

Practice Point

Clinical Practice Guidelines for the Management of Primary Hyperparathyroidism

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The aetiology of hypercalcemia is classified into two distinct categories: PTH-dependent (iPTH > 20 pg/ml) and independent (iPTH < 20 pg/ml)¹. PTH-dependent hypercalcemia can be caused by primary hyperparathyroidism (PHPT), tertiary hyperparathyroidism, lithium therapy, and familial hypocalciuric hypercalcemia (FHH). PHPT is the most common cause of PTH-dependent hypercalcemia. FHH can be distinguished from PHPT as the former is characterised by a 24-hour urinary calcium level of less than 100 mg/day and a strong family history. Sporadic PHPT cases represent about 90-95% of all cases, resulting from single parathyroid adenoma in 80% of cases and multiple gland involvement which is generally hyperplastic in 20% of cases². The remaining 5-10% of cases occur within familial inherited parathyroid disorders such as MEN1, MEN2A, MEN4, FHH, neonatal severe hyperparathyroidism (NSHPT), HPT-jaw tumour (HPT-JT) [CDC 73] mutation and familial isolated PHPT (FIPH).

PHPT is the third most common endocrine disorder in the West after diabetes mellitus and thyroid disorders³. Overall, about 1% of adults have PHPT in the West, and this figure rises to 2% in people older than 55 years. PHPT in India for the most part continues to be a symptomatic disease with skeletal, renal, pancreatic and cardiac manifestations with high serum calcium and PTH levels^{4,5,6}.

The management of PHPT can be described in 4 steps:

Step1 (Biochemical diagnosis): A diagnosis of PHPT is

established by the presence of persistent hypercalcemia (two albumin-adjusted serum calcium values > than upper limit of normal) and concomitant raised or inappropriately normal serum intact parathyroid hormone (iPTH). Biochemical evaluation also includes estimation of serum alkaline phosphate (ALP), serum phosphate and 24-hour urinary calcium.

Step 2 (Assessment of classical organ complications): After a biochemical diagnosis of PHPT, the patient should be assessed for classical organ complications. The two classical organ complications of PHPT include skeletal and renal disease. The skeletal disease is diagnosed by radiological survey (evidence of sub-periosteal resorption, fractures, salt and pepper appearance of the skull or brown tumour) and three site bone mineral density including lumbar spine, dual femur, distal radius (evidence of osteopenia or osteoporosis). Renal ultrasonography is performed for the diagnosis of renal disease in the form of nephrolithiasis and/or nephrocalcinosis. In addition, 24-hour urinary calcium and creatinine are estimated for evidence of hypercalciuria. In some patients, echocardiography is performed to assess diastolic and systolic dysfunction and aortic valve, mitral annular, septal and myocardial calcifications⁵.

Step 3 (Localization of parathyroid adenoma): After the biochemical diagnosis and assessment of classical organ

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complications, the next step is localization of parathyroid adenoma (PA). Localization techniques include USG neck, technetium-99m (99m Tc) sestamibi scan (MIBI) and in cases with negative or discordant USG and 99mTc, 4D-computerised tomography (CT) neck.

Step 4 (Parathyroid surgery): Patients in whom parathyroid adenoma is localized on two localization techniques, are referred to an endocrine surgeon for focused parathyroidectomy (PTX). Patients with doubtful lesions or nonlocalized lesions are referred for bilateral neck exploration.

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Figure 1: Flowchart for management of PHPT

