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the long-term effect induced by IL-6. To reconcile these apparently contradictory data, we reasoned that e-Src and IGFBP5 may be part of a third loop that negatively controls the e-Src/IL-6 activating cycle. Analysing the IGFBP-5 promoter, we recognized a responsive element for Runx2, a transcription factor indispensable for osteoblast differentiation that we found up-regulated in e-Src inhibited osteoblasts. To confirm this negative loop, we overexpressed Runx2 in osteoblasts and found an increase of IGFBP5 mRNA.

Conclusions: These results suggest that the balance and the timing between these opposite loops could account for a fine tuning regulation of osteoblast activity, and underscore a new role for IGFBP5 in the context of the signals that link e-Src and IL-6 pathways.

Disclosure of Interest: None declared.

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DELAYED UNION AFTER ATYPICAL SUBTROCHANTERIC FRACTURE UNDER ALENDRONATE TREATMENT: CASE REPORT
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Objectives: Atypical subtrochanteric fractures after long-term alendronate treatment, despite its rare occurrence, are difficult to handle. A meta-analysis of large clinical trials of bisphosphonates shows the frequency of atypical fractures at 2.3/10,000 patient-years. Delayed union of this fracture is one of the discussed topics. The aim of this study is to present a case of a patient on a long-term alendronate treatment with an atypical subtrochanteric fracture and delayed union.

Materials/Methods: Case study

Results: A 68-year-old woman after a sustained atypical fracture of right femur was admitted to Krakow Medical Centre after intramedullary nailing. Six months before fracture the patient had been complaining of pain in the right thigh. Six months after the surgery a delayed union was observed. Before the fracture the patient had been treated with alendronate for 6 years and had been under hormone replacement therapy for 10 years due to climacteric symptoms. Hyperthyroidism was treated surgically in 2007 and currently the patient is suffering from hypothyroidism. DXA BMD Neck (opposite side) = 0.633 g/cm², T-score = -2.0 SD, BMD L1-L4 = 0.897 g/cm², T-score = -1.4 SD. Vitamin D3 (25(OH)D = 17.0 ng/ml), decreased daily calcuria (1.8 mmol/24 h). Level of calcium, phosphorus, PTH in serum was normal. Increased level of ICTP and alkaline phosphatase was observed.

Conclusions: Taking into consideration the ASBMR Report (2010) the following risk factors of an atypical fracture were observed in our case: osteopenia at the time of bisphosphonate therapy commencement, a 6-year alendronate therapy (despite the lack of diagnosed osteoporosis), prior long-term hormone replacement therapy, vitamin D3 deficiency. A patient complaining of thigh pain should be evaluated for the risk of atypical fracture. Our case seems to support the possible delayed union after long-term alendronate treatment but concomitant medications should be taken into consideration as well.

Disclosure of Interest: None declared.

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THE EFFECT OF ALENDRONATE ON BONE METABOLISM IN TRPV5⁺/⁺ AND TRPV5⁻/⁻ MICE
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Objectives: To explore the effect of alendronate on bone metabolism in TRPV5⁺/⁺ and TRPV5⁻/⁻ mice.

Materials/Methods: In this study, the introduction of TRPV5 knock-out heterozygous mice, feeding and breeding in accordance with the requirements of SPF-class standards of animal feeding, there are three possible offspring phenotypes (TRPV5⁺/⁺, TRPV5⁻/⁻ and TRPV5⁻/⁺), selected the wildtype TRPV5⁺/⁺ and homozygous TRPV5⁻/⁻ and divided them into experimental groups and control groups separately. The experimental groups were given standard animal feed with alendronate weekly 2 mg/kg each time, the control groups were given standard animal feed only. 8 weeks later, 3 groups of data were collected from all the mice. One from biochemical analysis of bone metabolism in urine and serum samples; another from the femoral bone quantity based on application CT scanning; the third from analyzing the quantitative polymerase chain reaction of the calcium channel on the edge of osteoclast wrinkled surface which is extracted from mouse femur, the fourth from kidney, duodenum sample preparation.

Results: In experimental groups, the quantity of mRNA in TRPV5⁺/⁺ mice has increased, the thickness of the femur become more rich.TRPV5⁻/⁻ mice increased the thickness of the femur, while the TRPV5⁻/⁺ mouse femoral thickness become normal, alendronate significantly increased PTH hormone levels and expression of TRPV5⁻/⁻ mice. Alendronate led TRPV5 gene regulation of TRPV5⁺/⁺ mice, which will reduce absorption functions of osteoclasts of TRPV5⁻/⁻ mice.

Conclusions: Alendronate has different function to Alendronate inhibition of bone resorption achieved through the regulation of calcium channel TRPV5.

Disclosure of Interest: None declared.