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### Title

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## Acute Thyrotoxicosis in the Setting of Graves' Disease Following Cardiac Catheterization

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### Introduction

Use of contrast agents are necessary for diagnostic imaging, interventional radiology, and cardiac catheterization procedures. However, several of these contrast agents, such as Omnipaque, contain significant amounts of iodine that may contribute to temporary thyroid dysfunction. A high iodine load provided to a patient with preexisting multinodular toxic goiter or Graves' disease may trigger thyrotoxicosis, a hypermetabolic state often accompanied by severe autonomic dysfunction due to excess free thyroxine or free triiodothyronine. Individuals receiving iodinated contrast media may develop excess circulating thyroid hormones by the Jöd-Basedow phenomenon. We present a 62-year-old female with previously unknown history of Graves' disease developing acute thyrotoxicosis after cardiac catheterization.

### **Case Description**

A 62-year-old Russian woman with medical history significant for peripheral vascular disease, coronary artery disease, and bilateral carotid artery stenosis presented to the emergency department with chest pain, bilateral neck pain, and headache one day prior to planned carotid artery stenting. She was subsequently brought to the catheterization lab for percutaneous transluminal angioplasty of her right internal carotid artery with stent placement and received 90 mL of Omnipaque 300 mg/mL solution throughout the procedure. The operation was completed without complications. Eight hours following the procedure, she developed altered mental status, nausea and vomiting, hyperthermia up to 104.5°F, isolated systolic hypertension up to 238 mmHg, tachycardia up to 142 beats per minute, and hyperreflexia. Midazolam, acetaminophen, and cooling blankets were started and she was begun on an esmolol drip once transferred to the intensive care unit. Additional history obtained from the family at bedside included a vague history of overactive thyroid 5 years prior to presentation treated with radioactive therapy. The family noted, however, that her thyroid condition had persisted despite this therapy, and that she had symptoms consistent with thyrotoxicosis following prior catheterization procedures despite being treated. Additionally she had been prescribed methimazole, with poor medication adherence. Endocrinology suggested acute thyrotoxicosis following iodinated contrast administration with calculated Burch-Wartofsky score of 80 at the time. This scale assesses probability of thyrotoxicosis based on clinical factors without thyroid hormone levels. Levels greater than 45 highly

suggest thyroid storm. She was immediately begun on propylthiouracil, hydrocortisone, and propranolol to control peripheral T4 to T3 conversion. Aspirin was also held to prevent displacement of T4 from thyroxine binding globulin. She stabilized overnight and remained afebrile. Following transition to the general medicine service, she was weaned off hydrocortisone over the next 3 days and transitioned to methimazole. During this time, old records were consolidated with imaging suggestive of multinodular goiter and multiple cold nodules (Figure 1). She was discharged with close outpatient follow-up with an endocrinologist.

### Discussion

Iodinated contrast media is used with a multitude of radiologic procedures for greater absorption and scattering of x-ray radiation to increase CT attenuation and enhancement on the resultant CT image. Larger iodine concentrations distributed throughout the body provide greater contrast enhancement, with typical iodine loads containing approximately 13,500  $\mu$ g of free iodide and 15 to 60 g of bound iodine. The recommended daily intake of iodide, in comparison, is 150  $\mu$ g.<sup>1-3</sup> Administration of iodinated contrast media loads is associated with subsequent development of incident thyroid dysfunction or exacerbation of underlying thyroid pathology, with the sudden high iodide loads resulting in hypothyroidism by the Wolff-Chaikoff effect or hyperthyroidism by the Jöd-Basedow phenomenon.<sup>4</sup>

Typically, administration of excess iodine to otherwise healthy individuals results in the inhibition of oxidation of iodine for 48 hours prior to the normalization of thyroid hormone synthesis. However, in individuals more sensitive to iodine exposure, as in those with iodide deficiency or in patients with multinodular goiter containing autonomous nodules, excess iodide may result in a hyperthyroid state. Resultant thyrotoxicosis has been reported to follow a variable time course, ranging from decompensation within the first 24 hours to evolution over 12-weeks.<sup>5-6</sup>

Case reports have documented individuals previously treated with radioiodine therapy for multinodular goiter, developed iodinated contrast-induced thyrotoxicosis. These individuals were otherwise biochemically euthyroid prior to coronary angioplasty or imaging involving contrast loads of 35 g or greater. Subsequently, some developed exacerbations of congestive heart failure, respiratory compromise, and autonomic instability.<sup>7-8</sup> Many other case reports have described individuals without any known underlying thyroid dysfunction developing thyrotoxicosis following iodinated contrast exposure.<sup>9-11</sup> These cases were successfully treated with beta blockers, thionamide, and prednisone, with symptomatic temperature regulation and benzodiazepine therapy as needed to address ongoing agitation.

While the reported incidence of iodine excess-induced hyperthyroidism in both iodine deficient and iodine-sufficient areas is relatively low, monitoring thyroid function tests has been suggested for individuals at risk of developing thyrotoxicosis. Currently, manifest hyperthyroidism is an absolute contraindication to iodinated contrast media administration, while patients with Graves' disease, multinodular goiter, and thyroid autonomy are recommended to be closely monitored after iodinated contrast medium injection.<sup>2</sup> While prophylactic treatments are not routinely advised, perchlorate and thionamide administration to elderly patients with suppressed serum TSH or palpable goiter has been considered.<sup>12,13</sup> Because of side effects associated with these medications, however, follow-up thyroid function tests at 1 week and at 1-3 months after contrast exposure in these highrisk groups are preferable if otherwise asymptomatic.<sup>14</sup>

This case illustrates the potential for severe thyrotoxicosis following imaging or procedures with contrast loads. Caution is needed prior to ordering iodinated contrast media for CT imaging or angiography given the risk for life-threatening thyroid disease, especially in those with underlying endocrinopathies. Monitoring high risk individuals and recognizing symptoms of thyrotoxicosis following exposure to contrast are both imperative for rapid response to and resolution of such crises.



*Figure 1*: 2013 thyroid uptake scan after administration of iodine-123 demonstrating a diffusely enlarged gland with heterogeneous uptake indicating multinodular goiter. Discrete foci of decreased activity are present in the mid left lobe and in the inferior right lobe indicating cold nodules. Evaluation was limited by the patient not returning for 24-hour measurement.

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