Thyroid-S instructions

https://attentive.ru/item/thyroid-s-capsuly.html

Name and Strength of Active Ingredient

Each film-coated tablet contains thyroid extract 60 mg.

Product Description

Light brown round biconvex film coated tablet

Pharmacodynamics/Pharmacokinetics

Pharmacodynamics

Thyroid-S formula composition

Propylthiouracil 50 mg tablet
Methimazole (thiamazole) 5 mg tablet
L-Thyroxine sodium 50 mcg / 100 mcg tablet
Thyroid extract 60 mg tablet

Pharmacological action

Thyroid agents are natural preparations containing tetraiodothyronine (thyroxine, T) and triiodothyronine (T). The principle pharmacologic effect of exogenous thyroid hormones is to increase the metabolic rate of body tissue. Thyroid hormones affect protein and carbohydrate metabolism, promoting gluconeogenesis. increasing the utilization and mobilization of glycogen stores, stimulating protein synthesis, affecting lipid metabolism by decreasing hepatic and serum cholesterol concentrations. Thyroid hormones are also involved in the regulation of cell growth and differentiation. The hormones aid in the development of the brain and CNS and are involved with somatropin in the development of bones and teeth and in broad aspect of growth.

Thyroid hormones also exhibit a cardiostimulatory effect which may be the result of a direct action on the heart. Thyroid hormones also may increase the sensitivity of the heart to catecholamines and/ or increase the number of myocardial (3-adrenergic receptors. Thyroid hormones increase cardiac output, secondary to increased peripheral oxygen consumption. Thyroid hormones may increase renal blood flow and glomerular filtration rate in hypothyroid patients, resulting in a diuresis within 24 hours following administration.

Thyroid hormones will reverse the signs and symptoms of hypothyroidism and myxedema: in hypothyroid children, the hormones increase epiphyseal growth and bone ossification.

Pharmacokinetics

Absorption: Levothyroxine sodium is variably absorbed from the gastrointestinal tract (40-80%) following oral administration. The absorption is increased in fasting state (79-81%) and may be decreased by age and specific foods and drugs. Time to peak concentration in serum is 2-4 hours. Liothyronine sodium is almost completely absorbed from the gastrointestinal tract (about 95%) following oral administration. The absorption of hormones contained in the natural preparations is similar to that of the synthetic hormones. Thyroxine apparently undergoes enterohepatic circulation.

Levothyroxine sodium, thyroid, and thyroglobulin have a much slower onset and longer duration of action than liothyronine sodium. The full effects of levothyroxine sodium, thyroid, and thyroglobulin do not occur for 1-3 weeks following initiation of oral therapy, and effects are maintained for a similar period of time following discontinuance of the drugs.

Distribution: Thyroxine is distributed into most body tissues and fluids with highest concentrations in the liver and kidneys. Thyroid hormones do not readily cross the placenta and minimal amounts of thyroid hormones are distributed into milk.

Thyroxine and triiodothyronine are more than 99% bound to serum proteins, principally thyronine-binding globulin (thyroxine-binding globulin. TBG) and transthyretin (thyroxine-binding prealbumin, TBPA) and to a small extent albumin, whose capacities and affinities for the hormones vary. Thyroxine is more extensively and firmly bound than is triiodothyronine. The high affinity of thyroxine for TBG and TBPA is responsible for thyroxine's high serum concentration and slow metabolic clearance. Certain drugs and various pathologic and physiologic conditions can alter the binding of thyroid hormones to serum proteins and/ or the concentrations of the serum proteins that bind the hormones: these effects must be considered when interpreting the results of thyroid function tests.

Metabolism: About 85 % of thyroxine undergoes peripheral monodeiodination to form reverse triiodothyronine (reverse T3, rT3), which is calorigenically inactive. The metabolic fate of triiodothyronine is not clearly established. Triiodothyronine and reverse triiodothyronine undergo peripheral monodeiodination to form 3,3'- diiodothyronine.

Elimination: The usual plasma half-lives of thyroxine and triiodothyronine are approximately 6-8 and 1-2 days, respectively. The plasma half-lives of thyroxine and triiodothyronine are decreased in patients with hyperthyroidism and increased in those with hypothyroidism. Thyroxine is conjugated with glucuronic and sulfuric acids in the liver and distributed into bile; a portion is then hydrolyzed in the intestine and reabsorbed, and a portion reached the colon unchanged, where it is then hydrolyzed and eliminated unchanged in the feces. About 20-40% of thyroxine is eliminated in feces.

Indication

Use in the treatment of hypothyroidism

Recommended Dose

Dosage of thyroid agents must be carefully adjusted according to individual requirements and response. The age and general physical condition of the patient and the severity and duration of hypothyroid symptoms determine the initial dosage and the rate at which dosage may be increased to the eventual maintenance dosage.

Dosage should be initiated at a lower level in geriatric patients: in patients with long-standing disease, other endocrinopathies, or functional or ECG evidence of cardiovascular disease: and in patients with severe hypothyroidism. Adjustment of thyroid replacement therapy should be determined mainly by the patient's clinical response and confirmed by appropriate laboratory tests.

Initial dose for hypothyroid states, 60 to 300 mg daily. Usual maintenance dose is 30 to 125 mg daily. Thyroid extract 60 mg is usually considered equivalent to thyroglobulin 60 mg. levothyroxine sodium (T) 0.1 mg or liothyronine sodium (T) 25 |jg.

Laboratory monitoring

Thyroid function status must be assessed periodically in patients receiving thyroid agents as a guide to therapy. Selection of appropriate tests for the diagnosis and management of hypothyroidism or hyperthyroidism depends on patients-specific variables (e.g., signs and symptoms of thyroid disease, pregnancy, concomitant administration of drugs).

A combination of thyroid-stimulating hormone (TSH) assay plus free thyroxine (T) and/ or total or free triiodothyronine (T_3) assay usually is recommended to confirm a diagnosis of thyroid disease. TSH assay alone may be used initially to screen for thyroid disease and to monitor during drug therapy. Other thyroid function tests that may be used include total serum concentrations of T triiodothyronine resin uptake, free T index, and thyrotropin-releasing hormone (TRH) stimulation test.

Mode of Administration

THYROID-S tablet is administered orally.

Contraindications

1. Treatment of obesity or for weight loss

2. Patients with the presence of thyrotoxicosis and in acute myocardial infarction uncomplicated by hypothyroidism
3. Patients with uncorrected adrenal insufficiency because the drugs increase tissue demands for adrenal hormones and may precipitate an acute adrenal crisis in these patients
4. Patients with hypersensitivity to thyroid agents or any ingredient in the formulation

Warnings and Precautions

1. Patients receiving thyroid agents must be closely monitored and thyroid function status must be periodically assessed by appropriate laboratory studies.
2. Thyroid agents should be used with extreme caution and in reduced dosage in patients with angina pectoris or other cardiovascular disease, including hypertension.
3. Thyroid agents should be used with caution in geriatric patients since occult cardiac disease may be present.
4. Morphologic hypogonadism and nephroses should be ruled out before thyroid agents are administered.
5. When adrenal insufficiency and hypothyroidism exist concomitantly, adrenal insufficiency must be corrected by administration of corticosteroids before therapy with thyroid agents is initiated.
6. Thyroid agents may aggravate the intensity of previously obscured symptoms in patients with endocrine disorders, and appropriate adjustment of therapy for these concomitant disorders may be required.

Interactions with Other Medicaments

Oral anticoagulants

Thyroid agents may potentiate the hypoprothrombinemic effect of warfarin and other oral anticoagulants, by increasing catabolism of vitamin K-dependent clotting factors. When thyroid agents are administered to patients receiving oral anticoagulants, the prothrombin time should be determined frequently, and anticoagulant dosage adjusted accordingly, and patients should be observed closely for adverse effects.

It has been suggested that the dosage of the oral anticoagulant be reduced by one-third when thyroid therapy is started. No special precautions appear to be necessary when oral anticoagulant therapy is initiated on patients already stabilized on maintenance thyroid replacement therapy.

Antidepressants

Concomitant use of tricyclic (e.g., amitriptyline) or tetracyclic (e.g., maprotiline) antidepressants and levothyroxine may increase the therapeutic and toxic effects (e.g., increased risk of cardiac arrhythmias and CNS stimulation), possibly secondary to increased receptor sensitivity to catecholamines; onset of action of tricyclic antidepressants may be accelerated.

Antidiabetic agents

Hypothyroidism may reduce the severity of diabetes mellitus, resulting in decreased requirements of insulin or oral antidiabetics agents (e.g.. sulfonylureas). Administration of thyroid agents to patients with diabetes mellitus may cause an increase in the required dosage of insulin or oral antidiabetic agents. When therapy with thyroid agent is initiated or discontinued or when dosage of a thyroid agent is adjusted in diabetic patients receiving insulin or an oral antidiabetic agent, patients should be closely monitored and appropriate adjustments in dosage of insulin or the oral antidiabetic agent made accordingly if necessary.

Sympathomimetic agents

Parenteral administration of sympathomimetic agents (e.g., epinephrine) to patients with coronary artery disease may precipitate an episode of coronary insufficiency. Because this reaction may be enhanced in patients receiving thyroid agents, patients with coronary artery disease who are receiving thyroid agents should be carefully observed when catecholamines are administered.

Bile acid sequestrants

Bile acid sequestrants (e.g., cholestyramine resin, colestipol) bind thyroid agents in the gastrointestinal tract and impair their absorption. These agents should be administered at least 4 hours apart when the drug must be used concurrently.

Gastrointestinal drugs

Antacids (e.g.. aluminum hydroxide, magnesium hydroxide, calcium carbonate), simethicone, and sucralfate bind thyroid agents in the gastrointestinal tract and delay or prevent their absorption. These agents should be administered approximately 4 hours apart when the drugs must be used concurrently with thyroid agents.

Drugs affecting hepatic microsomal enzymes

Drugs that induce hepatic microsomal enzymes (e.g., carbamazepine, phenytoin, phenobarbital, rifampin) may accelerate metabolism of thyroid agents, resulting in increased thyroid agent dosage requirement.

Serum concentration of digitalis glycosides may be decreased in patients with hyperthyroidism or in patients with hypothyroidism in whom a euthyroid state has been achieved. Thus, therapeutic effects of digitalis glycosides may be reduced in these patients.

Growth Hormones

Excessive use of thyroid agents with growth hormones (e.g., somatropin) may accelerate epiphyseal closure. However, untreated hypothyroidism may interfere with growth response to growth hormone.

Xanthine Derivatives

Decreased clearance of xanthine derivatives (e.g., theophylline) may occur in hypothyroid patients; clearance returns to normal when the euthyroid state is achieved.

Other drugs

Cation-exchange resins (e.g.. sodium polystyrene sulfonate) and ferrous sulfate bind thyroid agents in the gastrointestinal tract and delay or prevent their absorption. Thyroid agents should be administered at least 4 hours apart from these drugs.

Concomitant use of ketamine with thyroid agents may produce marked hypertension and tachycardia: caution is advised when the drug is administered in patients receiving thyroid hormone therapy.

Pregnancy and Lactation

Thyroid agents do not readily cross the placenta, and clinical experience does not indicate any adverse effect on the fetus when thyroid agents are administered during pregnancy. Thyroid agent replacement therapy for hypothyroidism should be continued throughout pregnancy. In pregnant women dependent on thyroid replacement therapy, increased dosage may be required.

Although only minimal amounts of thyroid hormones are distributed into milk, thyroid agents should be used with caution in nursing women.

Undesirable Effects

Adverse reactions to thyroid agents result from overdosage and are manifested principally as signs and symptoms of hyperthyroidism including fatigue, weight loss, increased appetite, palpitations, nervousness, hyperactivity, anxiety, irritability, emotional lability, diarrhea, abdominal cramps, vomiting, elevated liver transaminase concentrations, sweating, tachycardia, increased pulse and blood pressures, angina pectoris, cardiac arrythmias, tremors, muscle weakness, headache, insomnia, intolerance to heat, fever, hair loss, flushing, decreased bone mineral density, impaired fertility, and menstrual irregularities.

Hypersensitivity reactions to excipients in formulations of thyroid agents have been reported rarely. Manifestations include urticaria, pruritus, skin rash, flushing, angioedema. various gastrointestinal symptoms (e.g., abdominal pain, nausea, vomiting, diarrhea), fever, arthralgia, serum sickness, and wheezing.

Overdose and Treatment

Complications of severe overdosage may include cardiac decompensation, cardiac failure, myocardial infarction, cardiac arrest, and possibly death secondary to cardiac arrhythmia or failure.

In the treatment of acute thyroid agent overdosage, symptomatic and supportive therapy should be instituted immediately. Treatment consists principally of reducing gastrointestinal absorption of the drugs and counteracting central and peripheral effects, mainly those of increased sympathetic activity. Initially, the stomach should be emptied by inducing emesis or by gastric lavage; activated charcoal or cholestyramine resin also may be used to decrease absorption. If the patient is

comatose, having seizures, or lacks the gag reflux, gastric lavage may be performed if an endotracheal tube with cuff inflated is in place to prevent aspiration of gastric contents. Oxygen may be administered, and ventilation maintained. If congestive heart failure develops, cardiac glycosides may be administered.

Measures to control arrhythmia, fever, hypoglycemia, or fluid loss should be initiated as necessary. (3-Adrenergic blocking agents (e.g., propranolol) are useful to counteract many of the effects of increased sympathetic activity. Large doses of antithyroid drugs (e.g., methimazole, propylthiouracil) followed in 1-2 hours by large doses of iodine may be administered to inhibit the conversion of thyroxine to triiodothyronine. Plasmapheresis, charcoal hemoperfusion, and exchange transfusion hafte been reserved for cases in which continued clinical deterioration occurs despite conventional therapy. Because thyroxine is highly protein bound, very little drug will be removed by dialysis.

Manifestations of overdosage are usually readily reversible following temporary discontinuance of therapy for 2-7 days and are obviated by a reduction in dosage.

Storage Condition
Store below 25°C and protect from light.

Dosage Forms and Packaging Available
Glass bottle of 500 tablets

Name and Address of Manufacturer
Manufactured by SRIPRASIT PHARMA CO.. LTD. SRIPRASIT Samut Sakhon, Thailand.
Tel. 0 2420 1632-5