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Case Report

Thyroid Hormone Resistance Syndrome: A Case Report with Literature Review

Genevieve Kela[1], Hamed Abdelma'aboud Mostafa[2], Hossam Gazya[3], Nourhan Ahmed[4], Ahmed Hassan[5,*]

1-Endocrine and Diabetes Division, UMass Chan Medical School - Baystate, Springfield, MA, USA
2-Faculty of Medicine, Al-Azhar University, Damietta, Egypt
3-Faculty of Medicine, Ain Shams University, Cairo, Egypt
4-Department of Nephrology, Suez Medical Complex, Ministry of Health and Population, Suez, Egypt
5-Department of Cardiology, Suez Medical Complex, Suez, Egypt

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ABSTRACT

Resistance to thyroid hormone receptor beta (THRβ) is a rare condition causing abnormal thyroid function tests (TFTs) characterized by elevated thyroid hormone levels with unsuppressed Thyroid Stimulating Hormone (TSH). Thyroid hormone action involves multiple steps, and mutations affecting these steps are key to understanding and managing thyroid disorders. We present a case of THRβ resistance associated with cardiac arrhythmia. A 40-year-old male with a history of atrial fibrillation (AF) was referred for evaluation of abnormal TFTs and thyroid nodules. TFTs revealed a normal TSH and elevated free thyroxine. Imaging showed a large, peripherally enhancing necrotic mass with calcification in the left thyroid lobe and a 0.8 cm hypodense area in the right lobe. Thyroid ultrasound confirmed bilateral nodules, with the largest in the lower pole of the left lobe. The fine-needle aspiration biopsy was benign (Bethesda category II). Inherited THRβ pathogenic variants cause thyroid hormone resistance, often resulting in an enlarged thyroid gland. Despite this resistance, patients may still show clinical signs of cardiac arrhythmias. Diagnosing thyroid hormone resistance helps avoid unnecessary treatment for asymptomatic patients.

1. Introduction

Thyroid hormone resistance (THR) is a rare genetic condition where tissues respond poorly to thyroid hormones [1]. Mutations in the thyroid hormone receptor beta (THRβ) gene cause about 85% of cases, with over 100 mutations identified. Neomutations account for the remaining 15%, meaning affected individuals may lack a family history of the disorder unless they pass it on to their children [2]. Researchers have classified THRβ into subtypes to improve understanding and treatment [3]. Clinicians describe it as "generalized" when patients are euthyroid and as "pituitary resistance" when hyperthyroid symptoms appear, as reported by Guo et al. [4, 5]. They use the term "isolated peripheral THR" when TSH decreases after high doses of liothyronine (L-T3) without hyperthyroid symptoms, documented in one patient [6]. Genetic classifications identify THR as either homozygous or heterozygous, with subtypes based on mutation characteristics [3]. Most cases involve heterozygous dominant-negative THRβ mutations, resulting in defective receptors. Only four homozygous cases have been reported, showing severe symptoms like growth restriction, vision and hearing impairments, and cardiac anomalies [7]. Although most patients with THR remain euthyroid, some show symptoms of hypothyroidism or hyperthyroidism [3]. Elevated free thyroid hormone levels with a non-suppressed TSH serve as the primary diagnostic indicator of tissue hyporesponsiveness [5]. Studying family members with similar thyroid test results can confirm the genetic basis of the disorder and reveal variability in thyroid hormone sensitivity [4]. Currently, no treatment can fully correct the defect in THR [6]. However, most individuals naturally compensate by increasing thyroid hormone production, often eliminating the need for medical intervention [7]. This article presents a case of THRβ with cardiac arrhythmia, confirmed through genetic testing.

2. Case Presentation

A 40-year-old man with a history of atrial fibrillation (AF) presented to the endocrinology clinic for evaluation of abnormal thyroid function tests (TFTs) and a thyroid nodule. He had recently been hospitalized for AF with a rapid ventricular response. He also had a history of receiving a two-month course of amiodarone treatment three years earlier. Initially, we found discrepant TFTs in which TSH was 1.375 (normal range: 0.55–4.78) and Free Thyroxine (fT4) was elevated at 2.76 (normal range: 0.89 – 1.76). Regarding imaging studies, a neck computed tomography (CT) scan showed a large, peripherally enhancing mass with central necrosis and calcification encompassing nearly the entire left thyroid lobe ($2.2 \times 3.6 \times 4.6$ cm). The scan also identified a 0.8 cm hypodense area in the right thyroid lobe. Thyroid Ultrasound: Bilateral thyroid nodules were present, with the largest nodule in the lower pole of the left lobe **(Figure 1)**. Regarding Fine Needle Aspiration (FNA) Biopsy, the left thyroid nodule showed Bethesda Category II findings, including benign-appearing follicular cells and abundant colloid. MRI brain ruled out TSH-secreting adenoma. He reported

* Corresponding author: Department of Cardiology, Suez Medical Complex, Suez, Egypt, Email: drahmedmhassan3@gmail.com

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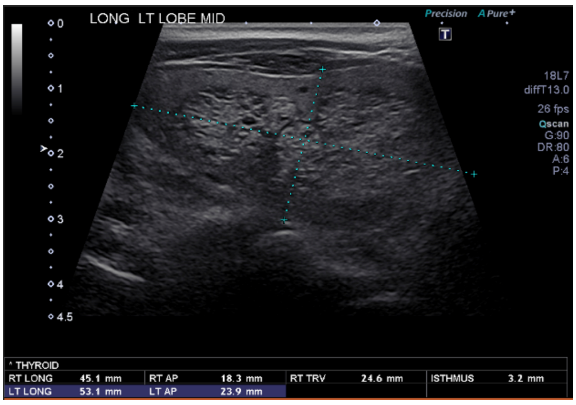

Figure 1: Thyroid ultrasound showed nodules bilaterally, with the largest one at the lower pole of the left lobe.

thyroid abnormalities in his son and brother but was unable to provide details. He was referred for genetic screening, which showed THRβ with a pathogenic variant: c.9628>G (p.Y321C)

3. Discussion

THR is a rare clinical syndrome marked by reduced sensitivity to thyroid hormone, primarily caused by mutations in the THRβ gene. THRβ affects approximately one in 40,000 individuals [8, 9]. Specific genetic mutations cause wide variations in its pathogenesis, influencing symptoms and signs. Refetoff et al. first described THRβ resistance in 1967, and Sakurai et al. identified the initial THRβ gene mutation in 1989 [3, 10, 11]. THR is characterized by elevated thyroid hormone levels with normal or slightly increased TSH levels [12]. Assay interference, such as interfering antibodies (anti-streptavidin, anti-ruthenium, heterophilic, or anti-T4/T3 antibodies), substances like biotin, treatments including heparin or nonsteroidal anti-inflammatory drugs, and changes in thyroid hormone transport proteins (albumin, transthyretin, and thyroxine-binding globulin) can cause these paradoxical biochemical findings. A TSH-secreting pituitary adenoma (TSH-oma) also serves as a significant differential diagnosis for THR [13, 3]. Campi et al. reported that 12% of 100 subjects with inappropriate TSH secretion were misdiagnosed, including a patient with THRβ resistance initially diagnosed with a TSH-producing tumor [14]. Several cases have highlighted the challenges of accurate diagnosis. Liao et al. documented a 54-year-old woman misdiagnosed with a TSH-oma, who showed no improvement after transsphenoidal surgery; genetic studies later confirmed THRβ [15, 16, 17, 18]. In Taiwan, Liu et al. described a 10-year-old boy with goiter misdiagnosed as having hyperthyroidism, but molecular studies eventually revealed THRβ [19]. Most patients showed no symptoms because excess thyroid hormone production compensated for tissue resistance [20]. When symptoms did appear, the primary clinical features of THRβ included goiter (66%-95%), emotional disturbances (60%), attention deficit hyperactivity disorder (ADHD) (40%-60%), sinus tachycardia (33%-75%), and hearing impairment [21, 22, 12]. Less common features included variable degrees of mental retardation, short stature with reduced subischial leg length, chronic constipation, and bradycardia [22]. In our case, initial TFTs showed normal TSH but elevated fT4 of 2.76. Imaging revealed a large mass in the left thyroid lobe with calcifications and additional nodules bilaterally. FNA biopsy indicated benign findings (Bethesda category II). Despite persistently elevated fT4 and normal TSH, the patient remained clinically euthyroid for 1.5 years. Repeat labs eventually showed elevated TSH and fT4, but the MRI ruled out a TSH-oma. Genetic screening identified a pathogenic variant in the THRβ gene, which explained the thyroid abnormalities. Ferrara et al. reported three THR cases with homozygous THRβ mutations causing mental retardation, tachycardia, goiter, hearing loss, and growth retardation [23]. Takeda et al. described a case with a complete absence of both THRβ alleles [24, 25], while Usala et al. documented an amino acid deletion causing THRβ [26]. Heterozygous cases showed variable symptoms based on tissue resistance and mutant TRβ protein expression [12]. THR has also been linked to congenital hypothyroidism, thyroid dysgenesis, and ectopic thyroid tissue, with five cases reported [27, 28, 29, 30, 31]. ADHD is present in 48-83% of patients, typically treated with standard methods. L-T3 has been effective in refractory cases, improving insomnia and hyperactivity[32, 33]. In 2017, Moran et al. reported a pediatric homozygous THRβ resistance case with dilated cardiomyopathy and thyrotoxicosis, treated with Triac and methimazole, leading to reduced thyroid hormone, normal TSH, and improved growth and cardiac function [34]. A large retrospective study revealed three key findings regarding THRβ resistance during pregnancy: A) higher miscarriage rates in pregnancies with THRβ resistance and unaffected fetuses; B) unaffected infants born to mothers with THRβ resistance had lower birth weights, possibly due to excess thyroid hormone crossing the placenta; and C) affected infants had normal birth weights due to impaired thyroid hormone sensitivity [22]. Treatment for pregnant women with THRβ resistance should be individualized, considering past pregnancy history and fetal genotype after amniocentesis [22]. Kahaly et al. examined the cardiovascular traits of patients with THRβ in comparison to those with hyperthyroidism, hypothyroidism, and euthyroid controls. Their study found that 26% of THRβ patients experienced tachycardia, 32% reported palpitations, 4% had dyspnea, 6% developed atrial fibrillation, and 4% exhibited mitral valve prolapse. These symptoms were less common and milder than in hyperthyroid patients. Additionally, the echocardiographic analysis revealed that the cardiac systolic and diastolic functions of THRβ patients were between those of hyperthyroid and euthyroid individuals [35]. THR treatment is individualized, as no curative therapy exists for THRβ defects [3]. Synthesized TH analogs and Roxadustat show promise in vitro but lack in vivo results [36], [37]. TSH suppression with high-dose T3 effectively reduces goiter size, avoiding surgery due to high recurrence rates [38]. Most THR-related thyroid nodules are benign, though some cases of papillary carcinoma require thyroidectomy and radioactive iodine, often resulting in poor outcomes. 3,3,5-Triiodothyroacetic Acid (Triac), combined with levothyroxine, beta-blockers, and calcium/vitamin D, has shown success in managing symptoms like tachycardia, attention deficits, and goiter [39, 40, 3].

4. Conclusions

THRβ resistance is an uncommon and often underdiagnosed endocrine disorder. Pathogenic variants in the THRβ gene cause resistance to thyroid hormone, typically presenting with elevated thyroid hormone levels and thyroid gland enlargement. Despite receptor resistance, patients may develop, likely due to elevated T4 and T3 concentrations affecting the heart, where Thyroid hormone receptor alpha (THRα) is predominantly expressed. Recognizing thyroid hormone resistance is essential to avoid unnecessary treatment in asymptomatic cases.

Conflicts of Interest

The authors declare no conflicts of interest.

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Informed consent

Obtained from the patient.

Large Language Model

None

Authors Contribution

GK Conceptualization; case identification; writing original draft. HGA Case presentation: introduction; writing—original draft. NAA Discussion: writing original draft. AHA Corresponding author; review. All authors reviewed and approved the final manuscript.

Data Availability

All data supporting the findings of this study are included in the article. Additional information is available from the corresponding author upon reasonable request.

Declaration

This case was previously presented as a poster at the Endocrine Society Meeting 2024. Poster abstract link: here

References

1. Refetoff S, Bassett JH, Beck-Peccoz P, Bernal J, Brent G, Chatterjee K, et al. Classification and proposed nomenclature for inherited defects of thyroid hormone action, cell transport, and metabolism. J Clin Endocrinol Metab. 2014;99(3):768-70. [PMID: 24823702, PMCID: PMC3942236, https://doi.org/10.1210/jc.2013-3393].
2. Lee JH, Kim EY. Resistance to thyroid hormone due to a novel mutation of thyroid hormone receptor beta gene. Ann Pediatr Endocrinol Metab. 2014;19(4):229-31. [PMID: 25654071, PMCID: PMC4316411, https://doi.org/10.6065/apem.2014.19.4.229].
3. Pappa T, Refetoff S. In: Resistance to thyroid hormone beta: A focused review. vol. 12;. p. 656551-1. [PMCID: PMCID, https://doi.org/10.3389/fendo.2021.656551.].
4. Dumitrescu AM, Refetoff S, Endotext KRF, Anawalt B, Boyce A, Chrousos G, et al.. Impaired sensitivity to thyroid hormone: defects of transport, metabolism and action. MDText.com, Inc;.
5. Singh BK, Yen PM. A clinician's guide to understanding resistance to thyroid hormone due to receptor mutations in the TRalpha and TRbeta isoforms. Clin Diabetes Endocrinol. 2017;3:8. [PMID: 28932413, PMCID: PMC5603052, https://doi.org/10.1186/s40842-017-0046-z].
6. Weiss RE, Refetoff S. Treatment of resistance to thyroid hormone–primum non nocere. J Clin Endocrinol Metab. 1999;84(2):401-4. [PMID: 10022391, https://doi.org/10.1210/jcem.84.2.5534].
7. Chiamolera MI, Sidhaye AR, Matsumoto S, He Q, Hashimoto K, Ortiga-Carvalho TM, et al. Fundamentally distinct roles of thyroid hormone receptor isoforms in a thyrotroph cell line are due to differential DNA binding. Mol Endocrinol. 2012;26(6):926-39. [PMID: 22570333, PMCID: PMC3355539, https://doi.org/10.1210/me.2011-1290].
8. Lafranchi SH, Snyder DB, Sesser DE, Skeels MR, Singh N, Brent GA, et al. Follow-up of newborns with elevated screening T4 concentrations. J Pediatr. 2003;143(3):296-301. [PMID: 14517508, https://doi.org/10.1067/S0022-3476(03)00184-7].
9. Tajima T, Jo W, Fujikura K, Fukushi M, Fujieda K. Elevated free thyroxine levels detected by a neonatal screening system. Pediatr Res. 2009;66(3):312-6. [PMID: 19542904, https://doi.org/10.1203/PDR.0b013e3181b1bcbd].
10. Refetoff S, DeWind LT, DeGroot LJ. Familial syndrome combining deaf-mutism, stuppled epiphyses, goiter and abnormally high PBI: possible target organ refractoriness to thyroid hormone. J Clin Endocrinol Metab. 1967;27(2):279-94. [PMID: 4163616, https://doi.org/10.1210/jcem-27-2-279].
11. Sakurai A, Takeda K, Ain K, Ceccarelli P, Nakai A, Seino S, et al. Generalized resistance to thyroid hormone associated with a mutation in the ligand-binding domain of the human thyroid hormone receptor beta. Proc Natl Acad Sci U S A. 1989;86(22):8977-81. [PMID: 2510172, PMCID: PMC298414, https://doi.org/10.1073/pnas.86.22.8977].
12. Ortiga-Carvalho TM, Sidhaye AR, Wondisford FE. Thyroid hormone receptors and resistance to thyroid hormone disorders. Nat Rev Endocrinol. 2014;10(10):582-91. [PMID: 25135573, PMCID: PMC4578869, https://doi.org/10.1038/nrendo.2014.143].
13. Donnars A, Leplat A, Grosheny C, Briet C, Illouz F, Bouzamondo N, et al. Clinically Symptomatic Resistance to Thyroid Hormone beta Syndrome Because of THRB Gene Mosaicism. J Clin Endocrinol Metab. 2022;107(9):e3548-52. [PMID: 35689814, https://doi.org/10.1210/clinem/dgac347].
14. Campi I. In: The Differential Diagnosis of Discrepant Thyroid Function Tests: Insistent Pitfalls and Updated Flow-Chart Based on a Long-Standing Experience. vol. 11; 2020. p. 432-2. [PMCID: PMCID, https://doi.org/10.3389/fendo.2020.00432.].
15. Carvalho Cunha N, Gomes L, Bastos M. Challenging diagnosis of resistance to thyroid hormone in a patient with pituitary adenoma. BMJ Case Rep. 2019;12(7). [PMID: 31326901, PMCID: PMC6663254, https://doi.org/10.1136/bcr-2019-229430].
16. Kong AP, Lam CW, Chan AO, Yiu SF, Tiu SC. Resistance to thyroid hormone in a Chinese family with R429Q mutation in the thyroid hormone receptor beta gene. Hong Kong Med J. 2005;11(2):125-9. [PMID: 15815068].
17. Suntornlohanakul O, Sriphrapradang C. Pituitary hyperplasia mimicking thyrotropin-producing pituitary adenoma in the patient with resistance to thyroid hormone: a case report. Int J Neurosci. 2022;132(2):207-11. [PMID: 32727243, https://doi.org/10.1080/00207454.2020.1803304].
18. Yu C, Zhao J, Yao J, Wang H, Shang H, Zhang R, et al. Pituitary resistance to thyroid hormone caused by a novel mutation (H435A) in the thyroid hormone receptor beta: A case report. Medicine (Baltimore). 2018;97(21):e10544. [PMID: 29794730, PMCID: PMC6392742, https://doi.org/10.1097/MD.0000000000010544].
19. Liu Z, Tsai WY, Lee CT. Resistance to thyroid hormone due to a novel THRB p.Val349Ala mutation in a Taiwanese boy. J Formos Med Assoc. 2020;119(10):1546-9. [PMID: 32505587, https://doi.org/10.1016/j.jfma.2020.05.035].
20. Liao W, Waisayanand N, Fanhchaksai K, Visser WE, Meima ME, Wejaphikul K. Resistance to Thyroid Hormone Beta Due to THRB Mutation in a Patient Misdiagnosed With TSH-Secreting Pituitary Adenoma. JCEM Case Rep. 2024;2(8):luae140. [PMID: 39091608, PMCID: PMC11291949, https://doi.org/10.1210/jcemcr/luae140].
21. Agrawal NK, Goyal R, Rastogi A, Naik D, Singh SK. Thyroid hormone resistance. Postgrad Med J. 2008;84(995):473-7. [PMID: 18940949, https://doi.org/10.1136/pgmj.2008.069740].
22. Onigata K, Szinnai G. Resistance to thyroid hormone. Endocr Dev. 2014;26:118-29. [PMID: 25231448, https://doi.org/10.1159/000363159].
23. Ferrara AM, Onigata K, Ercan O, Woodhead H, Weiss RE, Refetoff S. Homozygous thyroid hormone receptor beta-gene mutations in resistance to thyroid hormone: three new cases and review of the literature. J Clin Endocrinol Metab. 2012;97(4):1328-36. [PMID: 22319036, PMCID: PMC3319181, https://doi.org/10.1210/jc.2011-2642].

24. Takeda K, Balzano S, Sakurai A, DeGroot LJ, Refetoff S. Screening of nineteen unrelated families with generalized resistance to thyroid hormone for known point mutations in the thyroid hormone receptor beta gene and the detection of a new mutation. J Clin Invest. 1991;87(2):496-502. [PMID: 1991834, PMCID: PMC296336, https://doi.org/10.1172/JCI115023].
25. Takeda K, Sakurai A, DeGroot LJ, Refetoff S. Recessive inheritance of thyroid hormone resistance caused by complete deletion of the protein-coding region of the thyroid hormone receptor-beta gene. J Clin Endocrinol Metab. 1992;74(1):49-55. [PMID: 1727829, https://doi.org/10.1210/jcem.74.1.1727829].
26. Usala SJ, Menke JB, Watson TL, Wondisford FE, Weintraub BD, Berard J, et al. A homozygous deletion in the c-erbA beta thyroid hormone receptor gene in a patient with generalized thyroid hormone resistance: isolation and characterization of the mutant receptor. Mol Endocrinol. 1991;5(3):327-35. [PMID: 1653889, https://doi.org/10.1210/mend-5-3-327].
27. Grasberger H, Ringkananont U, Croxson M, Refetoff S. Resistance to thyroid hormone in a patient with thyroid dysgenesis. Thyroid. 2005;15(7):730-3. [PMID: 16053391, https://doi.org/10.1089/thy.2005.15.730].
28. Guo ML, Zheng X, Yang LX, Qiu YL, Cheng L, Ma SG. Coexistence of resistance to thyroid hormone and ectopic thyroid: ten-year follow-up. Arch Endocrinol Metab. 2016;60(6):601-4. [PMID: 27737329, PMCID: PMC10522167, https://doi.org/10.1590/2359-3997000000214].
29. Heather N, Hall K, Neas K, Potter H, Wiltshire E. Growth and development in a child with resistance to thyroid hormone and ectopic thyroid gland. Pediatrics. 2012;129(3):e817-20. [PMID: 22311990, https://doi.org/10.1542/peds.2011-0634].
30. Nakajima Y, Yamada M, Horiguchi K, Satoh T, Hashimoto K, Tokuhiro E, et al. Resistance to thyroid hormone due to a novel thyroid hormone receptor mutant in a patient with hypothyroidism secondary to lingual thyroid and functional characterization of the mutant receptor. Thyroid. 2010;20(8):917-26. [PMID: 20615127, https://doi.org/10.1089/thy.2009.0389].
31. Zhou Z, Yang C, Lv F, Liu W, Yan S, Zang H, et al. Novel THRB mutation analysis in congenital hypothyroidism with thyroid dysgenesis. J Cell Biochem. 2018;119(11):9474-82. [PMID: 30074255, https://doi.org/10.1002/jcb.27264].
32. Maruo Y, Mori A, Morioka Y, Sawai C, Mimura Y, Matui K, et al. Successful every-other-day liothyronine therapy for severe resistance to thyroid hormone beta with a novel THRB mutation; case report. BMC Endocr Disord. 2016;16:1. [PMID: 26754848, PMCID: PMC4709977, https://doi.org/10.1186/s12902-015-0081-7].
33. Weiss RE, Stein MA, Refetoff S. Behavioral effects of liothyronine (L-T3) in children with attention deficit hyperactivity disorder in the presence and absence of resistance to thyroid hormone. Thyroid. 1997;7(3):389-93. [PMID: 9226208, https://doi.org/10.1089/thy.1997.7.389].
34. Moran C, Habeb AM, Kahaly GJ, Kampmann C, Hughes M, Marek J, et al. Homozygous Resistance to Thyroid Hormone beta: Can Combined Antithyroid Drug and Triiodothyroacetic Acid Treatment Prevent Cardiac Failure? J Endocr Soc. 2017;1(9):1203-12. [PMID: 29264576, PMCID: PMC5686666, https://doi.org/10.1210/js.2017-00204].
35. Kahaly GJ, Matthews CH, Mohr-Kahaly S, Richards CA, Chatterjee VK. Cardiac involvement in thyroid hormone resistance. J Clin Endocrinol Metab. 2002;87(1):204-12. [PMID: 11788648, https://doi.org/10.1210/jcem.87.1.8170].
36. Shi Y, Ye H, Link KH, Putnam MC, Hubner I, Dowdell S, et al. Mutant-selective thyromimetics for the chemical rescue of thyroid hormone receptor mutants associated with resistance to thyroid hormone. Biochemistry. 2005;44(12):4612-26. [PMID: 15779888, https://doi.org/10.1021/bi0482349].
37. Yao B, Wei Y, Zhang S, Tian S, Xu S, Wang R, et al. Revealing a Mutant-Induced Receptor Allosteric Mechanism for the Thyroid Hormone Resistance. iScience. 2019;20:489-96. [PMID: 31655060, PMCID: PMC6806671, https://doi.org/10.1016/j.isci.2019.10.002].
38. Anselmo J, Refetoff S. Regression of a large goiter in a patient with resistance to thyroid hormone by every other day treatment with triiodothyronine. Thyroid. 2004;14(1):71-4. [PMID: 15009917, https://doi.org/10.1089/105072504322783876].
39. Anzai R, Adachi M, Sho N, Muroya K, Asakura Y, Onigata K. Long-term 3,5,3'-triiodothyroacetic acid therapy in a child with hyperthyroidism caused by thyroid hormone resistance: pharmacological study and therapeutic recommendations. Thyroid. 2012;22(10):1069-75. [PMID: 22947347, https://doi.org/10.1089/thy.2011.0450].
40. Groeneweg S, Peeters RP, Visser TJ, Visser WE. Therapeutic applications of thyroid hormone analogues in resistance to thyroid hormone (RTH) syndromes. Mol Cell Endocrinol. 2017;458:82-90. [PMID: 28235578, https://doi.org/10.1016/j.mce.2017.02.029].