting lipid profile in the two groups

Parameter	Hyperthyroid group (n = 61)	Control group (n = 61)	p value
TSH, mIU/L	0.05 ± 0.02	2.5 ± 0.8	<0.001
Free T4, ng/dL	3.8 ± 1.2	1.2 ± 0.3	<0.001
Free T3, pg/mL	8.5 ± 2.1	3.0 ± 0.5	<0.001
Total cholesterol, mg/dL	152 ± 25	185 ± 30	<0.001
LDL cholesterol, mg/dL	85 ± 20	115 ± 25	<0.001
HDL cholesterol, mg/dL	45 ± 10	50 ± 12	0.032
Triglycerides, mg/dL	110 ± 30	120 ± 35	0.154

TSH, thyroid stimulating hormone; T3, triiodothyronine; T4, thyroxine; LDL, low-density lipoprotein; HDL, high-density lipoprotein. Values are mean ± standard deviation; compared by unpaired t test.

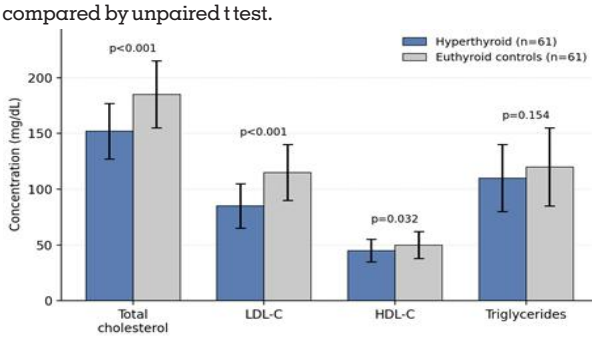

Figure 1. Baseline fasting lipid parameters in hyperthyroid

patients and euthyroid controls. Bars represent mean values; error bars represent one standard deviation.

Lipid differences across age, sex and body mass index strata

The reduction in cholesterol observed in hyperthyroidism was consistent across subgroups. Total cholesterol was lower in hyperthyroid patients in every age stratum, although the absolute gap narrowed in participants older than 45 years. LDL cholesterol was significantly lower in hyperthyroid patients of both sexes, with no meaningful difference in the magnitude of effect between men and women. HDL cholesterol was lower in hyperthyroid patients across all body mass index categories, reaching statistical significance in those with a body mass index below 20 kg/m² and between 20 and 25 kg/m², but not in those above 25 kg/m², where numbers were smallest (Table 3).

Table 3. Stratified comparison of lipid parameters between the two groups

Stratum	Hyperthyroid (mg/dL)	Control (mg/dL)	p value
Total cholesterol by age group			
18–30 years	148 ± 22	180 ± 28	<0.001
31–45 years	155 ± 26	188 ± 32	<0.001
>45 years	160 ± 30	190 ± 35	0.002
LDL cholesterol by sex			
Male	88 ± 18	118 ± 22	<0.001
Female	83 ± 21	112 ± 26	<0.001
HDL cholesterol by body mass index			
<20 kg/m²	42 ± 8	48 ± 10	0.045
20–25 kg/m²	46 ± 9	51 ± 11	0.028
>25 kg/m²	47 ± 12	52 ± 13	0.062

Values are mean ± standard deviation; compared by unpaired t test within each stratum.

Response to antithyroid drug therapy

Antithyroid drug therapy restored thyroid function in the great majority of patients: mean TSH rose from 0.05 ± 0.02 to 1.8 ± 0.6 mIU/L, FT4 fell from 3.8 ± 1.2 to 1.3 ± 0.4 ng/dL and FT3 from 8.5 ± 2.1 to 3.2 ± 0.6 pg/mL (all $p < 0.001$). This biochemical recovery was accompanied by a significant rise in all cholesterol fractions. Total cholesterol increased by 26 mg/dL (+17.1%) and LDL cholesterol by 23 mg/dL (+27.1%), both highly significant, while HDL cholesterol rose modestly by 3 mg/dL (+6.7%, $p = 0.041$). Triglycerides increased by 8 mg/dL, which did not reach statistical significance ($p = 0.092$). Post-treatment mean values approached, but did not exceed, those of the control group (Table 4, Figure 2).

Table 4. Thyroid function and lipid parameters before and after antithyroid drug therapy in the hyperthyroid group (n = 61)

Parameter	Before treatment	After treatment	Change (%)	p value
TSH, mIU/L	0.05 ± 0.02	1.8 ± 0.6	—	<0.001
Free T4, ng/dL	3.8 ± 1.2	1.3 ± 0.4	—	<0.001
Free T3, pg/mL	8.5 ± 2.1	3.2 ± 0.6	—	<0.001
Total cholesterol, mg/dL	152 ± 25	178 ± 28	+26 (+17.1)	<0.001
LDL cholesterol, mg/dL	85 ± 20	108 ± 23	+23 (+27.1)	<0.001
HDL cholesterol, mg/dL	45 ± 10	48 ± 11	+3 (+6.7)	0.041
Triglycerides, mg/dL	110 ± 30	118 ± 32	+8 (+7.3)	0.092

Values are mean ± standard deviation; compared by paired t test.

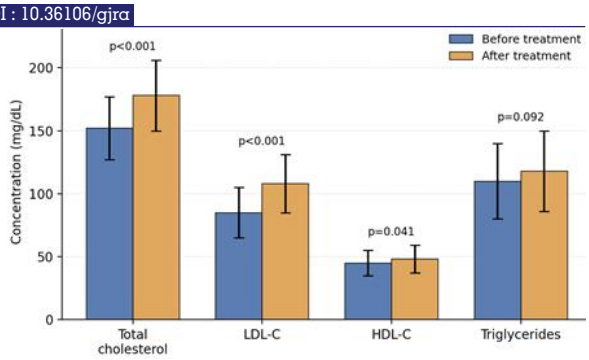

Figure 2. Fasting lipid parameters in the hyperthyroid group before and after antithyroid drug therapy. Bars represent mean values; error bars represent one standard deviation.

The magnitude of the rise in cholesterol was related to the duration of therapy. Patients reassessed at three months showed a mean total cholesterol of 170 ± 25 mg/dL, those at six months 180 ± 29 mg/dL, and those at twelve months 185 ± 30 mg/dL — the last being effectively identical to the control group mean. The increase in LDL cholesterol was seen in every age stratum, with slightly higher post-treatment values in older patients (Table 5).

Table 5. Change in cholesterol with duration of therapy and by age group

Subgroup	Before treatment (mg/dL)	After treatment (mg/dL)	p value
Total cholesterol by duration of therapy			
3 months	150 ± 23	170 ± 25	<0.001
6 months	153 ± 26	180 ± 29	<0.001
12 months	155 ± 28	185 ± 30	<0.001
LDL cholesterol by age group			
18–30 years	82 ± 18	105 ± 20	<0.001
31–45 years	86 ± 21	109 ± 24	<0.001
>45 years	90 ± 23	116 ± 26	<0.001

Values are mean ± standard deviation; compared by paired t test within each subgroup.

Correlation between TSH and lipid parameters, treatment response and tolerability

In the hyperthyroid group before treatment, pre-treatment TSH correlated significantly with total cholesterol ($r = −0.65$, $p < 0.001$) and LDL cholesterol ($r = −0.60$, $p < 0.001$), weakly with HDL cholesterol ($r = −0.25$, $p = 0.048$), and not at all with triglycerides ($r = −0.15$, $p = 0.245$). Cholesterol fractions were thus tightly coupled to the degree of biochemical thyroid derangement, whereas triglycerides were not (Table 6).

Fifty patients (82.0%) achieved biochemical euthyroidism at reassessment and were classified as responders, while 11 (18.0%) did not. Responders had significantly higher post-treatment total cholesterol (175 ± 26 vs 160 ± 22 mg/dL, $p = 0.034$) and LDL cholesterol (106 ± 21 vs 95 ± 18 mg/dL, $p = 0.041$) than non-responders, with no significant difference in HDL cholesterol (49 ± 10 vs 45 ± 9 mg/dL, $p = 0.128$) — that is, the lipid recovery tracked the biochemical recovery. Treatment was well tolerated: 51 patients (83.6%) reported no adverse effect, 5 (8.2%) developed a cutaneous rash, 3 (4.9%) nausea and 2 (3.3%) biochemical liver dysfunction. No patient developed agranulocytosis or required permanent discontinuation of therapy (Table 6).

Table 6. Correlation of pre-treatment TSH with lipid parameters, comparison of responders and non-responders, and adverse effects of therapy

Variable	Value	Comparator	p value
Correlation of pre-treatment TSH with:	r		
Total cholesterol	−0.65	—	<0.001
LDL cholesterol	−0.60	—	<0.001
HDL cholesterol	−0.25	—	0.048
Triglycerides	−0.15	—	0.245
Post-treatment lipids	Responders (n = 50)	Non-responders (n = 11)	
Total cholesterol, mg/dL	175 ± 26	160 ± 22	0.034
LDL cholesterol, mg/dL	106 ± 21	95 ± 18	0.041
HDL cholesterol, mg/dL	49 ± 10	45 ± 9	0.128
Adverse effects of therapy	n	% of 61	
None	51	83.6	—
Cutaneous rash	5	8.2	—
Nausea	3	4.9	—
Liver dysfunction	2	3.3	—

r, Pearson correlation coefficient. Responder status was defined as biochemical restoration of euthyroidism at reassessment.

DISCUSSION

This study documents, in a matched Indian cohort, three findings that are clinically relevant to the general physician. First, untreated hyperthyroidism was accompanied by a substantial and statistically significant reduction in total, LDL and HDL cholesterol, but not in triglycerides. Second, this pattern reversed with antithyroid drug therapy, the proportionate rise being greatest for LDL cholesterol. Third, the magnitude of lipid recovery paralleled the degree of biochemical recovery, both across treatment duration and between responders and non-responders.

Lipid profile in untreated hyperthyroidism

The 18% lower total cholesterol and 26% lower LDL cholesterol observed in our hyperthyroid patients are consistent with the classical description of thyrotoxic hypocholesterolaemia and with the mechanistic literature. Excess triiodothyronine increases hepatic LDL receptor transcription through the thyroid hormone response element of the receptor gene promoter, accelerating fractional catabolic rate of LDL, and simultaneously modulates cholesterol synthesis through sterol regulatory element-binding protein 2.[5,7] The classical prospective series of Kung and colleagues in hyperthyroid patients reported significantly lower total cholesterol, LDL cholesterol, HDL cholesterol and apolipoprotein B than in age-matched controls — a pattern essentially identical to ours, including the reduction in HDL.[13] Muls and co-workers similarly documented low apolipoprotein B and low LDL cholesterol before treatment that rose after therapy.[14]

The finding that HDL cholesterol was also significantly reduced deserves comment, since it is less consistently reported than the fall in LDL. Thyroid hormone excess increases cholesteryl ester transfer protein and hepatic lipase activity, both of which accelerate the catabolism and remodelling of HDL particles.[8,9] In our cohort, in which FT4 and FT3 were markedly elevated, this catabolic effect appears to have been sufficient to lower HDL detectably. The absence of a significant difference in triglycerides accords with the observation that lipoprotein lipase-mediated triglyceride clearance is normal or increased in thyrotoxicosis, so that increased hepatic very-low-density lipoprotein output is offset by increased peripheral removal.[7,10] Community-based and hospital-based comparative studies in other settings have reported the same overall pattern, with the most consistent signal in total and LDL cholesterol.[11,15,20]

Subgroup analysis showed that the cholesterol difference between cases and controls persisted in every age band, in both sexes and across body mass index categories, suggesting that thyroid status rather than body habitus or demography was the dominant determinant of the lipid pattern in this cohort. The slightly narrower gap in participants older than 45 years is compatible with the accumulation of competing determinants of cholesterol with age, but the numbers in that stratum were small and this observation should be regarded as hypothesis-generating. The strong female preponderance among our cases (68.9%) mirrors the well-documented sex distribution of hyperthyroidism.[3]

Lipid response to antithyroid drug therapy

Restoration of euthyroidism produced a rise of 26 mg/dL in total cholesterol and 23 mg/dL in LDL cholesterol in our patients. These increments are directionally identical to, though somewhat smaller than, the pooled estimates of the largest meta-analysis on this question, which reported increases of approximately 44 mg/dL in total cholesterol and 31 mg/dL in LDL cholesterol after treatment of overt hyperthyroidism.[12] Several factors may explain the smaller effect size in our cohort: a follow-up window as short as three months in some patients, a relatively young cohort with a low baseline body mass index, and the exclusive use of antithyroid drugs rather than radioiodine or surgery. The graded relationship we observed with treatment duration mean total cholesterol of 170, 180 and 185 mg/dL at three, six and twelve months respectively supports the first of these explanations and suggests that lipid values measured too early after starting therapy will underestimate the eventual steady-state value. Hussain and colleagues, studying lipid abnormalities in hyperthyroid patients before and after antithyroid drug therapy in an Indian tertiary care setting, similarly reported a rise in lipid parameters with restoration of euthyroidism.[16]

The comparison of responders and non-responders reinforces this interpretation. Patients in whom euthyroidism was biochemically achieved had significantly higher post-treatment total and LDL cholesterol than those in whom it was not, indicating that the rise in cholesterol is a marker of successful metabolic normalisation rather than an adverse drug effect. This distinction matters at the bedside: a patient whose cholesterol rises after starting carbimazole or methimazole is not developing drug-induced dyslipidaemia, but is returning towards his or her intrinsic lipid phenotype.

Relationship between thyroid function and lipids

Pre-treatment TSH correlated strongly with total and LDL cholesterol and weakly with HDL cholesterol, but not with triglycerides. The differential strength of these correlations is consistent with the mechanistic literature: cholesterol-carrying lipoproteins are directly regulated by thyroid hormone through LDL receptor expression and cholesterol synthesis, whereas triglyceride concentrations reflect the net balance of two opposing thyroid hormone effects and are therefore less tightly coupled to thyroid status.[7,8,11] It should be noted that within a cohort restricted to hyperthyroid patients, in whom TSH is uniformly suppressed and its dynamic range correspondingly narrow, correlation coefficients involving TSH must be interpreted with caution; FT4 or FT3 may be the more informative index of disease severity in such a population.

Clinical implications

Three practical points follow. First, a fasting lipid profile performed during untreated thyrotoxicosis is not a valid estimate of a patient's habitual lipid status and should not be used to make decisions about lipid-lowering therapy; it should be repeated once the patient is euthyroid, ideally not earlier than three to six months after starting treatment. Second, because LDL cholesterol showed the largest proportionate

increase after treatment (+27.1%), patients with additional cardiovascular risk factors merit formal risk assessment once euthyroid, particularly older patients in whom post-treatment values approached the control mean. Third, antithyroid drug therapy was well tolerated in this cohort, with 83.6% of patients reporting no adverse effect and only two instances of biochemical liver dysfunction, which supports its continued use as first-line therapy while reinforcing the standard practice of counselling patients about rash, sore throat and jaundice as reasons to seek review.[4-19]

STRENGTHS AND LIMITATIONS

The principal strengths of this study are the use of an age- and sex-matched control group, the exclusion of the common secondary causes of dyslipidaemia, and the availability of paired within-patient lipid measurements before and after treatment, which removes between-subject variability from the estimate of treatment effect. This was a single-centre study in an urban tertiary care hospital in one region of India, and no cardiovascular outcomes were assessed; the clinical significance of the observed lipid changes therefore remains an inference rather than a demonstrated finding.

CONCLUSION

Patients with untreated hyperthyroidism had significantly lower total, LDL and HDL cholesterol than matched euthyroid controls, with unchanged triglycerides. Antithyroid drug therapy restored thyroid function and reversed this pattern, with LDL cholesterol showing the largest proportionate rise; the increment was greater with longer treatment and was confined largely to patients who achieved biochemical euthyroidism. Therapy was well tolerated. The lipid profile of a thyrotoxic patient should therefore be interpreted as a manifestation of the thyroid state and re-assessed after euthyroidism has been established, at which point conventional cardiovascular risk stratification becomes meaningful. Larger multicentre studies with fixed follow-up intervals and cardiovascular endpoints are needed to determine whether the post-treatment rise in LDL cholesterol carries prognostic weight.

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