

Pathology

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The underlying aetiology of PHPT is usually a solitary parathyroid adenoma; however, in a small number of patients (2–4%) there are double adenomas. It may occur in a sporadic fashion or it can be familial (approximately 10%) (MEN type 1, type 4, type 2A or hyperparathyroidism–jaw tumour syndrome [HPT-JT]) in nature. John L Doppman, 1928–2000, radiologist, National Institutes of Health, USA, developed the technique of selective venous sampling for parathyroid localisation. is a history of prior neck irradiation. The underlying genetic pathogenesis of PHPT remains unclear. However, genes regulating the cell cycle, such as MEN1 and CCND1, have been recognised as playing an important role owing to the clonal nature of adenomas. Somatic mutations in MEN1, which encodes menin, occur in 12–35% of sporadic cases and rearrangements or overexpression of CCND1, which encodes cyclin D1, have been demonstrated in 20–40% of patients. Upregulation of cyclin D may lead to a clonal proliferation within the parathyroid glands. This does not alter the set point of calcium but the hyperplastic nature of the parathyroid cells - themselves causes excessive secretion of PTH. Multigland disease is less common, occurring in approximately 15% of patients. No clinical features differentiate single from multigland disease, although multigland disease is more commonly associated with familial syndromes such as MEN types 1 and 2A, as well as the chronic ingestion of lithium. Pathology

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