

## ACUTE SEVERE HYPERCALCEMIA: RAPID ONSET EPISODE IN PARATHYROID CARCINOMA

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Acute, severe hypercalcemia, usually defined as a serum calcium concentration greater than 14 mg/dl, is quite unusual, because most patients with hypercalcemia have PHPT, in which hypercalcemia is usually chronic and mild. Episode of acute, severe hypercalcemia may occur in PHPT while precipitating conditions are associated, in underlying malignancy, or in parathyroid carcinoma. Anyway the progression from slight to markedly severe hypercalcemia is generally slow, except for the evenience of rapidly progressive bone metastases.

P.B. 75 years, was admitted in our emergency room because of the onset of lethargy and subsequently of coma. This set of condition had started 24 hours before. Patient's past history was unremarkable, except for the presence of a mild congestive heart failure. Renal function was normal. Because of a short-lasting dyspnoic episode, the day before the admission the patient underwent a routine evaluation, in which a value of calcemia = 10.9 mg/dl was detected. In the previous years routine analysis were within the normal range. Laboratory evaluation in our hospital at the admission revealed: calcemia = 22 mg/dl, other parameters were normal. No precipitating factors were identified in patient history. Despite every therapeutic attempt (rehydration, diuretics, calcitonin, bisphosphonates, corticosteroid, dialysis), calcemia reached the level of 26 mg/dl and the patient died one day after admission. PTH levels were >1000 ng/dl. Histology revealed a huge mediastinal mass (Ø 8 cm), reported as parathyroid carcinoma. At the macroscopic appearance, a soft, brownish red nodule, apparently encapsulated, with hemorrhagic areas was noted. At the microscopic appearance, cellular elements, in trabecular rearrangement, seldom interrupted by fibrotic tissue, with capsular and vascular invasion. Cellular elements showed nuclear pleiomorphism and increase of mitotic index.

Parathyroid tissue abnormal growth does not primarily lead to space-occupying and pressure effect (such as for pituitary and thyroid glands), but it can be revealed only by abnormalities of human secretion. On the other hand, the molecular and genetic basis of the relationships between abnormal parathyroid cell growth and secretion (a proliferative defect and a set-point abnormality) still need to be elucidated.