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The Parathyroid Glands and Hyperparathyroidism: II

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I. INTRODUCTION

This manual is the second of a 2-part review on the parathyroid glands and hyperparathyroidism. The second part focuses on diagnosis and surgical treatment of hyperparathyroidism. A case patient is presented to illustrate important principles in the management of patients with hyperparathyroidism. The first part focused on embryology, anatomy, histology, and cell physiology of the parathyroid glands (*Hospital Physician General Surgery Board Review Manual*, Volume 6, Part 4).

II. CASE PATIENT I

PRESENTATION

A 57-year-old man is found to have hypercalcemia on blood work obtained before a laparoscopic cholecystectomy for symptomatic cholelithiasis. His medical history is pertinent for a known hiatal hernia, hypertension, gout, hypercholesterolemia, and peptic ulcer disease. He denies any history of bone problems, kidney stones, pancreatitis, neuromuscular or behavioral abnormalities, or head or neck irradiation. There is no family history of thyroid, parathyroid, pituitary, pancre-

atic, or adrenal diseases. In addition to his cholecystectomy, he has previously undergone left knee surgery for a fractured patella.

Physical examination reveals a blood pressure of 128/78 mm Hg and heart rate of 100 bpm. He has no palpable neck masses, and the rest of his physical examination is unremarkable. Laboratory studies reveal a serum calcium of 12.3 mg/dL, serum phosphorus of 2.3 mg/dL, chloride level of 101 mEq/L, alkaline phosphatase level of 88 IU/L, and intact parathyroid hormone (PTH) level of 113 pg/mL (normal, 10 to 65 pg/mL).

- What is the most likely cause of patient 1's hypercalcemia?

DISCUSSION

Primary Hyperparathyroidism

Primary hyperparathyroidism is one of the most common causes of hypercalcemia, accounting for 50% to 60% of cases diagnosed in the ambulatory setting and up to 27% diagnosed in the hospital.¹ Primary hyperparathyroidism and malignancy account for 80% to 90% of all causes of hypercalcemia,¹ although many other uncommon causes do exist (**Table 1**). Automated laboratory methods introduced in the late 1960s and early 1970s led to a dramatic increase in the recognition of this disorder. Approximately 100,000 people are newly