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REVIEW

ABC approach for the management of adults with hyperthyroidism: A practical strategy in primary care

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Abstract

Hyperthyroidism is commonly seen in primary care settings. However, the management of hyperthyroidism might be unclear to primary care doctors. Various guidelines have been published to assist clinicians in the management of thyroid disorders at various levels of care. The extensive coverage of these guidelines may not appeal to busy clinicians, and the guidelines do not focus on often resource-limited primary care settings. In this article, we aim to describe a practical guide for managing hyperthyroidism in primary care settings using an ABC approach.

Introduction

Primary care holds a central role in the management of hyperthyroidism through early detection and diagnosis and initial management of the condition. In Malaysia, a multicentre cross-sectional study has shown that the prevalence of overt hyperthyroidism is 0.6%, while that of subclinical hyperthyroidism (SH) is 2.8%. Among patients with a history of thyroid disorders, only 48.0% who are on antithyroid drugs (ATDs) have been found to be euthyroid.[1] Various guidelines have been published, including a recent one in Malaysia, to assist clinicians in the management of thyroid disorders at various levels of care.[2,3] However, the extensive coverage of these guidelines may not be appealing to busy clinicians, and the guidelines do not focus on often resource-limited primary care settings. Therefore, we aim to describe a practical approach for managing hyperthyroidism in primary care settings. For this purpose, we adopted local guidelines, especially the 2019 Clinical Practice Guidelines on the Management of Thyroid Disorders, with reference to international guidelines, research and textbooks to complete this review.

Management in primary care settings

Hyperthyroidism can be divided into overt hyperthyroidism (low thyroid-stimulating hormone [TSH] level with an increased free thyroxine [fT4] or free triiodothyronine [fT3] level) and SH (low TSH level with normal fT3 and fT4 levels) regardless of the presence or absence of the symptoms of the condition.[2] In general, the management of hyperthyroidism can be divided into an ABC approach: **A**scertain diagnosis, **B**est treatment option and **C**ontinuous assessment.

Overt hyperthyroidism

Thyrotoxicosis is characterised by the clinical signs and symptoms of inappropriate high thyroid hormone effects in tissues as a result of increased thyroid hormone levels.[2] Hyperthyroidism, which is a part of thyrotoxicosis, is characterised by an inappropriate increase in the synthesis of the thyroid hormone and its secretion by the thyroid gland.[2]

A. Ascertain diagnosis[2]

Once hyperthyroidism is suspected, the thyroid function test (TFT) including the assessment of the serum TSH and fT4 levels should be performed at the initial evaluation. The reference range for the TFT results may vary based on the method used for measurement and the local population. If the TSH level is lower than the reference range, but the fT4 level is within the normal range, the fT3 level should be measured. In general, a TSH level of <0.01 mU/L is considered to indicate TSH suppression, while a TSH level of ≥0.1 mU/L is considered low. Hyperthyroidism itself is not a diagnosis but a condition.[4] The TFT results can confirm the hyperthyroid state, but the cause of hyperthyroidism must be ascertained to determine the clinical management. The most

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common causes of hyperthyroidism are Graves' disease (GD), toxic multinodular goitre (MNG), solitary toxic adenoma (TA), transient thyroiditis and exogenous hyperthyroidism. The less common causes include drug-induced thyrotoxicosis, subacute thyroiditis (e.g. de Quervain's), hyperemesis gravidarum and post-partum thyroiditis (PPT). Rare causes are metastatic follicular thyroid cancer, struma ovarii, thyroblastic tumour, TSH-secreting pituitary adenoma, Marine–Lenhart syndrome, Hashimoto's thyroiditis, TSH-oma, gestational trophoblastic disease, Jod–Basedow thyrotoxicosis and L-asparaginase chemotherapy. **Table 1** shows the potential diagnosis for hyperthyroidism based on the TFT results.

Table 1. Possible aetiological diagnosis based on the thyroid function test results.[5,6]

TSH level	fT4 level	fT3 level	Possible diagnoses
↓	↑	↑	Overt hyperthyroidism (look for potential causes)
↓	Normal		Subclinical hyperthyroidism, central hypothyroidism, NTI, treatment with antithyroid drugs, early pregnancy, normal age-related variation in healthy elderly individuals, glucocorticoid and/or dopamine therapy or assay interference
↓	Normal	↑	Graves' disease, T3 hyperthyroidism, thyroid adenoma, ingestion of exogenous T3 or assay interference
↓	↑	↓	Hyperthyroidism with concurrent NTI, amiodarone therapy or ingestion of exogenous T4
↓	↓		Central hypothyroidism (hypothalamic or pituitary disorder)
↓	↓	↓	Sick euthyroidism or pituitary disease
↑/Normal	↑	↑	TSH-secreting pituitary adenoma, NTI, assay interference or drugs (thyroxine, amiodarone or heparin)

TSH, thyroid-stimulating hormone; fT4 thyroxine; fT3, free triiodothyronine; NTI, non-thyroidal illness; T3, triiodothyronine; T4 thyroxine.

Further investigations for the aetiological diagnosis of hyperthyroidism should be guided by clinical suspicion (**Figure 1**). When investigating thyroid nodules or nontoxic goitre, the serum TSH level should be measured. If the TSH level is beyond the reference range, the fT4 level should be assessed.[7] TSH suppression could indicate the presence of toxic and autonomously functioning nodules in the goitre. Hence, it is recommended to proceed with thyroid scintigraphy if available.

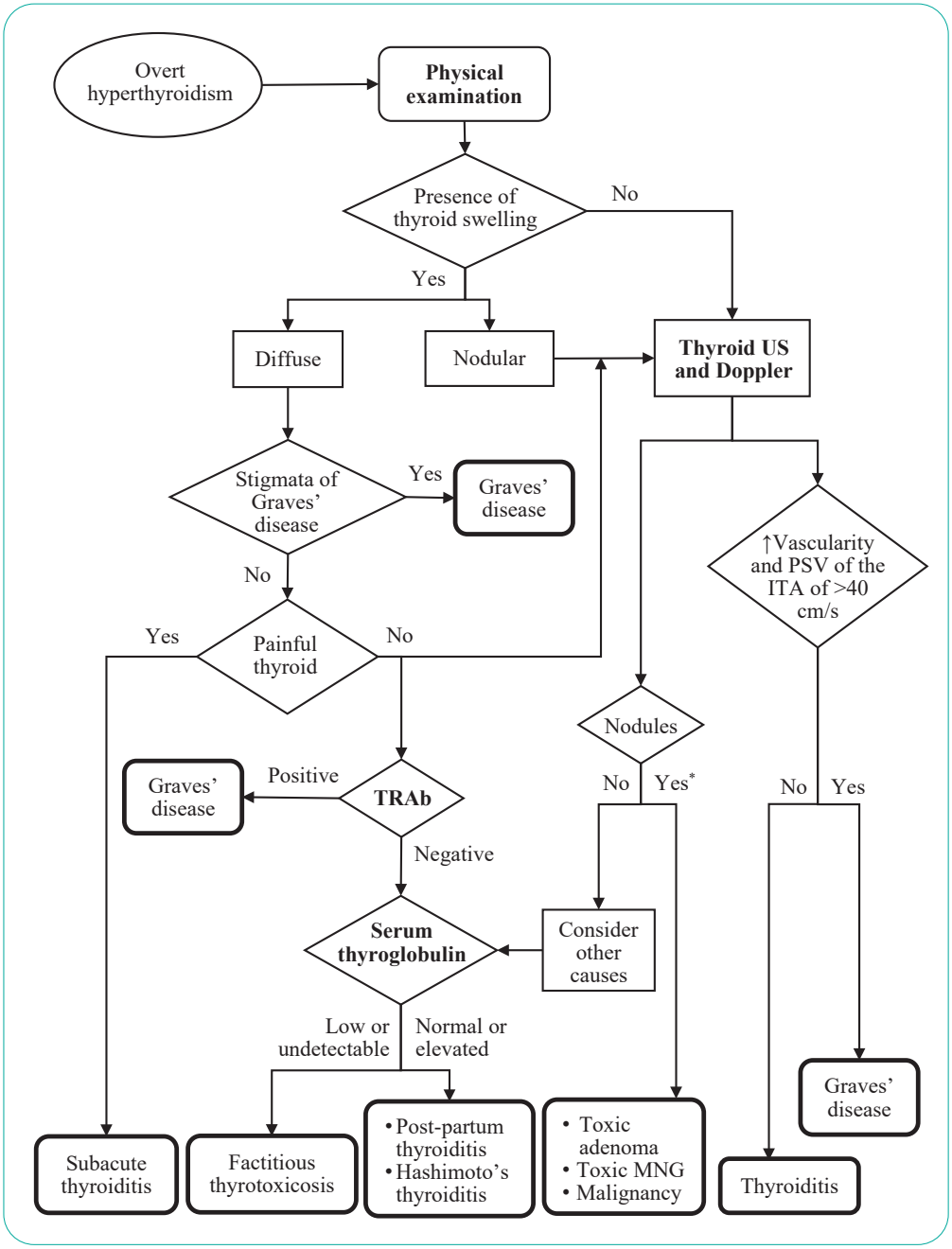

Figure 1. Algorithm for the aetiological diagnosis of hyperthyroidism in primary care settings.[2,6]

* US colour doppler can differentiate between benign and malignant thyroid nodules (through assessment of the vascularity pattern). Thyroid scintigraphy, if accessible, must be considered. US, ultrasound; PSV, peak systolic velocity; ITA, inferior thyroid artery; TRAb, thyroid-stimulating hormone receptor antibodies; MNG, multinodular goitre.

B. Best treatment option[2]

Patients with symptoms of thyrotoxicosis can be treated symptomatically with beta-blockers such as propranolol (10–40 mg every 8 h) or atenolol (25–100 mg daily). Other drugs can be used to treat adrenergic symptoms such as palpitation, heat intolerance, diaphoresis, tremors and stare.[8] Improvement in terms of irritability and exercise tolerance can also be seen with these medications.[9] Among beta-blockers, propranolol has the additional effect of stopping the conversion of inactive thyroxine to triiodothyronine (the active thyroid hormone) in the periphery of the body.[10] If beta-blockers are contraindicated or not suitable, non-dihydropyridine calcium-channel blockers such as verapamil and diltiazem are appropriate.

The choice of treatment depends on the underlying diagnosis, presence of a contraindication to a particular treatment modality, severity of hyperthyroidism and patient preference. In general, there are three modalities of treatment for hyperthyroidism, namely ATDs, radioactive iodine (RAI) and surgery (usually consisting of thyroidectomy) (**Table 2**). GD hyperthyroidism can be treated with any of these three modalities. Toxic MNG and TA should be treated with definitive treatment (RAI and thyroidectomy). Long-term ATD may be required in selected patients who refuse RAI therapy or have a contraindication to thyroidectomy.

Table 2. Choice of treatment for hyperthyroidism based on the clinical situation.[3]

Clinical situation	ATD	RAI	Surgery
Pregnancy	Yes !	No	↔ !
Comorbidities with increased surgical risk and/or limited life expectancy	↔	Yes	No
Inactive GO	↔	↔	↔
Active (moderate-to-severe) GO	Yes	Yes*	Yes
Liver disease	!	Yes	↔
Serious adverse effects of ATDs	No	Yes	↔
Previous operation or external irradiation on the neck	↔	Yes	!
Difficulty in accessing a high-volume thyroid surgeon	↔	Yes	!
High possibility of remission (particularly in women with mild disease, small goitre and negative or low TRAb titre)	Yes	↔	↔
History of periodic paralysis	↔	Yes	Yes
Right pulmonary hypertension or congestive cardiac failure	↔	Yes	!
Elderly patients with comorbidities	↔	↔	!
Confirmed or suspected thyroid cancer	-	No	Yes
Large thyroid goitre or nodules or with substernal or retrosternal extension or symptomatic compression	↔	-	Yes
Coexisting primary hyperparathyroidism requiring surgery	-	-	Yes

↔, acceptable choice; !, caution; -, not the first choice but may be acceptable depending on the clinical situation.

* When deciding for RAI therapy in GO, the disease activity and severity and other risk factors for GO progression need to be taken into consideration. ATD, Anti-thyroid drug; RAI, Radioactive iodine. GO, Graves' orbitopathy; TRAb, thyroid-stimulating hormone receptor antibody.

In primary care settings, hyperthyroidism is usually treated with ATDs to achieve the euthyroid biochemical state as soon and as carefully as possible. The medications that are generally available in primary care settings are carbimazole (CMZ) and propylthiouracil (PTU). CMZ is the preferred choice, as it can be taken once daily (OD) with less side effects. The initiation of CMZ therapy should be based on the level of fT4 relative to the upper limit (UL) of the reference range to decrease the risk of iatrogenic hypothyroidism, as recommended below.[3]

- fT4 level 1–1.5 times the UL: CMZ 5–10 mg OD
- fT4 level 1.5–2 times the UL: CMZ 10–20 mg OD
- fT4 level 2–3 times the UL: CMZ 30–40 mg OD (consider splitting to 20 mg twice daily)

PTU has a minor risk of severe liver injury in about 0.0001% of patients but can be prescribed in patients with a history of adverse effects of CMZ or in women who are currently pregnant or planning for pregnancy.6 PTU is usually given as a split dose two to three times daily. In the clinical setting, 1 mg of CMZ daily is equivalent to about 10 mg of PTU daily. The adverse effects of ATDs include common minor reactions such as pruritus or a limited, minor rash. Rarely, serious adverse effects such as agranulocytosis, liver injury or vasculitis can occur. Baseline full blood count (FBC) and liver function test (LFT) are recommended before commencing treatment. The FBC and LFT results are monitored only if patients are symptomatic (e.g. if they develop a sore throat or become jaundiced).[11]

C. Continuous assessment[2]

After initiation of ATDs, proper follow-up and adjustment of medication are important to achieve the goal of treatment. Initially, the treatment is guided by the levels of fT4 and

fT3, with the aim of titrating down the ATD dose until the levels decrease and eventually normalise. Initially, the fT4 and fT3 levels should be monitored at least every 2–6 weeks. Thereafter, the measurement of the fT3 level can be opted out in resource-limited settings (**Figure 2**). The serum TSH level may remain suppressed and low for several months after treatment is started and should not be used as a marker for adjusting the dosage of ATDs. ATDs should be continued for 12–18 months in a tapering dose and discontinued when the TSH level becomes normal at the time. Remission is achieved if the subsequent TFT results after 12 months of ATD withdrawal remain normal. **Figure 3** summarises the management of hyperthyroidism. Relapse of hyperthyroidism has been reported in approximately half of cases after stopping ATDs for 1–2 years. TSH receptor antibodies (TRAb) may be used to predict the risk of relapse and guide the definitive treatment of GD.

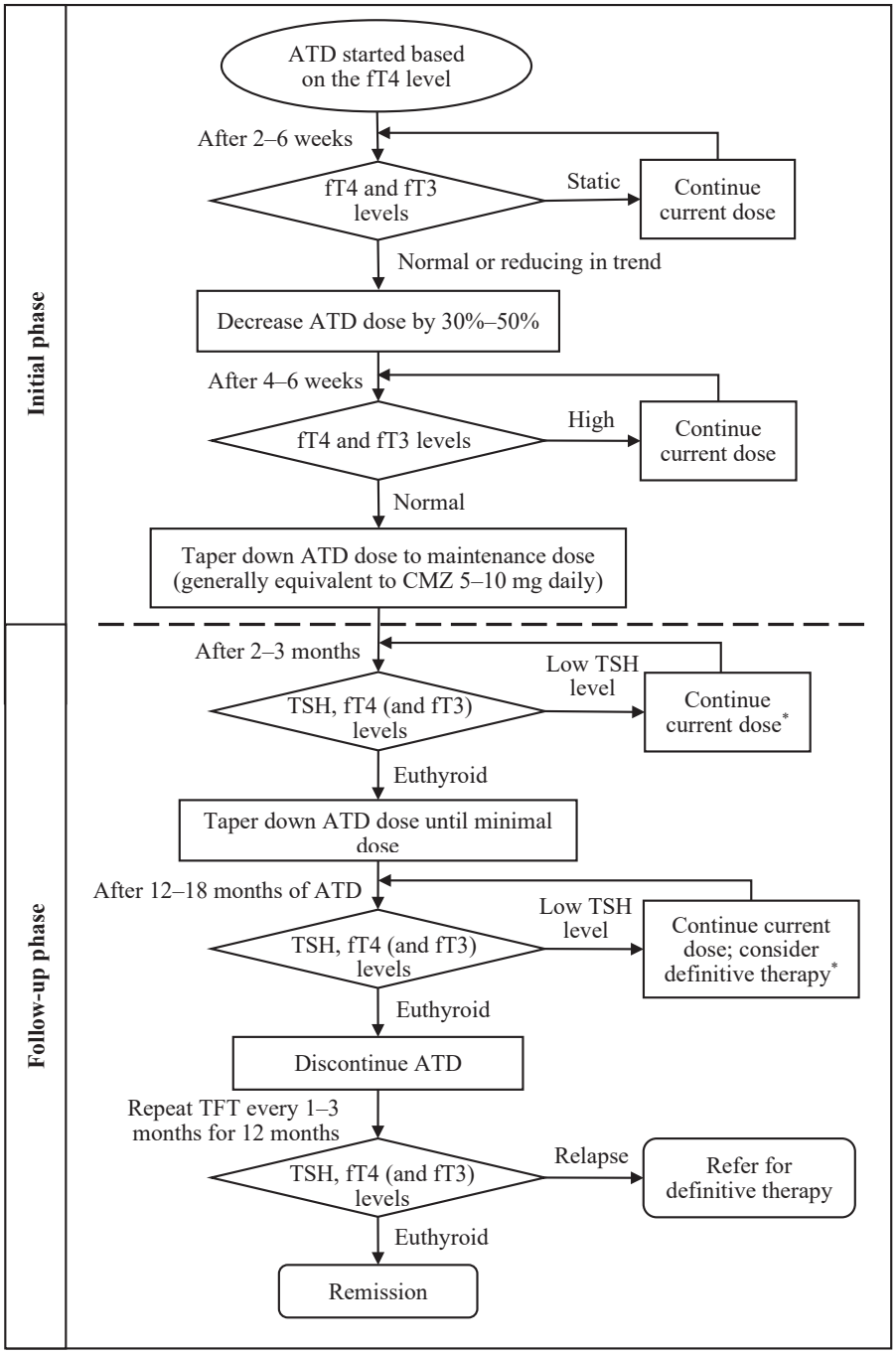

Figure 2. Algorithm for the management of hyperthyroidism with ATDs.
* Consider earlier monitoring of the TFT results.

MONITORING CHART FOR HYPERTHYROIDISM

Name:_____ **ID No.:** _____

Diagnosis: Graves' Disease/Hashimoto's Thyroiditis/Multinodular Goitre*/Toxic Adenoma*, Others:_____

Date of Test	Reference Range								
TSH									
T4									
T3									
Date of Review									
Weight									
Pulse Rate									
Medications									
Notes									

Baseline Investigations		
FBC	date	WCC: ANC: Hb: HCT: PLT:
LFT	date	ALT: AST: Total Bilirubin: ALP: Albumin:
Thyroid Ultrasound	date	
Thyroid Antibody	date	TRAb: (Reference:)
Others		

*Consider referral for further investigations and/or definitive therapy, see CPG on the Management of Thyroid Disorder.

Figure 3. Example of a monitoring chart for hyperthyroidism including serial test results, medication adjustments and investigations performed.

RAI and surgery should be considered in patients with toxic MNG or TA, failure to achieve the euthyroid biochemical state despite adequate treatment with ATD or relapse hyperthyroidism after an initial course of ATD treatment. Referral to a tertiary centre for hyperthyroidism can be considered in patients with comorbidities or in those who develop comorbidities. The pulse rate, blood pressure and temperature should be monitored during every doctor visit to assess for thyroid storm, atrial fibrillation, signs of fluid overload or cardiac failure.[11] The signs, symptoms and risk factors of osteoporosis (using the Fracture Risk Assessment Tool) and psychiatric disorders should also be assessed and managed accordingly.[11] Patients with active, moderate-to-severe or sight-threatening GO should be referred to ophthalmology for further assessment and management.

Subclinical hyperthyroidism[2]

SH is associated with an increased risk of atrial fibrillation, heart failure, and all-cause mortality, reduced bone marrow density and increased fracture risk. If the TSH level is low but with normal fT3 and fT4 levels, the TFT must be repeated within a few weeks in elderly patients aged more than 65 years, patients with risk factors or patients with a TSH level of <0.1 mU/L. For young patients without any risk factors or patients with a TSH level of 0.1–0.5 mU/L, the TFT can be repeated in 3–4 months. As in overt hyperthyroidism, the aetiological diagnosis should be identified. However, ATDs should be the first-line treatment for SH regardless of the aetiology. Treatment should be considered in patients with a substantially low TSH level, elderly patients aged more than 65 years or patients with comorbidities (**Table 3**). Treatment with RAI may be considered for persistent and worsening SH due to autonomous TA and MNG. If SH is treated, the management should follow the same basis as that for the treatment of overt hyperthyroidism. The major end point depends on the initial indication for treatment. Therefore, the decision for titration of treatment can be based on the symptoms of hyperthyroidism, bone density or reference range for the TSH level.

Table 3. Treatment and its indications in patients with subclinical hyperthyroidism.[2,3]

Clinical situation	TSH level of <0.1 mU/L	TSH level of 0.1–0.4 mU/L*
Age of ≥65 years	Treat	Consider treatment
Age of <65 years and presence of comorbidities (cardiac disease, osteoporosis, menopause but not on oestrogens or bisphosphonates and/or hyperthyroid symptoms)	Treat	Consider treatment
Age of <65 years and absence of symptoms	Consider treatment	Observe

*Depends on the lower limit of the reference range of the local laboratory.

Subacute thyroiditis[3]

Subacute thyroiditis is usually self-limiting and resolves spontaneously within 6 months. ATD and ablation are not typically used for subacute thyroiditis. In patients with subacute thyroiditis, including PPT and mild symptoms, beta-blockers and nonsteroidal anti-inflammatory drugs (NSAIDs) should be used for initial treatment. If patients fail to respond or present with moderate-to-severe pain and/or hyperthyroid symptoms, corticosteroids should be used instead of NSAIDs. Women with a history of PPT are at risk of developing permanent primary hypothyroidism within 5–10 years after the episode of PPT.[12] The TSH level should be monitored yearly in these women.

Hyperthyroidism in pregnancy[2,3,12,13]

A. Ascertain diagnosis

Clinical hyperthyroidism is confirmed when the TSH level is suppressed (<0.1 mU/L) or undetectable (<0.01 mU/L) along with an elevated fT4 or fT3 level.[2,12] The reference range for the TFT results for each trimester of pregnancy should be established by each institution using local data, as studies have shown discrepancies in the reference range for various populations.[2,13–17] However, when the trimester-specific fT4 level is not available, the non-pregnancy reference range can be used. If the TSH level is low (lower than the reference range), although this may be a feature of early pregnancy, thorough medical history-taking, physical examination and fT4-level assessment should be performed.[18] The approach for the aetiological diagnosis is similar to that in a non-pregnant adult, and the individual should be investigated accordingly. In the absence of a previous thyroid disease, stigmata of GD (goitre and ophthalmopathy) and TRAb, the diagnosis of gestational transient thyrotoxicosis (GTT) is more likely.

B. Best treatment option

If abnormal TFT results are attributed to GTT and/or hyperemesis gravidarum, the management includes supportive therapy and hospitalisation, if necessary, as GTT is usually self-limiting. Beta-blockers may be considered in these conditions if they are symptomatic, but ATD therapy should be avoided. Beta-blockers are frequently used in the early management of thyroiditis or during relapse and are weaned off once ATDs take effect, and there is clinical improvement, usually within 3 weeks.[18]

GD, TA and toxic MNG are usually treated with ATDs during pregnancy. However, if thyroidectomy is needed, it is best performed during the second trimester. If ATD therapy is needed, PTU is the preferred choice in the first trimester. After the first trimester, the decision to continue PTU or change to CMZ should be individualised and discussed with patients. However, CMZ is recommended when ATD therapy is initiated from the second trimester owing to the daily dosing and comparatively lesser side effects.[12] The FBC, LFT and TFT results should be monitored after 4 weeks of ATD initiation.[12]

C. Continuous assessment

For women with GTT, the TFT can be repeated at 14–18 weeks to confirm the normalisation of the fT4 level. In women treated with ATDs during pregnancy, the aim of treatment is to achieve the euthyroid state with the fT4 and fT3 levels at the upper end of the normal range for pregnancy. The lowest possible dose of ATDs (PTU: <150 mg/day; CMZ: <15 mg/day) should be used to avoid foetal hypothyroidism and goitre.18 The TFT should be conducted every 2–4 weeks following initiation of therapy. After initial stabilisation, the ATD dose is reduced as soon as possible (and is often stopped by the second trimester). Once the target is achieved, the TFT can be prolonged to 4–6 weeks. If needed, the same ATD therapy can be continued after the first trimester.[2,5] This is because ATDs are potentially teratogenic, and switching the ATD may lead to a period of inconsistent control.[2,5] If ATD therapy is stopped, the TFT should be conducted weekly during the first trimester and monthly thereafter.

TRAb monitoring during pregnancy is recommended to guide further management (**Table 4**). A TRAb level above three times the UL of the reference range is considered a risk factor of foetal hyperthyroidism, and foetal surveillance should be performed.[2] If the ATD-treated mother has high TRAb levels during late pregnancy, there is a risk of delayed neonatal hyperthyroidism, which warrants neonatal and postnatal monitoring.[3,5,13] If the mother has a history of RAI therapy or surgery for GD and has a high TRAb level, cautious evaluation of the foetus for hyperthyroidism is needed after 20 weeks of pregnancy, and ATD therapy may be started or adjusted accordingly.[2,13] If positive TRAb results become negative during pregnancy, reducing or stopping ATD therapy can be considered to avoid foetal hypothyroidism.[13]

Table 4. Management of pregnant mothers with Graves' disease and overt hyperthyroidism.[2,3,6,18]

Time of diagnosis	Current treatment	TRAb level measurement	Management
During pregnancy and not on treatment	During the first trimester	At diagnosis*	Begin PTU (150–400 mg/day)
	After the first trimester		Begin CMZ (15–40 mg/day)
Diagnosed and treated before pregnancy	Currently taking CMZ	At the first trimester or booking*	Switch to PTU or stop ATD (if on low dose of CMZ or PTU)
	In remission after stopping ATD	Not necessary	Perform the TFT to confirm euthyroidism
	History of RAI therapy or surgery	At the first trimester or booking*	Consider treatment if overt hyperthyroidism develops

* If the TRAb levels are low or undetectable, there is no need for further TRAb testing. If the levels are high, TRAb testing must be repeated at 18–22 weeks of gestation. If the levels are still high, and the mother is taking ATDs, TRAb testing must be repeated at 30–34 weeks of gestation.

After delivery, all babies born to mothers with hyperthyroidism should be referred to a paediatric team with the TFT conducted. In breastfeeding women, both CMZ and PTU can be prescribed with the lowest effective dose since the amount of transfer of these medications into the breast milk is small. Reliable contraception should be offered to women of childbearing age to avoid pregnancy until thyrotoxicosis is controlled.[6] If high doses of ATDs are required to achieve euthyroidism, definitive therapy is recommended before women become pregnant. Women with hyperthyroidism secondary to GD who desire pregnancy and whose condition is well controlled on CMZ should either be offered definitive therapy before they become pregnant or be switched to PTU before planning for pregnancy. Folic acid therapy should be offered to these women to reduce the risk of major birth defect, and follow-up should be conducted under a pre-pregnancy clinic.[12]

Thyroid storm[2,3]

Thyroid storm is a clinical diagnosis made in severely symptomatic patients with evidence of other systemic organ involvement. It is recommended to use the Burch–Wartofsky Point Scale in assessing patients for thyroid storm, as this scale is found to be more sensitive in detecting thyroid storm. The initial treatment approach of thyroid storm includes supportive treatment with airway maintenance, oxygen, intravenous fluids and cooling blankets, followed by heart rate control with beta-blockers or calcium-channel blockers.[8] Other measures include the initiation of ATD, inorganic iodide and corticosteroid therapies and treatment of the precipitating cause (**Table 5**). Patients should be referred to the hospital for further stabilisation and monitoring. All patients with thyroid storm should be planned for early definitive therapy either with RAI or thyroidectomy upon discharge.

Table 5. Initial management of thyroid storm.

Medication	Recommended dosage	Alternative
Propylthiouracil	500–1000 mg loading, then 250 mg every 4–6 h (may be given intravenously)	Carbimazole 60–80 mg/day
Propranolol	40–80 mg every 4 h	Esmolol infusion*
Hydrocortisone	100 mg every 8 h	Dexamethasone 2 mg every 6 h
Potassium iodide (saturated solution)*	Start only 1 h after ATD administration, 5 drops (0.25 mL or 250 mg) orally every 6 h	Lugol's solution*

* Usually not available in primary care settings

Conclusion

Hyperthyroidism is frequently seen in primary care settings. Early identification and diagnosis and appropriate management of hyperthyroidism are important to reduce the risk of complications. The simple ABC approach may be suitable and practical for managing hyperthyroidism in primary care settings.

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Author contributions

JJ conceptualised and designed the work, interpreted the relevant literature and drafted the manuscript. SFZ, GK, MZMI, MAMK, PK and TP interpreted the relevant literature and edited/revised the manuscript. All authors have read and approved the final version of the manuscript.

Review protocol registration

This review was registered in the National Medical Research Register (NMRR), Ministry of Health, Malaysia (NMRR ID-23-00141-FSV). The need for ethics approval was waived by the Medical Research Ethics Committee, as this study used publicly available data/information.

Conflicts of interest

None.

Funding

None.

How does this paper make a difference in general practice?

- Successful management of hyperthyroidism depends on ascertaining the correct diagnosis, employing the best treatment with continuous monitoring and facilitating follow-up to reduce the risk of complications.
- A practical approach to manage hyperthyroidism is vital in a resource-limited setting especially in primary care.
- An ABC approach may be appropriate and practical for managing hyperthyroidism in primary care settings.

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