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PAGET'S DISEASE OF BONE AS A RARE CAUSE OF HEADACHE

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Headache is one of the most common complaints encountered in clinical practice. Although primary headaches (such as migraine and tension-type headache) are most prevalent, timely identification of secondary headaches is essential, as they require distinct diagnostic and therapeutic approaches. This paper presents a clinical case of Paget's disease of bone with isolated skull involvement, manifested solely by chronic headache. Based on elevated serum alkaline phosphatase levels, further examination was performed in a 47-year-old patient initially diagnosed with tension-type headache. Magnetic resonance imaging (MRI), computed tomography (CT), and bone scintigraphy confirmed the diagnosis of Paget's disease of bone, after which pathogenetic therapy was initiated.

Keywords: headache, secondary cephalgia, Paget's disease of bone, alkaline phosphatase.

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Introduction. Headache is one of the most common reasons for seeking medical care [1, 8]. According to the Global Burden of Disease (GBD, 2022) study, the overall prevalence of headache was 52.0% (95% CI: 48.9–55.4), migraine – 14.0% (95% CI: 12.9–15.2), and tension-type headache – 26.0% (95% CI: 22.7–29.5). Chronic headaches lasting 15 or more days per month were reported in 4.6% (95% CI: 3.9–5.5) of cases [16]. Headache significantly reduces quality of life and often leads to temporary or permanent disability [14].

In most cases, primary headaches—migraine, tension-type headache, and

cluster headache—are observed. However, secondary headaches are not uncommon; these are symptoms of other, often more serious disorders, requiring specific diagnostic approaches [8].

Paget's disease of bone (PDB, also known as deforming osteitis) is a chronic, slowly progressive skeletal disorder characterized by focal abnormalities in bone remodeling [2]. Excessive osteoclastic resorption, coupled with disorganized osteoblastic bone formation, results in structurally weakened, deformed, thickened, and hypervascularized bone [3]. PDB typically affects older individuals, particularly men, and occurs in approximately 1–5% of people over 50 years old [5]. There is marked geographic variation, with the disease being more common in populations of European descent and relatively rare in Asians [11]. Although its exact cause remains unclear, PDB is thought to develop in genetically predisposed individuals, as a positive family history is reported in 5–40% of cases [9]. Environmental factors, including viral infections (especially paramyxoviruses) and vitamin D deficiency, have been proposed as contributing factors [3].

Clinically, the disease manifests with bone pain (73.8%), bone deformities (18.1%), hearing loss (7.9%), and patho-

logical fractures (5.7%). However, in up to 22% of patients, PDB may remain asymptomatic [15]. Neurological complications include hearing loss (76%), neck pain (2–5%), cranial nerve palsies (0.2–41%), and peripheral nerve involvement (2–5%) [13]. Although headache is frequently reported in PDB, it is a nonspecific symptom [4]. Typically, headaches are localized in the posterior head and are aggravated by increased intracranial pressure, such as during coughing or defecation. Possible mechanisms include skull hypervascularization, basilar impression, hydrocephalus, compression of trigeminal nerve roots, thickening of facial bones, and intracranial hemorrhage [7].

Thus, although rare, Paget's disease of bone should be considered in the differential diagnosis of chronic headache. We present a clinical observation of a patient in whom headache was the only manifestation of Paget's disease of bone.

Case Description. A 47-year-old man was admitted in August 2025 to the neurology department with persistent dull and aching headaches localized in the temporal and infraorbital regions (VAS score 7), unaccompanied by nausea or vomiting.

History of present illness. The patient had been experiencing headaches since

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2023, initially on the right side only, later involving both sides and the infraorbital area. The onset was associated with a remote head injury (without loss of consciousness). The headache was nearly continuous. Self-administration of nimesulide (2 tablets three times daily) provided moderate relief. There were no clear triggers or factors affecting the frequency or intensity. The patient denied nausea, vomiting, photophobia, phonophobia, and aura. He had not been under neurological supervision prior to this hospitalization.

Comorbidities: hypertension; chronic sinusitis with frequent exacerbations.

Medications: nimesulide 2 tablets three times daily.

Family history of headache: unremarkable.

Neurological examination: within normal limits.

Upon admission, based on the duration and nature of the headache, chronic tension-type headache complicated by medication-overuse headache due to prolonged use of simple analgesics (nimesulide) was suspected.

Laboratory findings: Routine blood and urine tests, coagulation profile, parathyroid hormone, serum protein electrophoresis, and tumor markers were within normal limits. Biochemical testing revealed elevated alkaline phosphatase of 183.0 U/L (reference range: 40–150 U/L). Given this abnormality, further evaluation was undertaken.

Ultrasound of the abdominal organs and prostate showed no pathology.

Brain MRI (FLAIR sequence) demonstrated foci of pathological bone remodeling in both parietal bones, accompanied

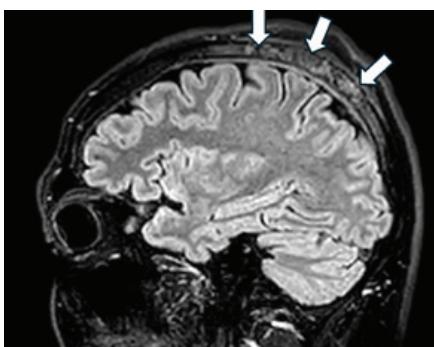


Fig. 1. Brain MRI (FLAIR sequence), sagittal slice – foci of bone remodeling in the parietal bones

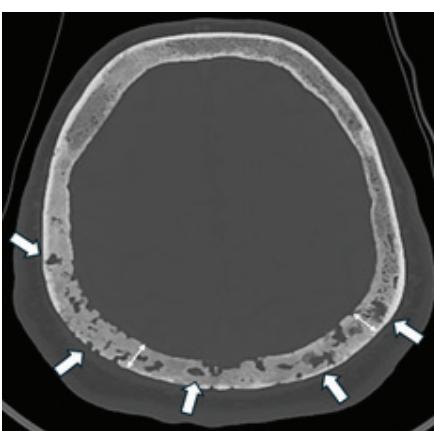


Fig. 2. Brain CT, axial slice – bone remodeling in both parietal bones.

by reactive changes in the adjacent dura mater and soft tissues (Fig. 1). Brain CT confirmed areas of pathological bone remodeling in the same regions (Fig. 2).

Whole-body bone scintigraphy performed three hours after radiopharma-

ceutical administration revealed physiological tracer uptake in the facial skeleton, sternum, scapular angles, spine, iliac wings, and shoulder and hip joints. A focus of increased radiotracer accumulation was identified in the parietal bones, more pronounced on the right (Fig. 3).

Sternal bone marrow aspiration, performed to exclude multiple myeloma, revealed no abnormalities.

A multidisciplinary consultation involving a neurologist and a rheumatologist concluded with a diagnosis of Paget's disease of bone with isolated skull involvement. Treatment with zoledronic acid 5 mg/100 mL intravenously once yearly was prescribed. The patient was referred for long-term rheumatologic follow-up.

Discussion. Paget's disease of bone is the second most common metabolic bone disorder, with a prevalence ranging from 1.5% to 8.8% [10, 17]. Neurological symptoms are reported in approximately 76% of patients [6]. Headache is a rare manifestation, described in only about 1.6% of cases; seizures are also uncommon [4, 7]. Radiographic methods, including CT, MRI, and bone scintigraphy, play a crucial role in diagnosis. Differential diagnosis may be required to distinguish PDB from metastatic lesions, lymphoma, or multiple myeloma [12].

The present case illustrates a rare but clinically important manifestation of PDB—chronic headache as the only symptom. Given the high prevalence of tension-type headaches, such cases can easily be misinterpreted as primary headache disorders. In this case, the elevated alkaline phosphatase level prompted further investigation, leading to the correct diagnosis through MRI, CT, and bone scintigraphy, and subsequent initiation of pathogenetic therapy.

Conclusion. Headache remains one of the most common reasons for medical consultation. Although the vast majority of cases (up to 90–95%) are due to primary headache disorders such as tension-type headache or migraine, clinicians must maintain vigilance for secondary causes. Secondary headaches may indicate serious underlying pathology and typically require specific diagnostic and therapeutic approaches. The presented case emphasizes the importance of comprehensive evaluation and consideration of Paget's disease of bone as a potential cause of chronic headache, particularly in middle-aged and older patients with elevated alkaline phosphatase levels.

The authors declare no conflict of interest.

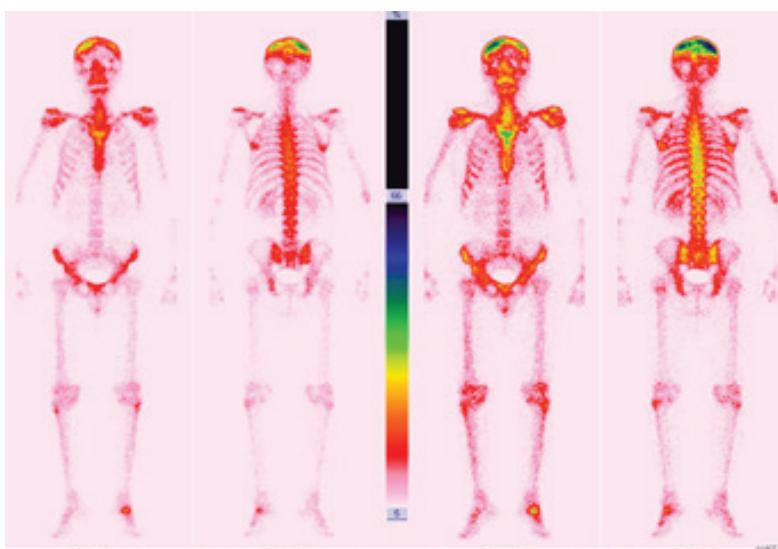


Fig. 3. Bone scintigraphy showing hyperfixation in the right parietal bone

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A CASE REPORT OF FREDERICK'S SYNDROME: COMPLETE TRIFASCICULAR BLOCK WITH ATRIAL FIBRILLATION

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This article presents a clinical case of a patient with newly diagnosed Frederick's syndrome, in this case persistent atrial fibrillation and complete trifascicular block, including proximal complete AV block, anterior hemiblock, and complete right bundle branch block. The possible mechanism of development of this condition and the treatment provided at Regional Clinical Hospital No. 1 in Tyumen, Russian Federation, are discussed.

Keywords: Frederick's syndrome; electrocardiogram; atrial fibrillation; trifascicular block; complete heart block; anterior hemiblock; complete right bundle branch block

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Introduction. As is often the case in medicine, Frederick's syndrome (FS) is an eponym. In 1904, the Belgian physiologist L.L. Frederick, during an experiment, established that in animals with atrial fibrillation (AF), the intersection of the His bundle causes regular contractions of the ventricles, despite the persistent arrhythmia in the atria [7]. In humans,

FS is characterized by a combination of complete atrioventricular (AV) block and AF or atrial flutter, which leads to a complete cessation of impulse conduction from the atria to the ventricles. Under these conditions, the ventricles are excited by the pacemaker from the AV node or ventricular conduction system, while chaotic contractions of individual muscle fibers occur in the atria. On an electrocardiogram (ECG), this manifests itself as both an f-wave and regular ventricular contractions. FS occurs in 0.6–1.5% of patients with AF [1,4]. Data on the epidemiology of FS are outdated and require updating. FS is mentioned in isolated English-language publications, which suggests the use of this eponym by physicians in the post-Soviet space rather than the extreme rarity of this pathology. The clinical presentation of FS may include episodes of loss of consciousness

(Morgagni–Adams–Stokes attacks), dizziness and weakness, as well as bradycardia.

Objective: to describe a clinical case of a patient with FS, with a trifascicular block against the background of AF.

Materials and Methods: A retrospec-tive analysis of the medical records of an inpatient in the arrhythmology depart-ment of the Tyumen Regional Clinical Hospital No. 1 was conducted. Data from clinical observation, laboratory tests, in-strumental diagnostics, and the surgical protocol were used.

Case Report. A 74-year-old man pre-sented to our emergency department with complaints of severe general weakness, dizziness, hypotension for 3 months, and syncope for 1 year. The last brief, unat-tended loss of consciousness, without bladder or bowel movement, occurred 2 months ago. There was no previous histo-

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