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# Sudden Death Due to Hashimoto's Thyroiditis :A Case Report and Literature Review

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***Abstract:*** *Sudden deaths caused by thyroid disease are rare in forensic pathology practice. Such deceased individuals often lack sufficient medical history to support their condition, and during the identification process, thyroid examination may be overlooked during autopsy. In August of a certain year, a student died at home. After a preliminary on-site investigation, homicide was ruled out. Through autopsy, it was confirmed that the cause of death was sudden death due to Hashimoto's thyroiditis.This case serves as a reminder that in some sudden death cases, especially those without relevant medical history before death, attention should be paid to thyroid examination, including histopathological examination, to avoid missed detection.*

***Keywords:*** *sudden thyroid death, Hashimoto's thyroiditis, Graves'disease*

## 1. Introduction

Female deceased, 14 years old. On the morning of August 31 of a certain year, around 5 a.m., the girl's mother saw her turn over when she got up. Around 6 a.m., when the girl's father called her to get up, he found she was unresponsive. He then called for rescue, and at 7:20 a.m., the doctor arrived and announced her death after attempts to save her.

## 2. Case presentation

### *2.1 Autopsy*

No hemorrhage was observed in the tissues beneath the scalp and bilateral temporal muscles, nor in the extradural, subdural, or subarachnoid spaces. Both sides of the brain exhibited congestion and edema, yet no damage or hemorrhage was detected on the surface or section of the brain, cerebellum, or brainstem. No skull fractures were identified. No bleeding was noted in the subcutaneous tissues or muscle groups of the neck. Bilateral goiter was present(Figure 1), with the left thyroid measuring 6.5cm×3.5cm×3.0cm and the right thyroid measuring 6.5cm×4.0cm×3.0cm(Figure 2). No fractures were observed in the hyoid bone, thyroid cartilage, or cricoid cartilage. The larynx showed congestion and mild edema, yet no foreign bodies were detected in the trachea or bronchial lumen. Scattered small patches of bleeding were observed in the tracheal mucosa. No bleeding was detected in the soft tissues of the chest wall, and no fractures were found in the clavicle, sternum, or ribs. There was 300mL of blood accumulation in the left thorax, while no blood accumulation or effusion was observed in the right thorax. The heart weighed 258.0g, with scattered punctate bleeding on the epicardium. A needle puncture was noted in the anterior wall of the left heart, with visible blood outflow. The wall thickness of the left ventricle was 1.2cm, while that of the right ventricle was 0.3cm.The left and right ventricular cavities are not dilated. The myocardium appears light red and has a medium texture. No abnormalities were detected in the heart valves. No significant abnormalities were observed in the coronary artery. Both lungs exhibited congestion, edema, emphysema, and scattered punctate hemorrhage. However, no masses or cavities were noted in the lung sections. The subcutaneous fat of the abdominal wall measures 3.0cm in thickness. There was no hematocele or effusion in the abdominal cavity, and no rupture of abdominal organs was observed. The gastric content, approximately 20mL, consists of

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brown liquid. The gastric mucosal folds are present, with extensive bleeding of the gastric mucosa but no ulceration or perforation noted. The intestinal serosa and mucosa are smooth, and no bleeding or necrosis was detected. The liver, spleen, pancreas, and both kidneys show congestion, with no visible damage on the surface or in the sections. There is bleeding at the tail of the pancreas. Additionally, a right ovarian cyst was observed. No injuries, bleeding, or fractures were detected on the back or spine.

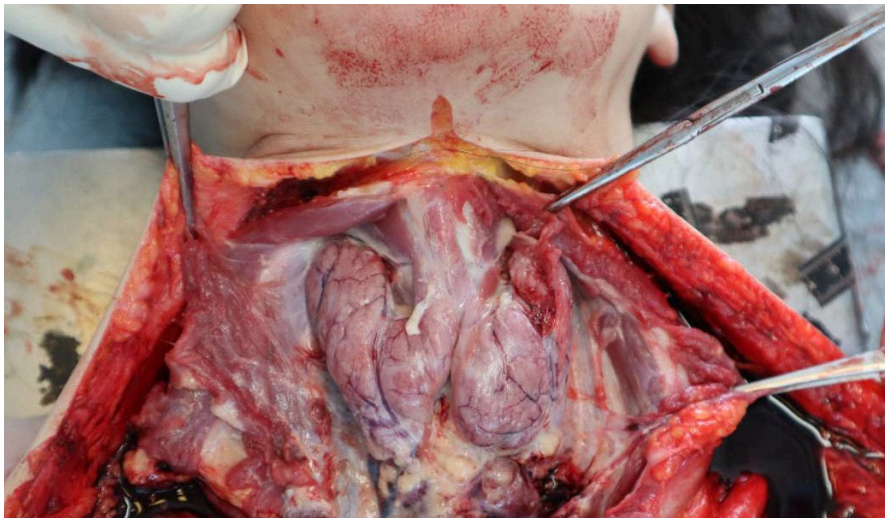

*Figure 1. Bilateral thyroid enlargement.*

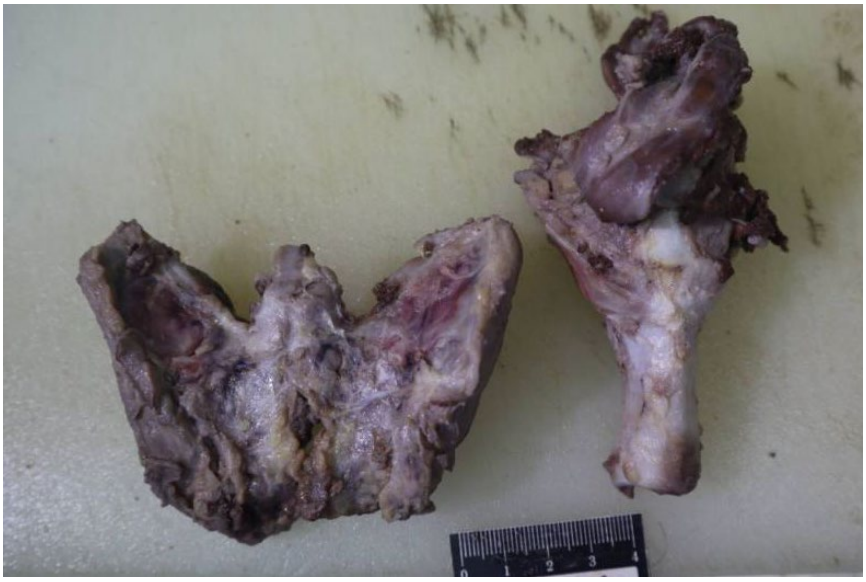

*Figure 2.Thyroid fixation with formalin.*

***2.2 Histopathological examination***

Subarachnoid small vessel congestion; The superficial cortex was loose, pyramidal cells swelled, and the pericellular and perivascular spaces widened. The subarachnoid space of the cerebellum exhibited small vessel congestion, the Purkinje cell layer was loose, the Purkinje cell nucleus disappeared, and the cytoplasm was red stained. Brain stem nerve cells swelled, the peripheral space widened, small blood vessels were congested, and the peripheral space widened. The epithelium of thyroid follicles became eosinophilic, interstitial lymphocytes infiltrated, part of thyroid follicles were destroyed, and glia decreased. Bronchiectasis,(Figure 3) congestion of small blood vessels in alveolar septum, thin alveolar wall, reduction of hair cell vascular bed, and expansion and fusion of alveoli. The bronchioles in the lower lobe of the right lung were infiltrated with lymphocytes. The interstitium of the upper and lower lobes of the right lung was widened, lymphocytes infiltrated, and no exudate was found in the alveolar cavity. The myocardial fibers were wavy, the myocardial longitudinal and transverse stripes were blurred, and eosin staining was enhanced. Lobules were still clear, a small amount of lymphocytes infiltrated in the portal area, and liver sinusoidal erythrocytes and Kupffer cells

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infiltrated. The red and white medulla were clear, the red blood cells in the splenic sinus were silted, and the sinus shore cells proliferated. The glomerulus exudes fibrin in the cystic cavity, the glomerular capillary cavity is empty, the proximal convoluted tubule is turbid and swollen, the medullary small vessels are congested, and no tubular casts were found. Focal autolysis of splenocytes, interstitial erythrocytes and plasma staining. The light yellow mucus adhered to the surface of gastric mucosa, and the small blood vessels in the submucosa were congested. Primary follicles in the ovarian cortex were scattered, and multiple cysts were seen, with light stained fluid gathering in the cystic cavity. The uterine glands are dense, the gland cells in the acini are columnar, and the interstitial red blood cells infiltrate in sheets.

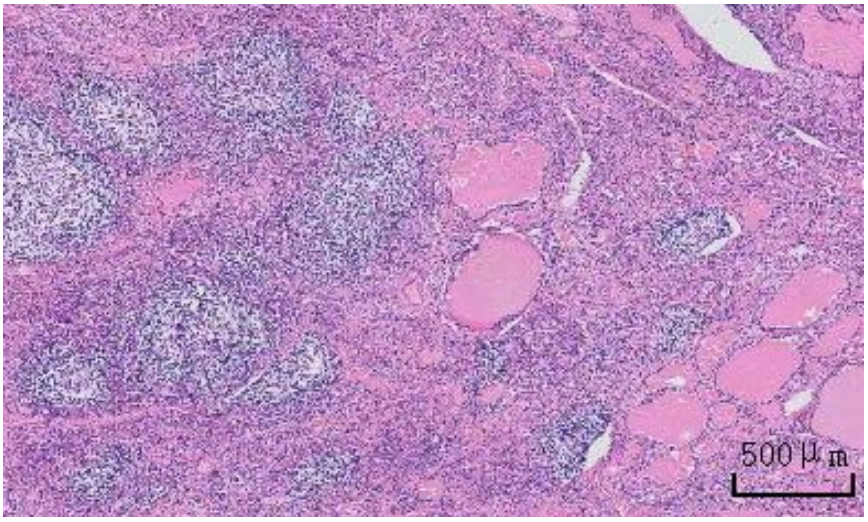


*Figure 3.Hashimoto's thyroiditis.*

### *2.3 Forensic pathological diagnosis*

1. Hashimoto's thyroiditis. 2. Peribronchiolitis; Interstitial pneumonia; Emphysema. 3. Myocardial ischemia (Heideihain staining); Fatty infiltration in the sinoatrial node area. 4. Spleen congestion. 5. Liver, kidney, and stomach congestion (mild). 6. Ovarian cysts. 7. Secretory endometrium. 8. Interstitial plasma infiltration of pancreas (dying or post-mortem changes).

## 3. Discussion

Hashimoto's Thyroiditis, also referred to as Chronic Lymphocytic Thyroiditis or Autoimmune Thyroiditis, is an autoimmune disorder affecting the thyroid gland. While its precise etiology remains incompletely understood, it is associated with genetic, environmental, and epigenetic factors [1]. Over the past three decades, the incidence of Hashimoto's Thyroiditis has escalated significantly[2]. Presently, it ranks among the most prevalent thyroid disorders[3]. Upon physical examination of patients with Hashimoto's Thyroiditis, the thyroid gland typically appears pale in color and firm in texture; it feels rubbery and exhibits a nodular surface with well-defined boundaries and an intact capsule. Histopathological analysis reveals diffuse chronic lymphocytic infiltration along with lymph follicle formation featuring a central focus. Additional findings may include varying degrees of fibrosis, tissue atrophy, and large follicular cells rich in eosinophilic granules—commonly known as Hurthle cells or Askanazy cells. In this particular case report involving a deceased individual who exhibited cyanosis of the lips and nail beds on both hands alongside bilateral thyroid enlargement: there were also scattered punctate hemorrhages observed within the pericardium. The lungs displayed signs of congestion, edema, and hemorrhage bilaterally; multiple organs showed diffuse congestion accompanied by edema and hemorrhage. Microscopic examinations confirmed features consistent with Hashimoto's Thyroiditis as well as bronchiolitis interstitial pneumonia and pulmonary edema. Additionally noted were myocardial ischemia along with fatty infiltration within both atrial walls and ventricular septum. No evidence was found indicating mechanical trauma or exposure to common toxic substances; these findings are consistent with sudden death attributed to Hashimoto's Thyroiditis.

In the existing literature, there have been sporadic reports of sudden death associated with Hashimoto's thyroiditis. Edston documented three cases of mortality linked to chronic thyroiditis, all

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involving young to middle-aged men. These fatalities occurred rapidly. Two patients had no significant past medical history, while the third had well-controlled Addison's disease. All three exhibited severe chronic thyroiditis characterized by extensive tissue destruction and follicular epithelial hyperplasia upon microscopic examination. The first patient presented with elevated triiodothyronine(T3) and tetraiodothyronine(T4) levels alongside low thyroid stimulating hormone(TSH); the second displayed low T3 and T4 levels with normal TSH; and the third had elevated T3 levels. Additionally, two of these patients tested positive for anti-thyroid antibodies[4].

Lorin et al. reported a case of sudden death attributed to Hashimoto's disease in conjunction with myocarditis. The deceased was a 40-year-old man who was found unresponsive on his sofa at home early one morning, with no prior medical history documented. Autopsy findings revealed increased weights of both the thyroid and heart, while histopathological analysis indicated lymphocytic myocarditis and lymphocytic thyroiditis. Postmortem biochemical examination demonstrated a slight elevation in TSH levels, alongside normal blood concentrations of T3 and T4, suggesting hypothyroidism[5]. Takahashi et al. described a case of death resulting from congestive heart failure due to thyroid storm associated with chronic lymphocytic thyroiditis. The deceased was a Japanese woman in her 30s who was discovered lifeless on a mattress. She had experienced fever, cough, and dyspnea for approximately two weeks preceding her death. Autopsy revealed mild enlargement of the thyroid gland, which histopathology identified as chronic lymphocytic thyroiditis. Blood biochemical analysis showed an extremely elevated concentration of T3 while the TSH level fell below detectable limits; anti-thyroid tissue autoimmune antibodies were also positive[6]. Radojevic et al. presented a case involving sudden death linked to Hashimoto's thyroiditis accompanied by thymic hyperplasia. The deceased was a 23-year-old male who died unexpectedly while cleaning his yard early one morning; he had no significant medical history noted prior to this event. Autopsy findings included an enlarged spleen and thymus consistent with thymic lymphoid constitution, along with other abnormal physical signs such as partial eyebrow loss, alopecia areata, and patent foramen ovale. Histopathological examination confirmed the diagnosis of Hashimoto's thyroiditis[7]. In summary, deaths associated with Hashimoto's thyroiditis occurred rapidly in these cases; some individuals exhibited no clinical symptoms prior to their demise. Histopathological examinations successfully identified corresponding changes within the thyroid tissue pathology across various cases examined post-mortem—biochemical tests occasionally indicated abnormal levels of thyroid hormones as well. Following autopsies that included histopathological assessments and toxicology testing, no overt signs indicating cause of death were discerned.

Therefore, when performing autopsies on corpses with unknown causes of death, we need to understand the pathogenesis and clinical manifestations of related diseases, pay attention to collecting relevant case details and medical history, and understand specific physical signs (emotional disorders, sleep disorders, memory loss, etc.). At the same time, we must not overlook the extraction of endocrine gland organs. Timely extraction of blood for biochemical examination has certain diagnostic significance. Literature indicates that the level of T4 in blood begins to decrease after 2.75 hours after death, and the content of TSH in serum remains stable for at least 24 hours after death[8,9,10].The T3 value changes greatly and cannot be used for auxiliary diagnosis.

## 4. Conclusion

Sudden deaths from thyroid diseases often occur swiftly and lack specific alterations and diagnostic markers. Hence, it's crucial to pay attention to thyroid examination and extraction during routine autopsies. The forensic examination presented in this case serves as a reference for handling similar causes of death.

## Abbreviations

T3=triiodothyronine;

T4=tetraiodothyronine;

TSH=thyroid stimulating hormone

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