

# Intractable Hiccups As a Presenting Symptom of Subacute Thyroiditis

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We describe an unusual presentation of a patient with hiccups. Two weeks before presentation, the patient experienced a sudden onset of hiccups lasting approximately 15 minutes per episode. The patient also reported symptoms of thyrotoxicosis during the same period of time. In euthyroid period hiccups is disappeared. We also discuss the mechanisms of hiccups in patient with thyroiditis.

**Key words:** Subacute thyroiditis, hiccups, phrenic nerve

## Introduction

Subacute thyroiditis has been termed granulomatous (giant) cell thyroiditis. It is thought to be caused by a viral infection of the thyroid gland and often following an upper respiratory illness. The mumps virus, coxsackie virus, influenza virus, echoviruses and adenoviruses may also be etiologic agents. Destruction of the follicular epithelium and loss of the follicular integrity are the primary events in the pathophysiology. Subacute thyroiditis is one of the several causes of low-uptake thyrotoxicosis. The laboratory findings vary with the phase of the disease. The characteristic feature is the appearance of pain in the region of the thyroid gland. Hoarseness, dysphagia and several symptoms of the thyrotoxicosis may be present. The thyroid gland is found to be enlarged, firm and tender on palpation. The disease usually subsides within a few months, leaving no residual deficiency of thyroid function.

Hiccups is a repeated involuntary, spasmodic contraction of the diaphragm accompanied by a

sudden closure of the glottis mediated by sensory branches of the phrenic and vagus nerves as well as dorsal sympathetic afferents. The principle efferent limb and diaphragmatic spasms are mediated by motor fibers of the phrenic nerve (1).

## Case Report

A 58 years-old male patient presenting 2-weeks-lasting intractable hiccups, weakness, fatigue, nervousness, pain on the anterior neck and sore throat. On his physical examination, sinus tachycardia (110/min), tremor, sensible and enlarged thyroid gland were found. His sedimentation rate was high (90 mm/h). Free thyroxine (fT4) and thyroid stimulating hormone (TSH) levels were 1.99 ng/dl (normal range: 0.92-1.9 ng/dl) and 0.025 mU/l (normal range: 0.4-4 mU/l), respectively. Anti thyroid peroxidase (anti-TPO) and anti thyroglobuline (anti-TG) antibodies were measured as negative. The Iodine uptake was low and the patient was diagnosed as the hyperthyroid period of subacute thyroiditis. In the scan there wasn't any sign of substernal enlargement of a goiter.

The phrenic nerve examination with Medelec Synergy EMG Machine (Oxford Instruments, Surrey, UK) determined the right and left latencies as; 8.45 ms and 6.6 ms respectively; and their amplitudes 0.2 mV and 0.5 mV respectively (2). We excluded all the other causes of hiccups (e.g, history of hiatal

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hernia, gastritis, reflux esophagitis, myocardial infarction). No significant diaphragmatic elevation was observed on the chest X-ray.

The patient was given a beta-blocker agent and low dose non-steroid anti-inflammatory agent as therapy. No medication was given for hiccups treatment. After two month's therapy, the patient was free of hiccups and the control thyroid function test results revealed that he was in euthyroid period (fT4: 1.1ng/dl, TSH: 0.45 mU/l).

His control examination of the phrenic nerve was revealed that the right and left latencies prolonged (10.4 ms and 8.1 ms, respectively) and the amplitudes decreased 0.1 mV and 0.1 mV, respectively as stable (0.1 mV).

### Discussion

The phrenic nerves lie along the lateral mediastinum and run from the thoracic inlet to the diaphragm. They course through the upper chest, medial to the mediastinal pleura and the apex of the right or left lung (3). The phrenic nerves provide motor innervation to the diaphragm and sensory innervation to the central intrathoracic and peritoneal surfaces of the diaphragm. They also innervate the pericardium and mediastinal pleura and mediate pain from these areas to the neck and shoulder.

The phrenic nerves can be compromised by enlarged lymph nodes, bronchial obstruction, pericardial disease, cardiomegaly, myocardial infarction, primary lung cancer and subphrenic disease. Injury to the phrenic nerves can occur from penetrating injury, surgery, and trauma from suboptimal placement of right-sided subclavian catheters or cardiac leads (4,5). Manifestations of phrenic nerve disease include diaphragmatic paralysis with elevation or persistent hiccups.

Phrenic nerve palsy secondary to benign thyroid enlargement is a rarely complication. Manning et al

(6) reported a case with phrenic nerve palsy without hiccups. The phrenic nerve may be stretched by a large goiter along its course in the neck, but the more likely, site of injury is the point at which it enters the thoracic cavity adjacent to the first rib.

In English literature, there is no hiccups developing due to the phrenic nerve stimulation by benign thyroid reasons (e.g.; inflammation, stretching) has been reported. Our case is the first in the literature.

The phrenic nerve may be stimulated due to intrathyroidal inflammation. And it is observed that, after the inflammation is treated, the hiccup disappears spontaneously.

In the control examination of the phrenic nerve was revealed that the right and left latencies prolonged and the amplitudes decreased as stable. These findings suggest that excitability of the phrenic nerve reduced in euthyroid period. In the literature there aren't any relationships with hyperthyroidism and hiccups.

We suggest that the cause of hiccups in our case is enlarged thyroid gland and/or inflammation. After non-specific therapy, the hiccups was disappeared and not repeated.

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