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# The Role of Thyroid and Parathyroid Metabolism Disorders in the Etiology of Sudden Onset Dizziness

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**Background:** The aim of this study was to evaluate thyroid and parathyroid functions as a cause of sudden onset dizziness (SOD) in patients who were admitted to the Emergency Department (ED).





**Material/Methods:** This study was conducted prospectively in 100 patients with sudden onset dizziness (SOD) admitted to the ED. Neurologic, ear-nose-throat, detailed neck examinations, serum calcium levels, thyroid function tests (TFT), and parathormone and thyroid ultrasounds were performed on all patients in our study.

**Results:** Thirty-seven (37%) females and 63 (63%) males were included in this study. Four patients (4%) had elevated serum TSH levels, 6 (6%) had decreased serum FT3 levels, 10 (10%) had decreased serum FT4 levels, 2 (2%) had elevated serum FT4 levels, and 2 (2%) had elevated serum parathormone levels. In 4 (4%) patients, the serum calcium levels were lower than normal, and 2 (50%) of these patients had symptomatic hypocalcemia. Thyroid ultrasound examinations showed multinodular goiter in 28 (28%) patients, 2 (2%) patients had thyroiditis, 12 (12%) had an isolated unilateral nodule, and 58 (58%) had normal thyroid tissues.

**Conclusions:** We suggest that detailed neck examination, TFT, and thyroid ultrasound examination should be considered in the diagnostic algorithms of SOD to provide rapid diagnosis and proper treatment for a patient in the ED.

**MeSH Keywords:** **Dizziness • Parathyroid Hormone • Thyroid Function Tests**

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## Background

Dizziness is one of the most common clinical complaints among patients who are referred to the Emergency Department (ED); it has both benign and serious etiologies and affects 20–30% of the general population. Approximately 2.6 million people who suffer from dizziness visit the ED annually in the USA [1]. There are tremendous costs associated with managing dizziness, mainly due to delayed diagnosis, and 86% of patients experience significant disruption in their daily activities [2]. Because dizziness is a symptom of many disorders, clinicians are often faced with difficulty in formulating a definitive diagnosis and effective symptom management [3]. Metabolic disorders are recognized by most authors as a source of balance disorders. Some endocrine diseases, such as hypothyroidism, may lead to balance disorders and can cause dizziness by affecting the vestibular system labyrinthine functions [4,5].

Early discovery of the etiology of dizziness can optimize the diagnosis and symptom management for patients, thereby decreasing costs, ED crowding, and visits to the ED. To date, there has been inadequate research to systematically investigate thyroid and parathyroid function disorders as etiologic factors for sudden onset dizziness (SOD) in patients presenting to the ED.

We examined whether thyroid and parathyroid function disorders played a role in patients who are referred to the ED with complaints of SOD.

## Material and Methods

### Study design

This study was conducted between in August 2013 and February 2014 at the Adiyaman University Medical Faculty Education and Research Hospital ED with the approval of the ethics committee (date/number: 05.02.2013/01-1.1). The study was performed prospectively with 100 SOD patients who experienced their first incidence of SOD. The patients were consecutively referred to the ED, complaining of dizziness that was diagnosed as SOD.

After the patients provided informed consent, a 5-mL sample of venous blood was obtained from the antecubital area of the arm before administering the treatment. Blood samples were studied with the Immulite 2000 analyzer using the chemiluminometric method, measuring serum levels of fT3 (range 1.8–4.6 pg/mL), fT4 (range 0.93–1.7 ng/dL), TSH (range 0.27–4.2 mIU/mL), anti-thyroglobulin antibody (anti-Tg) (range 0–7 IU/mL), antithyroperoxidase antibody (anti-TPO) (range 0–35 IU/mL), parathormone (PTH) (range 12–72 pg/mL), and serum calcium

([Ca<sup>2+</sup>]) (range 8.6–10.2 mg/dL). The serum [Ca<sup>2+</sup>] values were calculated in association with the serum albumin levels. Overt hypothyroidism was defined as elevated serum TSH levels with decreased serum fT3 and fT4 levels with presence of clinical signs of hypothyroidism. Subclinical hypothyroidism was defined as elevated serum TSH levels with normal serum fT3 and fT4 levels without clinical signs of hypothyroidism, or normal serum TSH levels with elevated serum fT4 levels without clinical signs of hypothyroidism. Overt hyperthyroidism was defined as decreased serum TSH levels with elevated serum fT3 and fT4 levels with together clinical signs of hyperthyroidism.

All detailed thyroid and parathyroid gland examinations and ultrasound imaging were performed by a general surgeon and a radiologist, respectively. According to thyroid ultrasonography, findings of tissue echogenicity of thyroid gland were divided into 3 categories: isoechogenic, hypoechogenic, and hyperechogenic.

Adult patients who experienced their first attack of SOD and presenting to the ED were included into study. The patients with SOD who had 1 or more of following disease conditions were excluded from study: undergoing thyroid surgery, any thyroid and parathyroid gland disease patients with any central and peripheral dizziness attacks before time, receiving some drugs affecting thyroid functions such as glucocorticoids, antipsychotics, oral contraceptives and proton pump inhibitors, and the patients who did not wish to participate in the study.

### Statistical analysis

National epidemiologic prevalence data were used to determine the frequency distribution of thyroid metabolism disorders. The general prevalence of thyroid metabolism disorders (TMD) and hyperparathyroidism are 3.4% and 0.03%, respectively, in Turkey [6,7]. The 1-sample z test was used to determine whether the hypothesized population proportion differed significantly from the observed sample proportion. NCSS v1.12 was used for the statistical data analysis. A p value of <0.05 was considered statistically significant.

## Results

A total of 100 patients were included in this study; 37% (n=37) were female, and 63% (n=63) were male. The mean age was 39 years old (19–69, SD±12, 38). The average values of TSH, fT3, fT4, and PTH are shown in Table 1. The results were: 2% (n=2) of the patients had elevated serum TSH levels with decreased serum fT3 and fT4 levels (overt hypothyroidism), 2% (n=2) had elevated serum TSH levels with normal serum fT3 and fT4 levels (subclinical hypothyroidism), 4% (n=4) had normal serum TSH levels with elevated serum fT4 levels (subclinical

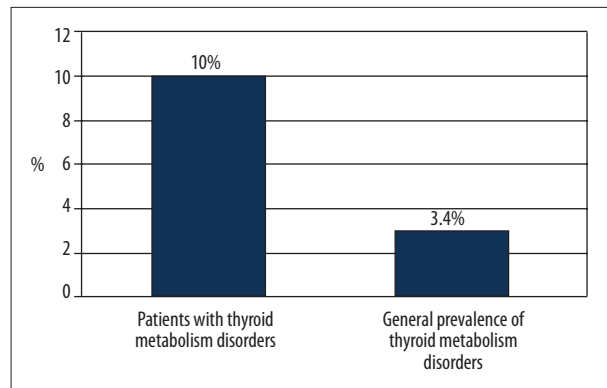
**Table 1.** The average values of TSH, fT3, fT4 and PTH.

TSH mIU/mL, mean (±SD)	1.77±1.11
fT3 pg/mL mean (±SD)	2.98±0.64
fT4 ng/dL mean (±SD)	1.15±0.27
PTH pg/mL mean (±SD)	43.92±15.96

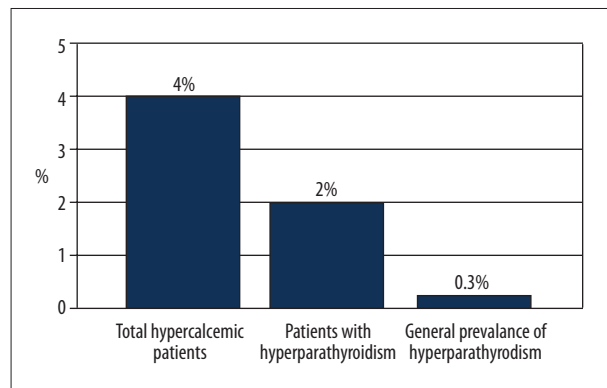
**Table 2.** Thyroid hormone, parathyroid hormone, serum Ca status, and ultrasonographic findings of SOD patients.

		n (%)
Thyroid status	Overt hypothyroidism	2 (2)
	Subclinical hypothyroidism	6 (6)
	Overt hyperthyroidism	2 (2)
	Subclinical hyperthyroidism	–
Parathyroid status	Hyperparathyroidism	2 (2)
	Hypoparathyroidism	–
Ultrasonographic findings	Multinodular goiter	28 (28)
	Nodular goiter	12 (12)
	Thyroiditis	2 (2)
Serum calcium status	Hypocalcaemia	2 (2)
	Hypercalcaemia	4 (4)

hypothyroidism), and 2% (n=2) had decreased serum TSH levels with elevated serum fT3 and fT4 levels (overt hyperthyroidism)

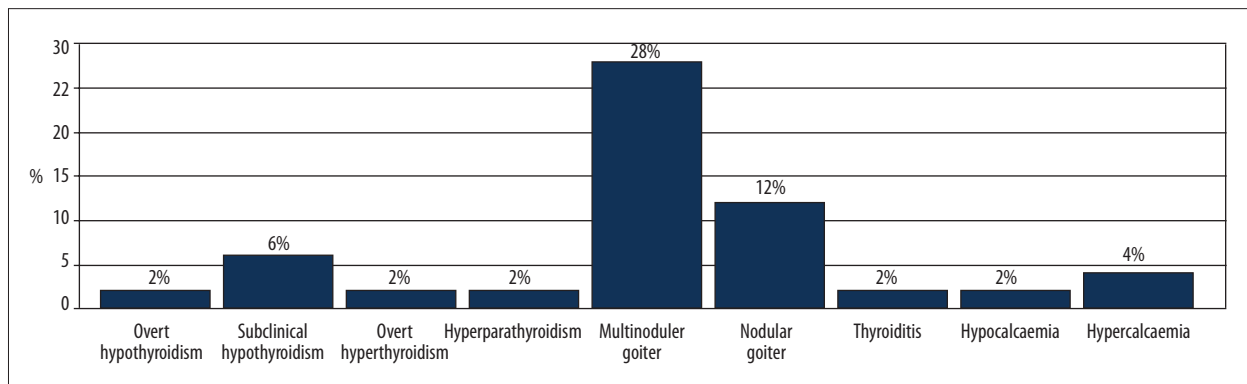


**Figure 2.** Comparison of patients with thyroid metabolism disorders and general prevalence of thyroid metabolism disorders.

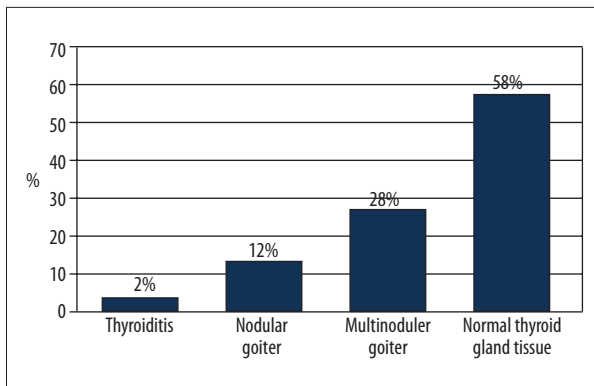


**Figure 3.** The comparison of patients with hypercalcemia, hyperparathyroidism, and general hyperparathyroidism prevalence.

(Table 2 and Figure 1). A total of 10% (n=10) of patients had TMD; this result was statistically significant compared with the general population prevalence (P=0.02) (Figure 2). Two percent (n=2) of the patients had elevated parathormone levels with elevated serum [CaP<sup>2+</sup>] levels, which were statistically significant compared with the general population prevalence (P=0.00) (Figure 3). Four percent (n=4) of the patients



**Figure 1.** Thyroid hormone, parathyroid hormone, serum Ca status, and ultrasonographic findings of SOD patients.



**Figure 4.** Thyroid gland ultrasound findings.

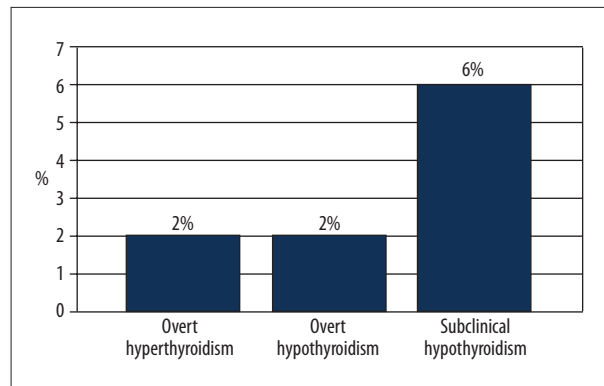
had corrected serum  $[Ca^{2+}]$  levels that were lower than the normal serum range. Thyroid gland ultrasound examinations showed that 28% ( $n=28$ ) of the patients had multinodular goiters, 2% ( $n=2$ ) had thyroiditis, 12% ( $n=12$ ) had an isolated unilateral nodule, and 58% ( $n=58$ ) had normal thyroid gland tissue (Figure 4). The anti-thyroglobulin and anti-topoisomerase levels were normal in all participants.

## Discussion

Dizziness is a common complaint among ED patients and can be caused by multiple diseases and conditions that involve nearly all of the body systems [8,9]. Metabolic events are the cause of approximately 10% of all dizziness complaints [1]. Metabolic disorders can cause dizziness resulting from labyrinthine system dysfunction, which plays a substantial role in the coordination of the balance system [5].

TMD, which is a common disease, and hypothyroidism occur more frequently than hyperthyroidism does. Thyroid hormone plays a role by affecting the maturation and function of the peripheral and central vestibular systems. It was experimentally demonstrated that the presence of alpha- and beta-specific receptors for thyroid hormone, which are present in the ears of mice, are essential for the maturation of the vestibular system [10]. Moreover, rat studies have suggested that thyroid hormones are responsible for the performance of prestin protein, which is directly linked to outer hair cell activity [11]. It has also been observed that neural stimulus conduction in the central vestibular system is impaired when thyroid hormone is absent [12].

Thyroid function abnormalities affect a considerable portion of the population. However, the prevalence depends on ethnic and geographical factors, particularly iodine intake [13]. In contrast to the national prevalence, which is 3.4% [6], we found that the prevalence of TMD is 10% percent in ED patients with SOD (Figure 2). We were surprised to find such a high incidence of TMD in our study population ( $P=0.02$ ). This finding might be an



**Figure 5.** Patients with overt hyperthyroidism, overt hypothyroidism, and subclinical hypothyroidism.

indicator of the necessity to study TFT among those with SOD in the ED because it is possible that TMD is an etiologic agent of SOD and could assist in diagnosis and treatment processes.

There are 2 clinical forms of TMD – hyper- and hypothyroidism – and they may exhibit an overt or subclinical pattern. The pattern of thyroid disorders may vary from society to society [13]. Hollowell et al. reported 0.3% clinical and 4.3% subclinical hypothyroidism and 0.5% clinical and 0.7% subclinical hyperthyroidism in the USA [14]. A study conducted by Kutluturk et al. [15] in northern Turkey demonstrated that the prevalence of hypothyroidism and hyperthyroidism was 4.3% (1.6% overt, 2.7% subclinical) and 5.5% (0.6% overt, 4.9% subclinical), respectively. In the present study, the rate of hypothyroidism was 8%, 2% of which was overt and 6% subclinical, and the rate of hyperthyroidism was 2%, which was entirely overt. These results are not compatible with the aforementioned study results (Figure 5). Our results demonstrated a significantly higher rate of hypothyroidism than did the results of Kutluturk et al. [15]. This variation may be due to studying TMD among SOD patients and the role of thyroid hormone on the vestibular system, as mentioned above. Therefore, we suggest that hypothyroidism should be kept in mind as a causative agent in patients with SOD who are admitted to the ED.

All patients with thyroid and parathyroid disorders were consulted by the general surgeon who conducted this study. We did not give any treatment for thyroid and parathyroid disorders to these patients in the ED because we only aimed to discover the etiology of SOD and there have been debates about the treatment of subclinical thyroid and parathyroid disorders [16]. After the patients were diagnosed with subclinical thyroid and parathyroid disorders, they were followed clinically by the general surgeon and/or an endocrinologist.

Assessing the serum anti-Tg and anti-TPO concentration levels assists in the diagnosis of chronic autoimmune thyroiditis (CAT), which is called Hashimoto's thyroiditis. These antibodies are

higher in number in CAT, at a rate of 95%–100% [13]. Although several studies have found that CAT is accompanied by diseases such as Ménière's disease and benign positional paroxysmal vertigo (BPPV) [17,18], debate remain about these conditions occurring together [19]. In our study, none of the patients with SOD had a higher level of anti-Tg or anti-TPO antibody titrations.

Previous studies have demonstrated that thyroid nodules are found by palpation in 4–8% of the general population, 19–67% of patients with the use of ultrasonography, and 50% of autopsy specimens [20]. In our study, we determined a rate of 42% of patients exhibiting ultrasonographic abnormalities, which is compatible with the data in the literature and included thyroiditis and both multinodular and nodular goiters (Figure 4). Thus, we suggest that thyroid gland ultrasonographic imaging should be performed on all patients who have been referred to the ED with SOD, even if palpation of the thyroid gland is normal and especially if the patient comes from an area of known low iodine levels.

In outpatient settings, hypercalcemia is typically mild and asymptomatic and is determined accidentally during routine laboratory monitoring. Serum  $[Ca^{2+}]$  levels are affected by some parathyroid diseases that are also endocrine disorders. The most common cause of hypercalcemia is hyperparathyroidism. A variety of factors can cause hyperparathyroidism, such as single (85%) or multiple (5%) benign parathyroid adenomas, diffuse parathyroid hyperplasia (10%), and paraneoplastic syndromes or exogenous intake [21,22]. The administration of some doses of parathormone to osteoporotic patients leads to dizziness. In a previous study, the infusion of 20 mg of exogenous human parathyroid hormone (hPTH) induced dizziness in a substantial number of patients [23]. The general prevalence of hyperparathyroidism is approximately 0.03% [7]. In this study, we identified that 2% of the patients had hyperparathyroidism with elevated serum  $[Ca^{2+}]$ . This is a higher rate than observed in the normal population, and it was statistically significant ( $p=0.00$ ) (Figure 3).

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Hypocalcemia occurs at a rate of 18% on secondary and tertiary care biochemical screening tests. It may be an asymptomatic laboratory finding or a life-threatening metabolic disturbance. Hypocalcemia is generally observed in patients with impaired function of the parathyroid glands. This is most common after thyroid or parathyroid surgery, but it is mostly idiopathic in young adults and occurs less often as part of a genetic syndrome, such as autoimmune polyglandular syndrome type-1 [24]. In our study, we excluded patients who had had thyroid surgery and known parathyroid disease, and we found that a total of 2 of 4 hypocalcemic patients had symptomatic hypocalcemia. This rate was lower than that found in the second and tertiary care results, as mentioned above, suggesting that this may be a cause of SOD in symptomatic hypocalcemic patients. Many neurological symptoms, such as fatigue, irritability, and numbness, may have been described as dizziness by the patients [25].

## Limitations

In our study, instead of a control group, the use of national prevalence data was preferred to compare study findings. The relationships between thyroid and parathyroid gland ultrasound findings and dizziness are not discussed here. Additionally, hyperthyroidism and hyperparathyroidism etiologic factor analyses were not conducted longitudinally.

## Conclusions

Thyroid and parathyroid disease play a role in dizziness etiology in different ways. We observed that TMD has a higher prevalence in SOD patients than in the normal population. Thus, detailed neck examination, TFT, and thyroid ultrasound examination should be routinely performed to provide a quick diagnosis and proper treatment for a patient who is admitted to the ED with SOD.

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