CASE REPORT

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Acute Suppurative Thyroiditis Secondary to Tuberculosis with Superimposed Bacterial Infection: A Case Report

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Abstract

Acute suppurative thyroiditis is a rare and potentially fatal condition. We present a case of an 18-year-old Malay female who presented with one-week history of painful right sided neck swelling, fever and odynophagia. Neck CT confirms ruptured multiloculated abscess with posterosuperior extension into prevertebral space. Pus and tissue cultured *Streptococcus anginosus* and *Eikenella corrodens* with positive TB PCR. She responded well to ampicillin/sulbactam and anti-tuberculosis treatment with no evidence of residual collection from ultrasound.

Key words: acute suppurative thyroiditis, thyroid abscess, thyroid tuberculosis, contrast induced thyroiditis

INTRODUCTION

Acute suppurative thyroiditis is a rare potentially life-threatening emergency with reported incidence of only 0.1-0.7% in the literature.[1,2] Due to the scarcity of cases, there are so far no comparative clinical studies, and management has been guided by published case reports and series. Mortality rate ranges from 3.7-12%.[3,4] Here we report a case of a young female with acute suppurative thyroiditis complicated with extensive thyroid abscess resulting from tuberculosis (TB) and secondary bacterial infection.

CASE

An 18-year-old Malay female, previously well, presented with one-week history of right sided painful neck swelling, fever, and odynophagia. Preceding this she denied any acute pharyngitis, constitutional symptoms, night sweats, or TB contacts. There were no hyper or hypothyroid symptoms. She denied any history of trauma to the neck. Clinical examination revealed a blood pressure of 118/72 mm Hg, pulse rate 89 bpm, and temperature 37°C. She was euthyroid, no orbitopathy or other evidence of Grave's disease. There was a right anterolateral neck mass measuring 4 x 5 cm, mild warmth and tender to palpation, however no fluctuant area. There was no cervical lymphadenopathy.

Laboratory investigations showed leucocytosis TWBC 17.6 x 10^9/L with neutrophil predominance and elevated inflammatory markers ESR 98 mm/hour, CRP 105.8 mg/L (<5.0). Negative HIV screening and fasting glucose of 5.1 mmol/L ruled out immunocompromised state. Her baseline thyroid function test (TFT) upon admission was TSH 0.13 mU/L (0.56-4.90) and FT4 21.1 pmol/L (1.5-22.7). Chest X-Ray showed clear lung fields. Neck ultrasound demonstrated heterogenous appearance of both thyroid lobes, with heterogenous hypoechoic mass occupying the right lobe, extending towards the left isthmus-thyroid junction (Figures 1A and 1B). Contrast-enhanced computer tomography (CECT) of the neck showed a multiloculated hypodense rim enhancing collection with the epicentre in the right thyroid lobe measuring collectively 3.5 x 4.6 x 5.4 cm. Posterosuperiorly, there was an extracapsular extension of the collection into the prevertebral space which measures 5.5 cm in craniocaudal length from the level of C2/3 until C6/7 with displacement of trachea to the left. In addition, there were multiple enhancing bilateral cervical lymph nodes in all levels, with the largest measuring 0.9 x 0.6 cm (Figures 2A and 2B). Fibreoptic laryngoscope showed patent airway and no pyriform sinus fistula. A repeat TFT three days after iodinated contrast media (ICM) exposure noted markedly elevated TFT almost three times upper limit with TSH of 0.098 mU/L (0.900 - 3.110) and FT4 36.3 pmol/L (7.8 - 13.2), suggestive of contrast induced thyroiditis. Hence, no anti-thyroid treatment was initiated.

She underwent a bedside fine needle aspiration which aspirated 5 cc of frank pus and a repeat ultrasound guided aspiration which aspirated 25 cc of pus. However, a repeat ultrasound showed persistence of the remaining collection at right thyroid bed thus a decision to proceed with right hemithyroidectomy and incision and drainage of right parapharyngeal abscess. Intra-operatively, the right thyroid gland weighed 12 g, the superior pole was found to be sloughy and adhering to the strap muscles. A total of 5 cc of pus was aspirated from the thyroid gland and parapharyngeal area. A drainage catheter was inserted and kept for six days which drained 10 – 30 mls of pus daily.

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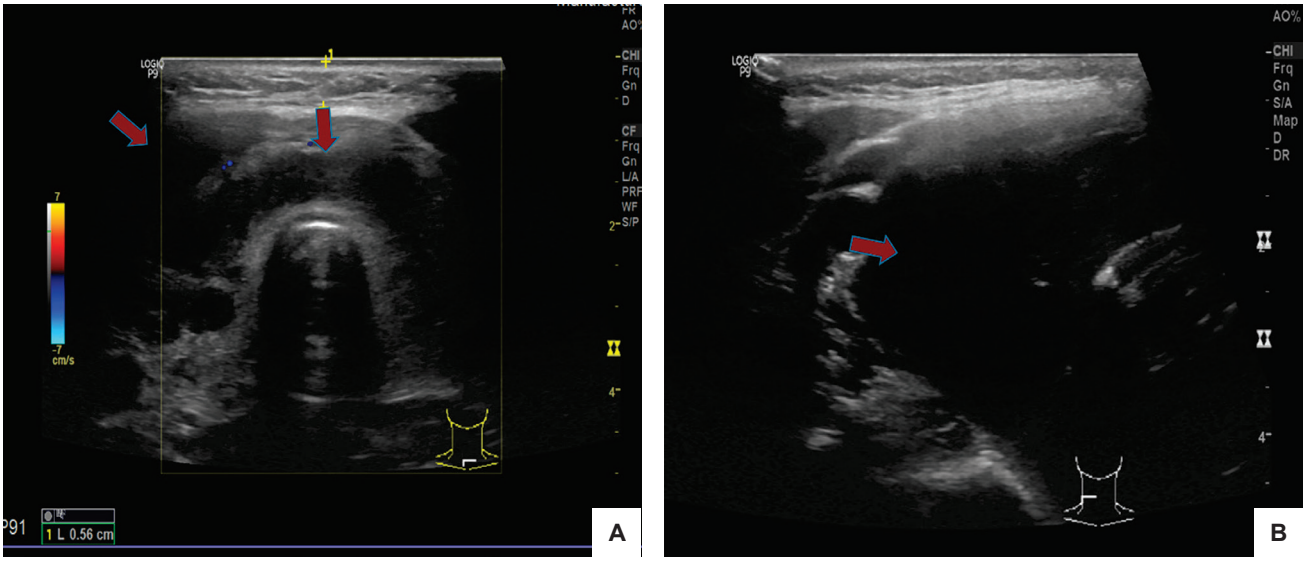

Figure 1. (A) Multi loculated hypoechoic collections arising from right thyroid lobe extending into the isthmus; **(B)** largest hypoechoic collection occupying the right lobe.

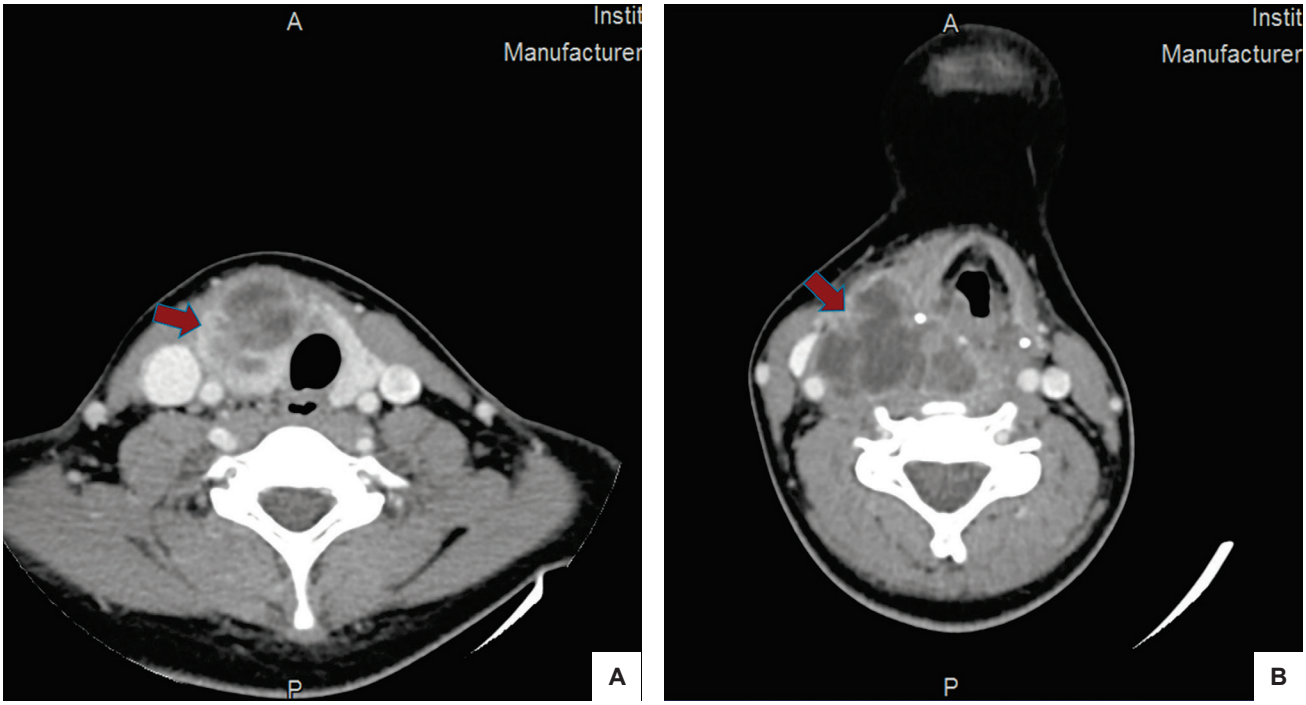

Figure 2. (A) Multi loculated hypodense rim enhancing collection at right thyroid bed, trachea deviated to the left. Left thyroid gland appears normal; **(B)** Collection extends posterosuperiorly into the prevertebral space.

Pus and tissue cultured *Streptococcus anginosus* and *Eikenella corrodens* both sensitive to penicillin G. Polymerase chain reaction (PCR) of the pus was also positive for *Mycobacterium tuberculosis* despite the acid-fast bacilli (AFB) smear being negative. However, pus and tissue culture did not isolate any mycobacterium tuberculosis (MTB). Tissue histopathological examination (HPE) demonstrated xanthogranulomatous inflammation with microabscess formation. No epithelioid histiocytes, granuloma formation nor Langhan's giant cells was seen. Sputum smear for acid fast bacilli sent twice were negative. She was discharged with oral Ampicillin plus Sulbactam 750 mg twice daily for four weeks (total duration six weeks) and combination of Isoniazid 75 mg, Rifampicin 150 mg, Ethambutol 275 mg and Pyrazinamide 400 mg, four tablets once daily based on the initial TB PCR result. A follow-up ultrasound neck five weeks after commencement of antibiotics and 18 days after initiation of anti-TB did not show any residual abscess collection. Outpatient TFT performed four weeks post-surgery indicate recovering TFT with TSH of 0.74 mIU/L (0.46-4.68) and FT4 6.8 pmol/L (10.0-28.2) without any anti-thyroid medication.

DISCUSSION

Acute suppurative thyroiditis (AST) is rare, as the thyroid gland is essentially resistant to infection attributed to its encapsulation, high iodide content, rich vascular supply,

and extensive lymphatic drainage.[5] Common etiological pathogens are staphylococcus and streptococcus with recent reported trend toward more atypical pathogens particularly in the immunocompromised host.[1] TB of the thyroid gland is an extremely rare extrapulmonary manifestation of TB even in areas where TB prevalence is exceedingly high with reported frequency of <1%.[6]

Raman et al., reported a case of primary TB of the thyroid gland presented with thyrotoxicosis symptoms associated with drenching night sweats, high grade fever and neck swelling.[7] Diagnoses was confirmed with fine needle aspiration (FNA) of the left thyroid nodule, which smeared positive for AFB and a subsequent positive TB culture. CT chest, abdomen and pelvis rule out other organ or lymph node involvement. He responded well to TB treatment for six months with complete resolution of symptoms. This case demonstrates that patients with thyroid TB may present with thyrotoxicosis although they are usually euthyroid.

Additionally, Falhammar and collegues published six case series of adult patients with AST.[8] The causative factors were identified as iatrogenic in two patients from FNA of thyroid gland and septicaemia from prostate biopsy in the other. Blood cultures were positive in three (*Streptococcus pneumonia, Streptococcus sanguinis, Pepto streptococci*), deep tissue culture in three (*Escherichia coli, Candida, Hemophilus influenzae*) and no positive culture at all in two. All patients were treated with antibiotics, three of them required drainage and all of them recovered from acute episode without reported recurrence during a mean period of seven years follow up. Our patient isolated *Streptococcus anginosus* and the less common gram-negative aerobes *Eikenella corrodens*. Her pus smear did not visualize any AFB and anti-TB was instituted earlier based on pus TB-PCR positivity. Following this pus and tissue culture failed to isolate MTB with no supporting evidence from tissue HPE. After consultation with infectious diseases physician, we decided to maintain her anti-TB treatment as she responded clinically to treatment.

Distinguishing AST from subacute thyroiditis is crucial as both may present similarly with preceding history of pharyngitis, fever, and painful thyroid swelling.[9] Both these entities are managed differently with an emphasis on early treatment for AST. Typically, ultrasound examination in the acute phase of AST shows a hypoechoic lesion spreading in or around the affected thyroid lobe. However, ultrasound may show an unclear hypoechoic area in the early acute phase which may lead to an erroneous diagnosis of subacute thyroiditis.[10] The most discriminating method for differentiating is FNA of the thyroid to detect pus and in the case of subacute thyroiditis, it is possible to observe multinucleated giant cell, granulomatous inflammation, and mononuclear cell infiltration.[1,9] In our case, the diagnoses was clear as ultrasound demonstrated classical finding of hypoechoic collection, FNA aspirated frank pus enabling prompt treatment with empirical antibiotics.

In AST it is important to consider an anatomical defect such as a pyriform sinus fistula which has a predilection to involve the left thyroid gland due to an atrophic right ultimo branchial body.[11] Means of diagnosing this may be with transnasal flexible fibreoptic laryngoscopy, CT scan with `trumpet manoeuvré and barium oesophagography.[1] Pyriform sinus fistula was not visualized from fibreoptic laryngoscope performed in our case, furthermore, the epicentre of abscess was located at the right thyroid gland instead of the usual typical left side.

Patients with acute thyroiditis are generally euthyroid. However, occasionally, the condition presents as destructive thyroiditis with thyrotoxicosis.[9,12] Serum levels of thyroid hormone can be transiently increased due to release of preformed thyroid hormone into the circulation resulting from the destruction of the thyroid follicles.[9] We deduce that the rapid increase of TFT in our patient from the baseline was partly contributed from exposure to ICM from CECT imaging.

Management of AST primarily focuses on antibiotic therapy and the need for invasive surgery and drainage or correction of a predisposing anatomic defect.[1] There are published cases on less invasive management not requiring open surgical drainage.[13] Ilyin et al., described two cases of successful nonsurgical management of a thyroid abscess with percutaneous 21-gauge needle aspiration under sonographic guidance with intra-thyroidal injection of antibiotic. Both patients recovered well with no recurrence during follow-up periods of 6 months and 5 years, respectively.[13] However, in our case open drainage with hemithyroidectomy was indicated because the thyroid abscess extended to the prevertebral space with tracheal compression and persistent collection post FNA. Paes et al., advocate a treatment duration of two weeks for thyroid abscess with no underlying anatomical defect.[1] We treated our case for a total of six weeks with antibiotics and continued anti-TB medication for six months.

CONCLUSION

This case exemplifies the importance of recognizing the rarer form of thyroiditis, distinguishing AST with other differentials particularly subacute thyroiditis and instituting treatment early to avoid fatal complications.

Ethical Consideration
Patient consent was obtained before submission of the manuscript.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure
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