

Refractory atrial fibrillation in an emergency surgical patient: a sign of untreated thyrotoxicosis

SHUYA KIYAMA and TAMOTSU YOSHIKAWA

Department of Anesthesia, Shizuoka Red Cross Hospital, 8-2 Otemachi, Shizuoka, 420 Japan

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Introduction

Diagnosis of thyrotoxicosis can be difficult in patients without a palpable goiter. The patient presented for emergency surgery with poorly controlled atrial fibrillation. History suggestive of thyrotoxicosis was obtained and antithyroid treatment was started simultaneously with general anesthesia. Early use of beta-blocker and an antithyroid drug prevented postoperative thyroid storm.

Case report

The patient was a 63-year-old woman who presented for emergency exploratory laparotomy due to acute abdominal pain. She had had an uneventful general anesthetic for lumbar laminectomy 5 years previously. She had not been on any medications except for an analgesic for her lumbago. She could not give any further medical history because of abdominal pain, dyspnea, and acute distress. Her peripheral pulse was irregular and her respiratory rate was 40 breaths per minute. Preoperative electrocardiogram showed atrial fibrillation with a rapid ventricular response, which had been unresponsive either to digoxin 0.25 mg or to verapamil 5 mg, i.v. Her temperature was 38.9°C and a surgeon attributed pyrexia to peritonitis. Goiter was not palpable.

An interview with the patient's family revealed that the patient had been experiencing finger tremor for the

past 2 years. The family also volunteered that she had become nervous and irritated recently. The patient had not sought any medical treatment for those matters. Presumptive diagnosis of untreated thyrotoxicosis was made and blood was sampled for later measurement of thyroid hormones. The patient was taken to the operating room without preanesthetic medication.

Right internal jugular vein and radial artery cannulae were positioned under local anesthesia. Initial hemodynamics were as follows: blood pressure 210/80 mmHg, central venous pressure 8 mmHg, and heart rate 165 beats per minute (bpm) in atrial fibrillation. Two intravenous bolus doses of propranolol 0.5 mg decreased the heart rate to 110 bpm without causing venous congestion. Methimazole 30 mg and methylprednisolone 1000 mg were also administered intravenously. Rapid sequence induction and orotracheal intubation with cricoid pressure was facilitated with fentanyl 0.1 mg, thiopental 150 mg, and succinylcholine 80 mg. Anesthesia was maintained with continuous infusion of fentanyl (0.075 mg/h), 0.5% isoflurane and 50% nitrous oxide in oxygen. The patient had a perforated duodenal ulcer, for which direct suture and omental patch were performed. Throughout the anesthetic, the cardiac rhythm was atrial fibrillation and the heart rate was controlled between 85 and 115 bpm. The temperature decreased to 37.4°C at the end of surgery. The patient was on a mechanical ventilator postoperatively and was successfully weaned 5 h later. Preoperative laboratory results became available on the 2nd postoperative day (POD). Both thyroxine (T_4) and triiodothyronine (T_3) were markedly increased: T_4 was $25.5 \mu\text{g}\cdot\text{dL}^{-1}$ (normal range; $5.0\text{--}11.5 \mu\text{g}\cdot\text{dL}^{-1}$) and T_3 was $8.0 \text{ ng}\cdot\text{mL}^{-1}$ (normal; $0.8\text{--}1.6 \text{ ng}\cdot\text{mL}^{-1}$). Antithyroid therapy with methimazole (120 mg per day i.v.) and beta-blockade with propranolol infusion ($1 \text{ mg}\cdot\text{h}^{-1}$) were commenced and cardiac rhythm reverted back to a normal sinus rhythm with a rate of 75 bpm on the 4th POD. The patient made an uneventful recovery.

Address correspondence to: S. Kiyama

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Discussion

The diagnosis of thyrotoxicosis is straightforward when hypermetabolic symptoms and signs are found in a young patient with a diffuse goiter. However, this is not the case when only nonspecific cardiovascular, gastrointestinal, and neurological symptoms are the primary manifestations in an elderly patient with very little thyroid enlargement [1]. This patient's preoperative major problem was a refractory atrial fibrillation. Atrial fibrillation is a common cardiac dysrhythmia and can be found in patients with mitral valve, pulmonary, pericardial disease as well as thyrotoxicosis and in patients with unknown etiology [2]. Digoxin and verapamil are the standard medical therapy for this dysrhythmia, but when these agents fail to control atrial fibrillation, the underlying etiology should be scrutinized. In this case, information from the family helped the correct diagnosis to be reached and appropriate therapy was started preoperatively.

Hyperthyroid patients should not undergo anesthesia and surgery except when it is life-threatening because of the risk of precipitating perioperative thyroid crisis [3]. Although the incidence of perioperative thyroid storm is now decreasing in patients undergoing elective thyroidectomy because of preoperative preparation with antithyroid drugs, thyroid storm can still occur when emergency surgery is performed in hyperthyroid patients [4]. Antithyroid drugs are the mainstay of treatment for most thyrotoxic patients in the medical out-patient setting. However, the role of parenteral antithyroid drugs remains unclear in patients presenting for life-saving emergency surgery. It takes a couple of weeks before the clinical effects of antithyroid drugs become apparent by inhibiting the biosynthesis of thyroid hormones. It is therefore unknown whether an early use of antithyroid drugs leads to better outcome in patients with thyrotoxic crisis.

On the other hand, acute beta-blockade has been recommended as the treatment of choice for thyrotoxic patients presenting for emergency surgery [5,6]. Although beta-blockade may bring symptomatic relief, its efficacy as a treatment of thyroid storm is still controversial [7]. Beta-blockade can precipitate overt cardiac failure, particularly in elderly thyrotoxic patients who may have coexisting cardiac diseases. Use of either central venous or pulmonary artery catheters should be considered in such cases to monitor hemodynamics. Another caveat in the use of beta-blockers is that they may mask clinical manifestations in patients with overlooked thyrotoxicosis and thus may delay correct diagnosis and appropriate treatment [8]. The risk of thyroid crisis can be still much higher if thyrotoxicosis is overlooked preoperatively.

Although the efficacy of intravenous antithyroid drugs with regard to morbidity and mortality and final

outcome in thyrotoxic crisis is unclear, the authors think that antithyroid treatment should be started as soon as possible for two reasons. Firstly, this is the only measure to inhibit synthesis of thyroid hormones. Although plasmapheresis has also been successfully performed to decrease plasma thyroid hormones in some cases of thyroid storm [9,10], this is not applicable at every institution. Secondly, there have been no serious adverse effects specific to the use of intravenous antithyroid drugs. Although one major adverse effect of these agents is agranulocytosis, the incidence of this complication after short-term intravenous administration is unknown. The starting dose of methimazole is 30 mg every 4–6 h as an intravenous bolus dose. When oral administration is feasible, intravenous methimazole can be switched to either methimazole or propylthiouracil orally. The latter theoretically offers more advantages by blocking peripheral conversion of T_4 to T_3 [11]. Lugol's solution is also beneficial to inhibit thyroid hormone release from the gland [6]. It is important that Lugol's solution should be given at least 1 hour after starting antithyroid drugs.

Other therapeutic measures for thyrotoxic crisis are mostly supportive. These measures include, beta-blockers and digoxin to control tachyarrhythmia, fluid management guided by invasive cardiovascular monitoring, and treatment of hyperthermia. Cases of thyrotoxic crisis which were initially misdiagnosed as malignant hyperthermia have been reported [12–14]. Monitoring of expired carbon dioxide and arterial blood gases can help differentiate these two life-threatening conditions.

In conclusion, thorough investigation for a cause of refractory preoperative atrial fibrillation was crucial in preventing full-blown thyroid storm in this patient. Although acute beta-blockade may provide immediate symptomatic relief, we emphasize the importance of early commencement of antithyroid therapy as well with proper preoperative diagnosis.

References

1. Davis PJ, Davis FB (1974) Hyperthyroidism in patients over the age of 60 years. *Medicine* 53:161–181
2. Aitkenhead AR, Barnett DB (1989) Heart disease. In: Vickers MD, Jones RM (eds) *Medicine for anaesthetists*. Blackwell, Oxford, pp 28–29
3. Stehling LC (1974) Anesthetic management of the patient with hyperthyroidism. *Anesthesiology* 41:585–595
4. Kiyama S, Yoshikawa T, Ozawa H, Koh H, Maki H, Tszaki K, Fukushima K (1993) Emergency laparotomy in uncontrolled thyrotoxic patient with preoperative fulminant hepatic failure. *J Anesth* 7:82–85
5. Feely J, Peden N (1984) Use of β -adrenoceptor blocking drugs in hyperthyroidism. *Drugs* 27:425–446
6. Blackshear PJ (1989) Thyrotoxic crisis. In: Rippe JM (ed) *Manual of intensive care medicine*. Little Brown, Boston, pp 399–403

7. Eriksson M, Rubinfeld S, Garber AJ, Kohler PO (1977) Propranolol does not prevent thyroid storm. *New Engl J Med* 296:263–264
8. Jones DK, Solomon S (1981) Thyrotoxic crisis masked by treatment with beta-blockers. *Br Med J* 283:659
9. Hermann J (1973) Plasmapheresis in the treatment of thyrotoxic crisis. *Acta Endocrinol [Suppl]* 173:22
10. Ashkar FS, Katims RB, Smoak WM, Gilson AJ (1970) Thyroid storm treatment with blood exchange and plasmapheresis. *JAMA* 214:1275–1279
11. Geffner DL, Azukizawa M, Hershman JM (1975) Propylthiouracil blocks extrathyroidal conversion of thyroxine to triiodothyronine and augments thyrotropin secretion in man. *J Clin Invest* 55:224–229
12. Bennett MH, Wainwright AP (1989) Acute thyroid crisis on induction of anaesthesia. *Anaesthesia* 44:28–30
13. Stevens JJ (1983) A case of thyrotoxic crisis that mimicked malignant hyperthermia. *Anesthesiology* 59:263
14. Peters KR, Nance P, Wingard DW (1981) Malignant hyperthyroidism or malignant hyperthermia? *Anesth Analg* 60:613–615