

BMSH4302 | Week 4: Diabetes and Metabolic Disease

Resource Sheet B

Hyperthyroidism and Graves' Disease

Your task: Read this sheet carefully. You will first discuss it with a partner who has the same sheet, then teach it to someone who has NOT seen it. Focus on what the condition is, what causes it, what the signs and symptoms are, and how it affects quality of life.

What is hyperthyroidism?

Hyperthyroidism means the thyroid gland is overactive. It produces too much thyroid hormone (T3 and T4). These hormones control the body's metabolic rate, so when levels are too high, everything speeds up. Heart rate, digestion, temperature regulation, and energy consumption all accelerate beyond normal. Think of it as the body's thermostat being turned too high.

What is Graves' disease?

Graves' disease is the most common cause of hyperthyroidism in the UK, accounting for 60 to 80% of cases. It is an autoimmune disease. The immune system produces antibodies called thyroid-stimulating immunoglobulins (TSI) that bind to the TSH receptor on the thyroid gland and continuously stimulate it to overproduce hormone. The thyroid essentially cannot switch off.

Compare this with Hashimoto's thyroiditis (the condition studied by the other group): in Hashimoto's, antibodies destroy the thyroid, leading to underactivity. In Graves', antibodies stimulate the thyroid, leading to overactivity. Both are autoimmune. The immune system attacks the thyroid in both cases, but the outcome is the opposite. Same mechanism, mirror-image results.

Prevalence and incidence

Hyperthyroidism (all causes) affects around 2% of women and 0.2% of men in the UK, with Graves' disease accounting for the majority of cases. Peak onset is between the ages of 30 and 60, although it can occur at any age, including in children. Women are 5 to 10 times more likely to be affected than men. The lifetime risk is approximately 3% for women and 0.5% for men. The worldwide incidence of paediatric Graves' disease appears to be rising.

Aetiology

In Graves' disease, B lymphocytes produce thyroid-stimulating immunoglobulins that continuously activate the TSH receptor. The trigger for this antibody production is not fully understood, but there is a strong familial component. Graves' disease is associated with other autoimmune conditions including Type 1 diabetes, rheumatoid arthritis, and vitiligo. Environmental triggers include stress (frequently reported before onset), smoking (which increases the risk and significantly worsens eye disease), pregnancy and the post-partum period, infection, and excess iodine intake. Other, less common causes of hyperthyroidism include toxic thyroid nodules and thyroiditis.

Pathology

Thyroid-stimulating immunoglobulins mimic TSH and bind to TSH receptors on thyroid cells, stimulating them continuously. The thyroid gland enlarges (forming a diffuse goitre) and overproduces T3 and T4. The pituitary gland responds by suppressing TSH production through negative feedback. Low TSH with high T3 and T4 is the key diagnostic pattern.

With excess thyroid hormone, metabolic rate accelerates across all body systems. Every cell is being told to work faster than normal. Importantly, the same antibodies can also attack tissues behind the eyes, causing Graves' ophthalmopathy (eye disease), and the skin on the shins, causing pretibial myxoedema. These are features unique to Graves' disease, not seen in other causes of hyperthyroidism.

Signs and symptoms

Because metabolism accelerates, symptoms reflect everything running too fast. Patients typically experience restlessness, anxiety, irritability, insomnia, and emotional instability. Weight loss occurs despite increased appetite because the body is burning calories faster than they can be consumed. Heat intolerance is common, with excessive sweating.

Cardiovascular effects are significant: heart rate increases (tachycardia), palpitations are common, blood pressure rises, and there is an increased risk of atrial fibrillation. If untreated, this can progress to heart failure. The skin is warm and moist, hair becomes thin and fine, and the palms may appear red. A fine resting tremor of the hands is characteristic.

Graves' ophthalmopathy affects more than one in three patients. The eyes appear to bulge (exophthalmos), feel gritty and sore, and patients may experience double vision and light sensitivity. In rare cases it can threaten vision. A visible goitre (enlarged thyroid) may be apparent at the front of the neck. Diarrhoea or frequent bowel movements occur because gut motility speeds up. Muscle weakness (particularly in the thighs and upper arms) and bone loss (osteoporosis) can develop.

The symptoms of hyperthyroidism closely mimic anxiety disorders. Palpitations, restlessness, tremor, insomnia, and irritability are features of both conditions. As a result, many patients, particularly women, are initially diagnosed with anxiety or panic disorder before thyroid function is tested.

Risk factors

Non-modifiable risk factors include female sex, age (peak onset 30 to 60 years), family history of thyroid or autoimmune disease, and the presence of other autoimmune conditions. Among modifiable risk factors, smoking is particularly significant. It increases the risk of developing Graves' disease and substantially worsens Graves' eye disease. Patients who smoke are more likely to develop ophthalmopathy and respond less well to treatment. Stress and excess iodine intake are also implicated.

Quality of life

Untreated hyperthyroidism poses serious health risks including heart failure, atrial fibrillation, stroke, and osteoporosis. Even when the condition is managed, many patients report ongoing fatigue and difficulty returning to their previous level of functioning. Anxiety, irritability, and emotional instability can damage relationships and work performance. Graves' ophthalmopathy causes significant psychological distress because of the visible changes to facial appearance.

There are three main treatment options. Anti-thyroid drugs (carbimazole or propylthiouracil) are taken for 12 to 18 months, but approximately 50% of patients relapse after stopping. Radioactive iodine destroys thyroid tissue and is effective, but most patients then become permanently hypothyroid and require lifelong levothyroxine. Surgery (thyroidectomy) is definitive but also results in lifelong hormone replacement. There is an irony to treating overactive thyroid disease: the most effective treatments often create an underactive thyroid instead.

Note: A more detailed version of this resource sheet is available on the Week 4 Moodle page for revision and exam preparation.