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Case Report

Off-Label Use of Teriparatide for Osteotomy Healing in an Adolescent with Osteogenesis Imperfecta Type VIII: A Case Report

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Abstract
Osteogenesis imperfecta (OI) type VIII is an autosomal recessive skeletal dysplasia caused by *P3H1* variants, resulting in defective collagen post-translational modification and increased bone fragility. Teriparatide (TPTD), a recombinant parathyroid hormone analogue, stimulates osteoblast activity and bone formation and is approved for the treatment of osteoporosis in adults. A 16-year-old male with genetically confirmed OI type VIII presented with delayed union with characterized by the absence of radiographic evidence of healing six months after femoral osteotomy and intramedullary rodding surgery. His prior management included intermittent bisphosphonate therapy which had improved bone mineral density. He had no recent history of fractures. Off-label therapy with TPTD (20 µg/day subcutaneously) was initiated and continued for six months. Although systemic markers of bone formation showed no remarkable changes, except osteocalcin, during therapy, serial radiographs confirmed successful bone union. The treatment was well tolerated, with no reported side effects; however, mild hypercalcemia with hypercalciuria, which persisted after discontinuation of TPTD therapy, was detected. Bisphosphonate therapy was resumed upon completion of TPTD treatment. This case represents the first documented use of TPTD to promote osteotomy healing in an adolescent with OI. The favourable clinical and radiologic outcome suggests a potential role for anabolic therapy in managing complex bone healing challenges in paediatric OI. Further research is warranted to evaluate safety and efficacy in this population.
Keywords: Hypercalcemia, nonunion, osteogenesis imperfecta, osteotomy, P3H1, teriparatide

What is already known on this topic?
Teriparatide (TPTD) is approved for the treatment of osteoporosis in postmenopausal women and men. Several studies and case reports have explored the use of TPTD in adult with osteogenesis imperfecta (OI). It has also been reported to promote healing in delayed union and atypical femoral fractures.

What this study adds?
This is the first reported use of TPTD for an unhealed osteotomy in OI, and in an adolescent with type VIII disease. TPTD facilitated bone healing after femoral osteotomy, supporting anabolic therapy as a promising option for complex bone conditions.

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Introduction

Osteogenesis imperfecta (OI) is a rare heritable skeletal dysplasia characterized by defects—most commonly in type I collagen synthesis—resulting in a broad clinical spectrum ranging from mild bone fragility to severe forms associated with perinatal lethality. Among its subtypes, OI type VIII is an autosomal recessive disorder caused by pathogenic variants in the *P3H1* gene (formerly known as *LEPRE1*), which encodes a prolyl 3-hydroxylase involved in the post-translational modification of collagen. This subtype is associated with profound skeletal deformities, growth deficiency, and increased susceptibility to fractures (1).

Teriparatide (TPTD), a recombinant parathyroid hormone analogue [PTH (1–34)], exerts anabolic effects by stimulating osteoblast activity and reducing osteoblast apoptosis, thereby enhancing bone formation (2). Its osteoanabolic action is most pronounced in the trabecular and endosteal cortical bone compartments (3). TPTD is approved for the treatment of osteoporosis in postmenopausal women, men, and patients with glucocorticoid-induced osteoporosis who have not responded to conventional therapies including bisphosphonates, calcium and vitamin D supplementation, or lifestyle modifications. In these populations, it has been shown to increase bone mineral density (BMD) and reduce the risk of both vertebral and non-vertebral fractures (4,5). TPTD has also shown promise in secondary osteoporosis associated with chronic conditions, including Duchenne muscular dystrophy (DMD), especially when glucocorticoid therapy is implicated (6). Owing to its potent anabolic effects, off-label use of TPTD has been explored in fracture healing, particularly in cases of non-union and atypical femoral fractures (AFFs), some of which involve patients with OI (7–9).

However, while TPTD has demonstrated efficacy in adult patients with OI, its use in pediatric populations remains controversial and off-label. Regulatory agencies such as the FDA and EMA continue to caution against its use in patients with open epiphyses or metabolic bone diseases associated with an elevated risk of osteosarcoma (10,11). Early preclinical studies in growing rats showed increased osteosarcoma incidence with high-dose TPTD, prompting a black-box warning (12). However, subsequent studies using supratherapeutic doses—up to three times the human equivalent—showed no evidence of malignancy (13). Despite these reassuring findings, the use of TPTD in children and adolescents remains conservative due to open epiphyseal plates and limited long-term safety data.

To our knowledge, there is no report evaluating TPTD treatment specifically for fracture or osteotomy healing in paediatric OI patients. We present here the first documented case of TPTD used to promote bone healing following femoral osteotomy in an adolescent with genetically confirmed OI type VIII, emphasizing the therapeutic rationale, clinical outcome, and associated limitations.

Case Report

The patient was born at term via spontaneous vaginal delivery to consanguineous parents as the ninth child of the family and presented with multiple fractures at birth. His family history was notable for an older brother with a similar condition characterized by recurrent fractures. At the time of evaluation, the brother was 26 years of age and had never received medical treatment and regular clinical follow-up. He was wheelchair-dependent due to severe shortening of left lower limb resulting from recurrent fractures.

During early childhood, the patient experienced six fractures. At six years of age, oral alendronate therapy (35 mg/week) was initiated at another medical center and continued for two years. He achieved independent ambulation 6 months after the initiation of treatment. However, bisphosphonate therapy was subsequently discontinued and the patient was lost to follow-up for the following three years.

The patient first presented to our clinic at 11 years of age. He had remained fracture-free for approximately 2.5 years but had sustained a femoral fracture one year before presentation. At that time, his height was 128 cm (–2.49 SD) and his weight was 36 kg (–0.3 SD). Following the initial evaluation, he was lost to follow-up but returned two years later after sustaining another femoral fracture. At reassessment, his BMD z-score had declined from -1.2 to -2.2 and progression of vertebral compression fractures was evident (Fig 1-A, -B). Oral alendronate therapy (70 mg weekly) was reinitiated at 13 years of age. Over the subsequent three years, his BMD improved markedly, increasing from 0.603 g/cm² to 1.064 g/cm². No further fractures occurred throughout subsequent follow-up.

However, due to femoral bowing and leg shortening resulting from previous fractures, his mobility was impaired. At the age of 16 years and 7 months, the patient underwent corrective femoral osteotomy with intramedullary telescopic rod fixation (Supp Fig). Alendronate was discontinued six weeks prior to surgery to reduce the risk of delayed healing. Postoperative radiographic follow-up was performed monthly for callus formation.

At 6 months postoperatively, there was no radiographic evidence of callus formation or bone union at the osteotomy sites (Fig 2-D1, -D2, -D3). Delayed union was diagnosed based on the persistent presence of sharp osteotomy margins, absence of periosteal callus formation, and lack of cortical bridging on serial anteroposterior and lateral radiographs, without any radiological sign of progressive healing throughout the 6-month follow-up period (14).

Although the FDA defines an established nonunion as a fracture persisting for at least 9 months without evidence of progressive healing for 3 consecutive months, fractures are often considered nonunions in clinical practice as early as 6 months after injury. Delayed healing can generally be identified between 3 and 6 months post-injury, with 6 months being the most commonly used threshold (15). According to these criteria, our patient at postoperative month 6 was more appropriately classified as having delayed union rather than established nonunion. However, given the complete absence of radiological progression, the lesion was considered at high risk of progressing to nonunion, and medical intervention was deliberated. Delayed union at osteotomy sites is a well-recognized complication in OI, particularly in patients who continue bisphosphonate therapy without a postoperative drug holiday (16,17).

Given the history of bisphosphonate use and poor healing, off-label anabolic therapy was considered. As TPTD has demonstrated efficacy in adult patients with delayed union and non-union fractures, its use was evaluated in our patient. Because TPTD is not approved for use in children with open epiphyses, radiographically confirmation of epiphyseal closure was obtained before treatment initiation. Following informed consent from the patient's parents, TPTD was initiated at the standard adult dose of 20 µg/day subcutaneously (approximately 0.32 µg/kg/day for the patient's body weight of 62.5 kg). The adult dose was selected because the patient was a post-pubertal adolescent aged 16 years and 7 months with radiographically confirmed epiphyseal closure, skeletal maturity, and body weight comparable to that of an adult. and because no validated paediatric dosing regimen for TPTD exists for this indication.

After six months of TPTD therapy, radiographic evaluation confirmed satisfactory healing at the osteotomy sites (Fig 2-F1, -F2). Early sings of healing were already evident by the third month of treatment, including progressive union and blunting of the previous sharp osteotomy margins (Fig 2-E1, -E2). The duration of treatment was not predetermined but was guided by serial radiographic assesssments, with progressive fracture union serving as the therapeutic endpoint. Treatment was discontinued at the sixth month once satisfactory radiographic union had been achieved, while also minimizing cumulative TPTD exposure given its off-label use in an adolescent patient.

During TPTD therapy, the patient reported no clinical adverse effects. Biochemical monitoring demonstrated an increase in osteocalcin levels, accompanied by mild hypercalcemia, hypercalciuria and, elevated serum $1,25(OH)_2$ vitamin D concentrations, and suppression of PTH levels (Table 1). Serum calcium increased from 9.9 to 10.5 mg/dL (reference: 8.4–10.2 mg/dL) by the sixth month of treatment and remained at 10.5 mg/dL 1.5 months after TPTD discontinuation. The urinary calcium-to-creatinine ratio increased from 0.28 to 0.37 (reference: <0.22 mg/mg), while serum $1,25(OH)_2$ vitamin D measured at the third month was 94.2 pg/mL (reference: 28–81.8 pg/mL). Concurrently, serum PTH was suppressed from 27.2 to 10.3 ng/L.

The hypercalcemia was mild and asymptomatic, requiring no therapeutic intervention, and TPTD treatment was completed as planned. Renal ultrasonography revealed no evidence of nephrolithiasis or nephrocalcinosis. The patient did not receive calcium or vitamin D supplementation during or after TPTD therapy. Following re-initiation of alendronate treatment, serum calcium and the urinary calcium-to-creatinine ratio normalized within approximately 1.5 months, decreasing to 9.5 mg/dL and 0.05 mg/mg, respectively.

After corrective surgery and complete union of osteotomy sites, the patient regained full mobility. He expressed satisfaction with the treatment, noting that he was able to resume normal daily activities without limitation.

Genetic analysis identified a homozygous pathogenic variant, *Leu149Arg* (c.446T>G), in the *P3H1* gene, confirming a diagnosis of OI type VIII. The same homozygous mutation was identified in his affected brother, while both parents were heterozygous carriers.

Discussion

We report the successful use of TPTD in an adolescent patient with genetically confirmed OI type VIII and delayed bone healing after corrective femoral osteotomy. To our knowledge, this is the first reported case of TPTD for delayed union in adolescent OI.

Delayed bone healing after osteotomy is a recognized complication in OI, attributed to defective collagen synthesis, impaired bone microarchitecture, and, in some cases, prior bisphosphonate exposure (18). These factors compromise bone remodelling and increase the risk of non-union or prolonged healing, particularly in severe OI subtypes. In our patient, despite adequate fixation and prior bisphosphonate response, there was no radiological evidence of callus formation at six months postoperatively.

TPTD has been shown to exert anabolic effects on bone by stimulating osteoblast differentiation, enhancing bone formation, and accelerating bone remodelling (19). Although much of the supporting evidence comes from experimental models and case series, TPTD has demonstrated potential as an adjuvant therapy in promoting fracture callus formation and enhancing bone consolidation (20). While its primary indications are the treatment of osteoporosis in postmenopausal women, men, and adults with glucocorticoid-induced osteoporosis, the off-label use of teriparatide has attracted interest in other challenging clinical contexts, including impaired bone healing. Furthermore, several studies have explored its use in adult patients with OI, suggesting potential benefits in improving BMD and eliciting an anabolic response, particularly in milder forms, such as OI type I (21–23).

Atypical femoral fractures (AFFs) are rare but serious complications associated with prolonged bisphosphonate therapy, often resulting from severely suppressed bone turnover. In such cases, TPTD has been used successfully to stimulate bone regeneration and promote fracture healing. The European Calcified Tissue Society (ECTS) has included TPTD among treatment options for AFFs and non-unions, even in patients with underlying skeletal dysplasia such as OI (9). Several case reports have documented its efficacy in resolving bisphosphonate-related AFFs in patients with OI, demonstrating improved radiographic and functional outcomes (7,24). The youngest reported patient to

receive TPTD in the literature was 21 years old and had OI type IV-B. Despite having a normal BMD three years earlier and no ongoing treatment, the patient sustained a low-trauma fracture characteristic of bisphosphonate-associated AFFs. Surgical fixation was performed with intramedullary nailing, followed by initiation of TPTD therapy. Notably, satisfactory fracture healing was observed at one-year follow-up (8). These findings suggest that TPTD may retain its bone-anabolic efficacy even in young adults with impaired bone remodelling capacity.

Beyond its role in managing AFFs, numerous reports have supported the utility of TPTD in promoting healing in cases of delayed union and non-union following osteotomy in adults (25,26) . However, there remains a significant gap in the literature regarding its application in patients with OI, particularly in paediatric or adolescent populations. To date, there are no randomized controlled trials evaluating the efficacy of TPTD in fracture or osteotomy healing, either in healthy individuals or those with OI. Most available data are derived from uncontrolled case series and observational studies, and it remains uncertain.

Importantly, the use of TPTD in paediatric population remains off-label and is approached with caution. While the black-box warning for osteosarcoma has been removed based on post-marketing surveillance and preclinical data (13), current FDA and EMA labels continue to advise against TPTD use in patients with open epiphyses (10,11). In our case, treatment was initiated only after radiographic confirmation of epiphyseal closure. Nevertheless, the persistence of mild hypercalcemia and hypercalciuria following treatment discontinuation underscores the importance of careful biochemical monitoring, particularly in children and adolescents with potentially altered calcium metabolism.

Radiographic evidence of bone union was observed a 6-month course of TPTD, and no serious adverse events occurred. An increase in serum osteocalcin levels was also observed during treatment; however, this finding may reflect the physiological process of fracture healing and, therefore, should not be interpreted as direct evidence of TPTD efficacy, particularly because other markers of bone formation were unavailable for assessment.

Another consideration is whether the healing observed at the osteotomy sites represents spontaneous late union rather than a treatment effect of TPTD. In our view, spontaneous healing is less likely, as serial monthly radiographs obtained during the 6 months preceding TPTD initiation demonstrated no progressive healing, with persistently sharp osteotomy margins and clearly visible osteotomy line. Following initiation of TPTD, progressive cortical continuity and gradual reduction of osteotomy line visibility became apparent within 3 months, with complete union achieved by 6 months. This temporal pattern, occurring after a prolonged period of radiographic stagnation and accompanied by an increase in serum osteocalcin, is consistent with the anabolic effects of TPTD, although the observed association remains correlational.

In addition, it should be noted that the presence of telescopic intramedullary rods may limit assessment of endosteal callus formation due to metallic artifact. Therefore, radiographic evaluation focused primarily on periosteal callus, cortical bridging, and obliteration of the osteotomy lines. This assessment approach is in line with the modified Radiographic Union Score (mRUST), which has been adapted and validated for the evaluation of femoral shaft fractures treated with intramedullary fixation devices (14).

This case has several limitations. As a single case report, it does not provide conclusive evidence regarding the efficacy or safety of TPTD, and the absence of a control group, together with the off-label use of TPTD, limits the generalizability of the findings. Spontaneous late union, although unlikely, cannot be entirely excluded. Biochemical monitoring was restricted to standard clinical laboratory markers, without advanced bone turnover assessment or histomorphometric analysis. In particular, procollagen type 1 N-terminal propeptide (P1NP) was measured only at baseline, and serial P1NP monitoring during TPTD therapy could not performed; therefore, longitudinal assessment relied primarily on osteocalcin and alkaline phosphatase as markers of bone formation.

Given the rarity of its therapeutic application, the patient's young age, and the underlying genetic diagnosis, this case provides valuable insight into the potential role of anabolic agents in managing complex bone disorders. However, the risk of hypercalcemia must be carefully considered, particularly in the paediatric population, even though it is uncommon in adults treated with TPTD (4). TPTD induces hypercalcemia in a dose-dependent manner, likely related to increased calcitriol levels, a pattern also observed in our case.

In conclusion, this case highlights a novel and potentially effective approach for managing delayed union in adolescents with OI. It underscores the need for further clinical studies to evaluate the safety, efficacy, and optimal duration of TPTD therapy in paediatric patients with osteoporosis. Until such evidence becomes available, carefully monitored and individualized use of TPTD may be considered in select cases when conventional therapies have proven ineffective.

Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

A.G organized the patient's medical records, planned the structure and methods for literature review, wrote the initial draft of the manuscript. S.T identified the case and conceptualized the report, analysed imaging studies and diagnostic results, critically reviewed and edited the manuscript. A.G and S.T prepared radiology images and accompanying figure legends. S.B.K, A.H.A, D.H, Z.Y.A and M.K.B performed the clinical examination and procedures. All authors reviewed the case details and confirmed the conclusions.

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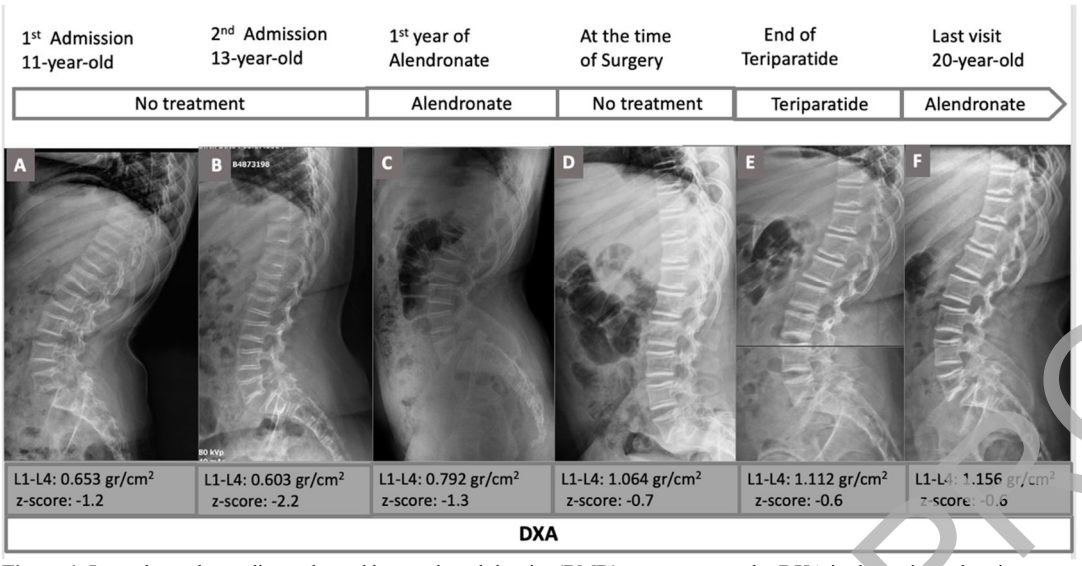


Figure 1. Lateral vertebra radiographs and bone mineral density (BMD) measurements by DXA in the patient, showing progression of the vertebra compression fractures and a decline in BMD over a 2-year untreated period (A, B). Improvement in BMD is observed after 1 year of alendronate treatment (C), followed by complete reshaping of the vertebral bodies and restoration of BMD at the time of surgery (D, age 16.5 year) and during subsequent follow-up

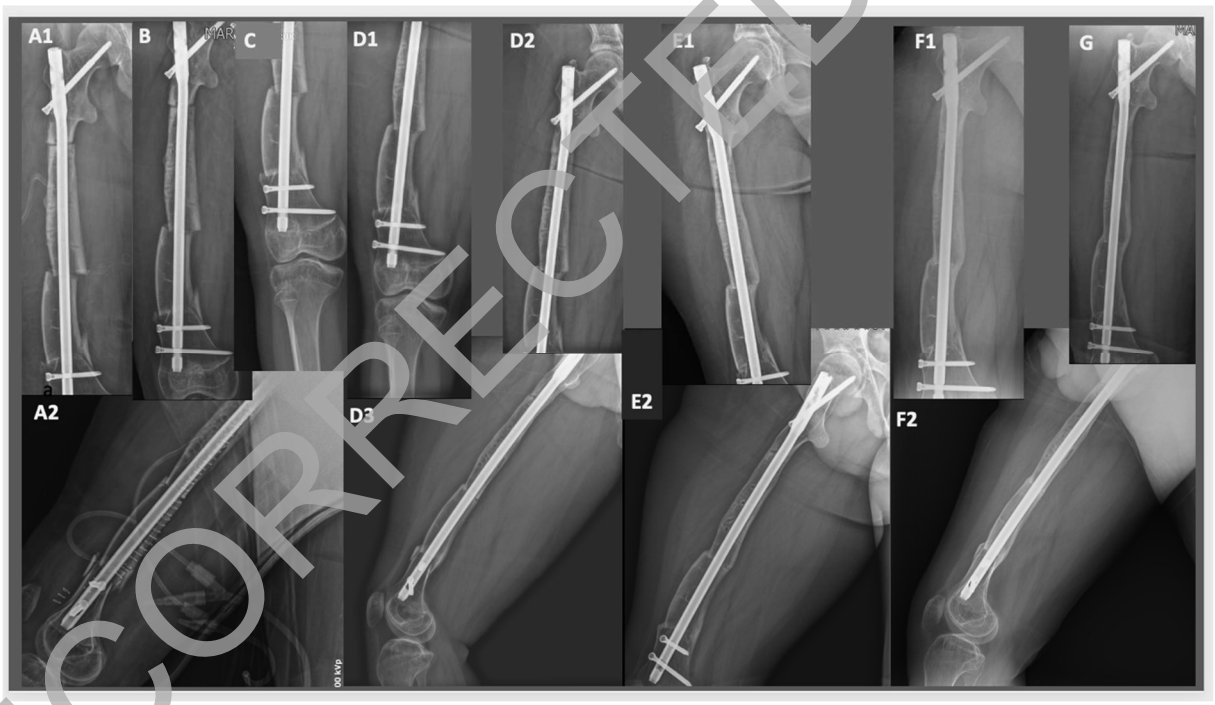
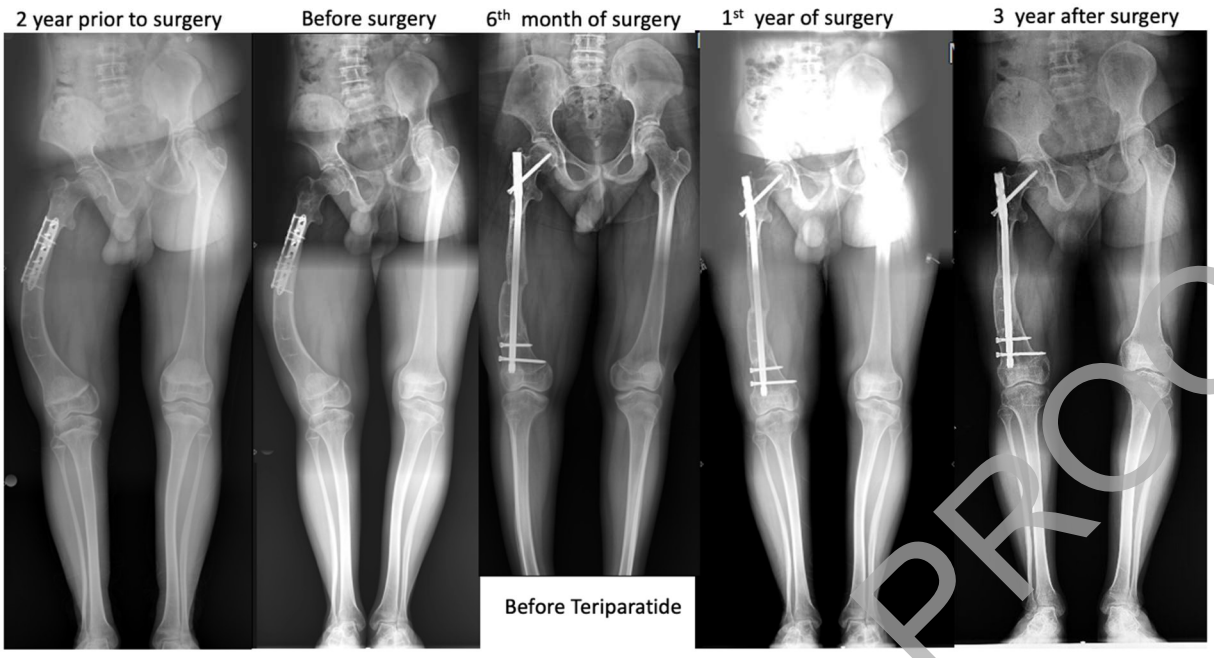


Figure 2. Post-surgical follow-up of the patient. Immediately after surgery (A1, A2), three months post-surgery (B), and four months post-surgery (C), persistent delayed union at the osteotomy sites is observed. At 6 months post-surgery (D1, D2, D3), prior to initiation of teriparatide treatment, delayed union persists. Progressive and complete healing of the osteotomy sites is observed three months (E1, E2) and sixth months (F1, F2) after the initiation of teriparatide, with continued consolidation six months after cessation of teriparatide treatment (G).



Supplementary Figure. Long-leg radiographs of the patient demonstrating right femoral bowing during the 2-year treatment period and after surgical correction during follow-up

Table 1. Comparison of biochemical markers during alendronate (ALN) therapy, before and after teriparatide (TPTD) treatment

Biochemical Marker	Under ALN	Pre-TPTD	3rd month of TPTD	6th month of TPTD	Post- TPTD (1.5 mo)	Under ALN[a]	Last visit
Osteocalcin (12-114 ng/mL)	19.1	34.0		50.2	21.1	31.5	28.5
Alkaline Phosphatase (49-139 U/L)	78	87.0	88	89	92	74	53
Parathyroid Hormone (15-65, 12-88* ng/L)	25.6	27.2	29.7	10.3	11.7	11.7	75.5*
25-OH Vitamin D (20-100 ug/L)	49.5	36.8	19.8	23.6			20.6
1.25 di(OH) Vitamin D (28-81.8 pg/ml)			94.2				
Calcium (Ca, 8.4-10.2 mg/dL)	9.5	9.9	9.6	10.5	10.5	9.5	9.4
Phosphate (2.9-5.1 mg/dL)	3.6	4.3	4.4	4.5	4.2	4.2	4.6
Magnesium (1.8-2.6 mg/dL)	1.9	1.8	2.0	1.9	2.2	2.2	2.1
C-telopeptide (0-0.85 □g/L)	0.4					0.249	0.237
Procollagen I, N-terminal (80-480 □g/L)	75.44						
U-Ca/Cre (<0.22 mg/mg)	0.07	0.28	0.09	0.37		0.05	0.04
TRP[b] (≥85%)	93.3	88.4		98.3		94.5	94.4
TMP/GFR[c] (2.2-3.6 mg/dL)	3.97	3.99		6.22		4.88	5.3

[a] 9 months after TPTD discontinuation, [b] Tubular Reabsortion of Phosphate,
[c] Tubular Maximum Reabsortion of Phosphate to Glomerular Filtration Rate