


Jod-Basedow phenomenon: Reactivation of thyroid eye disease after contrast computed tomography



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To report a case of Jod-Basedow phenomenon, where a patient with quiescent thyroid eye disease became active after a contrast computed tomography (CT). A patient with clinically inactive thyroid eye disease underwent a contrast CT, after which her clinical activity score increased for 1 to 6/10 with optic nerve dysfunction. She was managed medically with intravenous steroids, neomercazole and propranolol. Jod-Basedow phenomenon is a rare condition where thyroid eye disease reactivates after a large iodine bolus. It is important for clinicians to be aware of this when considering special investigations, such as contrast scans, where iodine is given in large doses.

Introduction

Thyroid eye disease (TED) is an autoimmune disease that represents the most common extrathyroidal manifestation of Graves' disease (GD).^{1,2} The annual incidence of GD is 16 out of 100 000 females and 3 out of 100 000 males, with visually significant GD in 3% – 5% of cases.¹ Jod-Basedow phenomenon is a rare cause of hyperthyroidism that can develop after an iodine bolus in a susceptible patient.^{3,4,5,6}

Iodine is a micronutrient that is required by the thyroid gland for synthesis of thyroid hormones and is found predominantly in coastal areas. The recommended daily dose of iodine ranges from 150 μg to 1100 μg for adults where approximately 70 μg – 80 μg is actively taken up by the thyroid for the synthesis of tetraiodothyronine (T4) and triiodothyronine (T3).^{7,8} Thyroid hormones undergo an iodine-dependent synthesis and are then released from the thyroid gland to have a systemic effect. The production and release of thyroid hormones are predominantly regulated by the thyroid-stimulating hormone (TSH) in the hypothalamic–pituitary axis. A negative feedback system by the circulating T3 and T4 decreases the release of TSH and thereby regulates the production of further thyroid hormones. Abnormal thyroid tissue may produce hormones independent of TSH or be stimulated by autoantibodies to produce excess thyroid hormones in a suppressed TSH environment.⁸

Sources of extra iodine may be iatrogenic and include vitamins, amiodarone, iodinated contrast medium and topical iodine. The average amount of free iodine in a single dose of iodinated contrast medium for computed tomography (CT) is 13 500 μg , making it more than ten times the recommended daily dose in a single bolus.^{4,7} The physiological response to excess iodine is the Wolff-Chaikoff effect, where there is a transient decrease in hormone production; however, some individuals develop hyperthyroidism, which is known as the Jod-Basedow phenomenon.^{3,7} This phenomenon is based on the premise that there are autonomously functioning areas of the thyroid gland resulting in an excess in hormone production when there is an iodine overload in the body.⁹ Amiodarone is one of the most common causes of this phenomenon, but there are a number of papers reporting this phenomenon after intravenous iodinated contrast administration.^{9,10} The average duration from intravenous contrast medium administration and onset of hyperthyroidism is 3–10 weeks. As this is a single bolus of iodine, it is generally considered to be a self-limiting disease.⁹ If a patient is considered to be at risk of this phenomenon, they can receive prophylactic methimazole or perchlorate.^{5,8}

Patients with Jod-Basedow phenomenon will typically present with systemic features of hyperthyroidism including palpitations, heat intolerance and tremors. Ocular signs include proptosis, lid swelling, optic nerve compression and diplopia.^{1,10} Management involves control of the systemic hyperthyroid crisis and management of ocular complications. Systemic management focuses on controlling the tachycardia (propranolol) and decreasing thyroid function with anti-thyroid agents (methimazole, carbimazole and propylthiouracil), while

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thyroid associated ophthalmopathy can be controlled with corticosteroids, radiotherapy and possible decompression surgery.¹

Our case report of a Jod-Basedow phenomenon after a contrast CT scan is the first in South African literature and highlights the danger of an iodine bolus in patients with TED.

Case report

A 33-year-old African female known with GD was jointly managed by the ophthalmology and endocrine departments at Charlotte Maxeke Johannesburg Academic Hospital (CMJAH), Johannesburg, South Africa. GD was diagnosed in August 2013 when she presented with a history of diaphoresis, tachycardia and significant unintentional weight loss. Thyroid scintigraphy revealed homogenously increased uptake of the tracer, in keeping with hyperthyroidism. Following medical management, she underwent radioactive thyroid ablation in December 2013. Subsequent hypothyroidism necessitated daily thyroid hormone replacement with eltroxin 100 µg.

Her condition remained inactive (clinical activity score 1/7)² and she was seen at ophthalmology in March 2015 for management of cosmetically unacceptable proptosis. Her visual acuity was 6/6 bilaterally. Other than proptosis, her ocular examination was normal. An elective orbital decompression was planned and she underwent a preoperative contrast CT of the orbits in May 2015 (Figure 1).

Two weeks after her CT, she presented with ocular pain and worsening proptosis. On examination, her visual acuity was 6/24 and 6/18 in the right and left, respectively, and her clinical activity score (CAS) had increased to 6/10. She had optic nerve dysfunction as evidenced by decreased perception of colour (7/10) and brightness (7/10) in the right and colour (8/10) and brightness (7/10) in the left. Her thyroid functions went from a hypothyroid to borderline hyperthyroid state after the CT (Table 1). She was admitted for intravenous steroid therapy (250 mg methyl-prednisolone 6-hourly) for three days, followed by oral corticosteroids. She was also recommenced on propranolol and high-dose neomercazole by endocrinology. The oral steroids were weaned over the course of a month and she remained clinically inactive. Her visual acuity improved to 6/6 and 6/7.5 in the right and left, and her optic nerve dysfunction resolved. She subsequently underwent a bilateral orbital decompression that improved the proptosis.

Discussion

Jod-Basedow phenomenon is a rare iodine-induced hyperthyroidism. Our case demonstrates this in a patient with TED who received a large iodine bolus after a contrast CT. She developed an increase in her CAS and her compressive optic neuropathy was resolved by medical therapy.

Our patient was known with GD with clinically inactive ocular disease for the preceding 17 months. After radioactive iodine ablation, she had hypothyroidism that was managed medically with oral thyroxine.¹¹ This can be seen by her

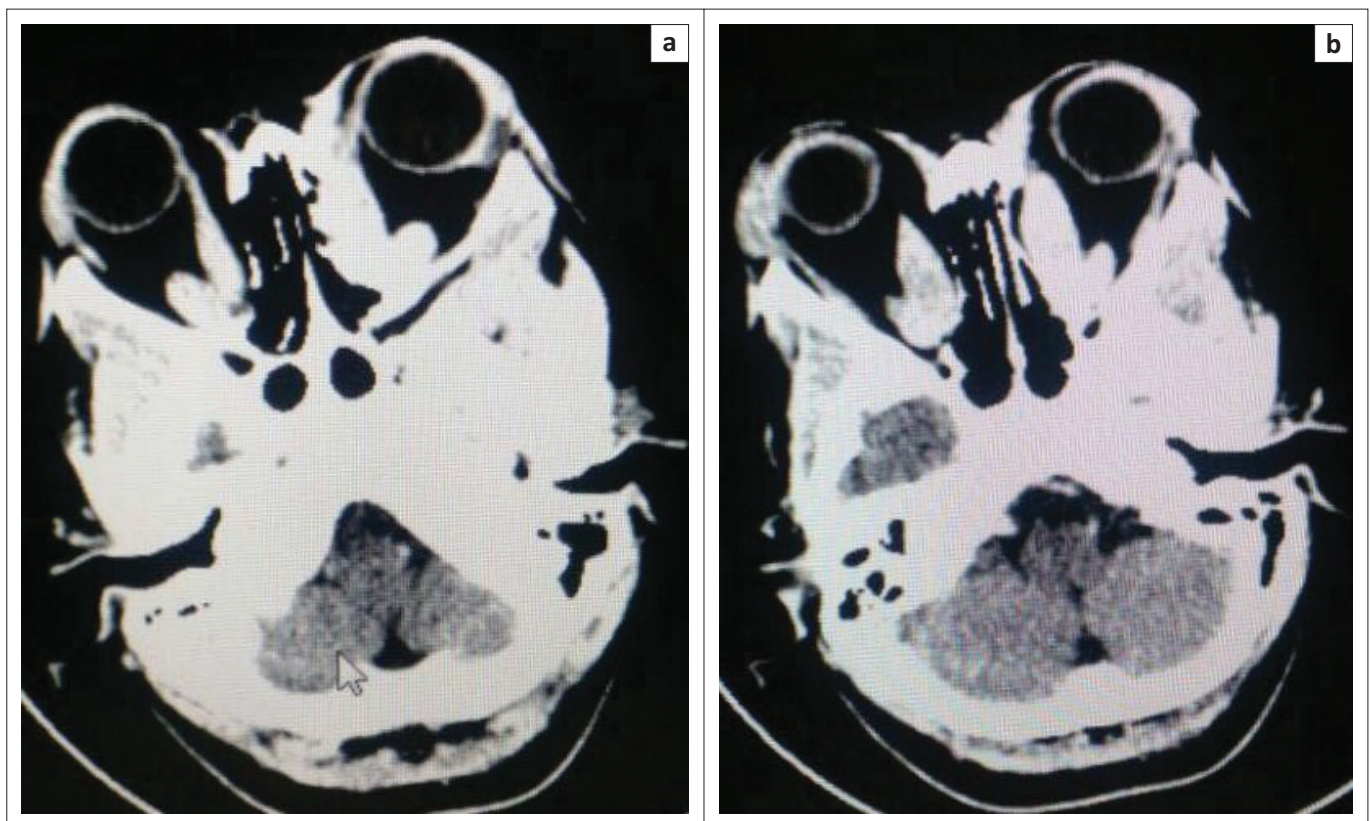


FIGURE 1: Computed tomography showing bulky extraocular muscles with sparing of the muscle insertions.

TABLE 1: Serial thyroid function tests that show the hypothyroid state before the computed tomography scan and the hyperthyroidism that developed after the computed tomography scan.

Date	TSH	T4
10 March 2015	149.3 [†]	4.6
18 May 2015	18.6	16.7
21 May 2015	7.0	16.4
07 July 2015	0.3	-
28 July 2015	0.5	20.3

TSH, thyroid-stimulating hormone; T4, free thyroxine.

TSH, Normal: 0.35 mIU/L–5.50 mIU/L; T4, Normal: 11.5 pmol/L–22.7 pmol/L.

[†], Computed tomography scan performed.

elevated TSH levels before that CT scan (Table 1). She had a cosmetically unacceptable proptosis and was being worked up for orbital decompression with a contrast CT scan.

The average dose of iodine in a single contrast scan is 13 500 µg, which is more than ten times higher than the recommended daily dose.^{4,7} This induced a hyperthyroid state with orbital congestion and compressive optic neuropathy. Her thyroid functions increased to a hyperthyroid state as reflected by her TSH decreasing from very high levels to the lower level of normal (Table 1). She was successfully managed medically with propranolol for the tachycardia and carbimazole for the hyperthyroidism. The optic nerve compression necessitated corticosteroid use to resolve the orbital congestion and inflammation. The patient responded well to this medication with improvement of her thyroid functions (Table 1) and resolution of her optic nerve dysfunction. She underwent subsequent orbital decompression that improved her proptosis.

Conclusion

Jod-Basedow phenomenon is a rare condition where TED reactivates after a large iodine dose. It is important for clinicians to be aware of this when considering special investigations, such as contrast scans, where iodine is given in large doses.

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Competing interests

The authors declare that they have no financial or personal relationships that may have inappropriately influenced them in writing this article.

Authors' contributions

F.I. contributed to the case report and images and R.H. contributed to the writing of the introduction and discussions in the article.

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