European Thyroid Journal

Eur Thyroid J 2014;3:65–68 DOI: 10.1159/000356852 Received: July 29, 2013 Accepted after revision: October 22, 2013 Published online: December 20, 2013

Thyrotoxicosis followed by Hypothyroidism due to Suppurative Thyroiditis Caused by *Nocardia brasiliensis* in a Patient with Advanced Acquired Immunodeficiency Syndrome

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What Is Known about This Topic

• Acute suppurative thyroiditis due to nocardiosis is extremely rare, with only 8 cases caused by *Nocardia asteroidies* or *Nocardia farcinica* having been reported. The only thyroid hormone abnormality documented prior to this report was subclinical hypothyroidism in 1 patient.

What This Case Report Adds

• This is the first documented case of acute suppurative thyroiditis due to *Nocardia* presenting with hyperthyroidism which, in addition, was followed by persistent hypothyroidism as a result of extensive gland destruction. This is also the first report of *Nocardia* thyroiditis caused by the species *Nocardia brasiliensis*.

Key Words

Suppurative thyroiditis · Nocardiosis · Hyperthyroidism · Human immunodeficiency virus · Acquired immunodeficiency syndrome

Abstract

Acute thyroiditis is an extremely rare complication of nocardiosis. We report a patient with hyperthyroidism due to suppurative thyroiditis caused by *Nocardia brasiliensis*. A 38-yearold Black male presented with features of thyrotoxicosis,

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© 2013 European Thyroid Association Published by S. Karger AG, Basel 2235–0640/13/0031–0065\$38.00/0 sepsis and airway obstruction. He had no evidence of underlying thyroid disease, but was severely immunocompromised as a result of acquired immunodeficiency syndrome. He had previously been diagnosed with pulmonary nocardiosis and also had nocardial abscesses on his anterior chest wall. Investigations revealed thyrotoxicosis, with a FT₄ of 43.2 pmol/l and a suppressed TSH <0.01 mIU/l. Serum anti-thyroperoxidase and anti-thyroglobulin antibodies were absent. Computed tomography scan showed a large abscess in the anterior neck involving the left lobe and isthmus, as well as inhomogeneous changes in the right lobe of the thyroid.

Roy Shires, BSc, MBBCh, PhD, FRCP Division of Endocrinology Chris Hani Baragwanath Academic Hospital PO Bertsham, Johannesburg (South Africa) E-Mail roy.shires@wits.ac.za The radioisotopic scan showed absent uptake of tracer in keeping with thyroiditis. Although the initial presentation was that of hyperthyroidism, destruction of the gland later resulted in sustained hypothyroidism, necessitating thyroid hormone supplementation. The hyperthyroidism can be explained by the release of presynthesized and stored thyroid hormone into the circulation as a result of inflammation and disruption of the thyroid follicles, and the subsequent hypothyroidism by the fact that much of the gland was destroyed by the abscess and the extensive inflammatory process. This is the first documented case of hyperthyroidism in a patient with acute suppurative thyroiditis caused by *Nocardia*.

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Introduction

Acute suppurative thyroiditis is rare, being far less common than the non-suppurative post-viral variety, de Quervain's disease. It occurs mainly in immunocompromised and elderly patients [1]. Possible reasons for the infrequency of suppurative infections of the thyroid include the gland's abundant blood supply and lymphatic drainage, and the antimicrobial action of iodine [2]. Most patients have a preexistent thyroid disorder, especially multinodular goiter [1–3]. Most infections are bacterial, but opportunistic infections also occur. Typically, patients are acutely ill with fever, dysphagia, and a tender thyroid mass, but presentation may be insidious in patients with opportunistic infections [1]. Management includes the administration of appropriate antibiotics and drainage of any abscesses [1, 2].

Nocardia are aerobic, branching, filamentous, Grampositive actinomycetes. Human nocardiosis occurs most commonly in immunocompromised hosts including acquired immunodeficiency syndrome (AIDS) [4]. The most frequent route of entry for *Nocardia* is the respiratory tract. Most organs can be affected [4, 5]. Infection due to *Nocardia brasiliensis* occurs less frequently than that caused by *Nocardia asteroides*, and commonly affects skin and soft tissue [5, 6]. Hematogenous seeding leading to disseminated disease is uncommon [6]. Therapy requires prolonged antibiotic administration, together with surgical intervention when indicated [4].

Thyroiditis caused by *Nocardia* is extremely rare and only 8 cases have been described in the literature [5, 7–13]. Of these, *N. asteroides* was cultured in 7 cases and *N. farcinica* in 1 case. Most of the reports made no mention of abnormal thyroid function at the time of presentation. Subclinical hypothyroidism developed in only 1 patient after 12 weeks [8]. Outcomes were poor and half of the patients demised [7, 10-12].

Case Report

A 38-year-old Black male presented to Chris Hani Baragwanath Academic Hospital with a 3-day history of a rapidly enlarging neck mass, dysphagia, dysphonia, and difficulty breathing. He complained of recent-onset diarrhea, significant weight loss, productive cough and night sweats. He had been managed by the hospital for AIDS as well as pulmonary nocardiosis. Non-compliance with medication had resulted in persistently low CD4 counts and a high viral load. The *Nocardia* infection had not improved despite the prescription of prolonged courses of appropriately selected antibiotics to which the organism was sensitive.

Physical examination revealed a wasted, pale, pyrexial patient with a hoarse voice and labored respiration. He had florid oropharyngeal candidiasis. A large, tender, fluctuant mass was noted on the left anterior aspect of his neck with overlying induration of the skin and displacement of the trachea to the right. There were bilateral crackles on auscultation of his chest. Two large abscesses on the upper aspect of his anterior chest wall were draining pus. The patient had a fine tremor and displayed generalized weakness, most pronounced in the proximal limb musculature.

Initial testing revealed a free thyroxine (FT₄) of 43.2 pmol/l (normal 12.0–22.0), a suppressed TSH <0.01 mIU/l (normal 0.27–4.20) and a free triiodothyronine (FT₃) of 3.1 pmol/l (normal 2.8–7.1). Serum anti-thyroperoxidase and anti-thyroglobulin antibodies were negative. Inflammatory markers were elevated with a leukocytosis of 13.7×10^9 /l and a C-reactive protein of 179.9 mg/l. CD4 count was 142 cells/µl and viral load, 69,064 RNA copies/ml.

The patient's chest radiograph showed bilateral infiltrates. Ultrasound revealed a large abscess in the anterior neck, shown by computed tomography to involve the left lobe and isthmus of the thyroid (fig. 1). The right lobe showed inhomogeneous changes. A technetium thyroid scan showed absent tracer uptake, in keeping with thyroiditis.

Aspiration of the abscess yielded purulent material, microscopy of which displayed branching filamentous Gram-positive organisms (fig. 2). *N. brasiliensis* was subsequently cultured and demonstrated extensive antibiotic resistance. Blood cultures and sputum microscopy and culture yielded no organisms. Tests for active tuberculosis were negative.

The patient was treated with targeted intravenous antibiotics. Aspiration of the abscess was followed by surgical drainage on the tenth hospital day, and the hyperthyroidism progressively abated. By day 19, the FT₄ had fallen to subnormal levels and was followed by a gradual rise in TSH levels. Hypothyroidism persisted and levothyroxine replacement was commenced on day 48 post-admission (fig. 3).

Discussion

This is the first documented case of hyperthyroidism in a patient with acute suppurative thyroiditis caused by *N. brasiliensis*. Our patient had no evidence of underlying



Fig. 1. Computed tomography scan of the patient's neck showing a large rim-enhancing collection ($45 \times 45 \times 52$ mm) in the left lobe and isthmus of the thyroid (arrow). The abscess extended around the trachea, compressing the esophagus. The right lobe showed an inhomogeneous pattern. There were significant paratracheal and jugular lymph nodes.

Fig. 2. Smear of abscess aspirate showing *Nocardia* species. Note the branching filamentous Gram-positive organisms. Kinyoun stain. $\times 100$.



Fig. 3. FT_4 and TSH levels from admission (day 0) to day 61. Levothyroxine replacement therapy was commenced on day 48.

thyroid disease, but was immunocompromised as a result of AIDS. Hyperthyroidism in an AIDS patient can be attributed to a number of processes including Graves' disease in the setting of the immune reconstitution syndrome, various antiretroviral drugs and complicating infections, such as cytomegalovirus. The absence of tracer uptake on the scan, his non-compliance with antiretroviral treatment, the presence of *Nocardia* cultured from thyroid abscess, and the absence of any other organism on culture all support the diagnosis of *Nocardia* thyroiditis in our patient.

While the initial presentation was that of hyperthyroidism, destruction of the gland subsequently resulted in hypothyroidism, requiring thyroxine supplementation.

Thyrotoxicosis Caused by *Nocardia* Thyroiditis

The majority of patients presenting with suppurative thyroiditis have normal thyroid functions in the absence of preexistent thyroid disease, but both hypothyroidism and hyperthyroidism have been reported [1, 2]. The mechanism of hyperthyroidism is probably similar to that occurring in subacute thyroiditis, in which presynthesized stored thyroid hormone is released into the circulation as a result of inflammation and disruption of the thyroid follicles [14]. Even when only part of the gland is affected, radioactive iodine uptake may be suppressed due, in part at least, to the inhibition of TSH secretion by the elevated circulating thyroid hormone [15]. As in our patient, the predominant hormone elevated is T₄. The normal T₃ levels are likely due to the effects of non-thyroidal illness on type 1 deiodinase (i.e. sick hyperthyroid). Following abscess drainage and resolution of the inflammation, the FT₄ level fell below normal and persisted without recovery, in contrast to the usual situation in non-suppurative de Quervain's thyroiditis. The reason for the failure to return to the euthyroid state in our patient can be explained by the fact that much of the thyroid gland was destroyed by the large abscess and the surrounding severe inflammatory process.

Acknowledgements

The authors thank Dr. Sharona Seetharam, Department of Microbiology, and Dr. Stephen Mphomane, Department of Radiology, Chris Hani Baragwanath Academic Hospital for their assistance.

Disclosure Statement

The authors have no conflicts of interest to disclose.

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