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Surgical Treatment of Tertiary Hyperparathyroidism: The Choice of Procedure Matters!

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In their recent study, Schlosser et al. [1] discussed the effect of parathyroidectomy (PTX) on kidney graft function in patients with tertiary hyperparathyroidism (tHPT). They observed a permanent decrease in glomerular filtration rate (GFR) in some patients, more relevant in those with more extensive parathyroid resection and more elevated preoperative levels of creatinine. They stated that subtotal PTX is the procedure of choice and that the risk of PTX must be weighed against the benefit of normalizing serum calcium by surgery. We think that both statements should be analyzed with caution.

After the correlation between decreased renal function and the drop of parathyroid hormone (PTH) was described [2], we reviewed our own cases of tHPT and primary hyperparathyroidism (pHPT). A mean transient creatinine increase of 39.5% occurred in tHPT. This mean was not different from that observed in pHPT (+30.8% increase), while kidney-transplanted patients undergoing nonparathyroid-related head and neck procedures did show a slight decrease in creatinine levels postoperatively [3]. In the same study we observed a mean increase in creatinine of +18.7% in patients undergoing thyroidectomy. When these patients were stratified according to presumed postoperative hypoparathyroidism (hypocalcemia or documented decreased PTH), creatinine increased 28.5% in those with presumed hypoparathyroidism, while it increased only 1.2% in those without hypocalcemia [3].

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These observations suggested that PTH, some PTH fragment, or eventually another parathyroid substance [4] may affect kidney function not only in hyperfunctioning parathyroids but also at physiologic states, which was previously demonstrated in an animal study [5].

Indeed, the observation of changes in renal function after parathyroid adenoma resection is not new. In 1958, Edvall [6] proposed that the hyperactivity of the parathyroid glands was followed by two different effects on renal function. He described functional changes (which included a marked increase in GFR in HPT) that rapidly subsided after removal of the overactive parathyroid tissue. The second effect was an organic tubular lesion. In his effort to establish if the renal disturbance of HPT was caused by excess PTH, hypercalcemia, stone formation, nephrocalcinosis, or pyelonephritis, he was inclined to consider damage caused by excess PTH and proposed that in cases where advanced renal damage was established, PTX was the possible cause of a "fatal" loss of "compensatory" renal function [6].

Considering that there may be other undesirable effects of excess PTH and that excessive PTH may damage the kidney, postponing parathyroid treatment or keeping elevated PTH levels may jeopardize renal function. Perhaps a parallel to contrast-induced nephropathy may be helpful in patient selection and protective measures can be used for those undergoing parathyroid surgery, including the proposal of subtotal PTX in selected patients with tHPT.

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