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Amiodarone-Induced Thyroid Dysfunction

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Amiodarone is often used to treat both ventricular and atrial arrhythmias. Although its cardiac side effects are less frequent than those associated with other antiarrhythmics, it has potentially marked effects on thyroid physiology; it can cause both thyrotoxicosis and hypothyroidism. Nursing care of patients receiving amiodarone includes recognizing and monitoring for these potential complications and teaching patients preventive measures to avoid

these problems. In this article, we review the pharmacokinetics of amiodarone and the thyroid dysfunctions that can occur as side effects of its use.

Case Study

W.W. was a 68-year-old man whose medical history included coronary artery disease with coronary artery bypass graft surgery, unstable angina pectoris, hypertension, hypercholesterolemia, benign prostatic hypertrophy with transurethral resection of the prostate, and atrial fibrillation. His first episode of atrial fibrillation had occurred about 3 years earlier; at the time, he said he felt "poorly" and had fatigue and shortness of breath. Amiodarone therapy was started. Shortly thereafter, his heart rhythm converted to normal sinus rhythm.

Now he had come to the hospital because of recurrent chest pain. He was admitted to rule out myocardial infarction and atrial fibrillation. He

stated that he did not feel well, and before admission, he had had an episode of unexplained profuse diaphoresis, rapid heart beat, and shortness of breath. He also said that he had lost about 4.5 kg (10 lb) in the preceding 2 months even though his appetite had been good and he had eaten well. His medications before admission included amiodarone 200 mg/d, metoprolol, enalapril, simvastatin, aspirin, folic acid, and warfarin. Upon admission, a 12-lead electrocardiogram showed normal sinus rhythm at a heart rate of 95/min with no acute changes; blood pressure was 140/70 mm Hg. The findings on physical examination were unremarkable. Admission laboratory results were all within the normal reference ranges. Initially, no serum levels of thyroid hormones were determined. Myocardial infarction and heart failure were ruled out, and his preadmission medications were continued.

Because Mr W. had somewhat vague signs and symptoms and a history of cardiac disease, he underwent a coronary angiogram. On the basis of the results, he had stenting of a vein graft to the diagonal coronary artery and stenting of the right coronary artery. On the day after placement of the stents, he continued to have intermittent diaphoresis



* This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. List the indications for amiodarone
2. Differentiate the signs and symptoms of amiodarone-induced hypothyroidism and amiodarone-induced thyrotoxicosis
3. Discuss the management of amiodarone-induced hypothyroidism and amiodarone-induced thyrotoxicosis

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Table 1 Laboratory tests of thyroid function

Test	Purpose	Reference range for adults ^{1,2}
Total thyroxine (T ₄), nmol/L (µg/dL)	Measure serum level of circulating T ₄	64-154 (5-12)
Free thyroxine (FT ₄), pmol/L (ng/dL)	Measure serum level of unbound biologically active T ₄	10-30 (0.8-2.3)
Free thyroxine (FT ₄) assay or index, thyroid hormone binding ratio (THBR)	Estimate serum level of FT ₄	96-396
Free triiodothyronine (FT ₃), pmol/L (pg/dL)	Measure serum level of circulating T ₃	3.7-6.5 (240-420)
Triiodothyronine (T ₃) uptake, proportion of 1.0	Determine proportion of T ₃ that does not bind to thyroxine-binding globulin in test serum	0.25-0.33
Reverse T ₃ , nmol/L (ng/dL)	Evaluate tissue utilization of T ₄	0.14-0.54 (9-35)
Thyrotropin-releasing hormone (TRH) stimulation test, mIU/L	Differentiate primary disorders from disorders associated with pituitary dysfunction	Significant increase from ~1 to 8 at 20 minutes and return to normal by 120 minutes
T ₃ resin uptake (T ₃ RU), proportion of 1.0	Evaluate unoccupied binding sites on TBG	0.28-0.41
Thyroid-stimulating hormone (TSH, thyrotropin), mIU/L	Measure serum level of TSH	0.3-5.0
Thyroglobulin, µg/L	Monitor disease and response to therapy	Up to 30
24-Hour uptake of radioactive iodine, %	Estimate (indirect) of the production of thyroid hormones	2 hours: 1-13 6 hours: 2-25 24 hours: 10-45
Thyroxine-binding globulin (TBG), mg/L	Assess serum level of FT ₄	13-30
Antibodies to thyroglobulin, thyroid hemagglutination test	Detect quantity of these autoantibodies	Negative to 1:20
Microsomal antibodies to thyroid peroxidase (TPO)	Detect quantity of these autoantibodies	Negative
Antibodies to the receptor for TSH	Detect quantity of these autoantibodies	Negative
Thyrotropin-binding inhibitor immunoglobulin (TBII)	Detect quantity of these autoantibodies	Negative

and episodes of not feeling well. His cardiac troponin I level after the stenting procedure had increased slightly to 5.32 µg/L. Because the procedure involved the stenting of a vein graft, this increase was not unexpected and was suggestive of a small nontransmural myocardial infarction. After the stenting, he was in a junctional rhythm with a heart rate between 47/min and 55/min and his blood pressure was stable. Preadmission medications were continued except for metoprolol because of his heart rhythm and rate.

On the next day, because of his persistent vague signs and symptoms, blood samples were obtained to

determine serum levels of thyroid-stimulating hormone (thyrotropin or TSH) and free thyroxin (FT₄). (See Table 1 for reference values for thyroid testing in adults.) His TSH level was low at less than 0.01 mIU/L, and his FT₄ level was elevated at 187 pmol/L (14.5 ng/dL), supporting a diagnosis of amiodarone-induced thyrotoxicosis (AIT). On that same day, atrial fibrillation developed with a ventricular rate of about 90/min. Blood pressure remained stable. Amiodarone was discontinued, and an endocrinology consultation was obtained.

On day 3 after the stenting, although 12-lead electrocardiography continued to show atrial fibrillation

with a ventricular rate in the 90s, W.W. started to feel better. His fatigue and diaphoresis abated, and he asked to be discharged. The cardiologist and the endocrinologist agreed to release him the following morning. His medications at the time of discharge were warfarin daily to maintain an international normalized ratio between 2.0 and 2.5, folic acid, aspirin, enalapril, simvastatin, clopidogrel, and nitroglycerin 0.4 mg as needed. Use of extended-release metoprolol was reinstated at a lower dose of 25 mg/d in hopes of controlling the ventricular rate while preventing episodes of bradycardia and/or junctional rhythm.

On the day of discharge, the level of free triiodothyronine (FT₃) was elevated at 7.8 pmol/L (504 pg/dL), the level of FT₄ was elevated at 32 pmol/L (2.5 ng/dL), the level of microsomal antibodies to thyroid peroxidase was elevated at less than 5 IU, and the level of antibodies to thyroglobulin was normal at less than 20 IU. Follow-up appointments were arranged with the cardiologist and the endocrinologist.

Approximately 5 hours after discharge, W.W. returned to the emergency department because of a high fever, flushing, diaphoresis, chills, heat intolerance, anxiety, and rapid heart rate. His temperature was 39.4°C. Physical examination revealed an irregular heart rate, flushing, and hyperreflexia. A 12-lead electrocardiogram showed atrial fibrillation at a ventricular rate of 130/min to 140/min. In order to convert the atrial fibrillation and/or control the ventricular rate, a total of 15 mg of intravenous metoprolol was administered over 15 minutes, resulting in 3 episodes of sinus pauses, each lasting approximately 8 seconds, and an intermittent junctional rhythm.

Although his blood pressure was stable, W.W. was admitted to the coronary care unit for further monitoring and observation. The medications he was taking at the time of discharge were continued, except for metoprolol, which was discontinued because of his response to the intravenous dose of the drug. A temporary pacemaker was placed at his bedside. The cardiologist thought that the junctional rhythm and sinus pauses might be due to sick sinus syndrome and anticipated that W.W. might need a permanent pacemaker if the signs and symptoms persisted. The endocri-

nologist revised the original diagnosis of AIT to include amiodarone-induced thyroiditis and ordered propylthiouracil 100 mg by mouth 3 times daily and prednisone 40 mg/d.

During the next few days, W.W. continued to stay in the coronary care unit. His vital signs remained stable. His face and neck continued to appear flushed. He intermittently experienced episodes of severe flushing, diaphoresis, anxiousness, heat intolerance, and mild tachycardia, and at times, he was fidgety. On day 3 in the coronary care unit, no further episodes of sinus pauses, sinus arrests, or junctional rhythms occurred. On the morning of day 4, the monitor indicated atrial fibrillation with a ventricular rate of about 150/min. Blood pressure remained stable. The rapid atrial fibrillation was accompanied by increased anxiety, but no other new signs or symptoms were noted. Lorazepam was started, and a slightly higher dose of metoprolol (25 mg every 8 hours) was reinstated. The serum level of FT₃ was normal at 5.2 pmol/L (334 pg/dL), the serum level of FT₄ was slightly elevated at 31 pmol/L (2.4 ng/dL), and the serum level of TSH was low at 0.01 mIU/L. Mr. W. continued to improve.

On day 5 in the coronary care unit, after he had been symptom-free for 24 hours, W.W. was transferred to a step-down unit. At that time, the dosage of metoprolol was increased to 50 mg every 12 hours, and digoxin 0.125 mg/d was started. He continued to do well and was discharged from the hospital on day 6. At the time of discharge, he was taking the following medications: aspirin, clopidogrel, propylthiouracil 100 mg 3 times daily, prednisone 20 mg every other day, metoprolol twice a day, digoxin, simvastatin, enalapril, war-

farin, and folic acid. At discharge, he remained in atrial fibrillation at a controlled ventricular rate. The final diagnosis was AIT without the destructive type of thyroiditis.

Mr W. has done well since discharge. By 4 weeks later, the results of all thyroid tests were normal, and they have continued to remain normal. Because amiodarone has such a long half-life, propylthiouracil was continued for another 4 months to prevent a relapse.

Seven months later, W.W. was taking prednisone 20 mg twice weekly and anticipated tapering it off completely. He continued to have atrial fibrillation and continued to complain of fatigue and lethargy. Subsequently, he was readmitted to the hospital for 3 days for the initiation of dofetilide therapy. During that admission, he was successfully cardioverted, became asymptomatic, and was discharged from the hospital.

Amiodarone

Amiodarone is effective in managing arrhythmias predominantly because of its class 3 (Vaughn-Williams classification) antiarrhythmic effects on the cell membrane. It suppresses potassium inflow and prolongs the cardiac action potential, thereby prolonging repolarization. In addition, amiodarone has class I, class II, and class IV activity; it suppresses sodium channels, causes non-selective beta-blockade, and blocks calcium channels, respectively. These class effects contribute to its ability to slow the heart rate and inhibit the atrioventricular node. In addition, amiodarone has some coronary and peripheral vasodilator properties.^{3,4}

Table 2 indicates the multiple uses and contraindications for use of

Table 2 Uses and contraindications for amiodarone therapy^{3,4}

Uses

- First-line agent in the treatment of ventricular fibrillation and pulseless ventricular tachycardia according to the Advanced Cardiopulmonary Life Support algorithms of the American Heart Association
- Treatment of stable ventricular tachycardia, both monomorphic and polymorphic
- Prevention of arrhythmias, especially in patients with left ventricular systolic dysfunction
- Treatment of refractory sustained atrial fibrillation
- Treatment of symptomatic atrial flutter
- Prevention of recurrence of paroxysmal atrial fibrillation and atrial flutter
- As an adjunct to the electrical cardioversion of supraventricular tachycardias

Contraindications

- Known hypersensitivity to amiodarone
- Pregnancy
- Lactation
- Severe sinus node dysfunction
- Marked sinus bradycardia
- Syncope
- Second- or third-degree heart block
- Cardiogenic shock

amiodarone. The medication is also associated with many potential drug-drug interactions because it inhibits most cytochrome P450 enzymes, which are involved in drug breakdown. Table 3 is a list of drugs with known interactions with amiodarone.

Amiodarone has numerous side effects. In addition to the potential adverse effect on the thyroid, it may cause other markedly adverse effects (Table 4). These effects strongly coincide with dosage and duration of therapy.

The elimination half-life of amiodarone and its metabolite, desethylamiodarone, are quite long. The half-life for amiodarone is approximately 6 to 7 weeks, and desethylamiodarone stays in the body for 8 to 9 weeks.⁶ Therefore, any adverse effect that occurs can take a long time to resolve.

Effects of Amiodarone on Thyroid Physiology

The amiodarone molecule is structurally similar to T₄ and contains 2

atoms of iodine per molecule.⁷ Amiodarone is approximately 37% iodine by weight; hence each 200-mg tablet contains about 75 mg of iodine, of which approximately 10% is released as free iodine after deiod-

ination. In comparison, the reference daily intake of iodine for adults is 150 µg.

An interesting phenomenon of the effect of amiodarone-induced thyroid dysfunction is that the effect may range from abnormal results on thyroid function studies without overt dysfunction to symptomatic thyrotoxicosis or hypothyroidism. (For a review of normal thyroid function, see Figure 1.) Signs and symptoms of alteration in thyroid function take longer to develop than do changes in serum levels of thyroid hormones. Figure 2 shows the initial effect of amiodarone on thyroid function, the Wolff-Chaikoff effect.

Typically, alterations in the concentrations of circulating thyroid hormones occur within days after treatment with amiodarone is started, and the alterations may change during the course of therapy. Almost immediately, an increase occurs in the serum levels of total T₄ and FT₄.

Table 3 Drug interactions with amiodarone

Drugs that may prolong the QTc interval

- Antiarrhythmic agents, including quinidine, procainamide, sotalol
- Antipsychotic agents, including haloperidol, risperidone, quetiapine
- Antinausea/antivomiting agents such as dolasetron, droperidol
- Azole antifungal agents
- Erythromycin
- Fluoroquinolones
- Halothane
- Macrolides
- Moxifloxacin
- Phenothiazines
- Tricyclic antidepressants
- Thiazide diuretics

Drugs that affect the metabolism of amiodarone

- Cimetidine
- HIV protease inhibitors
- Rifampin

Drugs whose metabolism is affected by amiodarone

- Inhibitors of 3-hydroxy-3-methylglutaryl coenzyme A (commonly called statins)
- Phenytoin
- Warfarin
- Digoxin

Table 4 Side effects of amiodarone

Cardiac side effects

- Hypotension
- Sinus bradycardia
- Heart block
- QRS and QT prolongation
- Depression of contractility (rare, but can occur in long-term therapy)

Noncardiac adverse effects (more common than cardiac side effects and related to dose and length of therapy)

- Hypothyroidism
- Thyrotoxicosis
- Pneumonitis, which can progress to pulmonary fibrosis; occurs in 10%-17% of patients treated with doses of 400 mg/d³
- Central nervous system side effects, such as proximal muscle weakness, peripheral neuropathy, and generalized neural symptoms; occur at a rate of 0.6%⁵
- Corneal microdeposits; develop in nearly all patients treated with amiodarone for longer than 6 months; however, signs and symptoms and impaired vision are rare
- Increased levels of liver enzymes; occur in 10%-20% of patients and usually respond to discontinuation or a reduction in dosage
- Bluish-tinged skin discoloration and photosensitivity; occur in >10% of patients after about 18 months of therapy; skin discoloration usually slowly reversible but can be irreversible
- Gastrointestinal signs and symptoms; most likely during the initiation of therapy^{3,5}

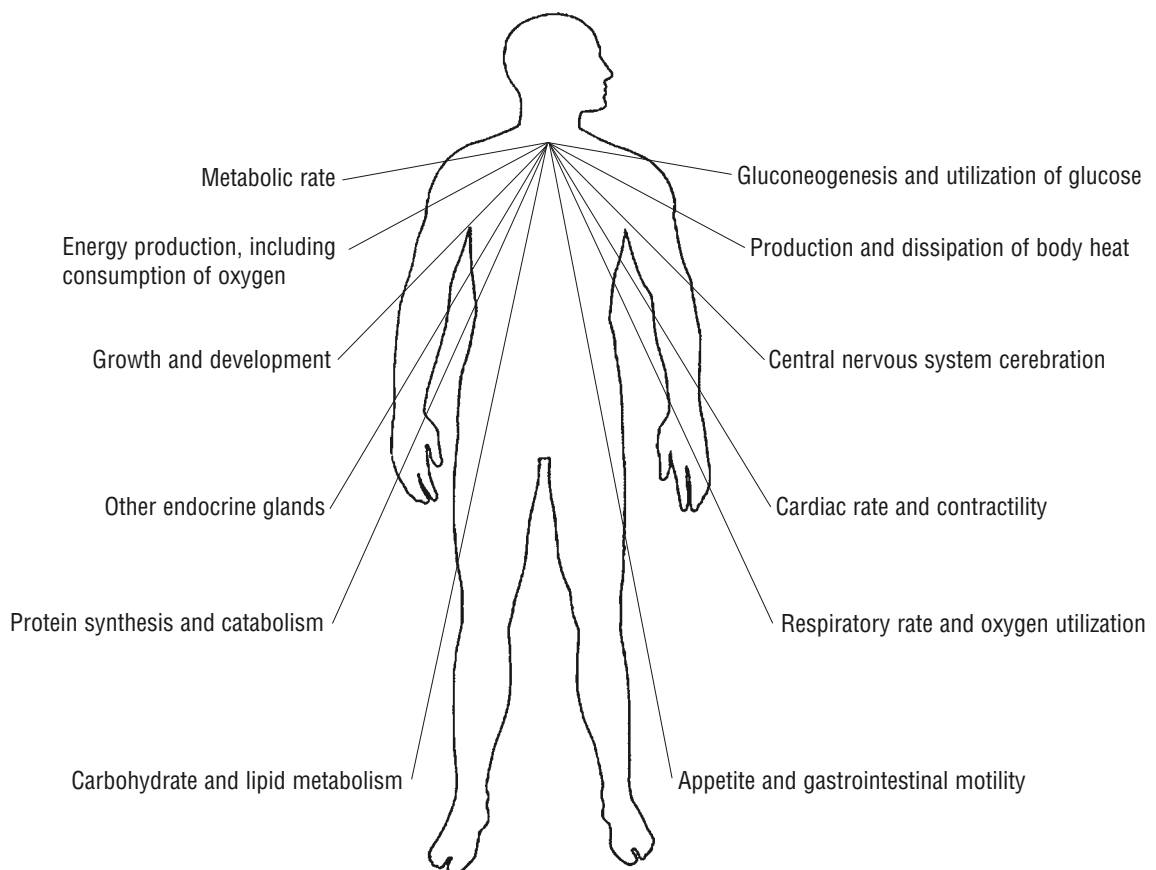
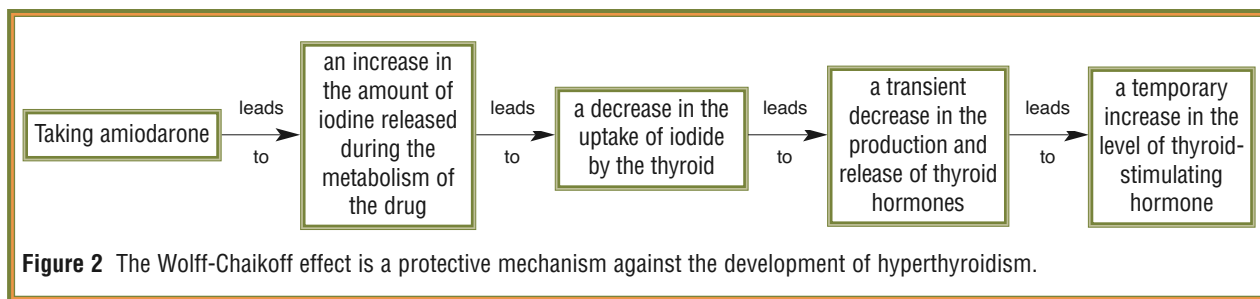


Figure 1 The highly vascular thyroid gland produces 3 hormones: triiodothyronine (T_3), thyroxine (T_4), and calcitonin, which is not affected by amiodarone. T_3 accounts for approximately 95% of the total hormone production by the thyroid. T_4 is the weaker hormone; it maintains the steady state and is converted to T_3 in the target tissues. T_3 is 5 times more potent than T_4 ; it also enters cells more easily and works much more rapidly. In the target tissues, normal thyroid function keeps these cellular activities in balance.



In fact, T₄ levels can increase by as much as 40%. This increase is an expected finding and does not in itself denote hyperthyroidism.⁷

Serum levels of T₃ decrease within days after the start of therapy because amiodarone inhibits the conversion of T₄ to T₃. Total T₃ levels are typically at the lower end of the normal reference range and continue to be low despite continued amiodarone therapy.⁸

TSH levels vary in response to amiodarone. Shortly after therapy is started, TSH levels increase because of the initial suppression of the thyroid hormones and the reduced negative feedback. TSH levels often return to normal with continuing amiodarone therapy.⁸

Despite changes in the results of thyroid function tests, most patients treated with amiodarone remain euthyroid. The incidence of amiodarone-induced thyroid dysfunction ranges from 14% to 18% and is related to environmental iodine adequacy versus iodine deficiency.^{9,10}

Amiodarone-Induced Hypothyroidism

Amiodarone-induced hypothyroidism (AIH) occurs more often in iodine-sufficient regions than in iodine-deficient regions and affects female patients and the elderly more often than it does male patients and younger patients.^{6,8,10} This difference

is partly due to the higher prevalence of preexisting autoimmune thyroid disease among women and the elderly. Women with preexisting antibodies to thyroid antigens who take amiodarone have a relative risk of 13.5 for the development of hypothyroidism.^{6,8} However, any patient with preexisting Hashimoto thyroiditis who receives amiodarone therapy is at risk for AIH. Conversely, AIH may also develop in patients with seemingly normal thyroid glands.^{6,8,10} Occasionally, spontaneous remission of AIH may occur, especially in patients without autoimmune thyroid disease.^{6,7}

Diagnostic Indicators

Similar to spontaneous hypothyroidism, AIH often is associated with vague clinical features (Table 5). Goiter rarely occurs.¹¹ Diagnosis of AIH is confirmed by laboratory tests that show decreases in the serum levels of total T₄ and FT₄ and an increase in the serum level of TSH.

Management

Management of patients with AIH depends on the need for amiodarone therapy. If the clinical manifestations are mild, and the patient is already on thyroid hormone replacement therapy, the dosage of levothyroxine

Table 5 Clinical manifestations of amiodarone-induced thyroid disturbances*

Amiodarone-induced hypothyroidism
Fatigue, lethargy
Diastolic hypertension
Bradycardia
Dyspnea
Cold intolerance
Dry skin, brittle hair and nails
Constipation
Weight gain
Delayed deep tendon reflexes, muscle weakness
Decreased appetite
Amiodarone-induced thyrotoxicosis
Supraventricular tachycardia, palpitations
Weight loss
Sweating, warm skin, heat intolerance
Low-grade fever
Muscle weakness
Fatigue, difficulty sleeping
Tremor
Occasional tenderness in the thyroid region
Mood swings

*Are consistent with thyroid diseases of other causes.

may be increased.^{6,8} When amiodarone is necessary for the treatment of a potentially life-threatening arrhythmia, thyroid hormone replacement therapy may be instituted. The initial dosage of levothyroxine is 25 to 50 µg/d. The dosage can be increased gradually until the signs and symptoms of AIH have resolved and the target serum level of TSH is normalized in the upper half of the normal reference range.⁷

If amiodarone is discontinued, the signs and symptoms of AIH may continue and the results of thyroid functions tests may not become normal for some time because of the drug's long half-life. Patients with underlying thyroid abnormalities and Hashimoto thyroiditis are less likely to achieve remission than are patients without these conditions. A short course (10-30 days) of treatment with potassium perchlorate (0.75-1 g/d), an ionic inhibitor, will hasten recovery. Potassium perchlorate inhibits thyroidal uptake of iodide and minimizes the inhibitory effect of intrathyroidal iodine content. Use of potassium perchlorate is limited primarily because of the potentially serious side effects, notably aplastic anemia and agranulocytosis.^{5-8,10,12} Potassium perchlorate may also be difficult for pharmacies to obtain.

Amiodarone-Induced Thyrotoxicosis

AIT is prevalent in iodine-deficient areas and predominantly occurs in men; the reported male-to-female ratio is 3:1.^{6,7,10} AIT may develop any time during amiodarone therapy or, similar to the situation with AIH, after the drug is discontinued. The development of AIH after discontinuation of amiodarone is a consequence of the drug, its metabolite storage, and its

slow release. AIT may develop in patients with or without preexisting thyroid abnormalities. The pathogenesis of the disease is complex and is not completely understood.

AIT in patients with preexisting thyroid abnormalities such as diffuse or nodular goiter or latent Graves' disease is called AIT type I. It is thought that the increased burden of iodine overload on the already abnormal thyroid gland leads to excessive synthesis and release of thyroid hormones.

AIT in patients with an apparently normal thyroid gland is called AIT type II. AIT type II may be related to destructive inflammatory thyroiditis caused by the increased iodine load. In vitro studies^{6-8,10} have shown that amiodarone and desethylamiodarone have cytotoxic effects on the thyroid cells, causing glandular damage and the subsequent leak of preformed thyroid hormones into the systemic circulation. Last, mixed forms of type I and II AIT can occur in which different features of the 2 types may coexist.

Diagnostic Indicators

Diagnosing AIT and differentiating between type I and type II can be challenging; the classical signs and symptoms of thyrotoxicosis may be absent because of the antiadrenergic activity of amiodarone and impaired conversion of T₄ to T₃. Common signs and symptoms that may occur with AIT are included in Table 5.

Laboratory testing in AIT reveals marked increases in the serum level of FT₄ (or high total T₄ and free thyroxine index); the serum level of FT₃ is elevated or normal in either type. The serum level of TSH is decreased or suppressed in both types.^{6-8,10}

Additional testing may be needed to differentiate between type I and

type 2. In type I, the serum level of interleukin-6, an inflammatory mediator, is normal or slightly elevated. In contrast, in type II, the serum level of interleukin-6 is markedly increased.^{7,8} In type I AIT, 24-hour uptake of radioactive iodine by the thyroid gland is normal to high. Conversely, in type II, uptake is low or none occurs. Color flow Doppler sonography shows increased vascularity in type I and normal vascularity in type II.^{6-8,10}

Management

Unlike management of patients with AIH, which is relatively easy to treat with thyroid hormone replacement therapy, the management of patients with AIT is more challenging and must be tailored to each patient and the type of AIT. In AIT type I, thionamides, either methimazole 40 to 60 mg or propylthiouracil 600 to 800 mg, are administered. Both drugs shut down the synthesis of T₃ and T₄. However, propylthiouracil may be preferred because it impairs peripheral conversion of T₄ to T₃ in the kidney and liver.^{4,6,10} In moderate to severe AIT type I, for more effective control, a short course (4-6 weeks) of potassium perchlorate may be added to treatment with either methimazole or propylthiouracil. In one study,⁷ potassium perchlorate was discontinued, and lithium carbonate (900-1350 mg) was administered along with thionamides. Lithium carbonate reduces the amount of thyroid hormones released from the gland and decreases thyroidal uptake of iodine. The result is a quicker return to euthyroidism.^{7,10,11}

In AIT type II, thionamides are not effective because hormone synthesis is not the issue. Simply discon-

tinuing amiodarone may reestablish euthyroidism. However, the return to normal thyroid function may take time because of the drug's long half-life. A long course of steroid therapy is another option. Steroids have membrane-stabilizing and anti-inflammatory effects and may be beneficial in inflammatory or destructive thyroiditis. The usual recommended dose is prednisone 15 to 80 mg/d for 7 to 12 weeks or dexamethasone 3 to 6 mg/d for 7 to 12 weeks.^{7,10,11}

In mixed forms of AIT, triple therapy with thionamide, potassium perchlorate, and prednisone may be used. When pharmacological treatments for AIT type II or the mixed form are unsuccessful and a patient requires continued amiodarone therapy, a total or near total thyroidectomy may be an alternative. This type of treatment allows continued use of amiodarone.^{7,10,11}

Nursing Responsibilities

Nursing responsibilities associated with the administration of amiodarone focus on monitoring the effects of the drug and teaching patients and their families. Nurses should be vigilant in checking for drug-drug interactions before amiodarone therapy is started and before the initiation of any additional medications for patients who are already taking amiodarone. Findings on electrocardiograms and QT/QTc intervals should be evaluated on a regular basis.

Nurses should also be vigilant in checking for drug-drug interactions when patients are receiving antithyroid medications. Aspirin is not recommended in thyrotoxic crisis, because it displaces thyroid hormones from the carrier proteins, thus increasing the levels of free hormones.¹

Patients receiving antithyroid medication are at risk for agranulocytosis. The person who prescribed the medication should be notified if fever or sore throat develops. Patients taking antithyroid medications at the time of discharge should receive education about calling their physician if either fever or sore throat develops.

Patients taking amiodarone at the time of discharge require significant teaching. Patients and their families need to know possible early indications of thyroid dysfunction and to report those signs and symptoms to a healthcare provider. Patients and their families should be taught about the signs and symptoms of hyperthyroidism, including increased temperature, warm skin, heat intolerance, palpitations, fast heart rate, diarrhea, weight loss, fatigue, mood swings, and difficulty sleeping. Patients and their families should also be educated about the signs and symptoms of hypothyroidism, including shortness of breath, fluid retention, weight gain, decreased appetite, constipation, dry skin, muscle weakness, cold intolerance, and brittle hair and nails. The importance of not taking any additional medications without first talking with the person who prescribed amiodarone should be stressed. Additionally, patients should avoid drinking grapefruit juice because it inhibits the metabolism of amiodarone.

Regularly scheduled examinations of organs often affected by the side effects of long-term amiodarone therapy are recommended. Education of patients and their families should emphasize the importance of regular thyroid, lung, and eye test-

ing. Commonly, patients undergo thyroid tests every 6 months.

Conclusion

Thyroid dysfunction is a well-known side effect of amiodarone. AIT or AIH may develop in patients taking amiodarone long term. Nurses provide optimal care for patients taking amiodarone by teaching the patients about early detection of side effects and by having a high index of suspicion for amiodarone-induced thyroid dysfunction in such patients.

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CE Test Test ID C0632: Amiodarone-Induced Thyroid Dysfunction

Learning objectives: 1. List the indications for amiodarone 2. Differentiate the signs and symptoms of amiodarone-induced hypothyroidism and amiodarone-induced thyrotoxicosis 3. Discuss the management of amiodarone-induced hypothyroidism and amiodarone-induced thyrotoxicosis

1. Amiodarone is *not* indicated for which of the following dysrhythmias?

- a. Atrial fibrillation
- b. Junctional tachycardia
- c. Ventricular tachycardia
- d. Ventricular fibrillation

2. Which of the following is an adverse effect of amiodarone?

- a. Hypotension
- b. QT shortening
- c. Sinus tachycardia
- d. Accessory pathway stimulation

3. Which of the following is the elimination half-life of amiodarone?

- a. 24 to 48 hours
- b. 72 to 96 hours
- c. 3 to 4 weeks
- d. 6 to 7 weeks

4. Which of the following amounts of iodine is present in each 200-mg tablet of amiodarone?

- a. 25 mg
- b. 50 mg
- c. 75 mg
- d. 100 mg

5. Amiodarone-induced hypothyroidism (AIH) occurs more often in which group?

- a. Male patients
- b. Female patients
- c. Young patients
- d. Obese patients

6. Which of the following is *not* a clinical manifestation of AIH?

- a. Cold intolerance
- b. Bradycardia
- c. Constipation
- d. Low-grade fever

7. The diagnosis of AIH is confirmed by which of the following laboratory tests?

- a. Decrease in total T₄
- b. Decrease in thyroid-stimulating hormone
- c. Increase in FT₄
- d. Increase in FT₃

8. Which of the following initial dosages of levothyroxine is indicated for the treatment of AIH?

- a. 0.25 µg daily
- b. 0.25 mg daily
- c. 25 µg daily
- d. 25 mg daily

9. Amiodarone-induced thyrotoxicosis (AIT) occurs more often in which group?

- a. Young patients
- b. Male patients
- c. Obese patients
- d. Female patients

10. Which of the following is *not* a clinical manifestation of AIT?

- a. Supraventricular tachycardia
- b. Heat intolerance
- c. Diastolic hypertension
- d. Weight loss

11. The diagnosis of AIT is confirmed by which of the following laboratory tests?

- a. Increase in FT₄
- b. Increase in thyroid-stimulating hormone
- c. Decrease in FT₃
- d. Decrease in total T₄

12. Which of the following medications may be administered for AIT type II?

- a. Propylthiouracil
- b. Methimazole
- c. Potassium perchlorate
- d. Prednisone

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

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