detected in women previously treated with this agent up to 19 months after discontinuation of therapy. The relationship between alendronate levels and both bone resorption and time of treatment cessation further indicates a residual effect of this drug in bone despite treatment discontinuation.

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PP411-T

Secondary osteoporosis associated with vitamin D deficit in young women – A therapeutic challenge

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Abstract: Background: Estrogen deficiency, glucocorticoid treatment, and increased PTH secretion are associated with bone loss irrespective of age. **Aim:** To present therapeutic challenges in four young women with secondary osteoporosis associated with vitamin D deficit. **Methods:** Serum intact PTH was measured by immunoradiometric assay, 25 OH vitamin D, osteocalcin and β crosslaps by radioimmunoassay, bone mineral density by DXA. **Case reports:**

Case 1: A.M., aged 27, underwent bilateral ovariectomy for ovarian carcinoma at the age of 22. Secondary osteoporosis (BMD L2–L4 = 0.712 g/cm ^{2}, Z score = −3.5 SD, T score = −4.1 SD) and vitamin D insufficiency (25 OH D = 11.09 ng/mL) were diagnosed. Zolendronate 5 mg and 1000 U/day vitamin D were administered.

Case 2: N.B., aged 18, with myasthenia gravis, 3 years treated with glucocorticoids developed iatrogenic Cushing syndrome with hypogonadotropic hypogonadism. Secondary osteoporosis (BMD L1–L4 = 0.488 g/cm ^{2}, Z score = −5.1 SD), and vitamin D deficiency (25 OH D = 7.69 ng/mL) were diagnosed. Osteocalcin was suppressed (6.81 ng/mL). Zolendronate 5 mg and 2000 U day vitamin D were administered.

Case 3: N.N, aged 39, diagnosed with primary hyperparathyroidism (corrected calcium = 12.8 mg/dL, PTH = 323.55 pg/mL) and Cushing syndrome due to ACTH independent bilateral macronodular adrenal hyperplasia, underwent parathyroid surgery and bilateral adrenalectomy. Pathology exam revealed right superior parathyroid carcinoma and left superior and inferior parathyroid adenomas. Secondary osteoporosis (BMD L2–L4 = 0.766 g/cm ^{2}, Z score = −4.2 SD, T score = −3.6 SD) and vitamin D insufficiency (25 OH D = 17.6 ng/mL) were diagnosed. Ibandronate and α calcidol were administered, with improvement of BMD after 1 year therapy (BMD L2–L4 = 0.833 g/cm ^{2}).

Case 4: I.S, aged 40, had tertiary hyperparathyroidism due to renal chronic failure (corrected calcium = 11.3 mg/dL, PTH = 1533 pg/mL) and vitamin D insufficiency (25 OH = 10.21 ng/mL). Bone turnover markers were markedly increased (β crosslaps > 6 ng/mL, osteocalcin = 273.3 ng/mL). Secondary osteoporosis was diagnosed (BMD L1–L4 = 0.848 g/cm ^{2}, Z score = −2.4 SD, T score = −2.8 SD). Parathyroid surgery was scheduled, preceded by cinacalcet treatment.

None of these patients experienced fractures during follow up. **Conclusion:** Osteoporosis screening is mandatory in young women with diseases affecting bone mineral density. Efficient treatment of both secondary osteoporosis and underlying disorder minimize osteoporosis complications.

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PP412-S

Identifying factors associated with patients making the link between a fragility fracture and osteoporosis

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Abstract: Introduction: Patients who associate their fragility fracture with osteoporosis are more likely to initiate osteoporosis treatment. It is not known, however, who is more likely to make this association. The purpose of this study is to examine baseline factors associated with patients making the link between their fragility fracture and osteoporosis at follow up. **Methods:** Data on a population-based cohort were collected as part of a provincial osteoporosis screening initiative targeting low trauma fracture patients over the age of 50. Osteoporosis screening coordinators collected data at baseline and treatment-naïve, previously undiagnosed patients were followed up at 3 and/or 6 months. Logistic regression was used to identify which modifiable and non-modifiable factors are predictive of patients making an association between their fragility fracture and osteoporosis at follow up. **Results:** At baseline, 93% (853/916) of patients did not believe their fracture could have been caused by having osteoporosis. Of these, only 8.8% (75) changed this perception at follow up. Adjusted analyses showed that the following baseline characteristics were associated with a transition to making the osteoporosis-fracture link at follow up: a previous fracture OR 2.2 (1.3–2.9), perception of bones as thin OR 7.1 (3.7–13.8) or uncertainty about the quality of bones OR 2.8 (1.5–5.4) at baseline. **Discussion:** Many fragility fracture patients do not associate their fracture with having osteoporosis and this perception stays constant over time in most cases. We identified the baseline characteristics predictive of making the osteoporosis-fracture link at follow up. These findings could be used to identify patients who are less likely to make the link and target interventions to this patient group by emphasizing the link between a previous fracture and osteoporosis and delivering a clear message about the quality of patient's bones as "thin".

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PP413-M

PTH 1–84 and bone turnover markers in severe post-menopausal osteoporosis

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Abstract: Introduction: Anabolic therapy appears to be a promising strategy in clinical osteoporosis. Full-length PTH (1–84) is a potent anabolic agent currently used in the treatment of this clinical context. Bone mineral density (BMD) changes measured by DXA and bone turnover marker (BTM) evaluation are established tools for monitoring the effects of anti-osteoporotic therapy. BTM also allows capturing early variations in bone metabolism, such as 30–200%, BTM increase or reduce within 3–6 months of treatment. Our purpose is to analyse BTM and BMD variations in patients treated with PTH 1–84, in the treatment of primary osteoporosis in postmenopausal women. **Methods:** We have measured the trend of parameters of bone metabolism (serum calcium, osteocalcin, and β-Ctx) in 51 women with severe postmenopausal osteoporosis (mean age 69.8 ± 8.3 years) treated with the anabolic drug PTH 1–84 at the baseline and after 6, 12 and 18 months of therapy. Indeed, we have measured hip and spine T-score at baseline and at the end of therapy. **Results:** In the 51 patients treated for 18 months, while serum calcium values remained on normal range (9.41 ± 0.4 mg/dl at the baseline, 9.76 ± 0.6 mg/dl after 6 months, 9.75 ± 0.7 after 12 months and 9.53 ± 0.6 at the end of therapy). Changes in T score were statistically significant, with an increase at the spine (−3.63 ± 0.8 at the baseline, −2.85 ± 0.7 at the end of the treatment, $p < 0.001$ Wilcoxon test) and less impressive, but not significant trend at the hip (−2.23 ± 0.6 at baseline, −2.11 ± 0.6 at the end of the treatment). Osteocalcin levels showed a significant increase after 6 months compared with the baseline, undergoing a further slight increase after 12 months of therapy and, although they fall down after 18 months, they still maintained at a level equal to 2.95 times above the baseline, testifying the strong anabolic effect of PTH. The levels of β-Ctx increased 2.49 times compared with the baseline, after 6 months of treatment. They still increased after 12 months (2.82 times compared with the baseline) and after 18 months. **Conclusion:** Results obtained in our clinical experience confirm the efficacy of treatment with PTH 1–84, testified by the statistically significant increase of T-score both at the spine and at the hip, besides the increase of β-Ctx and osteocalcin levels, indicating strong osteoanabolic activity. Thus PTH 1–84 demonstrates efficacy on high risk of fracture reduction in postmenopausal osteoporotic women.

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PP414-T

Treatment adherence and QoL with intermittent PTH therapy: Results from a multicentre Italian study after 6 months

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Abstract: This ongoing multicentre study was carried out to investigate the adherence to intermittent parathyroid hormone treatment and the effect on QoL in a typical Italian population eligible for drug reimbursement (i.e. a new vertebral or hip fracture following at least one year of anti-resorptive therapy or three severe vertebral fractures or two severe and one femur fracture). 204 female patients (mean age: 72.6 ± 8.3) were enrolled between March 2008 and April 2009. These patients were treated with PTH (PTH 1–34 or PTH 1–84) according to Italian legislation. Here, we report the results obtained at month 6.

The global percentage of patients who discontinued therapy was 15.2% (31/204).The percentage of patients adhering to therapy were 90.6% at month 1 visit, 97.4% at month 3 and 94.8% at month 6 visit. observed 8 new clinical fractures (4.1% on the first six months of observation (2 after 3 months and 6 after 6 months); 5 out of these fractures were clinical vertebral, 3 non-vertebral (2 ribs and one wrist).The impact of treatment on QoL showed a statistically significant improvement of 5.8 points on QUALEFFO – 41 score, as shown in the table.

Baseline (Mean ± SD)	1st month (Mean ± SD)	3rd month (Mean ± SD)	6th month (Mean ± SD)
55.7 ± 17.8	51.9 ± 17.5 ($p < 0.001$)	50.0 ± 17.2 ($p < 0.0001$)	49.9 ± 17.0 ($p < 0.001$)

There was also a statistically significant improvement of the VAS score (6.7 points).

Baseline (Mean ± SD)	1st month (Mean ± SD)	3rd month (Mean ± SD)	6th month (Mean ± SD)
49.5 ± 19.7	53.9 ± 18.9 ($p < 0.05$)	55.0 ± 17.6 ($p < 0.01$)	56.2 ± 19.4 ($p < 0.001$)

The data indicate that therapy with parathyroid hormone results in a significant improvement in patients QoL; this treatment is characterized by good compliance, with an adherence >90% after 6 months.

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PP415-S

Serum sclerostin levels decline in postmenopausal women with osteoporosis following treatment with intermittent PTH

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Abstract: Intermittent PTH treatment stimulates bone formation; animal studies have shown that PTH suppresses the production of sclerostin, an inhibitor of Wnt signaling. This study was therefore carried out in order to assess whether 18 months treatment with PTH decreases serum sclerostin in humans.

We investigated ten postmenopausal osteoporotic women (mean age 70.2 ± 9.9 years), previously treated with alendronate. They were randomly divided into two groups of five patients each; the first group was treated with 20 mcg of PTH (1–34) and the second one with 100 mcg of PTH (1–84) according to an open-label design. Fasting blood samples were collected at baseline and at 2, 4 and 24 h after hormone administration; the same protocol was followed at months 1, 6, 12, and 18. Serum sclerostin levels were measured at each time point by a sandwich-type ELISA, utilizing a biotinylated antibody/horseradish peroxidase-streptovidin for detection of the analyte (the intra and inter-assay CV were between 4 and 6% and between 5 and 7%, respectively). Data from both treatments were pooled, based on our previous results showing no difference in the biochemical results following administration of both forms of PTH (Calcif. Tissue Int. 2009).

Basal sclerostin levels were 29.42 ± 13.56 pmol/L. No significant acute changes of serum sclerostin levels were observed at 2, 4, and 24 h after administration of both PTH peptides. However, fitting a mixed effect regression model to the entire treatment period, we found a significant time effect ($p = 0.0012$) using the sclerostin level as the response variable and the month of drug administration as a single covariate. The estimate for the common slope of the single patient regression lines was equal to −0.1956, indicating that the sclerostin levels for both groups of patients had a monthly mean reduction of 0.1956 pmol/L.

This is the first study evaluating the effect of the two forms of PTH on serum sclerostin levels in postmenopausal women previously treated with alendronate. Our results indicate that long-term therapy with PTH (1–34) or PTH (1–84) is associated with a reduction in circulating sclerostin levels. This is a putative mechanism through which PTH performs its anabolic action.

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PP416-M

Baicalin inhibit osteoclastogenesis by suppressing MAPK and downregulating NFATc1 in RANKL-induced raw 264.7 cells

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Abstract: Osteoclasts are responsible for skeletal modeling and remodeling, but may also destroy bone in several bone diseases such as osteoporosis and rheumatoid arthritis. This study was carried out to determine the effect of flavonoid baicalin on osteoclastogenesis in RAW264.7 cells induced by receptor activator of NF-kB ligand (RANKL). We found that baicalin significantly inhibited the RANKL-stimulated tartrate-resistant acid phosphatase activity and the formation of multinucleated osteoclasts in dose dependent manner. Baicalin inhibited RANKL-stimulated activation of p-38 and ERK MAPK signaling, and inhibited RANKL-stimulated degradation of I_kB. And baicalin significantly decreased the expression of NFATc1 and cFos, the master regulator of osteoclast differentiation. In addition, baicalin decreased the mRNA expression of cathepsin K and cyclooxygenase 2 (COX2), involved in various pathophysiological processes such as inflammation. And baicalin decreased nitric oxide production induced by LPS. These findings indicate that baicalin may have therapeutic effects on bone destructive processes such as osteoporosis and rheumatoid arthritis. This research was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education, Science and Technology (#2010-0023679).

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PP417-T

Evaluation of adherence and persistence to teriparatide treatment in patients affected by severe osteoporosis: A multicenter observational real life study

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Abstract: Background: Osteoporosis is a chronic condition leading to an increased risk of developing fractures, with high morbidity and mortality in aging population. Efficacy of anti-osteoporotic treatment is based on drug potency but also on compliance and