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Predictive Factors of Early Postoperative Hypocalcemia After Surgery for Primary Hyperparathyroidism

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1. Abstract

Primary Hyperparathyroidism (PPH) surgical treatment is to date the most performed curative that definitively normalizes PTH serum levels. The main complication of this procedure remains the post operative hypocalcemia and the Hungry Bone Syndrom (HBS) resulting in death in extreme cases. The aim of the present review was to raise surgeons' awareness about detecting preoperatively patients at higher risk HBS by highlighting literature data about predictive factors of early postoperative hypocalcemia after surgery for primary hyperparathyroidism.

2. Review

Primary Hyperparathyroidism (PPH) is a frequent endocrinopathy whose symptomatology is related to a prolonged and excessive secretion of parathyroid Hrmone (PTH). A solitary adenoma is the cause in more than 80% of cases [1]. Surgery with removal of the pathological parathyroid gland (s) is the only definitive treatment that normalizes the serum calcium and PTH levels. Postoperative hypocalcemia is a frequent complication of parathyroidectomy with variable rates which can reach 70% depending on the series [2-4]. This can be temporary or permanent with a prevalence of 0 to 3.8% [2]. Given this fairly significant incidence, some authors recommend a systematic postoperative hospitalization of 24 to 72 hours [5].

While usually hypocalcemia is manifested by minor symptoms, such as perioral and extremity paraesthesia, more serious signs can occur such as attacks of general tetany, cardiac arrhythmias or myocardial dysfunction [6].

The objective of this review was to highlight predictive factors for developing hypocalcemia after PPH surgery and the means, if any, to prevent its occurrence.

The clinical signs of PPH result mainly from the effect of PTH on the skeleton, the kidneys and the digestive tract. During PPH, excess PTH results in 75 to 85% of cases of secretion by a solitary parathyroid adenoma, less frequently by multiple adenomatous or hyperplastic glandular damage or even parathyroid cancer [7].

The resulting hypercalcemia is due to an increase in the intestinal absorption of calcium, a mobilization of bone calcium with an increase in its renal tubular reabsorption.

Surgical excision of the affected parathyroid gland (s) represents the only treatment that definitively cures PPH with a significant success rate exceeding 90% [8]. However, hypocalcemia remains the most common complication postoperatively but which can be potentially dangerous. However, its pathogenesis remains multifactorial and the main mechanism proposed is an insufficiency of parathyroid function due to a slow and / or delayed functional recovery of the remaining parathyroid tissue [9].

In order to prevent the occurrence of this complication and to identify patients at risk for early management, several studies have attempted to identify clinical and biochemical markers that may be factors predicting the occurrence of postoperative hypocalcemia [1.9-12].

Various predictive factors for hypocalcemia after a parathyroidectomy have been identified, but their place remains differently assessed according to the authors. Thus, Mittendorf et al [13], reported that 42% of patients operated on by a parathyroidectomy developed hypocalcemia. In this same study, a bilateral cervical approach with a subtotal parathyroidectomy for glandular hyperplasia was the only factor predicting the development of postoperative hypocalcemia. The role of a bilateral cervical approach as the only factor responsible for a higher rate of hypocalcemia was confirmed in the meta-analysis conducted by Singh et al [14] including 82 studies and 6 randomized trials. In the same context, a history of cervical surgery, ablation of two parathyroid adenomas or thyroid surgery associated with lesion and / or accidental ablation of other healthy glands may be responsible for severe forms of hypocalcemia [11].

Others believed that they were more like biological markers. Indeed, in the study of Nasiri [12] as well as that of Zuberi [15], postoperative hypocalcemia is more frequent in the case of subnormal calcemia preoperatively and in the event of a high rate of PTH and PAL [12]. In the study by Yassine et al [16], the incidence of hy-

pocalcemia was 50% with a significant correlation between a high PTH level preoperatively which decreases significantly after surgery, a significant deficiency in vit D and the risk develop hypocalcemia.

Kaya et al [1] recommend more rigorous postoperative monitoring in the presence of glandular hyperplasia because of the need to identify all the glands with often the removal of three of them. This would be responsible for an often greater hypocalcemia than in case of adenoma [5].

However, the predictive value of biological markers seems more marked in HBS. This is defined as significant bone greed for calcium occurring in situations of intense remodeling; After hyperparathyroidism surgery, a rapid and significant fall in PTH of more than 85% of its initial value would be responsible for a rapid and intense bone remineralization responsible for a significant drop in calcium, mobilized from the blood sector to the sector intraosseous [2]. This phenomenon is all the more important as the preoperative bone demineralization is important [17]. A high serum PAL level would therefore be a reliable indicator of bone resorption and excessive bone turnover, which could predict an increased risk of early deep hypocalcemia [6,18]. This relationship has not, however, been demonstrated by Ellul [4].

Vitamin D plays an essential role in calcium metabolism. 1-25 (OH) 2 D3, the active metabolite of vitD, inhibits the synthesis and secretion of parathyroid hormone and thus slows bone resorption. It also improves the digestive absorption of calcium and phosphorus as well as the tubular reabsorption of calcium [19]. Low levels of 25-OHD is a factor admitted by several authors in the increase in the risk of hypocalcemia with a particular risk of development of a Hungry Bone Syndrom (HBS) [9,20]. However, systematic supplementation with vitD prescribed for prophylaxis postoperatively by some authors [2], has not been proved to prevent the occurrence of HBS even if it is started preoperatively [1,6]. In the study by Kaya [J], the incidence of HBS was 13.4% and the predictors of its occurrence were the high rate of PTH, PAL and significant osteoporosis preoperatively.

3. Conclusion

The predictive factors for post-parathyroidectomy hypocalcemia appear to be multiple and intricate. When possible, the identification of more exposed patients and the correction of parameters that may increase the risk of developing postoperative hypocalcemia should be done before or during the perioperative period. In other cases, rigorous surveillance with monitoring should be recommended.

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