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# Teriparatide in sequental treatment of osteoporosis in a patient with spinal muscular atrophy: a case report and literature review

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ABSTRACT

We report the case of a female patient with spinal muscular atrophy type 3c, low bone mineral density and multiple fragility fractures, successfully treated with teriparatide. She sustained a vertebral fracture at age 35 years while treatment naïve, and additionally, one vertebral fracture as well as an intertrochanteric right hip fracture during the 5-year treatment with oral bisphosphonates. A sequential 2-year treatment with teriparatide followed by a one-year treatment with oral bisphosphonate risedronate resulted in an overall 11-year fracture-free period and stable bone mineral density. Teriparatide is an osteoanabolic medication that effectively reduces vertebral and nonvertebral fractures in postmenopausal, male and glucocorticoid-induced osteoporosis, particularly in individuals at very high risk of fracture. In the context of neuromuscular disease, teriparatide proved effective in the treatment of osteoporosis in Duchenne muscular dystrophy. In contrast, the data for patients with spinal muscular atrophy are lacking. Further studies are needed to assess its role in this population.

**Patient consent Statement**

The patient provided written informed consent for the publication of anonymized clinical data and diagnostic and treatment details included in this case report. She was informed about the nature of the publication, understood the purpose of sharing her medical history for scientific advancement, and agreed without coercion. All ethical principles in accordance with the Declaration of Helsinki were upheld.

## Introduction

Spinal muscular atrophy (SMA) is a neurodegenerative disorder caused by a mutation in the gene *Survival motor neuron 1* (*SMN1*), leading to loss of SMN protein [1]. The incidence is 1 in 11.000 live-born children; the prevalence of the carrier is 1 in 47 [2]. A homologous variation of gene *SMN2* can partially replace SMN protein. An additional number of copies of the SMN2 gene affects the disease onset, severity, and survival [3]. SMA primarily affects alpha motor neurons in the spinal cord and brainstem, leading to weakness in the limbs, trunk and bulbar muscles. In severe forms, paralysis progresses to respiratory failure and, ultimately, death [4].

Bone impairment in SMA is an understudied area. Available data in children and adolescents with SMA indicate a high prevalence of low bone mineral density (BMD) and fractures [5–7]. Despite the recommendations for the care of SMA patients, which emphasise bone health care, data on the prevalence of osteoporosis, fractures, and treatment in adult SMA patients are lacking [8]. The pediatric population has limited knowledge of treatment with parenteral bisphosphonate (BP) and denosumab [9,10]. There is no data on treatment with oral BP and osteoanabolic medications in the SMA population.

An increased number of adult patients is expected as disease-modifying drugs will continue to prolong life expectancy and improve quality of life. More data are needed to address bone health in adults. We present the first case of an adult woman with SMA type 3c with low BMD and multiple fragility fractures sequentially treated with peroral BP and the osteoanabolic medication teriparatide. Additionally, we reviewed the known literature on osteoporosis and treatment in SMA patients.

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## Case report

A 40-year-old female with SMA type 3c was referred to the Endocrinology Department for evaluation and treatment of osteoporosis. As an infant, her motoric development was initially normal. At the age of 4 years, the first symptoms of muscle weakness appeared. Soon after she was diagnosed with SMA, genetic testing confirmed a homozygous deletion of 7 exons in *SMN1* and 4 copies of *SMN2*. She has been wheelchair-bound since the age of 15. She sustained a fracture of the Th12 vertebrae at the age of 35 years. According to national guidelines and insurance restrictions at that time, teriparatide treatment in Slovenia was indicated for patients with vertebral fractures sustained after at least one year of antiresorptive therapy and not for naive patients. She refused treatment with zoledronic acid (Zoledronic Acid). Denosumab was not yet available: Based on her preference, availability and insurance restrictions, treatment for osteoporosis was initiated with oral alendronate 70 mg QW, cholecalciferol 800 IU QD, and calcium carbonate 1 g QD. After one year of treatment, alendronate was changed to oral ibandronate 150 mg QM, as she wanted to reduce the frequency of administration from once weekly to once monthly.

At the age of 36 years, she sustained an intertrochanteric hip fracture during assisted movement from a wheelchair to a bed. The hip fracture was treated surgically with internal fixation. Because of respiratory failure and hemodynamic instability, she required prolonged intensive care, mechanical ventilation, and prolonged weaning post-surgery. Following an additional four years of ongoing treatment with ibandronate, the patient developed severe back pain without any high-energy injury. A spinal X-ray revealed degenerative changes and a Th7 vertebral fracture.

On presentation, she was 40-years-old, weighed 50 kg and had a segmental height of 166 cm. She reported maintaining regular dietary habits, including the consumption of dairy products. Her swallowing function was normal, and she had not previously been treated with corticosteroids. Menarche occurred at 12 years of age, and her menstrual cycles have always been regular. At the age of 30, she gave birth to a healthy girl. On neurologic evaluation, she could sit without support and displayed dorsal flexion against gravity and flexion of the elbows. She raised her hands to her forehead with compensatory anteflexion of the head. On the Revised Hammersmith Scale for SMA, she achieved 5 out of 69 points. Screening for multiple myeloma was negative, with a normal proteinogram. We also excluded hyperparathyroidism, hyperthyroidism, and osteomalacia. BMD, measured with the Hologic Discovery A densitometer, was significantly lowered. Assessment of BMD on the lumbar spine was not possible due to degenerative changes. Laboratory tests and BMD at the first visit are in Table 1.

Given the lack of therapeutic response to bisphosphonates and the patient's persistently high risk of subsequent fragility fractures, treatment was switched to subcutaneous teriparatide at a dose of 20 $^{\mu}$g once daily. Concurrently, the dose of cholecalciferol was increased to 1000 IU daily, while calcium carbonate supplementation was discontinued to reduce the risk of hypercalciuria. Teriparatide therapy was administered for the recommended duration of two years. This intervention, although not yet described in adults with SMA, was selected due to its osteoanabolic mechanism of action.

Upon completion of this treatment, and consistent with clinical guidelines for managing osteoporosis in high-risk individuals, oral risedronate was introduced at a dose of 75 mg on two consecutive days each month for one year. Given the subsequent reduction in bone turnover markers and the stabilization of bone mineral density, risedronate therapy was discontinued.

The patient was then followed for an additional eight years. There were no additional vertebral or non-vertebral fractures during treatment and follow-up, which lasted a total of 11 years. Data on treatment, laboratory, and BMD assessment are presented in Table 1. In the two-year teriparatide treatment, BMD was stable. BMD significantly declined on the femoral neck at four years (at the age of 47 years) and on the hip at eight years (at the age of 51 years) after discontinuation of risedronate (Fig. 1). The bone formation marker PINP increased markedly in the first year of treatment and then decreased in the second year. After treatment with risedronate, both PINP and bone degradation marker CTX decreased and started to increase only after 8 years. Calcium decreased significantly during teriparatide treatment and the first year of risedronate treatment. At all times of evaluation, normal phosphate, iPTH, and alkaline phosphatase levels were observed. In the eighth year of follow-up, with an additional decrease in BMD, we restarted the treatment with risedronate.

## Methods

Informed, written consent to participate and for the publication of any images, clinical data and possible other data was obtained from the patient. The study complies with local and ethical standards. Approval from the ethics committee was not required.

## Discussion

Given the history of multiple low-energy fragility fractures and significantly reduced BMD, the patient was considered to be at very high risk for additional fragility fractures. Sequential therapy with teriparatide for two years followed by risedronate for one year resulted in a fracture-free period during treatment and throughout an additional eight years of follow-up.

**Table 1**
Laboratory and dual X-ray absorptiometry measurements during treatment.

| Age (years) | Treatment | Corrected Ca mmol/l | P mmol/l | iPTH ng/l | CTX pmol/l | PINP µg/l | AP µkat/l | VitD nmol/l | BMD (FN) g/cm$^2$ | Z-score (FN) SD | BMD (TH) g/cm$^2$ | Z-score (TH) SD |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 36 | ibandronate (started) | 2.43 | 1.05 | - | 1962 | - | 0,61 | - | 0.435 | −3.5 | 0.491 | −3.6 |
| 40 | teriparatide (started) | 2.32 | 1.11 | 32.6 | 1043 | 23.7 | 0.64 | 93.0 | 0.453 | −3.3 | 0.505 | −3.4 |
| 41 | teriparatide (1 year) | 2.15 | 0.89 | - | - | 80.8 | 0.87 | 54.1 | 0.428 | −3.5 | 0.510 | −3.3 |
| 42 | teriparatide (2 years) | 2.10 | 0.86 | - | - | 57.4 | 0.91 | 63.5 | 0.423 | −3.5 | 0.500 | −3.4 |
| 43 | risedronate (1 year) | 2.06 | 0.79 | - | 256 | 17.9 | 0.71 | 107.2 | 0.434 | −3.4 | 0.505 | −3.3 |
| 44 | none | 2.17 | 0.93 | - | 256 | 15.2 | 0.77 | 133.2 | 0.397 | −3.7 | 0.495 | −3.4 |
| 45 | none | 2.03 | 0.87 | - | 326 | 17.9 | 0.76 | 82.0 | 0.403 | −3.6 | 0.513 | −3.2 |
| 47 | none | 2.23 | 0.85 | 44.0 | 474 | 18.5 | 0.85 | 140.7 | 0.380 | −3.6 | 0.491 | −3.3 |
| 51 | none | 2.31 | 0.90 | 34.0 | 542 | 25.7 | 0.89 | 73.6 | 0.357 | −3.6 | 0.474 | −3.3 |

AP – alkalic phosphatase; BMD – bone mineral density; Ca – calcium; CTX - C-terminal telopeptide of type I collagen; FN – femoral neck; iPTH – intact parathormone; P – phosphate; PINP – N-terminal propeptide of type I procollagen; SD – standard deviation; TH – total hip; VitD – vitamin D.

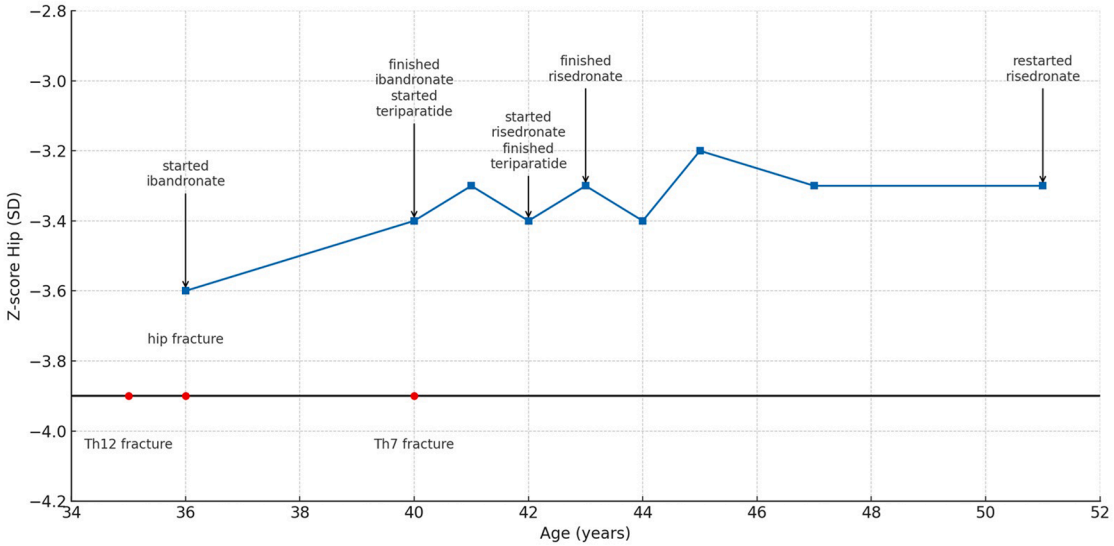


**Fig. 1.** Z-score for hip with treatment and fracture timeline.

Low BMD and fragility fractures were likely the consequence of muscle weakness and immobility, resulting in reduced mechanical loading. Our patient sustained a vertebral fracture while treatment naïve and additional vertebral and hip fractures during the 5-year treatment with oral bisphosphonates by the age of 40, highlighting the limited effectiveness of oral antiresorptive therapy in this context. Studies on pediatric and adolescent populations have demonstrated that patients with more severe subtypes of SMA have lower BMD, as reflected by lower Z-scores at younger ages [5,6]. Z-scores decrease during follow-up [5,7]. Additional risk factors for low BMD include increasing age and prolonged immobility [11,12]. Low BMD increases the risk of bone fractures. The most common being in the distal femur, followed by the lower leg, ankle, and arm [5,13]. Additionally, vertebral fractures are frequent in the pediatric population. They are usually asymptomatic and incidentally found with spine x-ray screening [6,7,14].

Fractures in the SMA population are usually treated conservatively with casts due to the increased risk of complications during anaesthesia and the risk of secondary operations and revisions. Conversely, during immobilisation, contractures may develop or worsen. Many patients lose their ability to walk or stand due to contractures or progressive muscle weakness. Because of pain and fear of further fractures, they limit activities and participation in social life [13].

Our patient developed post-operative complications and needed prolonged weaning from mechanical ventilation. Therefore, adequate primary and secondary fracture prevention treatment is essential. Therapy with parenteral BP zoledronic acid (Zoledronic Acid) is effective. However, an acute phase reaction was present in more than a third of SMA patients, and an atypical femur fracture was reported [9]. Denosumab is a potent antiresorptive agent. Data in the SMA population is scarce. Successful short-period treatment with denosumab was described in a case report of a child with SMA and fragility fractures [10].

Teriparatide is a recombinant form of human parathyroid hormone and is the first osteoanabolic drug available. It increases the number of osteoblasts by stimulating osteoblast formation and inhibiting apoptosis. This unique action promotes bone formation more than bone resorption [15]. Teriparatide treatment is associated with a 65 % relative risk reduction and a 9 % absolute risk reduction for vertebral fractures compared to placebo. Treatment also reduces the risk of non-vertebral fractures by 53 % [16]. The most significant benefit is in patients with a high burden of vertebral fractures. In patients with three or more vertebral fractures, teriparatide reduced the relative risk of vertebral fractures by 86 % [17]. Compared to risedronate treatment, in postmenopausal women with severe osteoporosis, treatment with teriparatide successfully reduced the incidence of major osteoporotic fractures consisting of vertebral, hip, forearm, or humerus fractures by 60 % [18].

Teriparatide efficacy was proven in postmenopausal women, men and glucocorticoid osteoporosis. The potential risk of complications with teriparatide treatment presents a risk of hypercalcemia and, consequently, hypercalciuria and nephrolithiasis. Hypercalcemia was reported in 5 % of the patients and was usually intermittent [15]. However, the evaluation of calciuria in SMA is limited due to muscle wasting and low creatinine.

Sequential treatment with BP after teriparatide is beneficial as it prevents bone resorption following the cessation of teriparatide's anabolic action. Oral BP maintained or increased BMD after one year of teriparatide treatment [19]. Treatment with BPs post-teriparatide can be omitted in rare cases, such as in pregnancy and lactation-associated osteoporosis [20]. Although peroral BPs are usually well tolerated, potential side effects could be more frequent in the SMA population due to specific health issues that these patients face, such as possible swallowing impairment and muscle vulnerability. Esophageal irritation and erosions can occur with oral BP therapy, particularly in patients with known gastroesophageal reflux disease or esophageal stricture. Also, there is a possibility of severe and incapacitating musculoskeletal pain that can occur at any point after the initiation of peroral or parenteral BP therapy. This severe musculoskeletal pain was distinct from the acute phase response [21]. Additional benefits could be achieved with a more potent antiresorptive denosumab. Treatment of postmenopausal women sequentially or concomitantly with teriparatide resulted in continued increases in BMD in 48 months. On the other hand, changing from denosumab to teriparatide led to a significant progressive bone loss [22].

In the context of neuromuscular disease, teriparatide proved effective in the treatment of osteoporosis in Duchenne muscular dystrophy (DMD). In a 20-year-old male patient with three vertebral fractures, treatment resulted in the prevention of additional fractures [23]. Additionally, in the prevention of vertebral fractures, a study of six DMD patients found that the fracture incidence of long bones was reduced from 0.84 per year before treatment to 0.09 per year over an 11.0 patient-year period [24]. Similar to our case report, bone formation markers in patients with DMD increased. However, the effects on BMD and Z-score are inconsistent and vary depending on whether BP is used as pretreatment. In the case report, the BMD and Z-score increased as the patient was treatment-naïve, and glucocorticoid treatment was

discontinued [23]. In our case, and in the study of DMD patients treated with BP, BMD remained stable. In DMD patients, the Z-score was reported to decrease [24]. The difference between the study and our data could be explained by not correcting children's reference Z-scores for height.

Our case report is limited by missing data on BTM and the inability to measure BMD in the lumbar spine, where changes occur most rapidly. The lumbar spine is also the site for the most accurate monitoring of the effects of osteoporosis treatment on BMD. In addition, treatment with ibandronate was chosen based on patient preference, although this agent is likely suboptimal for individuals at high risk of fractures. Ibandronate effectively prevented vertebral fractures in populations at lower fracture risk. Prevention of nonvertebral fractures was only proven by post-hoc analysis of a subgroup of patients with BMD T-score $< -3.0$ SD [25]. Risk assessment with BMD and BTM measurement has its limitations. Regarding our case, BMD does not assess bone quality or material properties and has limited sensitivity in individuals with prior fractures. BTM have no validated thresholds, have poor standalone predictive value, and single measurements are unreliable for risk stratification. The Fracture risk assessment tool (FRAX®) does not account for those younger than 40 with an increased risk of falls, multiple fractures, or immobilisation.

In conclusion, this case illustrates that spinal muscular atrophy can be associated with clinically significant osteoporosis and fragility fractures that may not be adequately managed with oral bisphosphonate therapy alone. Sequential treatment with teriparatide followed by risedronate was associated with a prolonged fracture-free period and stable bone health in our patient. Although this is a single case, it highlights the potential value of individualized treatment approaches and supports further investigation of osteoanabolic and sequential therapy strategies in adults with SMA. Larger studies are needed to determine the efficacy, safety, and optimal sequencing of such therapies in this specific population.

## Ethics approval and consent to participate

Consent to participate and written consent to publish this paper were obtained from the patient.

## Data availability

The data is available upon request.

## CRediT authorship contribution statement

**Matej Rakusa:** Writing – original draft, Investigation, Conceptualization. **Lea Leonardis:** Writing – review & editing. **Blaž Koritnik:** Writing – review & editing. **Andrej Janež:** Writing – review & editing, Supervision. **Mojca Jensterle:** Writing – review & editing, Supervision, Investigation.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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