PHT(1–34) for Surgical Hypoparathyroidism: A 2-Year Prospective, Open-Label Investigation of Efficacy and Quality of Life

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Context: Daily parathyroid hormone (PTH) (1–34) administrations can reduce the required total daily dose of calcium and calcitriol and restore normocalcemia in refractory hypoparathyroidism. However, most PTH(1–34) trials have been conducted on small cohorts including subjects with hypoparathyroidism of various etiologies, and quality of life (QOL) was not investigated.

Objective: To investigate the effects of 24-month PTH(1–34) treatment in a homogeneous cohort of adult subjects with postoperative hypoparathyroidism and to evaluate QOL changes.

Design: Prospective open-label study.

Setting: Italian multicenter study.

Participants: 42 subjects.

Intervention: Twice-daily PTH(1–34) 20 μg subcutaneous injection.

Main Outcome Measures: Calcium and vitamin D supplementation requirements, serum calcium, phosphate, and urinary calcium excretion (3, 6, 12, 18, 24 months). At baseline and at 6 and 24 months, QOL was evaluated by the RAND 36-Item Short Form (SF-36) Health Survey, covering eight domains of physical and mental health.

Results: Mean serum calcium concentration significantly increased from baseline to 3 months (7.6 ± 0.6 vs 8.9 ± 1.1 mg/dL, P < 0.001) and remained stable until the end of the study, despite reductions in calcium and vitamin D supplementation. Phosphate levels gradually decreased from baseline to 6 months (4.3 ± 1.1 vs 3.9 ± 0.6 mg/dL, P < 0.019), remaining stable until 24 months. Serum alkaline phosphatase and calcium excretion gradually increased from baseline to 24 months. Data from SF-36 showed a significant improvement in the mean scores of all eight domains (P < 0.001).

Conclusion: This study demonstrates the efficacy and safety of PTH(1–34) to treat adult patients with postsurgical hypoparathyroidism. PTH(1–34) may improve their mental and physical health. (J Clin Endocrinol Metab 103: 271–280, 2018)
Conventional therapy for hypoparathyroidism consists of calcium and calcitriol, but sometimes normal serum calcium cannot be maintained, and this approach may lead to nephrocalcinosis, nephrolithiasis, or renal insufficiency (1, 2). In January 2015, human recombinant parathyroid hormone (rhPTH) (1–84) was approved for the treatment of refractory hypoparathyroidism (3). Indeed, it has been well demonstrated that PTH(1–84) replacement therapy can reduce the demand for calcium and calcitriol and maintain stable serum calcium concentrations, with a good safety profile (4–6). Other studies have also demonstrated that twice-daily PTH (1–34) administrations were able to prevent postsurgical hypocalcemia (7) and reduce the required total daily dose of calcium and calcitriol and restore normocalcemia (8–11) in subjects with chronic hypoparathyroidism. In particular, Winer et al. (12, 13) have shown that teriparatide administered by a pump delivery system can also improve decrease the urinary calcium excretion and the markers of bone turnover with a smaller daily dose compared with multiple daily dosing regimens. However, most of the PTH(1–34) trials were conducted on small cohorts including subjects with hypoparathyroidism of various etiologies, and no clear quality of life (QOL) evaluation was performed.

We previously reported that treatment with PTH (1–34) for 6 months in a homogeneous cohort of 42 postsurgical hypoparathyroid subjects led to reduced supplemental calcium and 1,25-dihydroxyvitamin D requirements (14). Furthermore, we demonstrated that teriparatide might improve mental and physical health in this kind of subject (14). In this follow-up evaluation, we describe the effect of 24 months PTH(1–34) treatment on biochemical indices and QOL parameters in a homogeneous cohort of adult subjects with postoperative hypoparathyroidism.

Materials and Methods

Study population

The trial included 42 subjects (38 women, 4 men), with documented postsurgical hypoparathyroidism, treated with PTH(1–34) 20 μg twice a day. Nine Italian centers participated in this study. The diagnosis of hypoparathyroidism was established by the presence of serum calcium and PTH concentrations below the lowest normal limits (<10 pg/mL) on at least two previous occasions separated by an interval of ≥30 days. To be enrolled in this trial, subjects had to have met all the following inclusion criteria. Hypoparathyroidism should have been present for ≥1 year to establish a chronic state of PTH deprivation; each subject should have taken ≥2 g of elemental calcium (as calcium carbonate) together with ≥0.5 μg of calcitriol every day without reaching a normal calcium level, or they should have been intolerant to calcium carbonate; all patients had to be on a stable regimen of supplemental calcium carbonate and vitamin D for ≥3 months before enrollment. Of the 42 patients who provided written informed consent, 38 subjects had reached the 2-year time point and were included in this analysis at 12, 18, and 24 months.

Exclusion criteria

Patients were excluded if any of the following was present: they suffered from diabetes mellitus, severe chronic liver or renal (glomerular filtration rate <30 mL/min) diseases, Cushing syndrome, sarcoidosis, multiple myeloma, Paget disease, or rheumatic diseases such as systemic lupus erythematosus and rheumatoid arthritis; they had been on a bisphosphonate therapy within 2 years before study entry; they had taken, in the last 6 months, calcitonin, systemic corticosteroids, estrogen, raloxifene, fluoride, lithium, loop or thiazide diuretics, aromatase inhibitors, or other drugs that could interfere with calcium metabolism; they had taken PTH(1–34) or PTH(1–84) in the past; or they had serum magnesium levels below the lower limits or above the upper limits of normal on at least two previous occasions.

Study design

This is a 2-year, prospective, open-label study. Outpatient admissions occurred at 0, 3, 6, 12, 18, and 24 months. During the first admission, we evaluated the patients’ baseline status while they received calcium carbonate plus calcitriol. Height and body weight were measured to obtain their body mass index. At baseline, dietary calcium intake was ascertained by the use of a questionnaire (15). Subjects were instructed to self-administer a subcutaneous, twice-daily 20-μg injection of PTH (1–34) (teriparatide, Forsteo; Eli Lilly) in the abdomen at 8:00 AM and 8:00 PM and to rotate sites after each injection. Regardless of baseline calcium levels, the same starting dose of teriparatide was used in each patient. At each visit, blood sampling and 24-hour urinary collection were obtained.

The average value of the two pretreatment serum calcium determinations was used as the baseline calcium value. In addition to the defined time points, serum calcium was measured 15 days after patients started PTH(1–34) treatment to evaluate the opportunity to decrease the calcium and calcitriol supplementation. In particular, if the serum calcium remained stable or above the pretreatment level, supplemental calcium was reduced by 500-mg decrements until a goal of 1000 mg calcium supplementation was reached. After the calcium supplementation had reached 1000 mg daily, calcitriol was reduced by 0.25-μg decrements until the goal of stable serum calcium was reached. After the calcium and vitamin D supplementation had been reduced to 1000 mg and 0.25 μg daily, respectively, in subjects with serum calcium levels still ≥9 mg/dL, we continued to reduce the supplementation until eventual withdrawal. Serum calcium was measured 4 days after each supplementation therapy change to ensure stability of the serum calcium concentration. The main target was to bring the adjusted serum calcium back to its normal range (8.4 to 10.2 mg/dL), avoiding hypercalcemia. However, these steps were also made with the goal of maintaining serum calcium levels within the lower half of the normal range.

Assays

Serum calcium was measured by automated techniques, with a normal range of 8.4 to 10.2 mg/dL. Serum calcium was adjusted for albumin by the following formula: 0.8(4.0 – patient’s...
Calcitriol supplementation, m – Calcium supplementation, g/d 4.0 1.7 4.0 2
Urine calcium, mg/24 h 220 146 205 37
Body mass index, kg/m² 28.8 6.7 28.5 17.0
Age, y 55.8 10.4 54.0 34.0
Alkaline phosphatase, U/L 76 38 64 11
Serum creatinine, mg/dL 0.8 0.2 0.8 0.5
Uric acid, mg/dL 4.3 1.4 4.0 2.0
Serum phosphate, mg/dL 4.3 1.1 4.0 1.4
Serum calcium, mg/dL 7.6 0.6 7.8 6.1

Table 1. General Characteristics of the Sample

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>SD</th>
<th>Median</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
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<td>10.4</td>
<td>54.0</td>
<td>34.0–77.0</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
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<td>6.7</td>
<td>28.5</td>
<td>17.0–43.0</td>
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<td>Serum calcium, mg/dL</td>
<td>7.6</td>
<td>0.6</td>
<td>7.8</td>
<td>6.1–8.3</td>
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<tr>
<td>Serum phosphate, mg/dL</td>
<td>4.3</td>
<td>1.1</td>
<td>4.0</td>
<td>1.4–6.7</td>
</tr>
<tr>
<td>Uric acid, mg/dL</td>
<td>4.3</td>
<td>1.4</td>
<td>4.0</td>
<td>2.0–7.5</td>
</tr>
<tr>
<td>Serum creatinine, mg/dL</td>
<td>0.8</td>
<td>0.2</td>
<td>0.8</td>
<td>0.5–1.4</td>
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<tr>
<td>Alkaline phosphatase, U/L</td>
<td>76</td>
<td>38</td>
<td>64</td>
<td>11–191</td>
</tr>
<tr>
<td>Urine calcium, mg/24 h</td>
<td>220</td>
<td>146</td>
<td>205</td>
<td>37–696</td>
</tr>
<tr>
<td>Calcitriol supplementation, µg/d</td>
<td>0.80</td>
<td>0.30</td>
<td>0.75</td>
<td>0.50–1.25</td>
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<tr>
<td>Calcium supplementation, g/d</td>
<td>4.0</td>
<td>1.7</td>
<td>4.0</td>
<td>2–8</td>
</tr>
</tbody>
</table>

Results

Baseline characteristics

Our sample was made up of 42 patients with surgical hypoparathyroidism, and their baseline characteristics are reported in Table 1. Dietary calcium intake was ~800 mg/day elemental calcium. The mean age was 56 years (SD 10.4), and only four participants (9.5%) were men. At beginning of the study, the mean serum calcium concentration was 7.6 mg/dL (SD 0.6), and despite ongoing replacement therapy with calcium carbonate (mean 4 g/d, SD 1.7 g/d) and calcitriol (mean 0.8 µg/d, SD 0.3 µg/d), all patients had hypoparathyroidism, with serum calcium levels below the lowest normal value (<8.4 mg/dL).

Changes in biochemical parameters and calcium plus vitamin D supplementation

The mean serum calcium concentration rapidly increased after the initiation of teriparatide treatment, from 7.6 mg/dL (SD 0.6) to 8.9 mg/dL (SD 1.1) (P < 0.001) at 3 months, and then remained stable until the end of the study at 24 months (Fig. 1a). The percentage of participants with hypocalcemia changed from 100% at baseline to 29% at both 12 and 24 months (P < 0.001). The concurrent decrease in phosphate serum concentration was less dramatic, as shown in Fig. 1b: phosphate levels gradually decreased from baseline to 6 months (4.3 ± 1.1 vs 3.9 ± 0.6 mg/dL, P < 0.019). Compared with a baseline, the mean phosphate concentrations at 12 and 24 months were 3.9 mg/dL (SD 0.7) (P = 0.006 vs baseline) and 3.9 mg/dL (SD 0.6) (P = 0.01 vs baseline), respectively. Figure 2 reports the changes over time in the calcium × phosphate product (Ca×P). Compared with a baseline product of 31.8 (SD 1.1), at 3 months we observed an increase to 36 (SD 0.9) (P < 0.001). Over the rest of the follow-up time, the Ca×P product did not show statistically significant changes.

As shown in Table 2, calcium excretion increased from 220 mg/24 h (SD 146) at baseline to 239 mg/24 h (SD 108) at 12 months (P = 0.55 vs baseline) and 270 mg/24 h
(SD 126) at 24 months ($P = 0.047$ vs baseline, $P = 0.27$ vs 12 months). Similarly, serum alkaline phosphatase increased from 76 U/L (SD 38) at baseline to 133 U/L (SD 78) at 6 months ($P < 0.001$ vs baseline) and 157 U/L (SD 80), at 24 months ($P < 0.001$ vs baseline, $P = 0.085$ vs 6 months). Finally, uric acid increased from 4.3 mg/dL (SD 1.4) at baseline to 5.2 (SD 1.6) at 6 months ($P < 0.001$ vs baseline), to 5.6 mg/dL (SD 1.6) at 12 months ($P < 0.001$ vs baseline, $P = 0.13$ vs 6 months), and to 5.5 (SD 1.3) at 24 months ($P < 0.001$ vs baseline, $P = 0.82$ vs 12 months). Moreover, there were no significant differences in serum creatinine over the treatment period (Table 2).

The changes observed in the calcium concentration were mirrored by changes in the mean dose of calcium supplementation, which decreased from 4 g/d (SD 1.7) at baseline to 1.1 g/d (SD 0.8) at 6 months ($P < 0.001$) and then remained stable over the treatment period (Fig. 3a).
The same trend was observed for calcitriol supplementation: the average dose was 0.8 µg/d (SD 0.3) at baseline and 0.29 µg/d (SD 0.2) at 6 months ($P < 0.001$). The mean dose then increased to 0.41 µg/d at 12 months ($P = 0.015$ vs 6 months) and then remained stable ($P < 0.001$ 24 months vs baseline) (Fig. 3b).

**QOL evaluation**

The changes over time in SF-36 survey scores are reported in Table 3. Scores pertaining to all eight domains significantly increased at 6 months ($P < 0.001$ vs baseline for all the domains). At 24 months, there was a significant decrease in the PF score (78.4 ± 15.5 at 24 months vs 87.5 ± 10.6 at 6 months, $P = 0.01$) although this domain showed a significant improvement compared with baseline ($P < 0.001$). Similarly, the RE score was 62.2 (SD 26.1) at 24 months and 82.0 (SD 25) at 6 months ($P < 0.001$ vs both 6 months and baseline), and the GH score was 62.3 (SD 26.8) at 24 months and 69.8 (SD 16.3) at 6 months ($P < 0.001$ vs both 6 months and baseline). The overall physical health QOL score increased from 159.9 points (SD 56.4) at baseline to 307.2 points (SD 38.1) at 6 months ($P < 0.001$), and then slightly decreased to 279.9 (SD 76.89) points at 24 months, ($P = 0.035$ vs 6 months). The same pattern was seen for the MH QOL score, which increased from 153.4 points (SD 63.7) at baseline to 287.1 points (SD 54.6) at 6 months ($P < 0.001$) and then decreased to 270.7 points (SD 53.6) at 24 months ($P = 0.101$ vs 6 months). The physical and mental component summary showed a significant improvement over baseline values ($P < 0.001$).

**Safety data**

Patients were instructed to contact the study investigators between visits if any symptoms or adverse events occurred. No serious adverse events occurred during the study period. No subjects developed nephrolithiasis. Three mild adverse events led to treatment...
discontinuation: two subjects developed myalgia without elevation of muscle enzymes (after 7 and 8 months of treatment, respectively), and one subject experienced gastrointestinal illness (after 10 months of treatment). However, all these conditions resolved within 7 days of treatment discontinuation, and they did not necessitate hospitalization. Only one subject voluntarily stopped treatment (after 12 months of treatment). There were 11 asymptomatic mild to moderate episodes of hypercalciemia, caused by failure to reduce calcium carbonate and calcitriol supplements as recommended by the protocol. No hypercalcemic events necessitated hospitalization. Over the time, we recorded 32 asymptomatic episodes of hypercalciuria.

Discussion

This study demonstrated the efficacy of 2-year PTH (1–34) treatment in both increasing serum calcium and decreasing the need for calcium and calcitriol supplements.
in postsurgical hypoparathyroid patients. At the same time, PTH(1–34) therapy allowed a dramatic amelioration of patients’ QOL. These results were obtained in a cohort of 42 adult patients enrolled in a study on teriparatide in hypoparathyroidism. All patients reached 6 months of treatment, and only 4 patients dropped out of the study, at 7, 8, 10, and 12 months after treatment initiation, respectively, with 38 subjects completing the study (i.e., 24 months). Moreover, because all enrolled patients had postsurgical hypoparathyroidism, they represent a group with high homogeneity. Finally, the exclusion of hypoparathyroid children reduced possible confounders.

Altogether, these data offer good evidence of PTH (1–84) efficacy, and although our findings on teriparatide overlap, we believe that PTH(1–84) will become the drug of choice for managing chronic hypoparathyroidism. We have therefore decided to discuss our results on the basis of those obtained for PTH(1–84) and PTH(1–34) by comparing concordant results and contrasting discordant ones.

### Serum calcium, serum phosphate, Ca*P, and serum uric acid

In our study, serum calcium concentration rapidly increased after PTH(1–34), and it remained within the normal range for the disease throughout the study. This is a huge success for therapy with PTH(1–34), mirroring the results obtained in other studies with both teriparatide and PTH(1–84) (20). In particular, when PTH(1–34) is administrated as subcutaneous twice-daily injections, previous studies have shown that the daily mean dosage needed to get a good and stable calcium serum level were 46 μg/d (10) and 37 μg/d (8, 13), respectively. We achieved our aim using a similar dosage (40 μg/d).

Furthermore, as demonstrated previously by Clarke et al. (6) with the use of PTH(1–84), our study showed a significant reduction of phosphate levels after 6 months, and the achieved serum levels were maintained until the end of the study (Fig. 1b). Furthermore, Ca*P significantly increased and remained stable over the study period (Fig. 2). In our previous article (14), we hypothesized that delayed reduction of calcitriol supplements may have maintained higher intestinal absorption rate of alimentary phosphate, thus resulting in the slight increase in Ca*P (14).

We also observed a slightly significant rise in serum uric acid that confirms the well-known increase already described in the literature (Table 2) (21).

### Table 3. Changes in SF-36 Scores

<table>
<thead>
<tr>
<th>Domain</th>
<th>Baseline</th>
<th>6 Mo**</th>
<th>24 Mo***</th>
</tr>
</thead>
<tbody>
<tr>
<td>PF</td>
<td>55.5 (19.6)</td>
<td>87.5 (10.6)</td>
<td>78.4 (15.5)*</td>
</tr>
<tr>
<td>SF</td>
<td>38.4 (30.0)</td>
<td>69.8 (17.2)</td>
<td>71.8 (18.4)</td>
</tr>
<tr>
<td>RF</td>
<td>16.3 (27.5)</td>
<td>71.6 (20.9)</td>
<td>66.6 (37.5)</td>
</tr>
<tr>
<td>RE</td>
<td>37.3 (29.8)</td>
<td>82.0 (25.0)</td>
<td>62.2 (26.1)**</td>
</tr>
<tr>
<td>V</td>
<td>37.4 (11.7)</td>
<td>62.9 (16.3)</td>
<td>64.2 (22.1)</td>
</tr>
<tr>
<td>MH</td>
<td>40.3 (11.6)</td>
<td>72.4 (14.6)</td>
<td>72.5 (19.9)</td>
</tr>
<tr>
<td>BP</td>
<td>56.5 (25.8)</td>
<td>78.3 (18.9)</td>
<td>72.7 (20.9)</td>
</tr>
<tr>
<td>GH</td>
<td>31.6 (12.3)</td>
<td>69.8 (16.3)</td>
<td>62.3 (26.8)***</td>
</tr>
<tr>
<td>Physical component summary</td>
<td>159.9 (56.4)</td>
<td>307.2 (38.1)</td>
<td>279.9 (76.8)</td>
</tr>
<tr>
<td>Mental component summary</td>
<td>153.4 (63.7)</td>
<td>287.1 (54.6)</td>
<td>270.7 (53.6)</td>
</tr>
</tbody>
</table>

SDs in parentheses.

*P = 0.01 vs 6 months.

**P < 0.001 vs baseline for all domains.

***P < 0.001 vs baseline for all domains.

****P < 0.001 vs 6 months.
Urinary calcium excretion

Studies of PTH(1−34) administered by twice-daily injections did not show a significant decrease in 24-hour urinary excretion; instead, continuous PTH(1−34) delivery by insulin pump resulted in a >50% reduction in urinary calcium compared with twice-daily injections (13).

Trials with PTH(1−84) have demonstrated a trend to reduction of 24-hour urinary calcium excretion, even though a significant decrease of calciuria was not always achievable throughout previous studies (4, 5, 22–26). In our patients, urinary calcium excretion did not decrease, and at the end of the study it was significantly higher than baseline (Table 2). Even when patients developing hypercalcemia were removed from the analysis (only 3 subjects at 24 months), the increasing renal calcium excretion trend did not change (data not shown). This finding is in contrast with the observations obtained with PTH(1−84), and we proposed some potential explanations in our previous study on 6-month teriparatide treatment (14). All enrolled patients achieved serum calcium levels within the normal range, thus accounting for a possible higher glomerular filtered load of calcium. At the same time, increased bone turnover, reflected by increasing alkaline phosphatase values (Table 2), may have increased calcium mobilization from bones, resulting in higher delivery of renal calcium at the glomerulus level. Lower sensitivity of renal distal tubule cells to PTH(1−34) than to PTH(1−84) action is another possible explanation for the behavior of calcium excretion, but our data do not allow such a conclusion. Whatever the case, 2 years of PTH(1−34) treatment induced a slight increase of renal calcium excretion that has not been observed with PTH(1−84), and a possible additional risk of renal stones must be considered with this drug.

Supplements of calcium and calcitriol

Reduction of supplemental calcium and calcitriol in hypoparathyroid patients is generally considered one of the main goals of a therapy with exogenous PTH (20). Our results demonstrate that the mean daily calcium dose is dramatically reduced by PTH(1−34) therapy (Fig. 3a). In addition, daily supplements of calcitriol were significantly reduced by teriparatide from baseline to the end of the study, even though at both 12 and 24 months the required daily dose was slightly but significantly higher than that at 6 months (Fig. 3b). We hypothesize that, after 1 year of teriparatide, the adherence to two daily drug injections may have decreased, thus necessitating an adjustment of vitamin D supplements. Another possible explanation is that within the first semester of treatment, increased patient well-being may have led some patients to a self-reduction of calcitriol supplements that may not have been maintained throughout the study. Unfortunately, we did not accurately investigate adherence to treatment and therefore cannot give a definitive explanation for this finding. However, the important reduction in calcium and calcitriol supplements reduced the possible side effects of this therapy (hypercalciuria, hyperphosphatemia, nephrocalcinosis, urolithiasis, and ectopic soft tissue calcification) and the need for daily ingestion of large amounts of calcium, which can be very uncomfortable for patients.

QOL

Previous well-designed studies with teriparatide did not evaluate QOL by using SF-36, but Winer et al. (9) showed that fatigue (evaluated by the Multifaceted Assessment of Fatigue self-report questionnaire) was a common complaint in subjects treated with calcitriol and calcium, and several patients described greater endurance with PTH(1−34) therapy.

After 24 months of teriparatide treatment, physical and mental QOL scores were still significantly higher than at baseline, but some of them decreased significantly compared with the values obtained at 6 months (in particular the PF, RE, and GH scores). Also, when both the overall physical and MH-related QOL scores were considered, a similar V-shaped trend was found. Long-standing postsurgical hypoparathyroidism is a good model for studying QOL: patients who had normal serum calcium before surgery became dependent on large amounts of calcium and calcitriol after parathyroidectomy. By dramatically reducing the need for calcium and calcitriol supplements, PTH(1−34) has had a positive effect on patients’ QOL. It is reasonable to suppose that this change allowed higher expectations in patients, as demonstrated by the results of SF-36 scores at 6 months. Soon afterward, the routine twice-a-day injection of teriparatide may have become annoying, and some patients may have begun feeling it was unpleasant. In particular, the lowering of both PF and GH at 24 months can be a clear sign of patients’ changing thoughts on PTH(1−34) therapy. However, the stable increase in serum calcium and the dramatic reduction in supplemental calcium and calcitriol daily doses may be the main reason for QOL improvements, even though we cannot exclude the possibility that PTH itself may improve the QOL indices.

Limitations of the study

An important limitation of our study is the lack of a control group of hypoparathyroid subjects continuing calcium and calcitriol treatment. As stated in the enrollment criteria, hypoparathyroidism in these subjects was severe, so none of them could have entered the control group. Moreover, we did not evaluate the severity...
of hypocalcemia symptoms with validated scales, and therefore we are not able to give a numeric measure of their changes or to correlate them with QOL improvement. We also did not measure ionized calcium. Our study design may have led us to miss the detection of peak serum and urinary calcium levels if these occurred within 12 hours of PTH administration. Given that we did not collect empty drug boxes, we were unable to confirm patient adherence to treatment. Therefore, it is possible that some patients may have adjusted their own calcium and calcitriol supplementation on the basis of personal feelings rather than on researchers’ indications, thus modifying some of the study results, such as urinary calcium excretion.

Conclusions

Despite its limitations, this study demonstrates the effectiveness and safety of PTH(1–34) in the treatment of patients with postsurgical hypoparathyroidism. Moreover, PTH(1–34) treatment has durably improved mental and physical health in adult hypoparathyroidy subjects.

Acknowledgments

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Author Contributions: A.P., A. Santonati, and F.V. conceived and designed the trial; A.P., A. Santonati, G.T., D.M., S.M., D.B., A. Spada, B.R., T.D., and F.G. performed and received and designed the trial; A.P., A. Santonati, G.T., D.M., S.M., D.B., A. Spada, B.R., T.D., and F.G. performed and followed the trial; C.P. and A.P. analyzed the data; and A.P., F.V., C.P., and G.T. wrote the manuscript. All authors approved the final version of the manuscript.

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Disclosure Summary: The authors have nothing to disclose.

References