Death due to primary hyperparathyroidism

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Asymptomatic hypercalcemia is a common metabolic disorder, usually detected incidentally on routine biochemical screening. The most common etiologies are primary hyperparathyroidism (PHPT) and cancer. Primary hyperparathyroidism is characterized by excessive parathyroid hormone (PTH) secretion due to parathyroid adenoma (80% of cases), hyperplasia (15-20%), and carcinoma (1-2%). Primary hyperparathyroidism is usually mild and asymptomatic. Only a few patients with PHPT develop hypercalcemic crisis, a medical emergency characterized by severe hypercalcemia with serum calcium concentrations above 3.8 mmol/L and marked symptoms and signs of severe calcium intoxication. The symptoms are frequently nonspecific with gastrointestinal, renal, neuromuscular, cardiovascular, and central nervous system dysfunctions.[1] Here, we report a case of extreme hypercalcemia (serum calcium level of 6 mmol/L) associated with parathyroid adenoma, the patient died due to a cardiovascular complication (arrhythmia) of hypercalcemia.

A 45-year-old woman was admitted to our emergency department with a 20-day history of nausea, anorexia, vomiting, and muscular weakness. She denied any previous illness. There was no family history of any illness. On admission, she appeared dehydrated and extremely tired, almost apathetic, but had no focal neurological signs. Biochemical assays revealed extreme hypercalcemia, 24 mg/dL (6 mmol/L, normal range 2.2-2.6), confirmed by repeated measurements. Serum creatinine and urea levels were mildly elevated, indicating a slight renal affection. Electrocardiography showed a sinus tachycardia with a normal axis and QT interval. She received intravenous normal saline, loop diuretics, bisphosphonate, and calcitonin to decrease the calcium concentration. The initiated treatment was not successful to reduce the serum calcium level, and hemodialysis was performed. Her PTH level was found to be significantly elevated, 5670 pg/mL (normal range 10-65). Ultrasound examination revealed a parathyroid adenoma measuring 5 cm in diameter, with a cystic component. Surgical exploration was planned, but she died before operation due to a cardiovascular complication (arrhythmia).

Hyperparathyroid crisis, first documented in man in 1939, is a rare but often fatal condition.[2] Although the mortality rate has been reduced lately, as a result of earlier diagnosis and better intensive care management, patients are still dying due to inappropriate symptomatic treatment before surgical intervention or delay in surgery.[3] Our patient was admitted to the hospital with a short duration. She presented with all the typical symptoms and signs of a hypercalcemic syndrome. The highly elevated PTH concentration and the mass lesion that were found on the ultrasonography confirmed the diagnosis of PHPT. There is no clear correlation between serum calcium level, symptoms and mortality except at a very high calcium levels. In the literature, we found only few cases with high calcium levels (6.9-7.6 mmol/L) and resulted in death.[4] In cases of benign parathyroid disease, it is often unclear what leads to the exacerbation of the condition. Sometimes, rapid growth with partial necrosis or infarction of the parathyroid adenoma may be the reason. Acute cystic degeneration of preexisting parathyroid adenoma can also result in marked acute elevations of serum PTH and calcium levels.[1] We think that this also occurred in our case. As medical therapy alone rarely restores normocalcemia, surgical exploration of the neck and resection of the hyperfunctioning parathyroid tissue should be performed without further delay.[5] We also planned to operate on the patient, but she died before operation.

In summary, despite all advances in this field, hypercalcemic crisis in hyperparathyroidism still carries a significant risk of mortality, especially in patients with extremely high serum calcium levels.

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