

## Case report

## Plasma cell myeloma as one of the causes of bone pain in family medicine

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**ABSTRACT**

The article draws attention to the need to maintain oncological vigilance in the daily practice of a family doctor. Diagnosis and effective treatment of hypercalcemia and plasma cell myeloma are presented.

**Key words:** myeloma, hypercalcaemia, treatment

## CASE REPORT

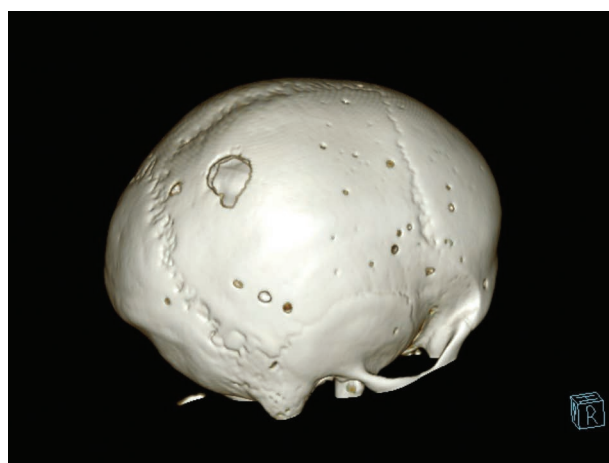
A 51-year-old patient with lumbosacral pain presented to her primary care physician. Pain management was carried out for 5 months. Due to the severity of back pain, the occurrence of headaches, she went to a neurosurgical clinic. A head CT scan was performed, which revealed numerous osteolytic foci. Pending the result of the examination, she was admitted to the neurology department due to the reduced muscle strength in the right lower limb. Magnetic resonance imaging of the spine revealed numerous osteolytic foci. The patient was admitted to the hematology department for further diagnosis. Laboratory blood serum tests showed (reference ranges are given in the brackets): normocytic anemia with the hemoglobin concentration of 11.1 g/dL (12–16 g/dL), creatinine 3.48 mg/dL (0.5–0.9), total calcium 18.2 mg/dL (8.5–10.5), ionized calcium 9.6 mg/dL (4–5.2), phosphorus 5.66 mg/dL (2.7–4.5), alkaline phosphatase 487 U/L (35–104). GFR estimate by MDRD was 13.9 mL/min/1.73 m<sup>2</sup>. The serum proteinogram showed: albumins 46.9% (52.0–65.1),  $\alpha_1$  4.8% (1.0–3.0),  $\alpha_2$  17.7% (9.5–14.4),  $\beta_1$  9.6% (6.0–9.8),  $\beta_2$  10.2% (2.6–5.8),  $\gamma$  10.8% (10.7–20.3), albumins 2.8 g/dL (3.1–5.2),  $\alpha$  10.3 g/dL (0.1–0.2),  $\alpha_2$  1.05 g/dL (0.6–1.2),  $\beta_1$  0.6 g/dL (0.4–0.8),  $\beta_2$  0.6 g/dL (0.2–0.5),  $\gamma$  0.6 g/dL (0.6–1.6). Serum and urine free  $\kappa$  light chains values were 7336.31 mg/L (3.3–19.4) and 12992.91 mg/mL (< 32.9), respectively. Serum and urine free  $\lambda$  light chains were 14.34 mg/L (5.71–26.3) and 13.97 mg/L (< 3.79), respectively. Bone marrow trephine biopsy revealed an interstitial infiltration of monotypic plasma cells: CD 138, CD 38, MUM1+, CD 117, CD79a+/-, CD56+/-,  $\kappa$ + constituting about 50% of the biopsy specimen. Morphologically, some of the tumor plasma cells were anaplastic. Bone marrow cytometry showed that 2.5% of nucleated cells were pathological plasma cells of the following phenotypes: CD38, CD138, CD56+/-, CD44+, CD27+/-, CD200+/-, CD45+/-, CD117+/-+. During hospitalization, computed tomography of the head, neck, chest, abdomen, and pelvis was performed, revealing numerous osteolytic foci, especially in the bones of the skull (fig. 1, 2) and the sternum, spine and ribs (fig. 3). Based on the clinical picture, additional investigations helped to diagnose  $\kappa$  light chain plasma cell myeloma.

Due to the hypercalcemic crisis, the patient was treated with intensive intravenous hydration from the moment of admission to the hospital. In addition, increased calcium excretion was stimulated by the supply of furosemide. Steroid therapy was implemented. However, despite renal failure, antiresorptive pamidronate disodium was administered to outweigh the risks. We observed the normalization of serum calcium and an improvement in renal function. A metastatic focus of the L5 vertebral body with a pathological fracture and the compression of the meningeal sac was the reason for single analgesic palliative radi-

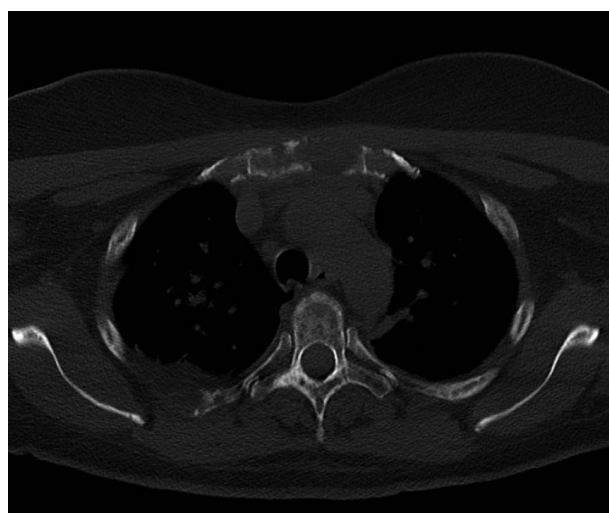
**Figure 1.** Computed tomography scan of the head in the axial plane in the bone window. Multiple osteolytic lesions of the skull bones.



**Figure 2.** Three-dimensional reconstruction of computed tomography in the volume rendering (VR) technique. Osteolytic lesions of the skull bones.



**Figure 3.** Computed tomography scan at the level of the aortic arch in the bone window. Uncountable osteolytic foci in the sternum, spine and ribs.



otherapy. Bortezomib, cyclophosphamide and dexamethasone were used to treat myeloma. Subsequently, an autologous bone marrow transplant was performed. A remission was obtained.

## DISUSSION

The main causes of hypercalcemia include primary hyperparathyroidism and hypercalcemia in the course of oncological diseases [1].

The mechanisms leading to hypercalcemia associated with malignant tumors are divided into three categories: humoral hypercalcemia caused by secreted factors (such as PTHrP, i.e., parathy-

roid hormone-related protein, or inflammatory cytokines), local osteolysis due to tumor invasion or humoral factors (only in case of impaired kidney function) and absorptive hypercalcemia due to excess vitamin D produced by cancer cells [2, 3].

According to various studies, hypercalcemia occurs in about 10–30% of patients with malignant tumors and may be a sign of a very poor prognosis and progression of malignancy. However, it should be remembered that the degree of skeletal involvement does not correlate with the degree of hypercalcemia. Knowing the diagnosis of the non-specific symptoms of hypercalcemia and rapid implementation of therapy can save lives, giving you the opportunity to seek its cause [4].

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### Authors' contributions:

All authors have equal contribution to the paper.

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