Acute Bacterial Thyroiditis Secondary To Urosepsis

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Acute bacterial thyroiditis (or pyogenic thyroiditis) is a rare, potentially life-threatening complication of bacterial infection elsewhere in the body, especially following an upper respiratory tract infection. We report a case of acute bacterial thyroiditis in a patient who sought treatment for urosepsis.

Case Report

An 85-year-old male visitor from out of state was brought to the emergency department with complaints of weakness, fever, and malaise for approximately 1 week. His history was notable for a large goiter, which had been present for at least 10 years and caused only mild dysphonia. The goiter had apparently never been treated. The patient initially denied dyspnea and dysphagia. On admission his temperature was 101°F; he had a large, firm nontender goiter and a markedly enlarged prostate. A urinalysis showed many bacteria and 5 to 20 white cells per high power field. His blood urea nitrogen (BUN) was 67 mg/dL, creatinine 2.8 mg/dL, and blood glucose 228 mg/dL (he had no known history of diabetes). There was a marked leukocytosis (white blood count $28.1 \times 10^3/\mu L$) and a mild normochromic anemia (hemoglobin 10.9 g/dL) on a complete blood count. Thyroid function studies showed a free thyroxine level of 2.4 ng/dL (normal 0.7 to 2.2 ng/dL) and a thyroid-stimulating hormone level of 0.1 μU/mL (normal 0.5 to 4.0 μU/mL).

Because we suspected urosepsis, the patient was admitted to the intensive care unit and prescribed intravenous cefotaxime, 1 g every 8 hours. He initially showed clinical improvement; however, 2 days after admission, his voice became quite hoarse, and he began to develop dysphagia and dyspnea. On physical examination, the goiter appeared unchanged from admission, but a lateral radiograph of his neck showed marked prevertebral soft tissue thickening. A computed tomographic (CT) scan of the neck showed a huge mass emanating from the thyroid and extending into the superior mediastinum with marked displacement of the trachea and esophagus. The patient was then endotracheally intubated and an otolaryngologist was consulted.

A needle aspiration of the thyroid mass recovered thick, purulent material. The patient was then taken to surgery for an incision and drainage of the thyroid mass. Approximately 150 mL of pus was drained; *Escherichia coli* was recovered from both the urine culture obtained on admission and from cultures of pus obtained from the neck mass. Blood cultures were also positive for *E. coli*. He was prescribed cefotaxime and gentamicin, and following a rather stormy postoperative course (primarily fever and diarrhea), he was discharged 15 days after admission in good condition. At the time of discharge his renal function studies had improved (BUN 24 mg/dL, creatinine 1.5 mg/dL). He was advised to contact his own physician in his home state for treatment of his goiter and prostatic hypertrophy.

Discussion

Although infections of the thyroid gland are rare, they are potentially life-threatening.[1] A review of the literature through 1993 found 224 cases reported since 1900. The condition occurs in all age groups, although women with pre-existing thyroid disease constitute the group most likely to develop thyroid infection. In childhood it is often linked to local anatomic defects. It is surmised that the ample blood supply and lymphatic drainage, high iodine content, and protective thyroid capsule contribute to the low incidence of infection.[2] Normal thyroid glands can be involved, but patients with underlying disorders are much more susceptible to infection. Infections usually arise from adjacent structures, such as the oropharynx or lymph nodes, or from congenital abnormalities, such as persistent thyroglossal duct or piriform sinus fistula; however, infection can also occur by means of hematogenous spread or after direct trauma.[3] Nodular goiters have also

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been reported to predispose to bacterial thyroiditis. There are reports of thyroid infections preceded by infection elsewhere in the body, including pyelonephritis, prostatitis, esophageal perforation, otitis, postpartum infection, and dental infection.

Common pathogens include *Streptococcus pyogenes*, *Staphylococcus*, and *S. pneumoniae*; less common organisms include salmonella, *Bacteroides*, *Haemophilus influenzae*, *S. viridans*, other streptococcal organisms, *Peptostreptococcus*, *Actinomyces*, *Echinococcus*, *Aspergillis*, *Klebsiella*, and *E. coli*.[4]

Clinical signs and symptoms include fever (92 percent), anterior neck pain (100 percent), tenderness (94 percent), warmth (70 percent), erythema (82 percent), dysphagia (91 percent), dysphonia (82 percent), and pharyngitis (69 percent).

Leukocytosis is often present; its absence could signify anaerobic infection; thyroid function tests are frequently normal. Studies useful in diagnosing acute suppurative thyroiditis include lateral neck radiographs, CT scan of the neck, sonography, and needle biopsy for organism identification.

Treatment in the initial phase should include intravenous antibiotics directed against staphylococci, streptococci, and anaerobes, changing antibiotics, if necessary, when culture and sensitivity reports are available. Improvement usually occurs within 48 to 72 hours, and complete resolution is often obtained in 2 to 4 weeks. Thyroid abscesses should be drained, and fistulae require excision to prevent recurrences.

Outcome is usually favorable, with complete recovery expected in most cases. One review listed a mortality rate of 6 to 8 percent, although most fatal cases had not been treated for the infection because of delays in diagnosis. Complications are unusual; they include vocal cord paralysis, hypothyroidism (usually transient), tracheal involvement, and recurrent infection.[5]

References

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