There were also significant positive correlations between IGF-1 and NTx with 60 ECs (2 h, $p<0.01$; day 1, $p<0.05$).
Conclusions: We found that one bout of severe ECs caused increases in OC and TRACP-5b, which promote increased bone metabolism. Our results suggest that contraction-induced IGF-1 may activate OC and NTx in acute response. In future, the mechanism of high bone turnover suggested by our result must be clarified in the context of the association between skeletal muscle and bone.

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GASTROINTESTINAL SECONDARY HYPERPARATHYROIDISM AND DECREASED BONE MINERAL DENSITY IN CELIAC DISEASE: A CLINICAL CASE

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Aims: The article covers the issue of development of osteoporosis and other disorders of BMD in patients with malabsorption syndrome, in particular with celiac disease.
Methods: The etiology and pathogenesis of decrease in BMD in patients with chronic diseases of the digestive system include, besides population-based risk factors, processes associated with disorders of vitamin D conversion, which leads to reduction of the absorption of calcium, magnesium and phosphorus in the intestine and increase of their excretion by the kidneys. As result hypocalcemia activates the parathyroid glands and secondary hyperparathyroidism, and against the background of chronic inflammation increases the activity of cytokines, having bone resorptive action.
Results: A 37-year-old woman with pain in the bones and joints, muscles, diarrhea, marked reduction in body weight (weight 38 kg with height 153 cm), general weakness and malaise was examined by doctors of various specialties (physician, gastroenterologist, neurologist), but the prescribed treatment was ineffective. It is known that at the age of 35 she had fallen and there was a fracture of the right hip joint which was treated by arthroplasty. As complaints were progressing patient was sent to hospital, where had firstly examined levels of calcium and phosphorus metabolism and PTH. Her calcium level have been low associated with 5-times-elevated PTH and normal phosphorous, which firstly initiated endocrinologist's consultation. The results of bone densitometry corresponds to severe osteoporosis (*Z*-criterion=−5.8). Scintigraphic pattern was assessed in favor of hyperplasia left parathyroid gland. Next, to clarify the causes and nature of malabsorption syndrome was conducted histological examination of small intestinal mucosa, which is fully confirmed the diagnosis of celiac disease (type Marsh IIIC). A gluten-free diet and calcium with vitamin D have been prescribed for treating celiac disease and osteoporosis. 2.5 years later the results of bone densitometry of the lumbar vertebrae is correct, the neck of the left femur - osteopenia (Z-criterion= −1.6), laboratory parameters within normal limits. In accordance with the results, treatment of osteoporosis was ended, but compliance with a gluten-free diet should be continued.
Conclusions: Given clinical case is interesting from the standpoint of difficulty diagnosing causes of osteoporosis that developed in the patient with secondary hyperparathyroidism on background malabsorption syndrome due to the classical form of celiac disease. Such situation requires participation in rehabilitation health care programs involving not only endocrinologists or rheumatologists, but also gastroenterologists and internists too.

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COMPENSATORY UPREGULATION OF THE VEGF PROTEIN EXPRESSION IN THE GROWTH PLATE OF TYPE 2 DIABETIC RATS WITH OSTEOPENIA

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Aims: Type 2 diabetes mellitus (DM) has been shown to induce osteopenia, osteoporosis, and impaired bone elongation in rodents. However, in growing rats, there are several systemic and local growth factors that try to increase bone length and body growth; therefore, it is hypothesized that, to overcome the detrimental effects of DM on endochondral bone growth, the growth plate chondrocytes should activate a compensatory mechanism to maintain bone elongation. The present study aimed to investigate BMD, bone length, growth plate morphology, and the expression of vascular endothelial growth factor (VEGF), a growth factor that stimulates vascular and bone cell invasions to promote bone elongation, in the growth plate of female growing Goto-Kakizaki (GK) diabetic rats.
Methods: Five weeks old wildtype (WT) and GK rats were fed ad libitum with normal diet for 11 weeks. After euthanasia, femoral and tibial lengths were measured. Changes of BMD in femora and vertebrae were analyzed by μCT. To demonstrate growth plate morphology, tibiae were removed, decalcified, sectioned, and processed for H&E staining. Total growth plate height and height of each growth plate zone, i.e., reserve zone (RZ), proliferative zone (PZ) and hypertrophic zone (HZ) were determined by histomorphometric image analysis. Expression of VEGF protein in the growth plate was demonstrated by quantitative immunohistochemistry. Data were compared by unpaired Student's *t*-test.
Results: We found osteopenia in the GK rats as indicated by low BMD in both femora and tibiae. Longitudinal bone growth was also suppressed with shorter bone length in the