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Original Research Paper

# Primary Hypothyroidism and Myopathy

Medical Science

KEYWORDS :

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## DEFINITION AND PREVALENCE

Hypothyroidism results from reduced effects of thyroid hormone on tissues. Hypothyroidism is more common in women and has a total prevalence of 1%to 2%, increasing with age (10% in adults >65 years) and it is found to be most common form of thyroid dysfunction.[1] Indian prevalence of undetected hypothyroidism is 3.47%. Hypothyroidism can be congenital or acquired, sub clinical or overt, and, according to the site of abnormality, primary(thyroid level) or secondary (pituitary or hypothalamic).[2] Iodine deficiency remains a common cause of hypothyroidism worldwide, and in areas of iodine suffiency, Hashimoto's thyroiditis and iatrogenic ( treatment of hyperthyroidism) remains most common cause.[3]

## CLINICAL MANIFESTATION

| | Symptoms | Signs |
|---|---|---|
| General | Weight gain, Cold intolerance, Fatigue, somnolence, hoarseness | Goitre , Hoarse voice |
| Gastrointestinal | Constipation | Ileus and ascitis (rare) |
| Cardiorespiratory | | Bradycardia, Pericardial and pleural effusions, Diastolic hypertension |
| Neuromuscular | Carpal tunnel syndrome, Muscle stiffness Deafness, Depression Psychosis (myxedema madness) | Delayed relaxation of tendon reflexes, Cerebellar ataxia, Myotonia |
| Dermatological | Dry skin, Dry hair, Alopecia, Yellowish skin | Myxedema, Carotenaemia , Vitiligo |
| Reproductive | Gynaecomastia | Menorrhagia, Infertility impotence |
| Ocular | | Periorbital odema , Loss of lateral eye brows |

Hypothyroidism can present as emergency in form of myxedema; it is often precipitated by other diseases. Patients have profound hypothermia, bradycardia, altered mental status, hyponatremia , pericardial effusion and typical skin and facial changes. Mortality is 100% if not treated. Without regard to the cause of hypothyroidism, neuromuscular and musculoskeletal manifestations can be observed in many patients with the condition. Manifestations can occur at any time in patients having hypothyroidism and it can be presented in any of the following form:

1.mylgia

2.muscle hypertrophy

3.proximal myopathy

4.rhabdomyolysis

5.myoedema

arising on the surface of a muscle, when struck with a percussion hammer. This phenomenon is characteristic of hypothyroid myopathy and lasts for 30 to 60 seconds.

Hypothyroid myopathy typically manifests as polymyositis-like myopathy with proximal muscle weakness and an increased Creatine kinase (CPK) level. However, it sometimes manifests as muscle enlargement (pseudo hypertrophy), in adults, this condition is called Hoffmann's syndrome. In children with hypothyroid disease (cretinism), a pattern of proximal weakness and diffuse muscle enlargement is known as Kocher-Debre-Semelaigne Syndrome.

## PATHOGENESIS

The pathogenesis of hypothyroid myopathy is not well understood. A variety of effects on cellular function and metabolism occurring in hypothyroidism may contribute to the development of muscle symptoms and abnormal muscle function. Thyroxine(T4) affects energy metabolism and T4 deficiency leads to abnormal glycogenolysis , mitochondrial oxidative metabolism, and triglyceride turnover, which in turn impair muscle function.

These effects are reflected in selective atrophy of Type II fibers, which are more dependent on glycolysis for their energy supply. Type I hypertrophy may be a compensatory response. With severe or prolonged oxidative damage, muscle cell injury and rhabdomyolysis may occur. The fact that the degree of weakness often does not correlate with the biochemical severity of hypothyroidism suggests that muscle injury, rather than impaired muscle function alone, plays a prominent role in some patients. Serum muscle enzyme elevations in hypothyroidism, which occur in the absence of weakness, myalgias, or structural muscle

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abnormalities, appear to be due to changes in muscle cell membrane permeability, although the basis for this change is unknown . Reduced clearance of CPK probably also plays a role and animal studies have also studied the role of thyroid hormone in gene expression of several skeletal muscle proteins, including myosin ATPase, AMP-activated protein kinase. The features described above are consequence of thyroid hormone deficiency. However, autoimmune hypothyroidism may be associated with signs and symptoms of other auto immune diseases, particularly vitiligo, pernicious anemia, addisons disease, alopecia aerata, and type 1 diabetes mellitus and less common associations include Celiac disease, dermatitis herpiiformis, chronic active hepatitis, Rheumatoid arthritis, Systemic Lupus Erythematosus(SLE), Mysthenia gravis and Sjogrens syndrome. [8]

**INVESTIGATIONS**

In the vast majority of cases, hypothyroidism results from an intrinsic disorder of the thyroid gland (primary hypothyroidism). In this situation, serum T4 is low and TSH is elevated, usually in excess of 20 milli international units per litre (mIU/L). Measurements of serum T3 are unhelpful since they do not discriminate reliably between euthyroidism and hypothyroidism. Secondary hypothyroidism is rare and is caused by failure of TSH secretion in an individual with hypothalamic or anterior pituitary disease. In severe, prolonged hypothyroidism, the electrocardiogram(ECG) classically demonstrates sinus bradycardia with low-voltage complexes and ST segment and T-wave abnormalities. Once clinical or sub clinical hypothyroidism is confirmed , the etiology is usually easily established by demonstrating the presence of anti-TPO(thyroid perioxidase) antibodies , which is present in > 90% of patients with autoimmune hypothyroidism & FNAC is used to confirm the presence of autoimmune thyroididtis.

**MANAGEMENT**

Treatment is with levothyroxine replacement. It s usually started with a low dose of 50 μg per day for 3 weeks, increasing thereafter to 100 μg per day for a further 3 weeks and finally to a maintenance dose of 100–150 μg per day. In younger patients, it is safe to initiate levothyroxine at a higher dose (for example, 100 μg per day), to allow a more rapid normalization of thyroid hormone levels. Levothyroxine has a half-life of 7 days so it should always be taken as a single daily dose and at least 6 weeks should pass before repeating thyroid function tests and adjusting the dose, usually by 25 μg per day. Patients feel better within 2–3 weeks. Reduction in weight and periorbital puffiness occurs quickly, but the restoration of skin and hair texture and resolution of any effusions may take 3–6 months. The dose of levothyroxine should be adjusted to maintain serum TSH within the reference range. To achieve this, serum T4 often needs to be in the upper part of the reference range or even slightly raised, because the T3 required for receptor activation is derived exclusively from conversion of T4 within the target tissues, without the usual contribution from thyroid secretion. Myopathy is completely reversible within six months of initiation of treatment.

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