International Journal of Research in Medical Sciences
Nirmala PAD et al. Int J Res Med Sci. 2024 Sep;12(9):3430-3433
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20242625

Case Report

Perioperative management of patient with newly diagnosed hyperthyroidism

Putu A. D. Nirmala*, Dewi C. Wulandari

Department of Internal Medicine, Wangaya Regional General Hospital, Denpasar, Bali, Indonesia

Received: 04 July 2024
Accepted: 16 August 2024

***Correspondence:**
Dr. Putu A. D. Nirmala,
E-mail: divya.nirmala@gmail.com

ABSTRACT

Hyperthyroidism is a clinical condition caused by increased synthesis and secretion of thyroid gland hormones that affect the whole body. Its multi-organ effects and complications require adequate management in perioperative period. A 50-year-old patient was consulted to internal medicine for perioperative management for ureteroscopy procedure. Patient reported bilateral flank pain, nausea, vomiting, palpitation, and history of weight loss. Patient also has history of diabetes mellitus, on gliquidone. Physical examination presented pale conjunctiva, tachycardia, and soft tremor. Thyroid gland was enlarged, tender on palpation, no bruits were found. Peripheral blood count and thyroid function evaluation showed elevated white blood count, severe anaemia, elevated blood urea nitrogen and creatinine, decreased thyroid stimulating hormone and elevated free thyroxine. Ultrasound of thyroid gland concluded multiple cystic nodules. Wayne's index is 20. Patient was administered 2 bags of packed red cells, insulin injection, propylthiouracil 3×200 mg, propranolol 3×10 mg, and hydrocortisone injection 2×100 mg prior to surgery. Patients with hyperthyroidism are recommended to be in euthyroid state prior to surgery. Hyperthyroid patients requiring emergent/urgent surgery are administered a combination of antithyroid drugs, corticosteroids, and beta-blockers to prevent thyroid storm. Propylthiouracil prevents production of thyroid hormones, corticosteroids inhibit hypermetabolism, and beta-blockers prevent sympathetic stimulation. Surgery was successful and thyroid storm did not occur.

Keywords: Hyperthyroidism, Perioperative, Surgery

INTRODUCTION

Hyperthyroidism is a disorder that involves a hyperactive thyroid gland.[1] It could lead to thyrotoxicosis, namely a clinical state characterized by excess serum and tissue concentrations of thyroxine (T4), triiodothyronine (T3), or both. It is divided into overt and subclinical thyrotoxicosis. The prevalence of thyrotoxicosis in the United States is estimated to be 1.2%, with 40% of cases being overt, and 60% being subclinical. The prevalence of overt hyperthyroidism is estimated at 0.2-1.4%, while subclinical is 0.7-1.4%. The estimated global prevalence of hyperthyroidism in iodine-sufficient countries is 0.2-2.5%.[2] One of the most frequently found cause of hyperthyroidism is Graves' disease, with multinodular thyroid disease coming after. Hyperthyroidism is also more prevalent in female than male, with a ratio of 8:1.[3,4]

Hyperthyroidism can cause numerous complications, one of them include thyroid storm. Thyroid storm is a form of severe uncontrolled hyperthyroidism with multi-organ failure. It is characterized with fever, tachycardia, atrial fibrillation, as well as central nervous system abnormalities.[2]

Hyperthyroidism or thyrotoxicosis can be found incidentally on perioperative assessment. Optimization of patient's thyroid status and general condition is vital to assert good surgical outcome and to prevent thyroid storm.[5] Our paper discusses a case of successful

perioperative management of patient with newly diagnosed hyperthyroidism in our centre.

CASE REPORT

A 50-year-old female was consulted to department of internal medicine by department of urology for preoperative assessment and management prior to ureteroscopy (URS) procedure. Patient was previously diagnosed with left ureter stone, hydronephrosis, and right kidney Staghorn stone. We assessed the patient with history taking, physical examination, and further testing. Patient has chief complaint of bilateral flank pain. Flank pain was reported since 3 months prior to admission, but worsened 2 weeks ago. Pain was said to come and go, and worsened with change of position. Patient also complained of nausea and vomiting. Nausea and vomiting occurred around the same time as flank pain. Patient also complained of palpitations from time to time, tremor, and also reported weight loss. Patient reported to have lost 10 kg within 3 months. Change in diet and quantity of meals was denied. Patient also reported history of diabetes mellitus since 2 years prior. Patient takes gliquidone tablet 30 mg, once daily. History of other chronic diseases such as hypertension, heart disease, kidney disease was denied. History of allergy of food and drugs was denied. History of fever was denied.

On admission, patient's condition was moderate and neurological status was alert. Vital signs showed blood pressure of 145/90 mmHg, heart rate 100 beats per minute (bpm), respiration rate 20 times per minute, body temperature 36°C, oxygen saturation 98% on room air, visual analogue scale 5/10. Vital signs were followed up by general physical examination. Upon physical examination, patient was found to have pale conjunctiva, tachycardia, and soft tremor. Patient also had enlarged thyroid gland (Figure 1). Thyroid gland was palpated and found to be soft in consistency, mobile, and is approximately 3 cm in diameter. No bruits were found on auscultation. Abdominal examination showed pain on left costovertebral angle, and tenderness on epigastrium. Thorax examination was within normal limits.

Figure 1: Patient's documentation showing enlarged thyroid gland.

We proceeded to conduct electrocardiogram (ECG), complete blood count, liver function test, kidney function test, urinalysis, and thyroid hormone evaluation. The ECG showed sinus tachycardia (Figure 2). Laboratory result showed leukocyte count of $19.22\times10^3/\mu l$ with increased neutrophil to lymphocyte ratio of 10.77, haemoglobin count of 6.0 g/dl, haematocrit 20.5%, platelet count $720\times10^3/\mu l$, blood urea nitrogen (BUN) 83 mg/dl, serum creatinine 1.9 mg/dl, sodium 125 mmol/l, potassium 4.3 mmol/l, chloride 103 mmol/l, random blood glucose 177 mg/dl.

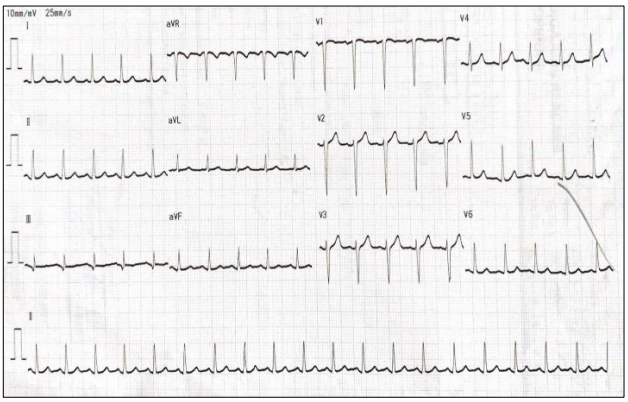

Figure 2: Sinus tachycardia in patient's ECG.

Thyroid stimulating hormone (TSH) was low at 0.20 mIU/l, free thyroxine (T4) was high at 1.65 ng/dl. Echocardiography showed normal dimension with concentric remodelling, ejection fraction 69%, normal valves. Ultrasonography (USG) of neck revealed mild enlargement of right thyroid gland, multiple cystic nodules with solid component and microcalcification, largest size of nodule 0.85×0.66 cm, echoparenchyme outside of lesion, no vascularization on parenchyme (Figure 2). Left thyroid gland showed enlargement, multiple cystic nodules with solid component and microcalcification, largest size of nodule 1.99×1.41 cm, echoparenchyme outside of lesion, no vascularization on parenchyme. No neck or submandibular lymphadenopathy was detected. Neck USG concluded multiple cystic nodules with solid component and microcalcification of right and left thyroid gland.

Patient was admitted to general ward with diagnosis of left ureter stone, hydronephrosis, right renal Staghorn stone, diabetes mellitus, anaemia, chronic kidney disease (CKD) stage III, and multiple thyroid nodules with hyperthyroidism. Patient had a total Wayne index score of 21, which is highly suggestive of toxic hyperthyroidism or thyrotoxicosis.

Patient was treated under the collaboration of three specialists namely urologist, endocrinologist, and cardiologist. Patient was ensured to be stable before proceeding to surgery, and was first administered with ceftriaxone 1 gram every 12 hours intravenously, levofloxacin 500 mg once daily intravenously. Patient was

ordered transfusion of packed red cells (PRC) 2 bags per day until haemoglobin is 10, with premedication of furosemide 40 mg intravenous injection, gliquidone 30 mg tablet once daily, ondansetron 4 mg injection thrice a day intravenously, and to avoid use of nephrotoxic drugs. For hyperthyroidism, patient was administered propylthiouracil (PTU) 200 mg tablet thrice a day, and propranolol 10 mg tablet thrice a day. Lugol was not administered because of unavailability at our centre.

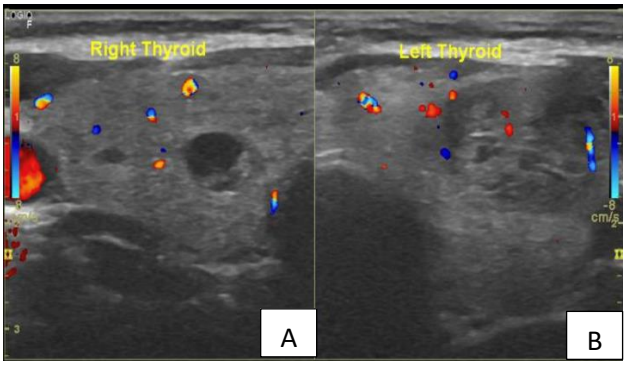

Figure 3 (A and B): Ultrasonography of the thyroid gland documenting multiple cystic nodules with solid component and microcalcification of both thyroid glands.

One day before surgery, patient's therapy was modified. PTU 200 mg was increased to 5 times a day. Patient's blood glucose also increased above normal limits, therefore gliquidone tablet was stopped and switched to insulin drip using syringe pump until blood glucose was under 200 mg/dl. Afterwards, drip was stopped and switched to insulin lispro injection 4 units subcutaneously. Patient was also conducted complete blood count after PRC transfusion and haemoglobin improved to 11.5 g/dl.

Surgery was successful, patient was stable, and thyroid storm did not occur. Therapy was modified again with PTU tapered down to 100 mg thrice a day, propranolol 10 mg thrice a day, hydrocortisone injection stopped, insulin lispro injection increased to 6 units subcutaneously thrice a day with meal, and insulin glargine injection 6 units subcutaneously at night before bedtime. Patient was discharged 3 days post-operation with PTU 100 mg tablet thrice a day, propranolol 10 mg tablet thrice a day, folic acid 2 mg tablet twice a day, glargine injection 6 unit subcutaneous once a day, and lispro injection 4 units subcutaneously thrice a day. Patient was scheduled for follow-up 3 days after discharge.

DISCUSSION

Hyperthyroidism is a condition in which there is an increase in synthesis and secretion of thyroid hormones by the thyroid gland. Thyrotoxicosis can also be caused by release of preformed hormones from thyroid due to inflammation, or excess T4 depletion. Causes of hyperthyroidism itself are usually Graves' disease, multinodular goitre, or toxic adenoma. Multinodular goitre is the second leading cause of hyperthyroidism in patients, with estimated incidence of 1.5-1.8 cases/100,00-persons/year worldwide.[2,6] Thyroid nodules can autonomously secrete thyroid hormones in excess that can exacerbate thyrotoxicosis. Prevalence of hyperthyroidism is higher in females than males, with 2% in women and 0.2% in men. Incidence also increases with age, iodine deficiency, and race. Toxic nodular goitre also accounts for 50% cases in iodine-depleted areas.[7,8] In our case our patient is a 50-year-old female, meaning patient is at higher risk for hyperthyroidism.

Hyperthyroidism can also be divided into two categories, namely overt or subclinical hyperthyroidism. Overt hyperthyroidism is defined as subnormal or undetectable TSH with increased levels of triiodothyronine (T3) and/or T4. Subclinical hyperthyroidism is defined as low or undetectable TSH levels and have T3 and T4 levels within normal limits. Both can lead to thyrotoxicosis and could be calculated using a scoring system, Wayne Index. Scoring interpretation are <11 for euthyroid/non-toxic, 11-19 equivocal, and >19 for toxic. Patient presented with palpitation, tiredness, excessive sweating, weight loss, palpable thyroid, hyperkinesis, pulse rate >90 bpm. Total score is 20 and patient classifies as toxic meaning patient shows signs of hyperthyroidism and thyrotoxicosis (Table 1).[4,6]

Perioperative management requires screening of multiple organ function. Other than routine laboratory testing, thyroid hormone function test could be done. Patients presenting with symptoms of thyrotoxicosis require screening. Other factors that could contribute are female patients over the age of 50, patients with goitres, type 1 diabetes, other autoimmune diseases, family history of thyroid disease, and use of medications such amiodarone, iodine, lithium. Our patient presents as a 50-year old female with goitre. Another reason to check thyroid function is because our patient presents with symptoms of thyrotoxicosis. It is to determine whether thyrotoxicosis is caused by hyperthyroidism or not. This will determine treatment plan of the patient.

Thyrotoxicosis is crucial to evaluate and manage properly before undergoing procedure. Uncontrolled thyrotoxicosis brings patient to risk of thyroid storm. Thyroid storm is a medical emergency characterized by severe thyrotoxicosis with tachycardia and hyperpyrexia. Precipitating factors include cessation of antithyroid drugs (ATD), infection, and surgery. Patients are usually recommended to be euthyroid for surgery as it adds additional risks. Complications that may occur are atrial fibrillation, ventricular dysfunction, heart failure, and even death. However, in cases where surgery is emergent, patients must be prepared adequately before procedure. Patients about to undergo surgery and anaesthesia face additional risks. Therefore, patient should be managed and observed accordingly to prevent thyroid storm with the administration of combined drugs.[1,2,5,6]

Table 1: Wayne's index scoring system for diagnostic approach of hyperthyroidism.[4]

Symptoms of recent onset and/or increased severity	Scores	Signs	If present	If absent
Dyspnea on effort	(+) 1	**Palpable thyroid**	(+) 3	(-) 3
Palpitations	(+) 2	**Bruit over thyroid**	(+) 2	(-) 2
Tiredness	(+) 2	**Exophthalmos**	(+) 2	-
Preference for heat	(-) 5	**Lid retraction**	(+) 2	-
Preference for cold	(+) 5	**Lid lag**	(+) 1	
Excessive sweating	(+) 3	**Hyperkinesis**	(+) 4	(-) 2
Nervousness	(+) 2	**Hands: hot**	(+) 2	(-) 2
		Hands: moist	(+) 1	(-) 1
Appetite increased	(+) 3	**Pulse rate >80 bpm**	-	(-) 3
Appetite decreased	(-) 3	**Pulse rate >90 bpm**	(+) 3	-
Weight increased	(-) 3	**Atrial fibrillation**	(+) 4	-
Appetite decreased	(+) 3			

Premedication in preparation for surgery in hyperthyroid patient includes ATD, beta-blockers, lugol iodide, and corticosteroids. Antithyroid drugs, namely thionamides (PTU and methimazole) block thyroid hormone synthesis. Its effect is also to control hyperthyroidism in postoperative period. Propylthiouracil is given at a dose of 100-150 mg every six to eight hours to reduce T4 to T3 conversion. Lugol is used to block organification of iodide and inhibit release of thyroid hormones. It is usually administered one hour after administration of ATD to avoid hormone surge, especially in patients with toxic adenoma or multinodular goitre. Beta-blockers are used to inhibit conversion to active thyroid hormone and also manages tachycardia, hypertension, and fever in patients. Propranolol can be administered at dose of 10-20 mg every eight hours. Corticosteroids are administered using stress-dose to prevent conversion of T4 to T3. Hydrocortisone injection at 100 mg is administered every 8 hours intravenously before surgery, and tapered over 3 days.[6,9,10] Our patient was given combination of drugs accordingly. Patient received PTU 200 mg thrice a day, propranolol 10 mg thrice a day, and hydrocortisone 100 mg injection thrice a day before surgery. Patient was also put under close monitoring and moved to intensive care unit (ICU) after procedure to ensure stable condition. After the procedure, dose of PTU was altered and decreased, and hydrocortisone was stopped. Lugol was not administered in our case as it is not available at our centre. However, with the combination of drugs available, patient's surgery was successful and patient is in stable condition.

CONCLUSION

Patients with hyperthyroidism are recommended to be in euthyroid state prior to surgery. If not, a combination of antithyroid drugs, corticosteroids, and beta-blockers are administered to prevent thyroid storm. Propylthiouracil prevents production of thyroid hormones, corticosteroids inhibit hypermetabolism, and beta-blockers prevent sympathetic stimulation. Surgery was successful and thyroid storm did not occur.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. McDermott MT. Hyperthyroidism – In The Clinic. Ann Int Med. 2020;172(7):ITC50-63.
2. Lee SY, Pearce EN. Hyperthyroidism: A Review. JAMA. 2023;330(15):1472-83.
3. Wemeau JL, Klein M, Sadoul JL,Briet C, Velayoudom-Cephise FL. Graves' disease: Introduction, epidemiology, endogenous and environmental pathogenic factors. Annales d'Endocrinologie. 2018;79(6):599-607.
4. The Indonesian Society of Endocrinology Task Force on Thyroid Diseases. Indonesian Clinical Practice Guidelines for Hyperthyoidism. J ASEAN Federation Endocrine Soc. 2012;27(1):34-9.
5. Terefe M, Bizuneh YB, Nigatu YA, Melesse DY. Perioperative management of the thyrotoxic patients: A systematic review. Ann Med Surg. 2022;81:104487.
6. Ross DS, Burch HB, Cooper DS, Greenlee MC, Laurberg P, Maia AL, et al. 2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis. Thyroid. 2016;26(10):1343-421.
7. Sharma A, Stan MN. Thyrotoxicosis: Diagnosis and Management. Mayo Clin Proc. 2018;94(6):1048-64.
8. Gilbert J. Thyrotoxicosis – investigation and management. Clin Med. 2017;17(3):274-7.
9. Himes CP, Ganesh R, Wight EC, Simha V, Liebow M. Perioperative Evaluation and Management of Endocrine Disorders. Mayo Clin Proc. 2020;95(12):2760-74.
10. Malhotra B, Bhadada SK. Perioperatve Management for Non-Thyroidal Surgery in Thyroid Dysfunction. Indian J Endocr Metab. 2022;26:428-34.

Cite this article as: Nirmala PAD, Wulandari DC. Perioperative management of patient with newly diagnosed hyperthyroidism. Int J Res Med Sci 2024;12:3430-3.