Clinical Reports

Fatal hyperparathyroid crisis

C.A. Keeling, M.J. Abrahamson and D.G.J. Harloe

Departments of Medicine and Anatomical Pathology, Groote Schuur Hospital and University of Cape Town, Observatory 7925, Cape Town, South Africa

Summary: A case of hyperparathyroid crisis presenting with a serum calcium level of 7.6 mmol/l is presented. The rarity and importance of recognizing the condition early is emphasized.

Introduction

Hyperparathyroid crisis, first documented in man in 1939, is a rare but often fatal condition complicating primary hyperparathyroidism, and often presents acutely with weakness, nausea, vomiting, an altered level of consciousness, elevated serum calcium and circulating parathyroid hormone (PTH) levels. A patient who presented with this syndrome and who was found to have a serum calcium of 7.6 mmol/l is discussed. We believe that this level of serum calcium due to a hyperparathyroid crisis is the highest yet recorded.

Case report

A 35 year old female presented with a four day history of abdominal pain, nausea, vomiting and increasing drowsiness. There was no previous medical history of note. Examination revealed an acutely ill woman who was dehydrated, pale (haemoglobin 10 g/dl), and drowsy. Blood pressure was 130/70 mmHg and the pulse was 120/minute and regular. A third heart sound was audible at the apex, but there were no other signs of congestive cardiac failure. Diffuse abdominal tenderness, marked meningism and generalized hypotonia were present, but there were no focal neurological signs.

Initial laboratory investigations revealed a serum calcium level of 7.6 mmol/l (normal 2.1–2.6), phosphorous 1.25 mmol/l (0.8–1.4), alkaline phosphatase 96 units (30–115), sodium 142 mmol/l (135–145), potassium 2.6 mmol/l (3.5–5.5), chloride 101 mmol/l (97–107), bicarbonate 27 mmol/l (22–30), urea 16.4 mmol/l (1.7–6.7), creatinine 167 µmol/l (75–115), total protein 86 g/l (60–80), albumin 37 g/l (35–50). An electrocardiograph showed a sinus tachycardia with normal axis and QT interval. X-rays of the chest, skull and abdomen were normal and showed no evidence of metastatic calcification. Lumbar puncture produced normal cerebrospinal fluid.

The patient was rehydrated and received routine hypocalcaemic agents including mithramycin and corticosteroids. Within 6 hours, the serum calcium level had dropped to 3.5 mmol/l. However, she became anuric, progressively hypotensive and stuporosed, and died 12 hours after admission.

Post-mortem examination revealed a parathyroid adenoma measuring 2 cm in diameter within the capsule of the lower pole of the right thyroid lobe. The 3 remaining parathyroid glands were small and atrophic. Metastatic calcification of the intracardiac vessels, myocardium, and walls of some central veins and reticulum fibres of the liver was noted. Nephrocalcinosis and evidence of early osteitis fibrosa cystica were also present.

Discussion

Hyperparathyroid crisis, a rare condition noted for its acute presentation and high mortality, was first described in 1939 by Hanes. Numerous reports have appeared since then emphasizing the need for immediate reduction of serum calcium levels with calcium-lowering agents, and immediate operative intervention if a prompt response to medical therapy is not obtained. In some series the mortality in unoperated cases approaches 100%, but with successful parathyroidectomy, mortality has been reduced to 24% or lower.

It appears from the literature that serum calcium
levels in patients presenting with hyperparathyroid crisis have been only as high as 5 mmol/l. We believe that the serum calcium level of 7.6 mmol/l in our patient is the highest yet recorded for this condition, and serves as a reminder that such high serum calcium levels are not only found in malignant conditions.

This case report also illustrates the importance of excluding rare metabolic conditions, such as this, in patients presenting with diminished level of consciousness, nausea, vomiting, abdominal pain, weakness and dehydration. Early diagnosis and prompt effective therapy contribute to improving the prognosis.

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References

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