Presentation

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PHPT is defined as hypercalcaemia in the presence of an unsuppressed and therefore relatively , or absolutely , elevated PTH level. Prevalence of the disease is reported to be 0.2–0.5%, with approximately 100 /uni00A0 000 new cases per year in the USA. The majority of PHPT is sporadic in nature. Familial disease can occur in multiple endocrine neoplasia (MEN) type 1 or type 2A or as a familial cluster. Patients usually present in the fifth or sixth decades and there is a female predominance with a ratio of 3:1. Patients are typically identified incidentally with an elevated total calcium or following routine assessment of bone densitometry (DEXA scan). Most patients will, however some vague constitutional symptoms, such as fatigue, muscle weakness, depression or some mild memory impairment on questioning. The presence of kidney stones remains the most common clinical manifestation of symptomatic PHPT . Between 15% and 20% of patients will have nephrolithiasis and over 40% of patients will have hypercalciuria. Increasingly , postmenopausal women present with significant osteopenia or osteoporosis in the distal one-third of the radius with a minimal reduction in the lumbar spine, which prompts further investigation. This distribution arises as PTH appears to be catabolic at cortical sites (distal one-third of the radius) and anabolic at cancellous sites (lumbar spine). PHPT may present with pancreatitis, although it is rarely seen in patients with milder forms of the disease. Common epidemiologically linked disorders, such as Sir James Paget , 1814–1899, surgical pathologist, Royal College of Surgeons of England. hypertension and peptic ulcer disease, are often encountered. Clinical examination is usually normal. Band keratopathy , pathognomonic of the disease and due to deposition of calcium phosphate crystals in the cornea, is now rarely identified. The di ff erential diagnosis of PHPT includes other causes of hypercalcaemia, which are usually readily distinguishable (Table 56.1). It is important to e xclude the presence of a widespread malignancy , in which patients will typically have other symptoms. The exception to this rule is multiple myeloma, in which hypercalcaemia can be the presenting /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF , have /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Plasma Ca Parathyroid Parathyroid concentration hormone glands 2+ Activation of Renal tubular Ca mobilised vitamin D absorption from bones 2+ of Ca 2+ Ca absorption in intestine 2+ Plasma Ca concentration Figure 56.2 The actions of parathyroid hormone. TABLE 56.1 Causes of hypercalcaemia. Endocrine Primary hyperparathyroidism Thyrotoxicosis Phaeochromocytoma Renal failure Secondary hyperparathyroidism Tertiary hyperparathyroidism Malignant Skeletal metastatic disease disease Multiple myeloma, lymphoma, leukaemia Solid tumours (PTH-related peptide mediated): lung, renal, squamous cell carcinoma of the head and neck, oesophagus, genital tract Nutritional Excessive vitamin D ingestion Vitamin A intoxication Milk-alkali syndrome Aluminium intoxication Granulomatous Sarcoidosis Tuberculosis Inherited disease Hypercalciuric hypercalcaemia Immobilisation Paget’s disease Drug related Lithium PTH, parathyroid hormone.

immunochemiluminometric assays for PTH can help to distinguish these conditions, as in malignancy PTH levels are typically suppressed. Presentation

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