**CASE REPORT**

**Association of Thyroid Disorder with Polymyalgia Rheumatica Suggesting Possible Common Underlying Aetiology**

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**KEY WORDS:** Thyroid storm, Autoimmune Thyroid Disorder, Polymyalgia Rheumatica, Giant Cells

**ABSTRACT**

Ours is a case of a 60 years old female patient with simultaneous onset of thyroid storm and polymyalgia rheumatica and responded very well to Glucocorticoids. There have been many previous reports suggestive of association of thyroid dysfunction (hyper or hypothyroidism) and polymyalgia rheumatica, so it is not simply a coincidence and suggests some common underlying aetiology, may be immunological. Here the patient had multisystem involvement including Central nervous system, cardiovascular system, musculoskeletal system along with metabolic derangements, all resolved with steroids.

**CASE REPORT**

A 60 years old female patient, retired professor; diabetic for almost 2 years, on Oral Hypoglycemic Agents, with acceptable sugar control, presented to emergency department with history of high grade fever, continuous, not responding to antipyretics, for almost 15 days associated with generalized weakness, joint pains (large joints like hip and shoulder joints, sacroiliac joints), continuous headache, nausea and vomiting; occasional irrelevant talking with abnormal behavior and Slurred speech for last 3 to 4 days.

For the same patient was hospitalized at some private hospital. Investigated and found to have Urinary Tract Infection and persistently high blood pressure. Chest X-ray and abdominal sonogram were normal. ECG showed sinus tachycardia with T inversion in aVL and V3 to V6. Laboratory investigations during hospitalization were suggestive of mild anaemia with normal total count and platelet count, ESR was high with normal Liver function and Renal function tests. Patient was treated with intravenous fluids, injectable antibiotics, antihypertensives, antithyroid drugs (Carbimazole and propranolol) were added.

As there was no improvement, patient was shifted to higher center. In emergency room, patient was primarily evaluated, she was conscious but confuse, had severe glossitis leading to dysarthria, dehydration, pallor, tachycardia, high blood pressure, few left sided basal crepitations on auscultation along with S3 gallop on cardiac auscultation was present. Abdomen was soft and non tender on palpation, no organomegally was there. On central nervous system examination, she had proximal weakness, power: 4/5 at ankle, knee, wrist and elbow, 2-3/5 at shoulder and hip joints. On local examination she had tenderness in thigh and upper arm muscles, movement restriction at shoulder and hip joints, may be due to joint pains. Laboratory investigations on admission showed mild anaemia, normal total count and platelet count, high ESR, high C-reactive protein, normal renal and liver function tests except hypoproteinemia and hyperfunctioning thyroid status. ECG showed multiple Ventricular premature beats with global ST-T changes. Her Urine examination was normal, Chest X-ray showed bilateral mid zone and lower zone patchy soft tissue opacities (Congestive changes), normal abdominal fluid examination, mild hyponatremia, herpes simplex serology negative. Her thyroid function tests showed hyperfunctioning thyroid and small lacunar infarcts were seen on CT Brain. Antithyroid drugs (Carbimazole and propranolol) were added.

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sonogram, ultrasonography of neck showed multinodular goiter, no lymphadenopathy. 2D Echo showed dilated Left Ventricle (LV), global LV hypokinesia, severe LV systolic dysfunction, LV Ejection Fraction: 10-15%, Grade II MR, severe PAH.

At this stage, provisional diagnosis which we kept in mind were:
- Fever under investigation

**CAUSE**

1) Urinary Tract Infection with sepsis (previous Urine complete examination showed pus cells)
2) Thyrotoxicosis presented with crisis
3) Atypical Bacterial/viral pneumonia leading to Systemic Inflammatory Response Syndrome (myocarditis / joint pains and myalgia? Proximal myopathy / encephalitis)
4) Underlying malignancy With
   - Possible Polymyalgia rheumatica
   - HTN with? Coronary Artery Disease? thyrotoxic cardiomyopathy
   - Type II Diabetes Mellitus

Patient was treated with BiPAP support, Guarded Intravenous fluids, injectable antibiotics, Insulin infusion according to blood sugars, Anti Failure treatment. Doses of antithyroid drugs were stepped up and as patient had cardiac failure as well as episodes of bronchospasm, propranolol was stopped and patient was put on cardioselective betablocker. But Fever remained persistent (continuous, up to 103°F) with persistent tachycardia and tachypneoa, altered sensorium, Proximal weakness and joint pains. To rule out atypical pneumonia, HRCT thorax was done which was suggestive of bilateral minimal pleural effusion with bilateral basal atelectasis. Further Laboratory investigations showed normal hematology except mild anaemia, Elevated ESR and CRP, Persistent hypercalcemia (Ca++: ~11-12), Low PTH (2°hypoparathyroidism), Hyperuricemia, Hypokalemia, Procalcitonin: negative, LDH/SGOT: borderline elevated, ANA / ANCA: negative, Pneumoslide panel: negative, Normal rest LFTs and Creatinine. Anti Microsomal Antibody to thyroid: 137 IU/L (N: < 5.61), S.Imunelectrophoresis: hypoalbuminemia associated with hypoproteinemia, Gamma globulin showed predominantly IgG Kappa type suggestive of malnutrition or mal absorption, Monoclonal Band not detected. Considering thyroid storm and associated polymyalgia rheumatica most common possibilities, steroids were started. Inj Dexamethasone 8mg/day intravenous was started in divided doses. Pt became afebrile within 2 hours, sensorium improved. A fresh episode of acute coronary insufficiency occurred on same day with Trop I: 7.75 (‘!!’). Anti failure treatment continued, low molecular weight heparin was added, Diuretics were uptitrated, allopurinol 100 mg 1 BD PO, biphosphonate injectable one stat dose was given to control hypercalcemia, potassium supplementation was given.

Pt improved, became afebrile within 2 days, Sensorium improved, S. Ca++ and Uric acid came to normal within 3 days. Patient was put on O2 mask with SpO2 98-99%, Pulse: 80-90/ min. Repeat 2D echo after 4 days showed improved EF ~ 25%. Her repeat thyroid function tests after two weeks showed Free T3: 1.8 mcg/dl (low), Free T4: 0.69 mcg/dl (low), TSH: 0.273 µIU/ml (low). Patient was discharged from hospital after 14 days of hospital course in hemodyanmically stable condition, no fever, improved sensorium, no joint pains and power grade 4+/5 in all 4 limbs. Dose of antithyroid medicines was reduced according to reports and oral steroids were continued up to 8 weeks in tapering doses.

**DISCUSSION**

Thyrotoxicosis or hyperthyroidism is a fairly common endocrine disorder in clinical practice and it is the clinical syndrome caused by an excess of circulating free thyroxine (T4) or free triiodothyronine or both. The interdependence of this thyroid disorder with disorders of the mind and emotions, as well as with mental disorders, is not being given the required consideration in many clinical settings. Similarly, many clinical symptoms suggesting thyrotoxicosis are sometimes overlooked, such as palpitations, agina, breathlessness, tremor, infertility, pruritus, bone pains, excessive sweating and insomnia. In addition, a lot of metabolic derangements can be secondary to thyrotoxicosis, such as hyperglycemia,
hypercalcemia, hypoparathyroidism, hypokalemia and hypcholesterolaemia.\(^{(1,4)}\) Despite the enormity of the several problems associated with a thyrotoxic condition, very inadequate attention is given to these problems in many clinical settings.

Although both hyperthyroidism (thyrotoxicosis) and hypothyroidism are the two common thyroid disorders, thyrotoxicosis is more prevalent. Therefore, thyrotoxicosis should be part of regular differentials in any clinical setting.\(^{(1)}\)

In old age sometimes even slightly elevated thyroid hormones may give picture of thyroid storm. In our case also T4 was only borderline elevated, but patient’s anti microsomal antibodies to Thyroid gland were significantly elevated, so one possibility is that the underlying immunological dysfunction may be responsible for crisis and also for the multisystem involvement. Patient also responded only after starting steroids, she became afebrile within hours and sensorium also improved.

Common causes of hyperthyroidism in the elderly are Graves disease (70-80%), thyroiditis, Toxic nodular goiter, Toxic thyroid adenoma and exogenous hyperthyroidism puberty (iatrogenic, factitious, iodine induced), secondary to hormones (trophoblastic tumors, pituitary tumors) and different clinical situations. Grave’s disease is an autoimmune disorder. There is a female preponderance of 10:1 and there may be a family history of thyroid disease or other autoimmune endocrine disease. Hyperthyroid Crisis (thyroid storm) is an uncommon medical emergency and is a life threatening exacerbation of thyrotoxicosis. It occurs in patients with untreated or poorly treated hyperthyroidism, in response to stress factors (infection, surgery, trauma), may be precipitated by administration of radiiodine, iodinated contrast agents, or withdrawal of anti-thyroid drugs. In thyroid storm, clinical features include those of severe thyrotoxicosis, seizure, coma, hyperpyrexia, dehydration, multi-system failure and death within a few hours or days.\(^{(2)}\)

An association of two diseases, particularly with a simultaneous onset, may indicate a common underlying aetiology. The evidence for an autoimmune basis for thyrotoxicosis is considerable and it has been associated with many other diseases thought to be autoimmune. Giant-cell arteritis is also considered to be a disease of altered immunity. That it should be associated with thyrotoxicosis is therefore not entirely unexpected.\(^{(3)}\) This association has clinical relevance and adds to other indirect evidence that giant cell arteritis is caused or perpetuated by an abnormal immune mechanism.

Polymyalgia rheumatica is considered to be a type of arthritis because it causes inflammation and swelling in the larger joints of the body, pain and stiffness predominate in the shoulder and hip girdles, along with systemic symptoms, such as fatigue, weight loss and low-grade fever. The inflammation is caused by cells of the immune system attacking the membranes lining the joint (synovium), but the reason for this attack is unknown. The condition is more common in people aged 50 years and over, with most sufferers diagnosed at around 70 years. Women are twice as likely to develop the condition than men. Diagnosis largely depends on ruling out other possible causes.\(^{(3)}\)

Laboratory findings are typically nonspecific. The most widely used acute phase indicator is ESR. Serologic studies (such as rheumatoid factor and antinuclear antibody tests) and muscle-related enzyme assays (such as creatine phosphokinase) are usually negative or normal. Occassionaly, liver-function test results are abnormal. There is no true muscle weakness nor evidence of muscle disease on electromyography (EMG) or on biopsy. Response to corticosteroid therapy is typically rapid and dramatic, and clearly helps to support the diagnosis.\(^{(3)}\) In fact, if the patient does not improve within 72 hours of treatment with a corticosteroid, such as prednisone (10-20 mg daily), a diagnosis of PMR may be incorrect.

In our case, we did put the possibility of thyroid storm and polymyalgia rheumatica after primary evaluation and laboratory investigations, but as patient was old aged and she had history of urinary tract infection during previous hospitalization, as well as patient was diabetic, so it is always safe to rule out underlying sepsis, before starting steroids. Sometimes this kind of picture may be present with some underlying malignancy.

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