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CASE REPORT

Klebsiella pneumoniae thyroid abscess complicated with esophagitis in a woman with newly diagnosed diabetes mellitus: A case report

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ABSTRACT

The thyroid is resistant to infection because of the anatomical and physiological characteristics of the gland, and even though diabetes can induce a compromised immune system, thyroid infection disease is scarcely encountered in diabetic patients. Thyroid abscess formation in an asymptomatic diabetic patient is an even rarer entity. We present a case of a previously asymptomatic diabetic patient showing clinical symptoms of painful swelling in the anterior neck followed by progressing dysphagia, who was later diagnosed with thyroid abscess as a result of *Klebsiella pneumoniae,* complicated with esophagitis caused by the same microorganism. To our knowledge, this is the first reported case of asymptomatic diabetes being clinically diagnosed with thyroid abscess as the first sign.

INTRODUCTION

Although the prevalence of thyroid diseases in diabetic patients is two- to threefold higher than in non-diabetic subjects[1], little attention is paid to infectious thyroid diseases in diabetics, as they are diagnosed in only a few diabetic patients; by contrast, autoimmune thyroiditis, Graves' disease and other endocrine disorders caused by thyroid hormones are most frequently involved. Here, we describe a patient who had escaped a diagnosis of diabetes because of the absence of any typical symptoms or common complications, perhaps for some time, until she presented with discomfort in the anterior neck and underwent a comprehensive examination that showed a blood glucose level above the diagnostic cut-off value. Additionally, the infection had spread to the esophagus, as confirmed by radiographical and etiological analyses.

CASE REPORT

A 41-year-old woman presented with a 10-day history of painful swelling in the anterior neck. Dysphagia, odynophagia and mild dyspnea followed. The patient reported being previously healthy, and denied a history of diabetes. Physical examination showed a fluctuating tender lump moving with deglutition in the thyroid region. Ultrasound showed a 4.6 × 3.2-cm hypoechoic area with a heterogeneous internal echo in the right lobe of the thyroid. Fine-needle aspiration of the fluctuant mass was carried out, and 1 mL of purulent fluid was obtained and sent for culture, which yielded *Klebsiella pneumoniae*. Using image-reformatting approaches, we discovered a low-density lesion bridging the abscess within the thyroid and the right wall of the cervical esophagus on an oblique computed tomography image (Figure 1), which appeared as the collection of encapsulated inflammatory material within the thyroid had broken open and the pus released then travelled into the esophagus. Esophagoscopy detected acute inflammation and a fibrin-covered ulcer located on the right wall at 18 cm from the incisors (Figure 2). The mucosal lesions were biopsied, and sent for pathological evaluation and culture. The pathology report showed necrosis and granulation tissue covered with large amounts of bacteria, which was determined to be *K. pneumoniae* by biopsy culture. Flexible laryngoscopic examination showed no sign of pyriform sinus fistula. Laboratory evaluation showed a leukocyte count of 6.13 × 10^9/L and an increased fasting plasma glucose level of 15.63 mmol/L. An additional

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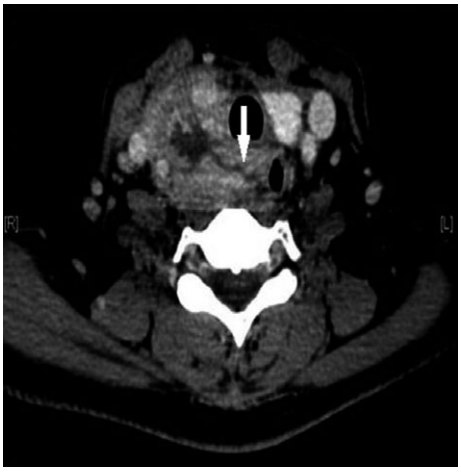

Figure 1 | Oblique computed tomography image showing an infection path through the abscess and the right wall of the cervical esophagus, suggestive of a ruptured abscess releasing pus into the esophagus.

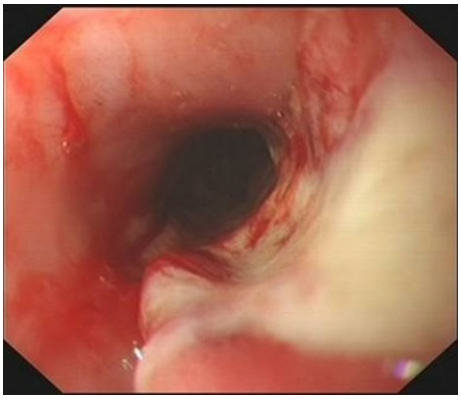

Figure 2 | Esophagoscopy showing a severe stenosis of the esophagus with a fibrin-covered ulcer at 18 cm from the incisors.

plasma glucose test on a separate morning showed 17.25 mmol/L and a hemoglobin A1c (HbA1c) test showed a HbA1c level of 10.8%. Thyroid function tests were normal, and there was no bacterial growth on blood culture. No pathological changes were found in computed tomography scan of the chest, abdomen and brain. Meropenem was given intravenously at a dose of 0.5 g three times per day for 10 days. Furthermore, in this asymptomatic patient, the diagnosis of diabetes could safely be made on the basis of her fasting plasma glucose concentrations of >7.0 mmol/L on two separate mornings and a HbA1c level of 10.8%. Therefore, subcutaneous injection of insulin was added to our intervention. At her most recent follow-up visit (approximately 7 months after discharge), repeat ultrasound showed a 0.64 × 0.81-cm area with a heterogeneous internal echo, and endoscopy exposed that the ulcer was almost undetectable. A barium esophagography was not carried out until these follow-up examinations showed a resolution of the acute inflammation, because it might have failed to show a fistula during the acute episode[2,3], but there was still no sign of pyriform sinus fistula.

DISCUSSION

In many cases, the route or source of thyroid abscess is not obvious. As in the present case, the patient had no pyriform sinus fistula, no history of thyroid disease or upper respiratory tract infection, no direct trauma and negative blood culture; also, no abnormal changes were found in the lung, liver or brain, where *K. pneumoniae* is likely to cause infection; however, her elevated fasting plasma glucose disclosed an underlying predisposition. It is a commonly held premise that diabetic patients are susceptible to infections. Although the patient denied any experience of clinical symptoms, such as intense thirst, excessive hunger, increased urine volume and weight loss, hyperglycaemia might already have been present for a long time, sufficient to cause pathological and functional changes that would result in an immunosuppressive state. Therefore, we are more inclined to consider that the infection originally took place in the thyroid, and the determinant of this rare infection appeared to be pre-existing diabetes in this patient.

However, this alone cannot fully explain abscess formation in this infection-resistant gland; thus, we must question whether diabetes could directly affect the defence mechanism within the thyroid gland itself. Bestetti *et al.*[4] showed structural alterations of thyroid tissues in diabetic mice, including significantly decreased numbers of mitochondria and lysosomes, flattened nucleus of thyroid follicle epithelial cells and dilated cisterns of rough endoplasmic reticulum. Another study discovered diffuse and strong staining of receptors for advanced glycation end-products in colloids of the thyroid gland[5]. Intracellular oxidative stress could be generated by advanced glycation end-products through receptors for advanced glycation end-products[6], and might be involved in both impaired responsiveness and enhanced apoptosis in lymphocytes that induce deficient immunity[7,8]. Both morphological and molecular changes in the thyroid tissues suggest that diabetes likely affects the local defence of the thyroid, making it relatively vulnerable to infection. Therefore, physicians should never ignore the possibility of infectious thyroid disease, though uncommon but with a tendency to occur, in their diabetic patients to avoid the delay of detection and appropriate management.

Diabetes might be partially responsible for this diffuse infection between the thyroid and the esophagus, because evidence has shown that diabetes can increase the permeability of connective tissue, with elevated hyaluronidase activity[9], allowing bacteria and their secreted toxins to travel through the tissue, thus facilitating the spread of infection. Esophageal complication is infrequent, but potentially life-threatening, and increased awareness might lead to a higher detection rate of early-phase infectious esophagitis, which can be healed without perforation or other severe outcomes.

Currently, there is a trend toward the identification of patients with thyroid abscess who might be best treated with less invasive strategies[2]. Given our patient's condition, an invaded esophagus increases the risk of surgical complications, such as esophageal rupture. Perioperative hyperglycemia poses an additional risk for infection among diabetics undergoing surgery. Some published case reports suggest that small areas of abscess can be successfully treated without the need for open surgical drainage[10], but additional clinical outcome data are necessary to justify this approach in the majority of cases. Our patient's marked and sustained clinical improvement provides more evidence for the validity of conservative treatment for thyroid abscess complicated with infectious esophagitis. Glycemic control is another indispensable intervention that would increase host defence, and improve prognosis.

In conclusion, diabetes played a key role in the facets of abscess formation in this unusual location and infection diffusion. Physicians should never ignore the possibility of infectious thyroid disease in diabetic patients, and careful detection of the surrounding structures is essential, because diabetes might promote the spread of infection.

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DISCLOSURE

The authors declare no conflict of interest.

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