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Surgery for secondary hyperparathyroidism

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Secondary hyperparathyroidism is a systemic metabolic derangement that leads to massive expansion of parathyroid tissue and overproduction of parathyroid hormone. Accurate biochemical diagnosis is crucial to establishing a plan of care for these patients. Indications and timing of surgical intervention once refractory to medical management are best established through multidisciplinary care including an expert parathyroid surgeon. Principles of careful parathyroid surgery including identification of all parathyroid tissue in a bloodless field allow for the appropriate management of parathyroid excision. Subtotal resection of parathyroid tissue and total parathyroidectomy with immediate parathyroid autotransplantation are equivalent options. Hypocalcemia is the most common postoperative occurrence and should be anticipated and managed aggressively.

Introduction

Homeostatic control of calcium level is centered in the parathyroid glands. In the normal state, 4 small glands, totaling 200 mg of tissue and residing in the central neck, are responsible for regulating calcium level in the blood in response to stimuli such as serum calcium level, serum phosphorous level, and serum vitamin D level.[1] This careful balance allows total and ionized calcium levels to remain in a rather tight concentration range, and thus bodily functions such as nerve conduction, muscle action potentials, bone metabolism, and coagulation, to name just a few, proceed in a normal fashion.

Overactivity of the parathyroid glands can either be driven from autonomy of the parathyroid cells or from stimulation from a dysregulation in calcium, phosphorous, or vitamin D. Parathyroid autonomy, in the form of single or multiple gland neoplasms, may be sporadic or driven by an inherited genetic mutation. This is known as primary hyperparathyroidism.[2] This is to be distinguished from the physiologically appropriate, though possibly exuberant, response of the parathyroid glands to alterations in the controlling factors of parathyroid hormone gene transcription and translation. The latter is termed secondary hyperparathyroidism. That is, the generation of hyperparathormonemia in response to altered systemic levels of determinants of parathyroid hormone production.[3]

Secondary hyperparathyroidism may result from any number of causes of low serum calcium. Renal tubular handling of calcium and phosphorous may become dysregulated such that there is a leak of calcium into the urine, or a retention of phosphorous. Either of these circumstances may secondarily increase circulating levels of parathyroid hormone. Finally, decreased bodily stores of vitamin D, either through malabsorptive states, decreased integumentary exposure to ultraviolet light, or decreased liver and especially renal production of metabolically active analogues of vitamin D can lead to higher levels of parathyroid hormone synthesis and secretion.[3]

Many pathophysiologic states leading to a secondary hyperparathyroidism are transient or remediable without the need for surgery. The prototypical example is vitamin D3 supplementation to remedy hypovitaminosis D–induced

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secondary hyperparathyroidism. Renal failure in contrast, however, may often not be correctable. The physiologic parameters seen with kidney dysfunction, such as hypocalcemia, hyperphosphatemia, and hypovitaminosis D, lead to, sometimes severe, secondary hyperparathyroidism. Those clinicians managing renal failure, most notably nephrologists, possibly in combination with endocrinologists, would often try multiple pharmaceutical agents in an attempt to bring down the secondary overproduction of parathyroid hormone. These drugs include vitamin D supplements (cholecalciferol and calcitriol), phosphate binders, and calcimimetic agents. It is often necessary, however, to perform a parathyroid resection as temporizing or permanent therapy for renal failure–induced secondary hyperparathyroidism.

Preoperative Diagnosis and Operative Indications

The initial consideration of parathyroid surgery for secondary hyperparathyroidism must commence with confirmation of the biochemical diagnosis. Parathyroid disorders are a diagnosis confirmed with laboratory data rather than imaging findings. Secondary hyperparathyroidism is diagnosed with an elevated parathyroid hormone in the setting of normal or low total serum or ionized calcium. The phosphorous levels are often elevated, and the vitamin D levels are low. It is not at all uncommon for the parathyroid hormone levels to be 10-80 times the upper limit of normal. In fact, in the absence of severe hypercalcemia, which could indicate a parathyroid carcinoma, only renal failure–induced secondary hyperparathyroidism produces parathyroid hormone levels of that magnitude.

Once the diagnosis of secondary hyperparathyroidism has been secured, a multidisciplinary discussion among the parathyroid surgeon, the nephrologist, and the endocrinologist most often yields the optimal patient care planning. Need for and timing of parathyroidectomy may be influenced by the possibility of renal transplantation. Further, while the sheer magnitude of parathyroid elevation itself may not drive indications for operation, it is unlikely that a parathyroid hormone level much beyond 10 times the upper limit of normal would respond favorably to medical intervention.[4] Other indications (Table 1) for parathyroidectomy as therapy for secondary hyperparathyroidism include evidence of progressive bone mineral density loss, metastatic vascular calcium-phosphate deposition leading to ischemic ulcers (calciphylaxis), bone fractures or tendon tears, severe myalgias or arthralgias, and severe uremic pruritus.[4]

Table 1 Indications for parathyroidectomy in secondary hyperparathyroidism

Failure or intolerance of medical therapy
Hypercalcemia
Intact parathormone > 800 pg/mL
Calciphylaxis
Osteoporotic/fragility/adynamic bone fracture
Tendon rupture
Unrelenting arthralgia or myalgias
Severe uremic pruritus

Preoperative Care

Preoperative cardiopulmonary risk assessment is necessary for all patients planned for parathyroidectomy for renal failure–related secondary hyperparathyroidism. Comorbid conditions are prevalent in this patient population, and medical optimization may allow avoidance of perioperative complications. Perioperative volume status must be carefully planned with dialysis timing in the preoperative setting mapped out in advance. Calcitriol supplementation, if not already on it, for at least 2 days before operation can potentially mitigate some of the anticipated postoperative hypocalcemia. Discussion, too, with the individual patient about potential prolonged hospitalization after operation is imperative given the significant possibility of severe hypocalcemia in the near term following the operation.

Imaging and Preoperative Localization

Secondary hyperparathyroidism is by its pathophysiologic nature a multiglandular parathyroid dysfunction. In this setting, then, minimally invasive operations that identify less than all 4 parathyroid glands play no role.[5] As such, the utility of preoperative parathyroid localization scans has been called into question.[6] Regardless of the extent of resection planned (to be discussed below), a bilateral neck exploration must be undertaken. High-resolution cervical ultrasonography may be performed by surgeon or radiologist in an attempt to identify the parathyroid glands preoperatively, though sidedness and conduct of the operation are not going to be significantly altered by the sonogram.[7] Ectopic or supernumerary parathyroid glands are unlikely to be identified with ultrasound given the limitations of the technique.

Nuclear medicine parathyroid scanning may be employed to identify the multiple parathyroid tumors, though this modality suffers decreased performance in a multiglandular setting.[8] A distinct advantage of nuclear medicine parathyroid scans is the ability to identify ectopic or supernumerary glands, of which preoperative knowledge can minimize the chances of operative failure or early disease recurrence.

Surgical Management

Parathyroidectomy is a functional surgical procedure to remove overactive parathyroid tissue. The extent of resection is dependent upon the specific parathyroid disease process. Secondary hyperparathyroidism is uniformly characterized by hyperplasia of all endogenous parathyroid tissue.[3] Therefore, the aim of any surgical approach for secondary hyperparathyroidism is to render the patient nearly euhormonal,