INTRODUCTION

Obesity, as defined by the BMI, is on the rise globally and in most sex-age groups is more than

30% in United States. Obesity prevalence has not altered much over past 12 years from 1999 to 2010. This trend in obesity prevalence is linear for men while it is not significant for women over this period.\(^1\)

The three major non-communicable diseases (NCDs) with highest mortality rate in Pakistan are cardiovascular diseases; malignancies and chronic respiratory diseases.\(^2\) All these NCDs have few common risk factors like smoking, alcohol intake, diabetes mellitus, hypertension, dyslipidemia, obesity, unhealthy & sedentary lifestyle; which can be changed resulting in prevention of these NCDs. Besides, studies have shown Asians to have a higher BMI than Europeans.\(^4\) Therefore, a lower cutoff value of BMI for overweight (23.0–24.9 kg/m²) and obese (>25.0 kg/m²) Asians has been suggested by the authorities of the international obesity task force.\(^4\) WHO figures reveal increasing prevalence of overweight and obese adults.\(^5\)
Growing evidence suggests association of altered thyroid function and obesity, a lasting state of low grade inflammation. Recent data has revealed leptin, an adipocyte hormone, to be a major factor linking obesity and thyroid autoimmunity. Recently, several clinical studies have evaluated the issue of hormonal changes associated with obesity.

The prevalence of subclinical hypothyroidism in Pakistani population is 5.4% compared to the prevalence of overt hypothyroidism that is 4.1% and compared to males, females are more affected. There are studies that reveal slightly elevated thyroid stimulating hormone level within reference range relating to slightly increased body weight. It has been reported that 100-fold changes in thyroid stimulating hormone can be caused by only two-fold changes in free thyroxine level and that a unit increase in thyroid stimulating hormone level can cause a 3% increase in the components of metabolic syndrome.

The present study was designed to determine the frequency of subclinical hypothyroidism in local adult obese population. Doing a thorough literature search, we found that very little data exists about the thyroid hormone levels and its dysfunction among local adult obese population. This study will be an attempt to determine the frequency of subclinical hypothyroidism in local obese adult population and the results of this study will be a guideline for devising future research strategies and identifying mechanisms of preventing subclinical hypothyroidism in patients with morbid obesity.

METHODS

This descriptive cross-sectional study was conducted at Hayatabad Medical Complex, Peshawar from March 2017 to August 2017. It was a. All patients aged between 18 and 60 years with BMI of more than 29kg/m² were included in the study. Patients on lipid lowering drugs, with renal failure, hepatic failure and already diagnosed cases of thyroid dysfunction were excluded.

All subjects were subjected to complete history and clinical examination to exclude effect modifiers. From all the patients, a blood sample was obtained and was sent to hospital laboratory to measure the serum TSH & Thyroxine levels (this was done free of cost from hospital laboratory). All the above-mentioned information including name, age, and sex was recorded in a pre-designed proforma and strict exclusion criteria was followed to control confounders and bias in the study results. All the investigations were done form hospital laboratory by single experienced microbiologist having minimum of five years of experience.

Data was stored and analyzed in SPSS version 20. Mean and standard deviation (SD) were calculated for quantitative variables like age, serum TSH and thyroxine levels. Frequencies and percentages were calculated for categorical variables like gender and subclinical hypothyroidism. Subclinical hypothyroidism was stratified among age and gender to see the effect modifications. Chi square was used and a value less than 0.05 were considered significant.

RESULTS

Total of 127 adult participants having a body mass index of more than 29kg/m² were observed to determine the frequency of subclinical hypothyroidism and results were analyzed. Age distribution among 127 patients was analyzed as; 44(34.6%) patients were <30 years, 45(35.6%) patients were in age range 30-40 years, 38(29.9%) patients were in age range above 40 years. Mean age of the study population was 34.5 ± 7.9 years. Out of 127 patients included in the study, there were 46.5% male and 53.5% female. Mean BMI was 32.05 ± 2.06. BMI was stratified into two classes i.e. BMI between 29.5-32.0kg/m² and BMI between 32.1-35.5kg/m².

All the patients were subjected to measurement of serum TSH and thyroxine levels. The mean serum TSH was 3.13 ± 1.10 mIU/L and mean serum thyroxine level was 1.08 ± 0.25ng/dl. Subclinical hypothyroidism was recorded in 15% of study population. Subclinical hypothyroidism was stratified with regards to age groups, gender and BMI categories.

Table-I: Summary of Patient Characteristics (n=127).

| Characteristics | Subclinical Hypothyroidism | p-value |
|-----------------|---------------------------|---------|
| Age             | Present | Not present |     |
| <30 years       | 0       | 44          | 0.001 |
| 30-40 years     | 13      | 32          |       |
| >40 years       | 6       | 32          |       |
| Gender          | Present | Not present |     |
| Male            | 6       | 53          | 0.159 |
| Female          | 13      | 55          |       |
| BMI             | Present | Not present |     |
| 29.0-32.0 kg/m² | 6       | 56          | 0.103 |
| 32.1-35.5       | 13      | 52          |       |
DISCUSSION

Rising weight gain and obesity is rapidly becoming a grave problem globally due to its well-established association with diabetes, hypertension, heart diseases, ischemic stroke, and several types of malignancies. On one hand, sedentary lifestyle leading to a disparity between consumption and utilization of energy causes weight gain while on other hand, thyroid dysfunction leading to impaired resting energy expenditure (REE) balance is an established entity leading to weight gain and obesity.

In our study, the mean BMI was 32.05 ± 2.06 with mean TSH was 3.13 ± 1.10 mIU/L and mean serum thyroxine level was 1.08 ± 0.25ng/dl. Hypothyroidism was recorded in 15% of the patients in our study. Like these findings, Michalaki et al. also found low occurrence of the patients in our study. Like these findings, Michalaki et al. found low occurrence of the patients in our study. Like these findings, Michalaki et al. found low occurrence of thyroid impairment in obese patients with elevated TSH. Likely, by summing up the past studies, two different theories arise showing relationship of thyroid function and obesity. Subclinical hypothyroidism is highly prevalent in our population with BMI of more than 29kg/m². Further studies are recommended on relationship between thyroid functions and BMI and its effect on cardiovascular functions.

CONCLUSION

Subclinical hypothyroidism is highly prevalent in our population with BMI of more than 29kg/m². Further studies are recommended on relationship between thyroid functions and BMI and its effect on cardiovascular functions.

REFERENCES

1. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. JAMA. 2012;307(5):491-497. doi:10.1001/jama.2012.39.
2. World Health Organization. Burden: mortality, morbidity and risk factors. Chapter 1. In: Global status report on noncommunicable diseases 2010. Description of the global burden of NCDs, their risk factors and determinants. World Health Organization. April 2011. Available from URL: http://www.who.int/entity/nmh/publications/ncd_report_chapter1.pdf.
3. World Health Organization, Western Pacific Region. The International Association for the Study of Obesity and the International Obesity Task Force. The Asia-Pacific perspective: redefining obesity and its treatment. Sydney, Australia: Health Communications Australia Pty Limited; 2000. [Cited on Mar 22, 2015]. Available from URL: http://www.diabetes.com.au/pdf/obesity_report.pdf.
4. Bahadur S, Yousaf M, Ayaz HM, Sohail Z, Rehman AU, Baloch S, et al. Self-reporting of obesity, overweight and health risks among 1st year MBBS students of Rehman medical college, Peshawar. Khyber Med Univ J. 2013;5(2):24-27.
5. Samir N, Mahmud S, Khuwaja A. Prevalence of physical inactivity and barriers to physical activity among obese attendants at a community health-care center in Karachi, Pakistan. BMC Res Notes. 2011;4(1):174. doi: 10.1186/1756-0500-4-174.
6. Duntas LH, Biondi B. The interconnections between obesity, thyroid function, and autoimmunity: the multifold role of leptin. Thyroid. 2013;23(6):646-653. doi:10.1089/thy.2011.0499.
7. Rotondi M, Magri F, Chiovato L. Thyroid and obesity: not a one-way interaction. J Clin Endocrinol Metab. 2011;96(2):344-346. doi: 10.1210/jc.2010-2515.
8. Fox CS, Pencina MJ, D'Agostino RB, Murabito JM, Seely EW, Pearce EN, et al. Relations of thyroid function to body weight: Cross-sectional and longitudinal observations in a community-based sample. Arch Intern Med. 2009;169(6):587-592. doi: 10.1001/archinte.169.6.587.
9. De Moura Souza A, Sicieri R. Association between serum TSH concentration within the normal range and adiposity. Eur J Endocrinol. 2011;165:11-15. doi:10.1530/EJE-11-0261.
10. Kitahara CM, Platz EA, Ladenson PW. Body fatness and markers of thyroid function among US men and women. PLoS One. 2012;7:e34979. doi: 10.1371/journal.pone.0034979.
11. Fatourechi V. Subclinical hypothyroidism: an update for primary care physicians. Mayo Clinic Proc. 2009;84(1):65-70.
12. Waring AC, Rodondi N, Harrison S, Kanaya AM, Simonson EM, Miljkovic I, et al. Thyroid function and prevalent and incident metabolic syndrome in older adults: the Health, Ageing and Body Composition Study. Clin Endocrinol (Oxf). 2012;76(6):911-918. doi: 10.1111/j.1365-2265.2011.04328.x.

Grant Support & Financial Disclosures: None.
13. Nguyen DM, El-Serag HB. The epidemiology of obesity. Gastroenterol Clin N Am. 2010;39:1-7. doi: 10.1016/j.gtc.2009.12.014.

14. Abdullah A, Peeters A, de Courten M, Stoelwinder J. The magnitude of association between overweight and obesity and the risk of diabetes: a meta-analysis of prospective cohort studies. Diabetes Res Clin Pract. 2010;89:309-319. doi: 10.1016/j.diabres.2010.04.012.

15. Field AE, Coakley EH, Must A, Spadano JL, Laird N, Dietz WH, et al. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. Arch Intern Med. 2001;161:1581-1586. doi: 10.1001/archinte.161.13.1581.

16. Strazzullo P, D’Elia L, Cairella G, Garbagnati F, Cappuccio FP, Scalì L. Excess body weight and incidence of stroke: meta-analysis of prospective studies with 2 million participants. Stroke. 2010;41:e418-426. doi: 10.1161/STROKEAHA.109.576967.

17. World Cancer Research Fund/American Institute for Cancer Research. In: Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: AICR. 2007.

18. Al-Adsani H, Hoffer LJ, Silva JE. Resting energy expenditure is sensitive to small dose changes in patients on chronic thyroid hormone replacement. J Clin Endocrinol Metab. 1997;82:1118-1125. doi: 10.1210/jcem.82.4.3873.

19. Reinehr T. Obesity and thyroid function. Mol Cell Endocrinol. 2010;316:165-171. doi: 10.1016/j.mce.2009.06.005.

20. Reinehr T, de Sousa G, Andler W. Hyperthyrotropinemia in obese children is reversible after weight loss and is not related to lipids. J Clin Endocrinol Metab. 2006;91:3088-3091. doi: 10.1210/jc.2006-0095.

21. Michalaki MA, Vagenakis AG, Leonardou AS, Argentou MN, Habeos IG, Makri MG, et al. Thyroid function in humans with morbid obesity. Thyroid 2006;16:73–78. doi: 10.1089/thy.2006.16.73.

22. Spencer CA, Hollowell JG, Kazarosyan M, Braverman LE. National Health and Nutrition Examination Survey III: thyroid-stimulating hormone (TSH)-thyroperoxidase antibody relationships demonstrate that TSH upper reference limits may be skewed by occult thyroid dysfunction. J Clin Endocrinol Metab. 2007;92:4236-4240. doi: 10.1210/jc.2007-0287.

23. Biondi B. Thyroid and obesity: an intriguing relationship. J Clin Endocrinol Metab. 2010;95:3614-3617. doi: 10.1210/jc.2010-1245.

24. Knudsen N, Laurberg P, Rasmussen LB, Bulow I, Perrild H, Ovesen L, et al. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. J Clin Endocrinol Metab. 2005;90:4019-4024. doi: 10.1210/jc.2004-2225.

25. Araujo RL, Andrade BM, Padron AS, Gaidhu MP, Perry RL, Carvalho DP. High-fat diet increases thyrotropin and oxygen consumption without altering circulating 3,5,3’-triiodothyronine (T3) and thyroxine in rats: the role of iodothyronine deiodinases, reverse T3 production, and whole-body fat oxidation. Endocrinology. 2010;151:3460-3469. doi: 10.1210/en.2010-0026.

Author’s Contribution:
Abdul Sami conceived, designed and did statistical analysis & manuscript writing.
Malik Faisal Iftekhar & Muhammad Abdur Rauf did data collection and editing of manuscript.
Akhter Sher did review and final approval of manuscript.