Turkish Journal of Internal Medicine

ÇALIŞMA-İNGİLİZCE ÖZET ÖRNEĞİ

Comparison of The Framingham Risk Scores and The Factors Affecting The Score According to Body Mass Indices in Obese Patients

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Background: Although obesity has been shown to increase cardiovascular disease risk, body mass index (BMI) is not yet included in cardiovascular risk scoring systems. This study aimed to evaluate the relationship between Framingham risk scoring, which is a marker of obesity, and Framingham risk scoring in individuals with obesity and to evaluate and compare the factors affecting obesity.

Material and Methods: 55 female patients admitted to Bursa Uludağ University Faculty of Medicine Department of Internal Medicine Endocrinology and metabolic diseases outpatient clinic between January 2010 and January 2015 were included in the study. The patients were divided into 2 groups according to their BMI: Group 1 (n=25) with a BMI of 40-50 kg/m^2 and Group 2 with a BMI of >50 kg/m^2 (n=30). We compared cardiac risk score, factors, and metabolic syndrome (MS) criteria in both groups.

Results: Total cholesterol, HDL cholesterol, triglyceride, and fasting blood glucose values of both groups were comparable ($p>0.05$). 24% of the first group, 36.7% of the second group, and 30.9% of the whole group smoked. There was no statistically significant difference between both groups in terms of smoking ($p>0.05$). Mean systolic blood pressure (SBP) was significantly lower in the first group compared to the second group. There was no statistical difference between the two groups in mean diastolic blood pressure (DBP) values. Five patients (20%) from the first group and 9 (30%) patients from the second group took medication for hypertension.

The Framingham risk score in the first group was statistically significantly lower than the second group ($p<0.05$). Framingham risk score correlations were correlated with Framingham risk score ($p<0.001$), smoking ($p<0.001$), total cholesterol ($p<0.001$), SBP ($p<0.01$), HDL cholesterol. No correlation was found ($p>0.05$). The first group had an average of 2 MS criteria, and the second group had 3. There was a positive correlation between Framingham's risk score and the number of MS components ($p<0.05$). In our study, female patients with different levels of obesity; There was no difference between mean lipid and fasting glucose values. While DBP values were similar in both groups, SBP increased with increasing obesity degree. The Framingham risk score, a cardiovascular risk predictor, increased with increasing obesity. There was a correlation between the Framingham risk score and the parameters that formed it, age, total cholesterol, SBP, and cigarette smoking, but not with HDL. There was a correlation between the presence and number of MS components and the Framingham risk score.

Conclusions: We showed in our study that increased BMI increased the risk of cardiac disease, and MS contributed to the increased cardiac risk. Losing weight can reduce the risk of cardiac events secondary to obesity and MS and increase the person's quality and duration of life.

VAKA SUNUMU-İNGİLİZCE ÖZET ÖRNEĞİ

A Case of Autoimmune Polyglandular Syndrome Presenting with Latent Autoimmune Diabetes

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Background: Autoimmune polyglandular syndrome (OPS) is a group of autoimmune diseases characterized by the failure of at least two endocrine glands in hormone production. OPS type 1 is seen in childhood and is diagnosed with hypoparathyroidism, Addison's disease, and candida infection involving the skin or mucous membranes. OPS type 2 (Type 1 diabetes with Addison's disease or autoimmune thyroid disease), OPS type 3 (Autoimmune thyroid disease with other autoimmune diseases other than Addison), and OPS Type 4 (Addison's disease with one or more organ-specific Type 1 and type 2 autoimmune diseases that do not include major components seen in OPS). When we look at the subgroups of OPS type 3, Autoimmune thyroiditis is accompanied by type 1 diabetes in type 3A, pernicious anemia in type 3B, vitiligo in type 3C, and collagen tissue diseases in type 3D. We wanted to share a case of OPS type 3A+3B with latent autoimmune diabetes and pernicious anemia accompanying autoimmune thyroiditis.
Case Report: 38-year-old female patient was applied to the external center for 1 month due to pain in the legs, drinking too much water, frequent toileting, and weakness. Fasting blood glucose was found 383 mg/dL, thereby metformin, vildagliptin, gliclazide, and alpha-lipoic acid treatments were initiated for diabetes and diabetic neuropathy. In her next control, hemoglobin A1c (HbA1c) was detected at 16 mmol/mol, and she was hospitalized immediately. Gliclazide treatment was discontinued, and insulin glargine and insulin aspartate was initiated. C-peptide was sent with the suspicion of adult's latent autoimmune diabetes (LADA) in a young patient with polyuria, polydipsia, and HbA1c 16 mmol/mol, c-peptide 0.8 µg/L and GAD 65 antibody were found as >250 IU/mL. The patient was diagnosed with LADA, and thereby vildagliptin, and metformin treatment were discontinued. Pancytopenia was detected in whole blood count and deficient vitamin B12 levels in the laboratory. Subsequent peripheral smear confirmed pernicious anemia, and intravenous B12 therapy was initiated. Subclinical hypothyroidism was observed with anti-thyroid peroxidase positivity. Thyroid ultrasonography showed that thyroid gland dimensions were normal, contours were smooth, parenchyma echogenicity was decreased and heterogeneous. The patient was diagnosed with Hashimoto's thyroiditis and started levothyroxine therapy. She was screened for other autoimmune diseases, including autoimmune hepatitis, myasthenia gravis, vitiligo, sarcoidosis, and collagen tissue diseases. None of the further laboratory or clinical investigations suggest any other autoimmune disease, but only granular 1/100 anti-nuclear antibody and a basal cortisol level of 6.6 µg/dL were detected. Subsequent 1 µg ACTH stimulation test excluded the presence of adrenal insufficiency as well.
Conclusions In conclusion, the association of autoimmune diseases should be considered in young patients with diabetes, and LADA must be suspected. In such cases, further investigation of autoimmune thyroiditis, adrenal insufficiency, myasthenia graves, etc., should be performed to support the diagnosis.

ÇALIŞMA-İNGİLİZCE TAM METİN ÖRNEĞİ

Coexistence of Medullary and Papillary Thyroid Carcinomas Detected Incidentally

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ABSTRACT
Background: Papillary thyroid carcinoma (PTC) and medullary thyroid carcinoma (MTC) are extremely rare and constitute less than 0.5% of all thyroid malignancies. In this study, the prevelance and characteristics of patients with simultaneous PTC and MTC diagnoses were evaluated.
Material and Methods: Patients with MTC who were followed up in Uludag University Endocrinology Clinic were retrospectively analyzed and evaluated with literature reviews.
Results: 31 patients diagnosed with MTC were analyzed retrospectively. Coexistence of MTC and PTC were detected in 8 patients. 7 were women and 1 was male. The average age of the patients was 60.1 in the study. Total thyroidectomy was applied to all patients. In addition, central lymph node dissection was performed in 4 patients, and lateral lymph node dissection was performed in 1 patient. None of the patients had an accompanying germline RET mutation. 1 patient was not alive. There was only one patient with distant metastases and was having a tyrosine kinase inhibitor.
Conclusions: Although our results support the coincidental existence of MTC/PTC, physicians should be aware should be aware of the coexistence of these thyroid malignencies to avoid possible misdiagnosis.

Keywords: *Papillary thyroid carcinoma, medullary thyroid carcinoma, thyroid cancer, germline RET mutation.*

Introduction
The incidence of thyroid cancer, the most common cancer of the endocrine system, has increased over the years. Thyroid cancer is the 12th most common cancer with 2.9% of all new cancer cases in the U.S.[1] Papillary thyroid carcinoma (PTC), originating from thyroid follicular epithelial cells, is the most common form of thyroid cancer which accounts for about 70% of thyroid malignancies.[2] Medullary thyroid carcinoma (MTC) develops from parafollicular cells that express calcitonin, and accounts for 1-2% of all thyroid cancers.[3] Coexistence of PTC and MTC is extremely rare, accounts for less than 0.5% of all thyroid malignancies.[4] Here, we report 8 cases of co-existing papillary and medullary thyroid carcinomas with histopathological features, imaging and laboratory findings, outcomes and impact of treatment with the data in the literature.

Material and Methods
We retrospectively analyzed data of 31 patients with medullary thyroid carcinoma between 2012 and 2020 at our institution. The information collected included age, gender, type of surgery, histopathological findings (tumor localization, maximal diameter, capsule invasion, lymphovascular invasion, lymph node involvement and number, tumor stage), presence of RET protooncogene, time since diagnosis of thyroid cancer, previous and current sonographic findings, treatments received for thyroid malignancies, and related comorbidities. Calcitonin, thyroglobulin and anti-thyroglobulin levels were measured with local methods and commercial kits. The staging of thyroid cancers were re-evaluated based on Tumor–Node–Metastasis (TNM) cancer staging system by the new, 8th editions of the relevant Union for International Cancer Control (UICC) and American Joint Committee on Cancer.

Results
The mean age of all patients (MTC and MTC/ PTC) was 53.7±12.01 years at the time of diagno-sis. The mean age of only MTC patients (23/31) was 50.6±10.6 years. Eight of 31 patients (25%) with MTC had

PTC and MTC simultaneously. Overall, median age was 64.5 years (range, 41- 83) in MTC/PTC group, and seven of eight par-ticipants were female, five patients among 8 were treated in Uludag University hospital. Median fol-lowup for these patients was 25 months, with a range of 16-104 months. In 7 of the 8 patients, the maximal diameter of PTC was 10 mm or less. In 7 patients the largest tumor diameter was 23.5±18.5 mm for MTC and 6.75±4.6 mm for PTC. 3 pa-tients had lymph node involvement. Calcitonin, CEA and thyroglobulin were respectively 380 pg/ mL (range: 2-16072), 24.6 mcg/L (range: 1-551.6), and 9.2 mcg/L (range: 0.19-25) preoperatively. Same blood tests were performed after surgery and measurements of serum calcitonin, CEA and thyroglobulin were 2 pg/mL (range: 2-10071), 2.5 mcg/L (range: 0.9-419.3), and 0.27 mcg/L (ran-ge: 0.13-1.4), respectively. Fifty percent of patients (50%, 4/8) had received adjuvant RAI treatment and one patient received tyrosine kinase inhibitor (TKI) therapy.

Discussion

Three previous studies have reported that mean age of only MTC and MTC/PTC patients were respectively 48.2±16.9, 44.5±12.6, median age 44.3 (range: 43-45.7) years for only MTC and 49.9±13.9, 53.5±6.5, median age 50.2 (range: 44.6-55.8) years for MTC/PTC patients.[5-7] In these studies, MTC/PTC patients were older than only MTC patients at the time of diagnosis. The median age in our study was also higher. Despite low numbers, similar to previous studies reported, the median age of patients with MTC/PTC was higher than that of patients with only MTC in our study. Several studies have shown different frequencies of coexistence of MTC and PTC between 3.6% and 19%. The higher frequency (25%) in our study can be explained by the fact that the patients have been diagnosed in recent years and the frequency of PTC has increased significantly in the last 2 decades.[8] In this study, the mean follow-up period of the patients was 25 months. In the previous studies, follow-up time was 32 months (range: 0-261) and 49.1±33.4 months, respectively. The medical records were reviewed from 1996 to 2006 and from 1992 to 2014 by these studies.[5,9] Follow-up period was shorter in this study because most of our patients diagnosed lately. For instance, six of 8 patients with MTC/PTC were diagnosed in 2018 and later. PTC diameter of seven patients was equal or less than 1 cm. In Limh et al.'s study[8], the largest tumor diameter was equal or less than 1 cm in 32.5% of patients with PTC. The fact that mPTC was detected in most of the patients in our study can be explained by the advanced age of the patients. In addition, the reason for this high rate may be related to the fact that fine needle aspiration biopsy is not performed for nodules smaller than 1 cm detected by thyroid ultrasound before surgery. In our study, only 2 patients were diagnosed with PTC preoperatively by FNAB. Similarly, in the Korean study, 9 of 10 patients had mPTC.[6]

In conclusion, although our results support the coincidental existence of MTC/PTC, physicians should be aware of the coexistence of these thyroid malignancies to avoid possible misdiagnosis.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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VAKA SUNUMU-İNGİLİZCE TAM METİN ÖRNEĞİ

Focal Segmental Glomerulosclerosis with Sjogren Syndrome: A Case Report

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ABSTRACT
Cushing's disease (CD) constitutes most common cases of adrenocorticotropic hormone (ACTH) Sjögren's syndrome is a chronic lymphoproliferative disease. Sjögren's syndrome is rarely complicated by focal segmental glomerulosclerosis. Here, we presented a 39-year-old female patient with a history of Sjögren's syndrome for 5 years and diagnosed with focal segmental glomerulosclerosis.
Keywords: *Sjogren syndrome, focal segmental glomerulosclerosis, nephrotic syndrome.*

Introduction

Primary Sjögren's syndrome (pSS) is a chronic, slow-progressing, autoimmune and lymphoproliferative disease. The syndrome's main symptoms are xerostomia and keratoconjunctivitis sicca as a result of chronic inflammatory infiltration of the salivary and lacrimal glands. The exocrinopathy can be encountered alone (pSS or in association with other autoimmune disorders, the three most common ones being rheumatoid arthritis, systemic lupus erythematosus, and progressive systemic sclerosis (secondary SS).[1] Several systemic features have also been described; the presence of autoantibodies against the ubiquitously expressed ribonucleoprotein particles Ro (SS-related antigen A - SSA) and La (SSB) underline the systemic nature of SS. The original explanatory concept for the pathogenesis of pSS proposed a specific, self-perpetuating, immunemediated loss of acinar and ductal cells as the principal cause of salivary gland hypofunction.[2] SS-associated renal disease usually consists of tubulointerstitial nephritis (75% of patients) and glomerulopathy, such as membranous proliferative glomerulonephritis.[3,4] Focal segmental glomerular sclerosis (FSGS) as a dominant pathological finding is found rarely in patients with pSS. Although rare, glomerulonephritis may accompany membranoproliferative glomerulonephritis, membranous nephropathy, and focal mesangioproliferative glomerulonephritis. In our case, FSGS, which is associated with pSS, was considered suitable for the presentation because of its rare occurrence.

Case Report

A 39-year-old woman presented with bilateral leg edema. Urinary examination revealed increased urinary protein levels, and blood tests revealed hypoalbuminemia; thus, she was diagnosed with nephrotic syndrome. She reported a history of pSS diagnosed 5 years earlier. Physical examination of the bilateral lower extremities revealed pitting edema. Urinary examination revealed urinary protein levels of 4700 mg/day, no hematuria. Blood tests revealed the following results: serum albumin 2.6 g/dL, blood urea nitrogen 20 mg/dL, creatinine 0.6 mg/dL, aspartate aminotransferase 22 U/L, alanine aminotransferase 13 U/L, and LDL cholesterol: 215 mg/dL. Anti-SS-A and anti-SS-B antibody test results were positive. Tests for antinuclear antibodies showed a positive result. A kidney biopsy was performed, and the specimen included 23 glomeruli, with 3 showing segmental glomerulosclerosis, detected. There were six glomeruli in the immunofluorescence specimen. Immunofluorescence staining showed no glomerular deposition of immunoglobulin (IgG, IgA, and IgM), C3, or fibrinogen. Based on these findings, she was diagnosed with FSGS. The current patients did not show any electrolyte disturbances in the blood. The blood pH level of the patient did not show acidemia. Hepatitis markers were negative. Histopathological findings in kidney biopsy compatible with FSGS. Steroid (oral prednisolone at a dose of 15 mg/day) and cyclosporine (2x100 mg/d) therapies were initiated. Four weeks after initiation of steroid and cyclosporine therapy, the creatinine levels increased; hence, cyclosporin was discontinued. After 2 months of treatment initiation, urinary protein levels decreased to 303 mg/day and creatinine 0.6 mg/dL, and her leg edema disappeared. Steroid treatment was continued with 7.5 mg/day oral prednisolone. Angiotensin-converting enzyme inhibitor (ramipril) was added to her regimen to decrease urinary protein levels.

Discussion

In the current case, the patient developed nephrotic syndrome secondary to FSGS. Typical renal complications associated with pSS are tubulointerstitial nephritis and renal tubular acidosis. Glomerular diseases manifested by nephrotic syndrome are infrequent in these patients. Although secondary FSGS could be attributed to several etiopathogenetic factors such as familial, viral, drug-induced, structural, and functional responses (nephron depletion and hemodynamic changes)[5], this patient showed no obvious findings that could have resulted in secondary FSGS. Therefore, we could not conclusively establish an association between pSS and FSGS. Goules et al.[6] and Maripuri et al.[7] reported only 5 cases of severe proteinuria or nephrotic interval out of 60 patients who underwent kidney biopsy in his studies. Unfortunately, Kurihara et al.[8] reported that there had been no investigations or case reports that concern the prognosis and clinical response of the cases with FSGS and pSS. Therefore, to discuss the appropriate treatment or prognosis, further accumulation of similar cases with current patients is necessary. Because there are few cases of glomerular diseases coexisting with pSS, it is difficult to describe the best treatment options for each glomerular disease.[8] Treatment for renal involvement primarily includes the administration of corticosteroid, and a few patients receive other immunosuppressants such as cyclosporine, cyclophosphamide, mycophenolate mofetil, and rituximab. Jasiek et al.[9] and Ren et al.[10] reported that 5 cases of 95 patients and 4 cases of 130 patients developed end-stage renal disease. In conclusion, limited data are available regarding renal involvement and prognosis in patients with pSS.

Conflict of Interests

Authors declare that there are none.

Acknowledgment

This study has been presented in 17th Uludag Internal Medicine National Winter Congress, 6th Bursa Family Medicine Association National Congress, 11th Uludag Internal Medicine Nursing Congress, 5–7 March 2021, Bursa, Turkey.

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