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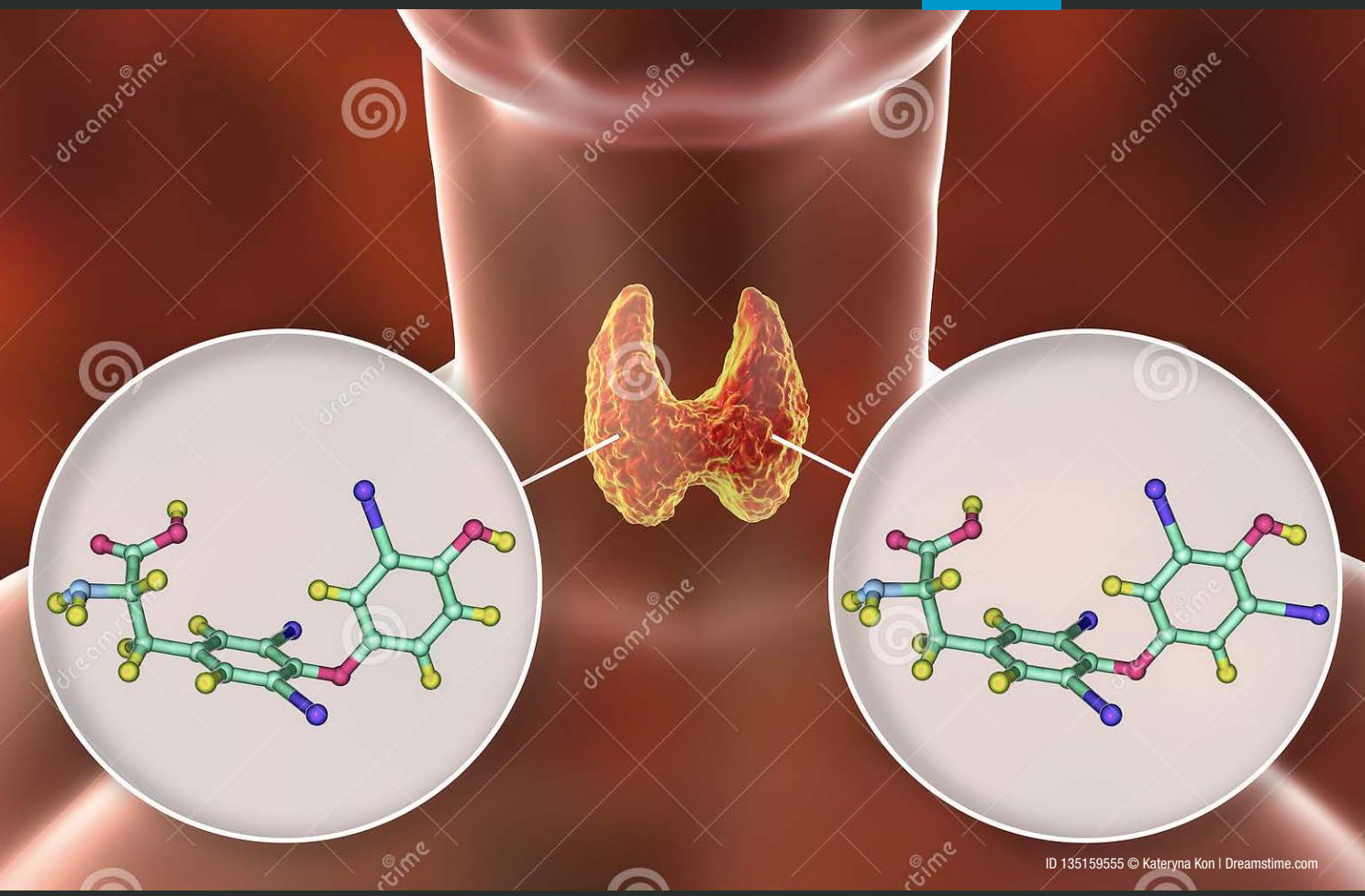

Dental management of patients with Graves' disease

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Dental management of patients with Graves' disease

Abstract

This article will highlight the oral disease risk factors present in patients with Graves' disease and help restructure the dental management for these patients. Graves' disease is an autoimmune disorder affecting thyroid function and is the most common cause of hyperthyroidism, which is the overproduction of thyroid hormone. Graves' disease positively correlates with periodontitis and can also cause a greater susceptibility to dental caries, osteoporosis of the maxilla and mandible, burning mouth syndrome, and Sjögren's syndrome. Collaboration between dental professionals and endocrinologists is needed to ensure optimal oral and systemic health for patients with Graves' disease and to accelerate the prevention and detection of the associated oral diseases. Dental professionals should be aware of the symptoms of Graves' disease and adverse reactions to treatment options. A shorter dental recall interval for patients with Graves' disease is beneficial for preventing and arresting multiple dental diseases.

Educational objectives

Upon completion of the course, participants will be able to:

1. Describe the definition, etiology, symptoms, and treatment for Graves' disease
2. Identify the clinical oral manifestations of patients with Graves' disease
3. Define systemic, oral, and mental health implications for patients with Graves' disease
4. Define recommended treatment and recare intervals for patients with Graves' disease

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What is Graves' disease?

Graves' disease is an autoimmune disorder in which the immune system overstimulates the body's thyroid. The thyroid gland is enlarged when antibodies called thyroid-stimulating immunoglobulins (TSI) attach to its receptors and stimulate its production of too much thyroid hormone. The exact etiology of thyroid mechanisms leading to thyroid autoimmunity is not yet clear. It is believed that it arises from genetics, epigenetics, and environmental factors. As the genetic contributors are more closely studied, it has been revealed that up to seven genes can be involved. The more affected genes a person has, the more likely thyroid autoimmunity will occur. Environmental factors include iodine excess or deficiency, bacterial or fungal infections such as hepatitis C, smoking, or stress, and environmental factors from coal, crude, and gasoline.[1]

Having too much thyroid hormone in circulation causes an increase in metabolism and is commonly known as hyperthyroidism.[2] The immune system creates antibodies that alert the thyroid to become more active, resulting in the gland producing an excess of thyroid hormones.[3] Graves' disease is the most common cause of hyperthyroidism and thyrotoxicosis, accounting for 20 to 50 cases per 100,000 individuals annually.[3] Thyrotoxicosis is the clinical state of excessive amounts of thyroid hormones such as triiodothyronine (T3) and thyroxine (T4), which are two of the main hormones released into the bloodstream by the thyroid.[4] T4 and T3 are the primary hormones that circulate in normal function.[5]

The usual range of T4 for adults is 5.0–11.0 ug/dL, and for T3 it's 100–200 ng/dL; blood tests that show higher ranges for T3 and T4 indicate hyperthyroidism.[5]

Another important hormone involved in Graves' disease and hyperthyroidism is thyroid-stimulating hormone (TSH). The pituitary gland secretes TSH, which, in individuals with normal thyroid function, binds to the TSH receptor site on the thyroid and facilitates the release of T3 and T4.[6] However, in Graves' disease, the immune system secretes TSIs that compete for the TSH receptor site. Subsequently, the pituitary gland decreases the amount of TSH output because the TSIs have stimulated the thyroid to produce hormones.[6] **(Table 1)** Additionally, TSH levels are abnormally low in patients with hyperthyroidism. The average value for TSH in the blood ranges from 0.40 to 4.50 mIU/mL, and

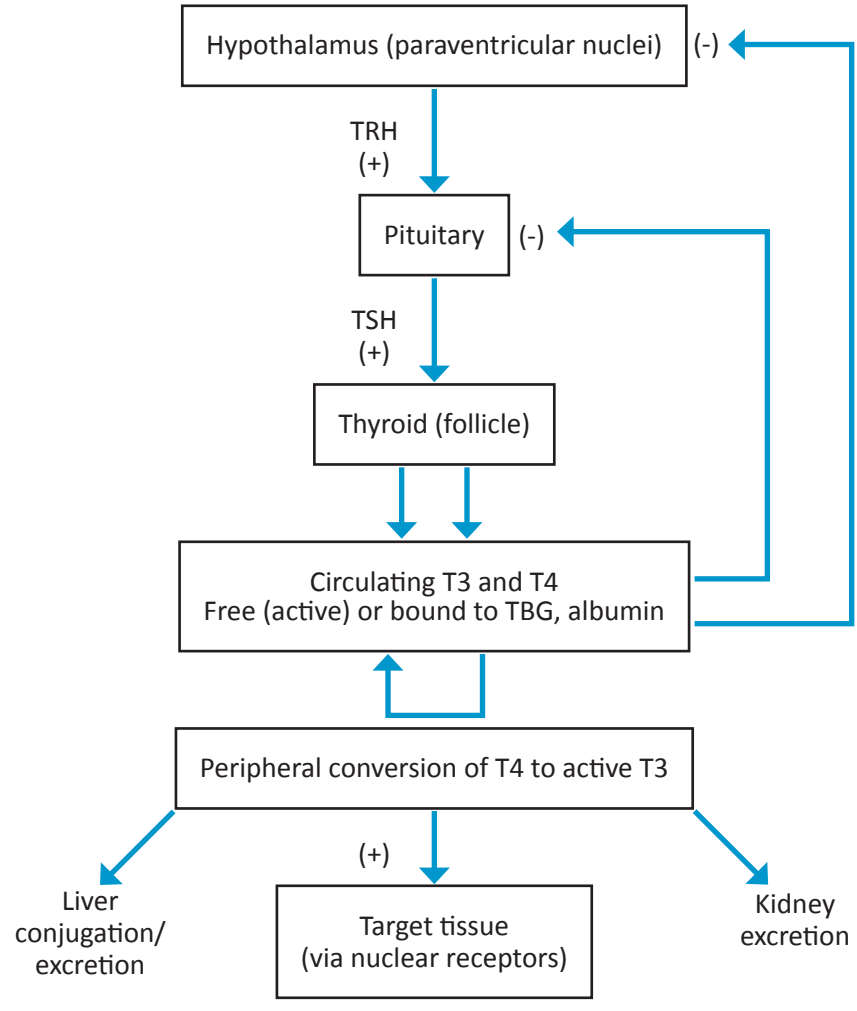

TABLE 1: Flowchart of thyroid hormone regulation[8]

FIGURE 1: Symptoms associated with Graves' disease[8]

any value under this range is considered hyperthyroidism.[5] It is also worth noting that there are two main types of hyperthyroidism: overt hyperthyroidism is defined as below normal TSH levels with higher than normal T3 and T4 levels, and subclinical hyperthyroidism is characterized as TSH levels below the standard limit with standard T3 and T4 levels.[7]

Symptoms associated with Graves' disease

Graves' disease can manifest differently in each patient, depending on the severity of the disease and when hyperthyroidism develops **(figure 1)**.[8] The primary physical signs of hyperthyroidism are tachycardia, hypertension, fine tremors, a palpable goiter (enlarged thyroid), hair loss, and muscle weakness.[9] The most common extrathyroidal symptom is exophthalmos or eye bulging. Less common are dermopathy and acropachy. Dermopathy is described as red, swollen skin on the feet and shins, while acropachy refers to clubbing or swelling of the fingers and toes.[9]

What causes Graves' disease?

Autoimmune responses target the thyroid more frequently than any other organ, but the exact cause of Graves' disease is still unknown.[10] It is believed to be a combination of genetic susceptibility, epigenetics, and environmental factors.[10] Studies have long recognized the critical role of genetics in the causation of thyroid autoimmune diseases, and several genes are associated with the development of Graves' disease.[1] Among these, thyroglobulin and CD40 are significant discoveries in advancing the understanding of Graves' disease. Thyroglobulin serves as a precursor to thyroid hormones T3 and T4.[1] Due to Graves' disease being characterized by the development of TSI antibodies, thyroglobulin has been recognized as a primary antigen and precise target gene in thyroid autoimmunity.[1] CD40 is critical to both innate and adaptive immunity. The antigen-presenting cells, including B cells, express this molecule, and it plays a role in activating B-lymphocytes so they can produce antibodies.[11] Among many other autoimmune diseases, whole-genome scanning has shown a strong link between CD40 and Graves' disease.[11]

Studies show that epigenetic changes contribute to disease etiology and that the effects are hereditary and not part of the genetic sequence.[1] These effects have now been extended to include long-lasting effects on gene expression. Gene expression can be modified epigenetically through changes in DNA methylation (detoxification), protein modification patterns, and microRNA competition.[1] Seventy percent of autoimmune thyroid disease is thought to be genetic. For those who have a genetic susceptibility, a multitude of factors may trigger the onset of Graves' disease.[12] The identified environmental risk factors include medications (interferon-alpha and amiodarone), viral infections (hepatitis C), stress, smoking, excess iodine or iodine deficiency, sex, age, preexisting autoimmune disorders, pregnancy, and pollutants.[1]

How is Graves' disease diagnosed?

When Graves' disease is suspected, a commonly used method for diagnosis is accurate for most cases.[3] In addition to the clinical signs and symptoms, blood is drawn from the patient to measure the concentration levels of thyroid hormones currently in circulation; this first test can rule out or show a positive hyperthyroidism diagnosis.[3] If hyperthyroidism is confirmed, a second analyzation is used to test for the presence of TSI. This test will confirm or deny the suspected Graves' disease diagnosis because these antibodies have a 99% sensitivity and specificity for Graves' disease.[3] If no amount of TSI is present, a final test will examine the thyroid on iodine uptake; if it absorbs an increased amount in both nodules, the patient has a positive diagnosis for Graves' disease.[3] This testing process is summarized in **table 2**.[3]

Treatment methods

Standard treatment options for patients with Graves' disease include antithyroid medication (ATD), radioactive iodine treatment (RAI), and partial or whole thyroidectomy.[13] According to a recent survey comparing regional treatment methods for Graves' disease, the use of antithyroid medications is the most common treatment for Graves' disease patients in the US, followed by RAI.[13] The survey also reviewed a study examining RAI outcomes to predict future results. The analysis was comprised of 576 Graves' disease patients, and after the first dose of RAI, 77% displayed hypothyroidism, and that percentage grew to 86.4% after the second dose.[14] Another option for treatment of Graves' aside from ATD and RAI is thyroidectomy, either total thyroidectomy, where the entire thyroid is removed, or subtotal thyroidectomy, where only a portion of the thyroid is removed.[13]

Nearly 100% of patients who undergo a total thyroidectomy do not have reoccurrence of hyperthyroidism. Unfortunately, this can result in hypothyroidism, which can be controlled by medications.[13] However, a study done between 2005–2011 to evaluate the long-term outcomes of subtotal thyroidectomies showed that of the 415 Graves' disease patients examined, hyperthyroidism recurred in 28.7% of the patients, 19.3% gained euthyroid (normal thyroid function), and hypothyroidism developed in more than 50% of patients.[15] Antithyroid drugs that are used to treat Graves' disease include methimazole and propylthiouracil (PTU).[16] In addition to ATDs, beta-blockers are commonly used to alleviate the hypertensive and cardiac effects of Graves' disease.[16] Methimazole in excessive doses is also known to cause hypothyroidism, though there is little information regarding the percentage of hypothyroidism due to antithyroid drug therapy.[17]

Beta-blockers: Metoprolol and propranolol are often prescribed to alleviate the side effects of Graves' disease, including anxiety, excessive sweating, tremors, palpitations, and tachycardia.[3] The disadvantages of this class of medication are that it does not influence the course of the disease and it should be used with caution in patients with asthma, congestive heart failure, Raynaud's syndrome, and bradyarrhythmia.[3] Calcium channel blockers should be considered as an alternative in patients with chronic obstructive pulmonary disease (COPD).[3]

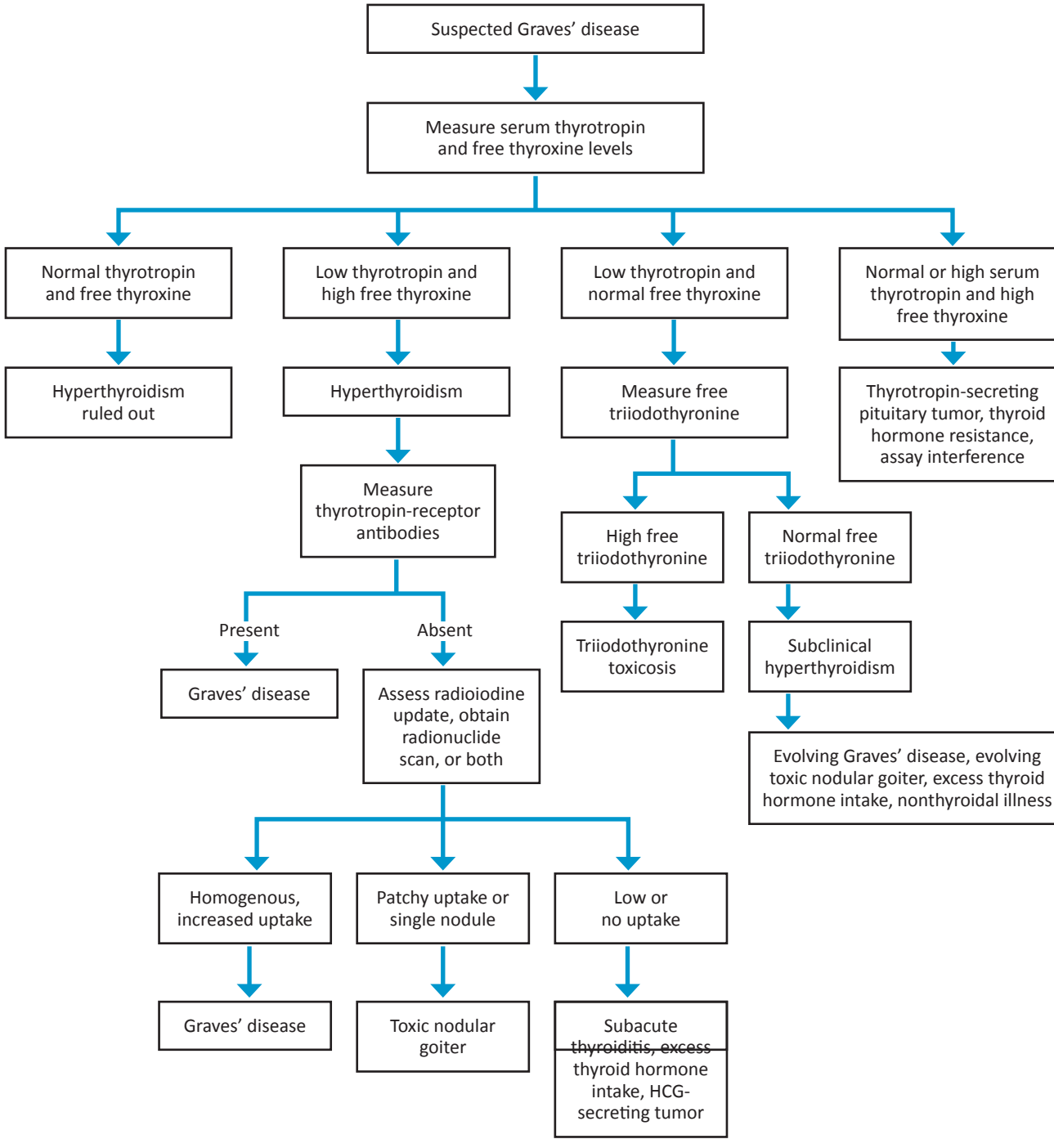

TABLE 2. Testing process for Graves' disease[3]

Antithyroid drugs: Methimazole and propylthiouracil block thyroid hormone synthesis.[3] Patients are given either a single dose to be taken daily or medication is used in conjunction with thyroxine to prevent hypothyroidism.[3] The advantages to antithyroid medication treatment are that there is a low chance of developing hypothyroidism, there is no radiation hazard, and there is no surgical risk.[3]

Radioactive iodine: The amount of iodine therapy given is dependent upon the size of the thyroid and the amount of uptake.[3] This therapy involves radiation that causes damage to thyroid cells to eliminate hyperthyroidism.[3] It is usually an outpatient procedure with a single dose and it effectively reduces the size of the thyroid.[3] It does have potential radiation hazards, and eventually, hypothyroidism develops in most patients.[3]

Thyroidectomy: A thyroidectomy is a surgical procedure in which most, if not all, of the thyroid gland tissue is removed.[3]

Recurrence of hyperthyroidism is infrequent, there is no radiation exposure, and patients experience rapid relief of their symptoms.[3] This is the most expensive form of therapy, and hypothyroidism is the goal.[3] It cannot be overstated that smoking cessation is crucial.[3] The efficacy of medical therapy is decreased in smokers.[3]

Graves' disease manifestations within the oral cavity

Oral manifestations of Graves' disease include increased susceptibility to caries and periodontal diseases, maxillary and mandibular osteoporosis, accelerated eruption of teeth, enlargement of extraglandular thyroid tissue (lingual thyroid), burning mouth syndrome, and Sjögren's syndrome, which is a chronic immune disorder that causes dry mouth.[18]

The increased risk of periodontal diseases can be attributed to interleukins, specifically interleukin 6, involved in the inflammatory response present in patients with Graves' disease.[19] "Inflamed periodontal tissues also demonstrate higher levels of IL-6. IL-6 has been shown to stimulate osteoclasts and thus contribute to the alveolar bone loss seen in periodontitis."[20] The results of a 2018 study show that there is a positive correlation between Graves' disease and periodontitis due to the shared increase of interleukin-6 present in both disorders.[21] The study consisted of 120 participants, 30 of which were healthy, 30 with Graves' disease alone, 30 with periodontitis alone, and the remaining 30 with Graves' disease and periodontitis together.[21]

The purpose of the study was to identify the correlation between Graves' disease and periodontitis by examining the levels of IL-6 and TNF-α in both disorders. The conclusion states, "The level of IL-6 and TNF-α in GD [Graves' disease] plus CP [periodontitis] group were significantly higher than those in CP group or GD group, indicating that there is an interaction between periodontitis and Graves' disease in immune mechanism."[21] Further evidence to support the increased prevalence of periodontal diseases present in patients with Graves' disease can be attributed to an imbalance of reactive oxygen species (ROS), also called free radicals, and antioxidants circulating within the body.[22] The imbalance of ROS and antioxidants is referred to as oxidative stress.[22] In periodontal diseases, the number of antioxidants within the periodontal tissue is inadequate compared to the amount of ROS, meaning there is increased oxidative stress, which increases periodontal destruction.[20] Periodontal destruction can be prevented with high enough levels of antioxidants to fight off the free radicals or ROS; however, in patients with Graves' disease, there is increased oxidative stress, so in turn, Graves' disease patients have a higher incidence of periodontal destruction.[23]

Patients with Graves' disease have an increased risk of developing caries.[24] However, it is unclear whether the risk for dental caries is based on the predisposition of the disease itself or side effects from commonly prescribed medications for the disease.[24] It is known that Graves' disease places a patient at a higher risk for developing Sjögren's syndrome, which can cause xerostomia, and in turn, makes the oral cavity more prone to dental caries and periodontitis.[24]

Oral manifestations of various Graves' disease treatments

Understanding the process of radioactive iodine (RAI) ablation and thyroidectomies is important for dental clinicians because these treatments frequently result in Graves' disease patients developing hypothyroidism. If a patient develops hypothyroidism, thyroid replacement drugs are usually necessary for the patient's lifetime.[13] Levothyroxine is a commonly prescribed thyroid replacement medication. It has potential side effects such as difficulty swallowing, irregular heartbeat, irregular breathing, tremors, irritability, and swelling of the eyes, face, lips, throat, and tongue.[25] Patients with hypothyroidism may also have inadequate wound healing.[18] Aside from causing hypothyroidism, other side effects associated with RAI can include salivary gland dysfunction and xerostomia.[26] A 2013 study examining the salivary gland function post-RAI in 213 patients concluded that 20% of the salivary glands tested were dysfunctional, and 16.4% of patients reported xerostomia five years following RAI therapy.[26]

As stated, beta-blocker medications such as propranolol are commonly prescribed to patients with Graves' disease to alleviate hypertensive and cardiac side effects.[16] However, beta-blockers have been directly linked to xerostomia, which can increase a person's rate of tooth decay.[27]

Thioamides are commonly prescribed to patients with hyperthyroidism; typical examples include methimazole and PTU.[16] Thioamides can pose a risk for inadequate wound healing and increased susceptibility to oral infections in rare cases due to a severe agranulocytosis reaction in 0.5% of patients taking methimazole.[18] Also, PTU is known to cause hypoprothrombinemia (a deficiency of the blood-clotting substance prothrombin) and may place a patient at risk for hemorrhage. Therefore, patients who take PTU need to be thoroughly examined before invasive dental procedures.[18] Other possible drug interactions that clinicians should be aware of include NSAIDs and any analgesic containing acetylsalicylic acid (ASA).[18] ASA is contraindicated in patients with hyperthyroidism because it interferes with the binding effects of T3 and T4. This can result in more free-floating T3 and T4, which can increase symptoms of thyrotoxicosis.[18]

Dental interventions

Patients with Graves' disease can present challenges to dental professionals, and it is essential to make appointment modifications to avoid possible complications. The use of thyroid collars during radiograph exposure prevents aggravation of Graves' disease and helps avoid a thyroid disorder.[28] The effects of thyroid hormones on the sympathetic nervous system can cause an increase in blood pressure and heart rate in patients with Graves' disease hyperthyroidism.[28] This can cause a delay in dental treatment by allowing time for the patient's blood pressure to lower, a longer duration of applying localized pressure to stop bleeding, and more frequent rinsing to stop bleeding.[28] If the patient presents as hypertensive, they should be referred to their primary care physician for evaluation and treatment.[28] Additionally,

dental professionals are responsible for being aware of drug side effects and being familiar with the symptoms and signs of hormone toxicity.[28]

Head, neck, and oral examinations: During the intraoral and extraoral examinations, it is essential to keep in mind the signs and symptoms of Graves' disease because the patient may be undiagnosed. Under normal function, the thyroid is undetectable when palpated, and the patient will most likely not experience any tenderness.[29] Notable extraoral findings include a palpable gland, asymmetrical lobes, enlarged or goitered lobes, and the presence of nodules.[29] An additional extraoral concern is eye bulging, as this could be exophthalmos. Intraorally, one of the most common side effects of Graves' disease is xerostomia or dry mouth.[29] This condition can be manifested by antithyroid prescription medications or Sjögren's syndrome, an autoimmune disease that affects the lacrimal and salivary glands, causing dry eyes and dry mouth.[29]

According to caries management by risk assessment (CAMBRA), patients with dry mouth are automatically placed at extreme risk for developing dental caries.[30] With saliva being the primary mode of keeping the oral cavity clean, having a minimal flow rate allows more time for bacteria to harbor and cause tooth decay and demineralization. Because of this, dental caries and periodontal disease appear to develop and progress more rapidly in patients with Graves' disease.[28] Burning mouth syndrome is another intraoral manifestation; this condition is described as a painful burning sensation in the mouth.[28] Burning mouth and Sjögren's syndrome are more common in people with thyroid disorders.[28] Head and neck and oral examinations are crucial for every dental visit as these assessments can save a patient's life.[20]

Gingival and periodontal assessments: Drug-induced gingival enlargement is a predisposing factor for periodontal disease and rarely occurs in patients who take beta-blockers.[31] Nonetheless, 75% of patients prescribed calcium channel blockers experienced an overgrowth of gingiva and higher rates of plaque and calculus.[31] Bone loss is amplified in patients with hyperthyroidism due to a long-term excess of thyroid hormone.[2] Because of this, osteoporosis may occur, affecting the rate of alveolar bone loss; this is important to keep in mind when reviewing patient radiographs.[2] A rare but life-threatening side effect of patients taking thioamides, such as methimazole, is agranulocytosis.[2] This disorder is described as a significant loss of circulating white blood cells, which causes increased susceptibility to infections and poor wound healing.[2] Orally, agranulocytosis resembles necrotizing ulcerative gingivitis, and manifests as excessive bleeding from the gingiva and rapid destruction of the supporting tissues of the teeth.[2] These patients should be given an immediate referral for treatment and testing before performing dental treatment.[2]

Nutritional counseling: Guidelines for maintaining a healthy diet can be found through the MyPlate initiative, previously known as MyPyramid.[32] The recommendations are designed to control obesity, cancer, diabetes, cardiovascular disease, hypertension, and osteoporosis.[32] In patients with Graves' disease, osteoporosis is the risk factor of most concern that can be somewhat prevented with proper nutrient intake.[33] Magnesium and calcium are important minerals for bone health. Good sources include green leafy vegetables, and nuts such as Brazil nuts and almonds.[33] Dairy is an additional source of calcium; however, many experts do not suggest dairy consumption. Many ethnic groups have low rates of osteoporosis while also having a low dairy consumption.[33] Vitamin D is also beneficial for bone health, and the best source is sunlight exposure. Patients should speak with their doctor when contemplating dietary supplements.[34]

Foods to potentially limit or avoid for patients with Graves' disease are iodine and gluten products.[35] Iodine is required for proper thyroid function, but excess intake can contribute to hyperthyroidism.[35] A food source high in iodine is kelp, and two cases have shown women presenting with hyperthyroidism and multinodular goiter following a high-kelp diet.[35] Research shows that there is a link between thyroid-harming antibodies and gluten intolerance.[36] Studies have shown that a patient with Graves' disease could have up to 4.5 times greater risk of developing celiac disease than that of a healthy individual.[36] Two case studies have been documented of women who were diagnosed with celiac disease after not responding to their Graves' disease medications, and they responded to medications after starting a gluten-free diet.[37] Education on a gluten-free lifestyle or a referral to a reputable dietician would benefit Graves' patients.[37]

Short recare intervals: Patients who have either a visibly reduced salivary flow or who take medications known to cause xerostomia are automatically placed at moderate risk for caries development. It is recommended these patients receive a dental exam every four to six months for reevaluation of their caries risk.[38] As stated, beta-blockers, commonly prescribed to patients with Graves' disease, are known to cause xerostomia; therefore, patients with Graves' disease on chronic beta-blocker therapy have a moderate risk of developing caries.[38] This moderate risk does not consider the patient's dental examination; CAMBRA defines high risk for developing caries as carious lesions present on the patient's radiograph, white spot lesions, or restorations within the last three years.[38] In other words, patients with Graves' disease will most likely have at least a moderate risk for caries due to medications. Still, they could potentially have a high or even extreme risk for caries dependent upon their dental examination.[38]

There is weak evidence supporting the effectiveness of preventing periodontal disease by using shorter recall intervals for patients without periodontitis; however, the positive correlation between Graves' disease, periodontitis, and dental caries can be beneficial when determining a patient's recall interval.[38]

Stress reduction protocols: For dental patients with Graves' disease, it is essential to note that hyperthyroidism causes increased anxiety, which can potentially trigger a thyroid storm. A thyroid storm, also called thyrotoxic crisis, is an acute state that presents as an

exaggerated appearance of hyperthyroidism. Symptoms of a thyroid storm include tachycardia, heart failure, arrhythmia, fever, gastrointestinal symptoms, and central nervous system manifestations such as agitation, delirium, psychosis, or coma.[6] Graves' disease that goes undiagnosed can predispose a patient to a thyroid storm, which has a high mortality and morbidity rate.[6]

Stress reduction protocols may be helpful for patients undergoing surgery or those who appear stressed or anxious during routine dental treatment.[18] Stress reduction protocols for patients with Graves' disease can include scheduling their dental procedures early in the day due to the increased levels of cortisol in the morning, using measures to reduce pain and anxiety such as local anesthetics (with or without epinephrine depending on blood pressure levels), giving nitrous oxide or antianxiety medications, and frequently monitoring blood pressure throughout the appointment.[38]

Interprofessional collaboration: Bidirectional communication between the patient's endocrinologist, dentist, and dental hygienist allows for optimal treatment for patients with Graves' disease.[18] The dentist should be updated on the patient's antithyroid medication and any adverse effects of the drugs.[18] It is also beneficial for the dental team to know about alternative Graves' disease treatments such as RAI and thyroidectomies due to the potential adverse reactions the treatments may display. Aside from the dental team, it is beneficial for the endocrinologist to have a baseline knowledge of oral manifestations of the disease and therapies to assist a patient's oral and overall health.[18]

Conclusion

Research shows that there is indeed a positive correlation between Graves' disease and periodontitis due to the shared immune mechanisms and increased oxidative stress present in both disorders. With the various oral disease risk factors considered, modifications during the dental appointment may be indicated when treating a patient with Graves' disease. Due to this correlation, patients with Graves' disease could benefit from a shorter preventive dental treatment recall interval. When determining a recall interval for patients with Graves' disease, dental professionals should keep in mind the link between periodontitis and Graves' disease as well as the moderate caries risk due to commonly prescribed medication.

However, upon further research regarding short intervals to prevent periodontal diseases, there is an overall lack of evidence to support the benefits of patients who do not have periodontitis receiving routine treatment in increments such as three and four months. Dentists and dental hygienists should be familiar with potential alternative therapies for Graves' disease to monitor oral or systemic health changes. Endocrinologists should be aware of oral manifestations of Graves' disease. Dental and medical professionals working together and collaborating with the patient allows for optimal health care for the patient.

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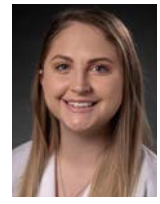

Jayci Jones, RDH, is a graduate of Collin College. She is passionate about the dental management of Graves' disease because she has lived with the symptoms of the disease for two years. Jayci is motivated to research the oral effects of autoimmune diseases for the benefit of her clinical patients and herself. She was the Hu-Friedy Golden Scaler recipient of her class and hopes to work in periodontal and community clinics to educate patients on the impact of oral-systemic health.

Lindsey Cunningham, RDH, is an honors graduate from the Collin College dental hygiene program in the Dallas, Texas, area. She is passionate about community dentistry, periodontics, and the oral-systemic link. After being diagnosed with Graves' disease in 2018, Lindsey's passion for learning more about the etiology of the disease grew exponentially. Her goal is to spread awareness to hygienists to help other patients who are living with Graves' disease.

Kandice Swarthout, RDH, LPC, is a full-time dental hygiene educator in Texas. She is the owner of Inspired Education & Wellness, where she is a speaker, writer, and private practice therapist. She combines her clinical dental and mental health experience to help other health-care professionals have a fulfilling work-life experience. Kandice is an approved provider for human trafficking training for health-care professionals. Contact Kandice at kandiceswarthout.com or on social media @ thecounselorhygienist.

Notes

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QUESTIONS

1. Graves's disease is:
   - A. An autoimmune disorder that understimulates the thyroid
   - B. An autoimmune disorder that overstimulates the thyroid
   - C. A disease that shrinks the thyroid
   - D. None of the above

2. The thyroid gland is enlarged when antibodies called ___ that attach to its receptors and stimulate too much thyroid hormone.
   - A. Estrogen
   - B. T3s
   - C. T4s
   - D. TSIs

3. Dental management of patients with Graves' disease includes all of the following except:
   - A. Nutritional counseling
   - B. Interprofessional collaboration
   - C. Skipping periodontal assessments
   - D. Stress reduction protocols

4. The usual range for T3 in adults is:
   - A. 5.0–11.0 ug/dL
   - B. 11.0–13.0 ug/dL
   - C. 1.0–5.0 ug/dL
   - D. .5–1.5 ug/dL

5. TSH is an important hormone in Graves' disease and is excreted by the:
   - A. Thyroid
   - B. Hypothalamus
   - C. Pituitary
   - D. Kidneys

6. TSH levels are abnormally ___ in patients with hyperthyroidism.
   - A. High
   - B. Low
   - C. Wavering
   - D. Unchanged

7. All of the following are symptoms of Graves' disease except:
   - A. Fine tremors
   - B. Enlarged goiter
   - C. Hair loss
   - D. Double vision

8. Autoimmune responses tend to target which gland more than any other?
   - A. Thyroid
   - B. Pituitary
   - C. Salivary
   - D. Pancreas

9. All of these are potential causes of Graves' disease except:
   - A. Genetics
   - B. Epigenetics
   - C. Ethnicity
   - D. Environmental factors

10. There is a ___ risk of developing Graves' disease due to genetic background.
    - A. 50%
    - B. 70%
    - C. 90%
    - D. 10%

11. Which of the following is the most common treatment method for Graves' disease in the United States?
    - A. Thyroidectomy
    - B. Antithyroid medications
    - C. Radioactive iodine therapy
    - D. Synthetic thyroid hormone

12. All of the following Graves' disease treatment methods may result in hypothyroidism except:
    - A. Beta-blocker medications
    - B. Antithyroid medication
    - C. Radioactive iodine therapy
    - D. Total thyroidectomy

13. There is approximately a ___ recurrence rate of hyperthyroidism after a total thyroidectomy.
    - A. 0%
    - B. 50%
    - C. 7%
    - D. 15%

14. Of the 576 Graves' disease patients examined, what percentage displayed hypothyroidism after the first dose of radioactive iodine therapy?
    - A. 86.4%
    - B. 77%
    - C. 56%
    - D. 100%

15. All of the following are commonly prescribed medications for Graves' disease patients except:
    - A. Methimazole
    - B. Propranolol
    - C. Propylthiouracil
    - D. Lisinopril

16. Beta-blocker medications can be used to alleviate which of the following symptoms?
    - A. Dermopathy
    - B. Exophthalmos
    - C. Goiter
    - D. Hypertension

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QUESTIONS

17. What method of diagnosis is used to test for Graves' disease?
    A. Urine sample
    B. Biopsy
    C. Blood sample
    D. Ultrasound

18. The efficacy of medical therapy for Graves' disease is significantly lessened if the patient:
    A. Smokes tobacco
    B. Does not exercise
    C. Drinks alcohol
    D. Eats an unhealthy diet

19. According to a study done between 2005–2011, what percentage of patients gained euthyroid, or normal thyroid function, following a subtotal thyroidectomy?
    A. 50%
    B. 28.7%
    C. 6%
    D. 19.3%

20. Advantages of antithyroid medications include all of the following except:
    A. No surgical risk
    B. No radiation risk
    C. Low chance of developing hypothyroidism
    D. Increased wound healing

21. Oral manifestations of Graves' disease include all of the following except:
    A. Increased susceptibility to caries
    B. Delayed tooth eruption
    C. Burning mouth syndrome
    D. Maxillary and mandibular osteoporosis

22. Graves' disease places a patient at a higher risk for developing ___, which can cause xerostomia.
    A. Diabetes mellitus
    B. Fluorosis
    C. Aphthous ulcers
    D. Sjögren's syndrome

23. If a patient develops hypothyroidism following Graves' disease treatment, thyroid replacement drugs are usually necessary for:
    A. The patient's lifetime
    B. 3 to 6 months
    C. 2 years
    D. 24 months

24. Which is contraindicated in patients with hyperthyroidism because it interferes with the binding effects of T3 and T4?
    A. Methimazole
    B. Ibuprofen
    C. Acetylsalicylic acid
    D. Acetaminophen

25. Under normal function, the thyroid is ___ when palpated, and the patient will most likely not experience any tenderness.
    A. Firm
    B. Pea sized
    C. Mobile
    D. Undetectable

26. What percent of patients prescribed calcium channel blockers experienced an overgrowth of gingiva and higher rates of plaque and calculus?
    A. 50%
    B. 75%
    C. 45%
    D. 90%

27. Which of the following is a rare but life-threatening side effect of thioamides?
    A. Osteoporosis
    B. Necrotizing ulcerative gingivitis
    C. Agranulocytosis
    D. Hypoprothrombinemia

28. Potential foods for patients with Graves' disease to limit or avoid are ___ and ___ products.
    A. Iodine and gluten
    B. Gluten and nut
    C. Soy and iodine
    D. Salted and soy

29. Patients with Graves' disease will most likely have at least what risk for caries due to medications?
    A. Low
    B. Moderate
    C. High
    D. Extreme

30. Research shows that there is ___ correlation between Graves' disease and periodontitis.
    A. No
    B. A negative
    C. Binary
    D. A positive

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Dental management of patients with Graves' disease

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1. Describe the definition, etiology, symptoms, and treatment for Graves' disease
2. Identify the clinical oral manifestations of patients with Graves' disease
3. Define systemic, oral, and mental health implications for patients with Graves' disease
4. Define recommended treatment and recare intervals for patients with Graves' disease.

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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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