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# THE EFFECT OF STEROID THERAPY ON BONE TISSUE IN RHEUMATIC DISEASES (DESCRIPTION OF A CLINICAL CASE)

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**Abstract**

Joint and bone pain is the most common symptom of most joint pathologies of inflammatory autoimmune nature. In patients with diagnosed rheumatic disease undergoing hormonal therapy, it can be regarded as an exacerbation of the underlying disease, and not as a symptom of secondary osteoporosis.

The article presents a clinical case of a patient with mixed connective tissue disease (systemic lupus erythematosus + juvenile idiopathic arthritis), who was referred for hospitalization due to deterioration of health in the form of severe arthralgia and myalgia against the background of glucocorticoid therapy.

***Keywords:*** *arthralgia, glucocorticosteroids, osteoporosis.*

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**Резюме**

# ВЛИЯНИЕ СТЕРОИДНОЙ ТЕРАПИИ РЕВМАТИЧЕСКИХ ЗАБОЛЕВАНИЙ НА СОСТОЯНИЕ КОСТНОЙ ТКАНИ (ОПИСАНИЕ КЛИНИЧЕСКОГО СЛУЧАЯ)

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Боли в суставах и костях – самый распространенный симптом большинства суставных патологий воспалительного аутоиммунного характера. У пациентов с диагностированным ревматическим заболеванием, находящихся на гормональной терапии, могут быть расценены как обострение основного заболевания, а не как симптом вторичного остеопороза.

В статье представлен клинический случай пациента со смешанным заболеванием соединительной ткани (Системная красная волчанка + Ювенильный идиопатический артрит), который был направлен на госпитализацию в связи с ухудшением самочувствия в виде появления выраженных артралгий и миалгий на фоне получаемой глюкокортикоидной терапии.

***Ключевые слова:*** *артралгии, глюкокортикостероиды, остеопороз.*

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**Түйіндеме**

# РЕВМАТИКАЛЫҚ АУРУЛАР КЕЗІНДЕ СТЕРОИДТЫ ЕМНІҢ СҮЙЕК ТІНІНЕ ӘСЕРІ (КЛИНИКАЛЫҚ ЖАҒДАЙДЫ СИПАТТАУ)

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Буындар мен сүйектердегі ауырсыну – қабынулық аутоиммундық сипаттағы бірлескен патологиялардың көпшілігінің ең көп таралған симптомы. Ревматикалық ауру диагнозы қойылған гормональді ем қабылдайдын науқастарда екіншілік остеопороз емес негізгі аурудың өршуі ретінде бағалануы мүмкін.

Мақалада алынған глюкокортикоидты ем аясында айқын артралгия және миалгия түрінде жағдайының нашарлауына байланысты ауруханаға жатқызуға бағытталған дәнекер тінінің аралас ауруы бар науқастың клиникалық жағдайы (жүйелі қызыл жегі + ювенильді идиопатиялық артрит) ұсынылған.

**Түйінді *сөздер:*** *артралгиялар, глюкокортикостероидтар, остеопороз.*

***Дәйексөз үшін:***
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**Introduction**

Joint and bone pain is one of the most common symptoms associated with autoimmune inflammatory joint pathologies. In patients diagnosed with rheumatic diseases who are undergoing corticosteroid therapy, these symptoms may be mistakenly interpreted as an exacerbation of the primary disease rather than a manifestation of osteoporosis.

This article presents a clinical case involving a patient with mixed connective tissue disease (Systemic Lupus Erythematosus and Juvenile Idiopathic Arthritis), who was hospitalized due to worsening symptoms, including pronounced arthralgia and myalgia, while receiving glucocorticoid therapy.

As is well known, this autoimmune pathology, characterized by clinical and immunological features of mixed connective tissue disease, presents with diverse manifestations occurring in various combinations; however, the predominant feature is the musculoskeletal syndrome due to the overlap of clinical signs with those of rheumatoid arthritis.

Osteoporosis (OP) is a metabolic skeletal disorder characterized by reduced bone mass, deterioration of bone microarchitecture, and, consequently, an increased risk of fractures from minimal trauma [5]. Osteoporosis can be classified as either primary or secondary. Primary osteoporosis develops independently, whereas secondary osteoporosis arises as a result of various diseases, conditions, or the use of certain medications.

The most frequent cause of secondary OP in children is the prolonged use of glucocorticosteroids, which, even in low doses, can lead to a significant reduction in bone mineral density (BMD). Glucocorticosteroids (GCS) suppress osteoblast activity and promote bone resorption. As a result, bone density and mass decrease, and bone architecture is altered. One of the key risk factors for steroid-induced osteoporosis is a high daily dose of GCS (more than 7.5 mg) and prolonged use.

**Relevance.** Patients of all ages and sexes undergoing long-term glucocorticoid therapy are considered at high risk for developing osteoporosis. The public health significance of osteoporosis is largely due to its consequences—namely, skeletal fractures. Bone is a metabolically active tissue in which continuous remodeling occurs, involving the resorption of old bone and the formation (ossification) of new bone tissue.

Prolonged use of GCS for the treatment of rheumatic diseases (RD) inhibits bone formation and increases resorption, reducing serum calcium concentrations. This can lead to secondary hyperparathyroidism, simultaneously stimulating osteoclasts and inhibiting osteoblasts. These effects, along with protein catabolism-induced reduction in protein matrix components, may result in impaired bone growth in children and adolescents and the development of osteoporosis in pediatric patients of all ages. In addition, GCS therapy decreases calcium absorption in the intestines, increases urinary calcium excretion, impairs ossification processes, and reduces endogenous estrogen synthesis—an important hormone for bone strength.

Maintaining stable plasma calcium levels is essential for proper physiological function and is tightly regulated by calcium homeostasis mechanisms. Vitamin D plays a crucial role in bone maintenance and growth by enhancing

calcium absorption in the intestines. Patients receiving GCS therapy should undergo regular monitoring of serum electrolyte and vitamin D levels. Additionally, bone mineral density (BMD) measurement is essential for diagnosing steroid-induced OP. In pediatric patients, BMD is assessed using the Z-score (the number of standard deviations above or below the mean for age-matched individuals). A normal Z-score in children ranges from +2.5 to -0.9 standard deviations. Values below -1 SD indicate decreased bone density (osteopenia), while scores below -2.5 SD are diagnostic for osteoporosis.

Epidemiological studies [9] have shown that the most rapid bone mass loss occurs within the first year of initiating corticosteroid therapy. Lumbar spine BMD may decrease by up to 30% within six months of treatment, and proximal femur BMD may decrease by 14% over 12 months. Although the rate of bone loss typically slows thereafter, it remains significantly higher than the rate of age-related physiological loss. Osteoporotic fractures occur in 30–50% of patients undergoing long-term systemic GCS therapy. The most common fracture sites include the ribs, vertebrae, and diaphyses of long tubular bones. At a daily GCS dose of 5 mg or more (in prednisone equivalents), the relative risk of fractures compared to the general population increases 1.9-fold overall, 2-fold for hip fractures, and nearly 2.9-fold for vertebral fractures. The greatest loss of bone mass occurs during the early stages of the disease, likely due to both high inflammatory activity and the use of high GCS doses. Thus, this patient category requires early screening for OP and timely initiation of anti-osteoporotic therapy.

In osteoporosis, clinical manifestations are often subtle and asymptomatic until the condition is advanced. Bone tissue serves as a reservoir and compensates for reductions in key parameters of phosphorus-calcium metabolism in the bloodstream by releasing essential components to maintain homeostasis [4].

**Objective**

To highlight the importance of a differential diagnostic approach in evaluating complaints of arthralgia in a patient with a rheumatic disease undergoing glucocorticoid therapy.

**Clinical Case Summary**

Patient B., 16 years old, was admitted to the Pediatric Department of the University Hospital at NAO "Semey Medical University" with complaints of bone pain; arthralgia in the elbow, knee, and ankle joints; muscle pain; morning stiffness lasting 20–30 minutes; general weakness; and recurrent oral ulcers.

**Medical history**: Since 2015, the patient has been followed for a diagnosis of Mixed Connective Tissue Disease, including Systemic Lupus Erythematosus (SLE) (manifesting as dermatitis, vasculitis, stomatitis, nephritis, lymphadenopathy, myositis, arthralgia, cheilitis) and Systemic Juvenile Idiopathic Arthritis, with a chronic relapsing course. For the past 10 years, the patient has been receiving Methylprednisolone (Meticor), initially at 8 mg/day and increased to 12 mg/day over the past 3 years, in combination with Hydroxychloroquine (Immard) 400 mg/day.

Over the last 6 months, symptoms worsened, prompting hospitalization for suspected disease exacerbation and administration of pulse therapy with methylprednisolone.

**Life history**: Second child in the family, with age-appropriate growth and development. No history of fractures. Positive family history: rheumatoid arthritis in older sister and maternal aunt; SLE in maternal grandmother.

**Physical examination**:

- Height: 150 cm; Weight: 78 kg; BMI: 34.67 (obesity class I).
- General condition: moderate severity due to musculoskeletal symptoms.
- Excess subcutaneous fat on abdomen and flanks; mild gynecomastia.
- Musculoskeletal: Tenderness over humeral and femoral bones and muscles. No joint deformities.
- No lymphadenopathy or peripheral edema.
- Cardiovascular: HR 98 bpm, rhythmic, muffled heart sounds.
- Respiratory: Vesicular breath sounds, no wheezing.
- Abdomen: Liver palpable +1 cm; spleen not palpable; no tenderness.
- Normal bowel and urinary habits.

**Laboratory and instrumental findings**:

1. CBC (21.05.2025): Hb – 152 g/L; WBC – 5.82 x$10^9$/L; RBC – 4.86 x$10^{12}$/L; PLT – 280 x$10^9$/L; ESR – 10 mm/h.
2. Urinalysis: Sp. gravity – 1025; RBCs – 2/μL; WBCs – 2/μL; protein – negative.
3. ECG (22.05.2025): Sinus arrhythmia (HR 85–92 bpm); signs of ventricular repolarization abnormalities.
4. Echocardiography: Normal heart chamber dimensions; valve structures intact; LVEF – 62%.
5. Ophthalmologic exam: Retinal angiopathy; ametropia.
6. Multislice computed tomography (MSCT) of the chest:The thoracic cage is of normal shape and symmetrical. The lungs are well-aerated and in full contact with the chest wall. No areas of pathological consolidation are detected. The major bronchi are patent and free of pathological inclusions. No pleural effusion is observed. The mediastinum is midline and of normal width. No pathological inclusions are identified in the anterior, central, or posterior compartments. The heart is of normal configuration with non-enlarged borders. The great vessels are unremarkable. The thoracic aorta is not dilated and is uniformly opacified with contrast. The pulmonary artery is not enlarged. Degenerative changes are noted in the thoracic spine.
7. Esophagogastroduodenoscopy (EGD) Findings:The esophageal mucosa is matte pink and glistening. The esophagogastric junction is visualized at 40 cm from the upper incisors; the cardiac rosette closes completely.The stomach is of normal shape and contains approximately 70.0 ml of gastric juice in the fasting state. The gastric body folds are tortuous, longitudinally oriented, and unfold uniformly upon insufflation. Antral peristalsis is active, rhythmic, and visible across all walls up to the pylorus. The gastric mucosa appears pink and glistening. In the antral region, it is edematous and focally hyperemic, with flat and linear erosions of round and polygonal shape, measuring 2–4 mm, covered with fibrin and surrounded by a rim of hyperemia. On palpation, the mucosa is soft, elastic, and mobile. The pylorus is not deformed, closes completely, and opens adequately. The duodenal bulb is not deformed, oval in shape, with a pink, velvety mucosa. The exit from the

bulb is unobstructed. The postbulbar segments are passable and show no abnormalities.

8.Bone densitometry: Total body Z-score = -0.3 SD – consistent with osteopenia for age.

9.Calcium-phosphate metabolism:

- Total calcium: 2.47 mmol/L (normal)
- Ionized calcium: 0.82 mmol/L (↓)
- Inorganic phosphate: 0.88 mmol/L (low-normal)
- Vitamin D: 12.0 ng/mL (deficiency, normal range: 30–100 ng/mL)

The patient with a known history of SLE and systemic JIA presents with symptom exacerbation and features of steroid-induced side effects, including obesity, osteopenia, vitamin D deficiency, and erosive gastritis. Current management involves re-evaluation of immunosuppressive therapy, pulse corticosteroid therapy, gastroprotection, vitamin D supplementation, and bone health monitoring.

**Discussion**

Due to the onset of pronounced pain in the bones, joints, and muscles, as well as the appearance of small ulcers in the mouth—interpreted as characteristic ulceration of the oral mucosa resulting from lupus activity—the boy was hospitalized in UG JSC "SMU" for adjustment of cytostatic therapy and an increase in the glucocorticoid component. However, the absence of fever, normal condition of the skin (i.e., no erythema), lack of articular syndrome or multi-organ involvement, and normal hematological parameters led to a decision to perform a differential diagnosis of arthralgia and myalgia. Moreover, intensive therapy is used in patients with autoimmune diseases, including mixed connective tissue disease, in the presence of severe immune complex pathology with nephrotic syndrome, severe cerebrovasculitis, cytopenias, systemic vasculitis. All of these listed complications were not present in this patient. In this regard, the development of secondary osteoporosis in this patient was suspected.

To this end, it was decided to conduct blood tests for calcium, phosphorus, and vitamin D levels, as well as to perform dual-energy X-ray absorptiometry (DEXA). The patient's total calcium and phosphorus levels were within the normal range—presumably due to compensatory leaching from the bones—while ionized calcium levels were found to be significantly decreased. There was also a noted deficiency in vitamin D, which may additionally be attributed to the fact that patients with systemic lupus erythematosus are contraindicated from ultraviolet exposure, i.e., being out in the sun.

Based on the onset of pronounced bone, joint, and muscle pain, along with the appearance of oral ulcers—regarded as characteristic ulceration of the oral mucosa due to systemic lupus erythematosus (SLE) activity—the patient was hospitalized at the University Hospital of the NJSC "Medical University of Semey" for adjustment of cytostatic therapy and intensification of glucocorticoid treatment. However, the absence of fever, the normal condition of the skin (no erythema), the absence of joint syndrome and multi-organ involvement, as well as normal hematological parameters, prompted a differential diagnosis of the musculoskeletal syndrome. This was particularly relevant given that intensive therapy is typically indicated in patients with autoimmune diseases, including mixed connective tissue disease, in the presence of severe immune complex pathology such as nephrotic syndrome, severe cerebrovascular involvement, cytopenias, or systemic vasculitis. None of these complications were present in this patient.

As a result, the development of secondary osteoporosis was suspected. Accordingly, laboratory tests were ordered to assess serum calcium, phosphorus, and vitamin D levels, and dual-energy X-ray absorptiometry (DEXA) was planned.

The coated tongue and presence of isolated ulcers were found to be a result of erosive gastritis, diagnosed through esophagogastroduodenoscopy and also associated with steroid therapy.

Based on the clinical, laboratory, and instrumental findings, it was decided not to change the treatment for the underlying disease. A concomitant diagnosis was made: *"Drug-induced osteoporosis without pathological fracture. Glucocorticoid-induced myopathy."*

A gradual reduction in glucocorticoid dosage was planned, along with recommendations to enhance the diet with calcium-rich foods or to prescribe calcium and vitamin D in pharmaceutical form, administered in age-appropriate dosages.

In accordance with the guidelines of the American College of Rheumatology (ACR), at discharge the patient will be recommended to undergo regular bone mineral density monitoring once every six months until stable bone density values are achieved [5].

**Conclusions**

To identify the cause of joint and muscle pain, it is essential—taking into account the nature of the therapy being administered—to distinguish a flare of the underlying disease from the likely development of secondary osteoporosis in such patients. Bone pain is a hallmark sign of osteoporosis, ranging in intensity from mild to severe, and can present in various localizations and degrees of expression.

Thus, a mandatory component of treatment for rheumatic diseases is careful monitoring of potential complications of glucocorticoid therapy and the application, whenever possible, of the minimum therapeutically effective glucocorticoid dose.

Prolonged glucocorticoid therapy necessitates the periodic administration of vitamin D and calcium supplements to enhance bone tissue mineralization, thereby serving as a preventive measure against hormone-induced osteoporosis and muscular dystrophy. Timely recognition of the clinical signs of osteoporosis is essential for the appropriate management and referral of the patient, as well as for the prevention of this complication as one of the possible outcomes of the underlying disease.

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