



Managing concurrent thyrotoxicosis and acute appendicitis: a case report and perioperative considerations

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Abstract

Thyrotoxicosis can mimic acute abdominal pathology and may progress to thyroid storm when combined with surgical sepsis. A 47-year-old woman with poorly controlled Graves' disease presented with right iliac fossa pain, fever, tachycardia, and biochemical thyrotoxicosis. Her Burch-Wartofsky score was 60, indicating high storm risk. Ultrasound and CT confirmed tip appendicitis. She was admitted to intensive care and treated with beta-blockade, high-dose thionamides, Lugol's iodine, corticosteroids, and antibiotics before undergoing urgent laparoscopic appendicectomy. The appendix was inflamed but not perforated, and histology showed appendicitis. She recovered without perioperative cardiovascular instability or progression to storm. This case shows the importance of distinguishing true surgical causes of abdominal pain from thyrotoxic presentations, assessing storm risk objectively, and stabilising hyperthyroid patients before urgent surgery. Early endocrine optimisation and coordinated multidisciplinary care can allow safe operative management despite severe thyrotoxicosis.

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Introduction

Thyrotoxicosis is a common endocrine disorder, most often due to Graves' disease, toxic nodular goitre, or thyroiditis, and produces a hypermetabolic state affecting multiple organ systems^{1,2}. Elevated triiodothyronine (T3) and thyroxine (T4) upregulate β -adrenergic receptors and enhance catecholamine sensitivity, leading to tachycardia, widened pulse pressure, and tremor². Thyroid hormones also increase cardiac inotropy and chronotropy while reducing systemic vascular resistance, raising cardiac output by up to 300%². Severe untreated thyrotoxicosis may progress to thyroid storm, an acute decompensation with fever, tachyarrhythmias,

neurological disturbance, and multi-organ failure, carrying a reported mortality of 10-30%^{3,4}.

Acute appendicitis is among the commonest surgical emergencies worldwide and arises from luminal obstruction with subsequent bacterial overgrowth, transmural inflammation, and possible perforation⁵. Thyrotoxicosis can both mimic and precipitate acute abdominal pathology: thyroid storm has been reported to present with "pseudo-peritonitis" in the absence of intra-abdominal disease, whereas surgical stress in a hyperthyroid patient may trigger storm^{3,6}. Only a small number of cases describing true appendicitis complicated by thyrotoxic crisis have been published⁶. This report



describes a patient with Graves' thyrotoxicosis and acute appendicitis with a high Burch-Wartofsky score, managed surgically after rapid endocrine optimisation, and sets this in the context of existing evidence^{1-3,6}.

Case Report

A 47-year-old woman with Graves' hyperthyroidism and hypertension presented with five days of worsening abdominal pain, initially diffuse, then localising to the right iliac fossa, with nausea, vomiting, and anorexia. She described weight loss despite increased appetite, palpitations, heat intolerance, irritability, and irregular heavy menses. Carbimazole and propranolol had been prescribed two months earlier, but adherence was poor. She smoked 10 cigarettes daily and chewed miraa; there was no relevant family or surgical history.

On examination, she was anxious and diaphoretic, with a temperature of 38.5°C, a heart rate of 128 beats/min, a blood pressure of 166/71 mmHg, and a respiratory rate of 22/min. She had a fine tremor, warm, moist skin, moderate exophthalmos, and a diffuse grade 2 goitre with a bruit. Abdominal examination showed right iliac fossa tenderness with guarding and rebound, and hypoactive bowel sounds. Neurological examination was normal (Glasgow Coma Scale 15).

Blood tests showed white cell count $8.0 \times 10^9/L$, C-reactive protein 2 mg/L, and mild cholestatic liver enzyme elevation, a pattern recognised in thyrotoxicosis². Thyroid function was markedly abnormal: TSH <0.00083 mIU/L, free T4 53.95 pmol/L and free T3 >30.7 pmol/L, confirming severe thyrotoxicosis. Thyroid receptor antibodies were >40 U/L and anti-thyroid peroxidase antibodies >1000 IU/mL, consistent with active autoimmune Graves' disease and high relapse risk¹⁻³. Electrocardiography showed sinus tachycardia. Echocardiography demonstrated preserved left ventricular function with mild mitral and tricuspid regurgitation.

Ultrasound of the abdomen demonstrated a non-compressible, thickened tubular structure in the right iliac fossa with increased Doppler flow, in keeping with acute appendicitis. Contrast-enhanced

CT confirmed tip appendicitis with reactive mesenteric lymphadenopathy and no perforation or alternative cause of pain.

A Burch-Wartofsky Point Scale score of 60 was calculated, with contributions from fever, tachycardia, gastrointestinal symptoms, agitation, and the precipitating intra-abdominal infection, exceeding the threshold strongly suggestive of thyroid storm⁷. Although she had no heart failure or encephalopathy, she was considered at high risk of imminent storm.

The patient was admitted to intensive care. Intravenous beta-blockade (labetalol infusion) was commenced, and antithyroid therapy with propylthiouracil plus Lugol's iodine was given, together with corticosteroids, in accordance with guidance for emergent management of severe thyrotoxicosis¹⁻³. Broad-spectrum intravenous antibiotics were started. Once heart rate and temperature had improved and haemodynamics were stable, urgent laparoscopic appendicectomy was undertaken under general anaesthesia with invasive monitoring. Anaesthetic agents that stimulate the sympathetic system were avoided; short-acting beta-blockers and vasopressors were used cautiously to manage fluctuations.

Intra-operatively, the appendix was inflamed and thickened at the tip without perforation or pus. Appendicectomy proceeded uneventfully. Postoperatively, she was observed in high-dependency care, with continuation of beta-blockade, antithyroid medication, and a steroid taper. She did not develop thyroid storm or cardiovascular instability. Oral carbimazole and propranolol were reintroduced when tolerated. She mobilised on day 1 and was discharged on day 3. Histology showed chronic appendicitis with superimposed active inflammation ("acute on chronic" changes). At the three-week review, she remained clinically stable on antithyroid therapy.

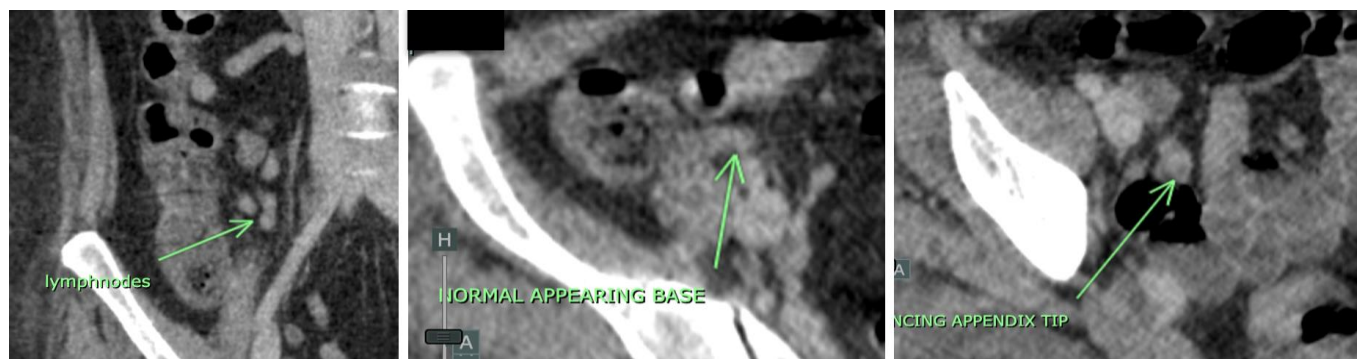


Figure 1: Abdominal CT imaging at presentation. (a) Sagittal view showing lymph node enlargement. (b) Axial view showing a normal appearing appendix base. (c) Enhancing the tip of the appendix

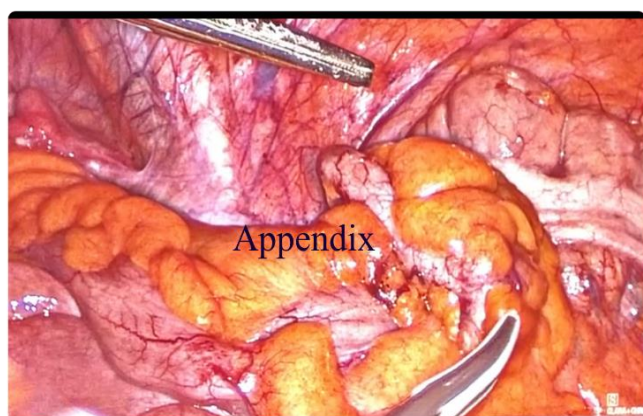


Figure 2: Laparoscopic view of an inflamed appendix with adjacent caecum and terminal ileum (hyperaemic) visible.

Discussion

This case demonstrates the diagnostic and perioperative problems posed by acute appendicitis in a patient with poorly controlled Graves' thyrotoxicosis. Thyroid hormone excess can cause abdominal pain, vomiting, and altered bowel habit, occasionally with peritoneal signs, and has led to unnecessary laparotomy in reported series^{3,6,10,11}. Conversely, true intra-abdominal sepsis may precipitate thyroid storm in hyperthyroid patients^{3,6}. In this patient, localised right iliac fossa tenderness, supportive ultrasound and CT findings, and subsequent histology confirmed genuine appendicitis rather than thyrotoxic "pseudo-peritonitis". Combined presentation of acute appendicitis with impending thyroid storm appears uncommon. Mohseni et al. described a patient with Graves' disease who developed appendicitis complicated by a storm, requiring intensive endocrine rescue and surgery⁶. Another report

detailed ruptured appendicitis in unrecognised Graves' disease, in which iodinated contrast contributed to severe thyrotoxicosis¹. Historical cases suggested a possible association between untreated hyperthyroidism and suppurative appendicitis, but evidence remains limited³. Our case adds a further example and supports maintaining a high index of suspicion for surgical pathology in hyperthyroid patients with focal peritonism, while simultaneously evaluating for storm^{1,3,6,10,11}.

Markedly raised TRAb and anti-TPO antibody titres in this patient confirmed active autoimmune disease and indicate a high risk of recurrence with medical therapy alone, as well as eventual hypothyroidism after definitive treatment¹⁻³. Incorporating antibody testing into preoperative assessment helps clarify aetiology, inform counselling, and guide long-term planning, including radioiodine or thyroidectomy.

Perioperative management followed principles from endocrine emergency guidance^{1-3,7}. For elective procedures, rendering patients biochemically euthyroid is advised, but urgent surgery should not be unduly delayed when there is clear surgical sepsis. In such cases, rapid optimisation with beta-blockers, high-dose thionamides, iodine, and steroids can reduce the risk of storm sufficiently to allow timely operation¹⁻³. Modern anaesthetic techniques, careful drug selection, and invasive monitoring further mitigate risk². In our patient, this combined approach allowed appendicectomy without progression to storm despite a Burch-



Wartofsky score in the “storm” range⁷. Finally, the case illustrates the value of objective scoring systems. The Burch-Wartofsky scale provided a structured assessment and supported the decision to treat as an impending storm even in the absence of heart failure or coma⁷. In practice, any hyperthyroid patient with acute illness and a high score should receive full storm therapy while the underlying precipitant is addressed.

Overall, this report shows that acute appendicitis can occur in the context of severe Graves’ thyrotoxicosis with a high calculated storm risk, and that with prompt recognition, rapid endocrine stabilisation and coordinated anaesthetic and surgical care, operative management can proceed safely^{1-3,6,7,11}.

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Generative Artificial Intelligence Transparency (GAIT)

Statement: ChatGPT Plus was used in August 2025 to check and edit the case report, supplement the literature review, and edit small sections of the manuscript text for clarity (writing, review, & editing). We, the authors, retain final responsibility for our work.

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