

## CASE REPORT

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### Thyrotoxicosis: Postmortem Diagnosis in an Unexpected Death

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Thyrotoxicosis, the clinical manifestation of extreme thyroid gland hyperfunction, is readily diagnosed and amenable to therapy. Although physicians recognize the lethal potential of thyrotoxicosis, it does not appear as a diagnosis in several series of unexpected deaths [1,2]. Coe [3], in 1973, discussed the difficulties in making this diagnosis postmortem. This report presents a case of clinically unrecognized thyrotoxicosis presenting as an unexpected death.

#### Case Report

The subject, a 24-year-old Caucasian male, was found dead, lying across the bed in his residence, fully clothed. Although the apartment was in considerable disarray, there was no evidence of foul play, and money was found on his person. He had last been seen alive six days earlier when he had talked with a neighbor. The degree of postmortem change was consistent with his having been dead in a warm room for five or six days. No medical history was discovered. He had been a university graduate student in good academic standing until about one year prior to death. After leaving the university he had been employed as a service station attendant. His driver's license, issued the previous year, recorded his weight at 175 lb (79 kg).

At autopsy the subject weighed 80 lb (36 kg). Cachexia was extreme. Subcutaneous fat was scant. Old white striae were present over the iliac crests, consistent with the subject having been considerably heavier at one time. Postmortem changes included diffuse greenish discoloration, mummification of the hands and feet, and maggot infestation of the head. The only significant anatomic abnormality, other than cachexia, was a symmetrically enlarged thyroid gland weighing 87 g.

Analysis of cardiac blood revealed a protein-bound iodine (PBI) level of 71.2  $\mu\text{g}/\text{dl}$  (normal range, 4.0 to 8.0  $\mu\text{g}/\text{dl}$ ) and  $T_4$  (competitive protein binding) of 36.0  $\mu\text{g}/\text{dl}$  (normal range, 5 to 15  $\mu\text{g}/\text{dl}$ ). Although there was considerable autolytic change, microscopic sections of the thyroid gland exhibited a pattern of diffuse hyperplasia. Given the extreme cachexia, prominent thyroid gland hyperplasia, and the chemical evidence of marked thyroid gland hyperactivity, the cause of death was certified as being thyrotoxicosis.

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### Discussion

Untreated severe hyperthyroidism is certainly uncommon, and death from thyrotoxicosis is rare. The prolonged hypermetabolism terminates with cardiac arrhythmias and heart failure, and PBI levels in excess of 50  $\mu\text{g}/\text{dl}$  occur. Since exogenous sources of iodine can falsely elevate the PBI, a more specific assay for thyroid hormone is needed to confirm the diagnosis.

In this case, the PBI was markedly elevated and the  $T_4$ , which measures total serum thyroxine by competitive protein binding, was in the hyperthyroid range. There is evidence that  $T_4$  values tend to fall after death [3], although the effects of specimen decomposition on the assay system are not known. The chemical values in this case are not in themselves diagnostic; however, they strongly support the anatomic diagnosis of marked thyroid hyperplasia and the conclusion that death was caused by thyrotoxicosis.

Thyrotoxicosis should be considered in the differential diagnosis of unexpected death, especially of young and middle-aged adults with enlarged thyroid glands, thyroid nodules, or otherwise unexplained weight loss.

### References

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