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Case report

Plasma Cell Dyscrasia Presented with Hypercalcemia, Intracranial Mass and Lytic Bone Lesions in a Geriatric Patient

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Abstract

The presence of an intracranial mass, hypercalcemia, lytic bone lesions, and height loss suggesting a pathological fracture in the vertebra led to the diagnosis of plasma cell dyscrasia at the diagnosis stage of our case. The purpose of this case report is to raise awareness about intracranial plasmacytomas and multiple myeloma with intracranial growth.

Keywords: intracranial plasmacytoma, intracranial involvement in plasmacytomas, intracranial mass, Intracranial plasma cell tumors

Introduction

Intracranial involvement in plasmacytomas and multiple myeloma is rarely reported in the literature [1]. While the presence of an intracranial mass at the diagnosis stage of our case caused diagnostic difficulties at the beginning, the presence of hypercalcemia, lytic bone lesions and loss of height in the vertebra led us to consider the diagnosis of plasma cell dyscrasia. Diffuse lytic bone lesions, intracranial mass and hypercalcemia in our case were thought to be due to multiple plasmocytic infiltration of the skeleton.

Case report

When the 72-year-old female patient was admitted to the emergency department with the complaints of weakness, intermittent consciousness, general condition disorder, serum calcium value was 12.7 mg/dl, and cranial computerized tomography examination revealed intracranial mass and lytic bone lesions. The patient was hospitalized in the outer center intensive care unit to investigate the etiology of malignancy and a PET-CT examination was performed. Laboratory examinations of the patient revealed glucose 174 mg/dL, urea 44 mg/dL, creatinine 0.8 mg/dL, uric acid 9.8 mg/dL, calcium 12.7 mg/dL, albumin 3.5 g/dL. The patient was referred to our internal medicine clinic to investigate the etiology of hypercalcemia and disseminated lytic bone lesions. The patient had diagnoses of hypertension and diabetes mellitus. In the physical examination of the patient, her neurological examination was normal; the patient was conscious, orientated and cooperative. Respiratory sounds were normal. On cardiac examination S1, S2 and rhythm were normal, there was no additional sound, there was no murmur. Abdominal examination was normal, hepatosplenomegaly was not detected. In laboratory examinations; hemoglobin 11.3 g/dL, hematocrit 31.4%, MCV 92 Fl, platelet 423.000, leukocyte 14.900, neutrophil 7800, albumin 4g/dL, globulin 2.7 g/dL, glucose 169 mg/dL, urea 43 mg/dL, creatinine 0.9 mg/dL, AST 14 U/L, ALT 18 U/L, LDH 184 U/L, total bilurubin 0.48 mg/dL, direct bilurubin 0.1 mg/dL, Na 141 mmol/L, K 3.7 mmol/L, Cl 101 mmol/L, calcium 12.3 mg/dL, phosphorus 3.6 mg/dL, Mg 1.2 mg/dL, CRP 2.8 mg/dL were detected. Parathormone (PTH) was 13.6 pg/L and 25 OH D vitamin level was 42.74 pg/L. Hyperparathyroidism was excluded in the patient with low PTH levels. Intracranial mass and lytic bone lesions were detected in the outer center, diffuse lytic lesions were observed in direct cranial radiographs. Hydration and diuretic treatment were started for the patient to treat hypercalcemia. The missing electrolytes were replaced. The patient underwent a malignancy scan, a contrast computed tomography examination was performed. The computerized tomography examination revealed a 44x33 mm mass lesion with a heterogeneous enhancement showing growth towards the petrous apex in the left lateral of the clivus, hypodense lesions in the clavicle and vertebrae, right occipital and right temporal bone squamous mucosa. Heterogeneity and multiple hypodense appearances were detected in thoracic bone structures. It was thought that the height losses observed in the T8, T11, L1 and L4 vertebrae were secondary to the pathological fracture. Intense hypodensities were observed in the pelvic bones, the largest in the pelvic region, which are considered to be the left wing. It was learned from the patient's history that he had a total abdominal hysterectomy and bilateral

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salpingo-oophorectomy operation. Breast examination of the patient was considered normal. Breast ultrasonography and mammography were requested for screening, BIRADS 2 benign findings were detected. Serum immunoglobulin levels were low, albumin/globulin reversal was not detected. (IgA: 0.26 g/L, IgM: 0.17 g/L, IgG: 4.94 g/L, total IgE: 10.8 IU/ml). Multiple diffuse bone lytic lesions and a slightly increased 18 FDG uptake in the right lung hilar millimetric lymph node were reported in the patient's PET-CT scan at the outer center (SUV MAX: 4.1). In the thorax computed tomography examination of the patient, active parenchymal pathology was not observed, while a millimetric lymphadenopathy-compatible appearance was observed in the right hilar region. In order to investigate hypercalcemia etiology, the patient was consulted with chest diseases clinic, sarcoidosis was not considered. Osteoporosis was not detected in bone densitometry. Bone marrow showed 75% cellularity in aspiration. Bone marrow biopsy was hyper cellular and shows 50 percent involvement by abnormal appearing CD38 positive plasma cells (Figure 1-3).

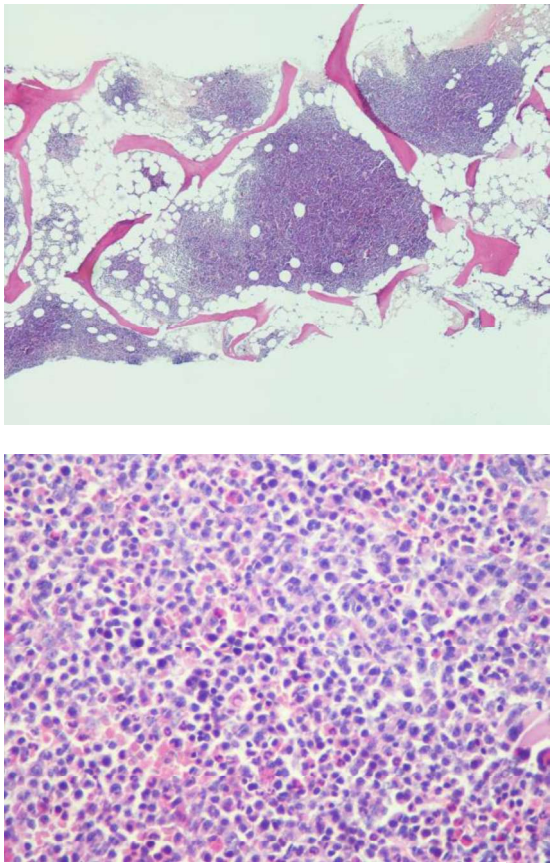

Fig. 1, 2. Sheets of plasma cells, hematoxylin and eosin (40x and 400x)

Almost all plasma cells are kappa positive (Figure 4). With Lambda, there are positivity in a few cells (Figure 5). Based on the morphological and immunohistochemical findings bone marrow biopsy reported as plasma cell neoplasia and recommended to evaluate the case in terms of myeloma with its clinical, laboratory and radiological findings. As a result, pathological

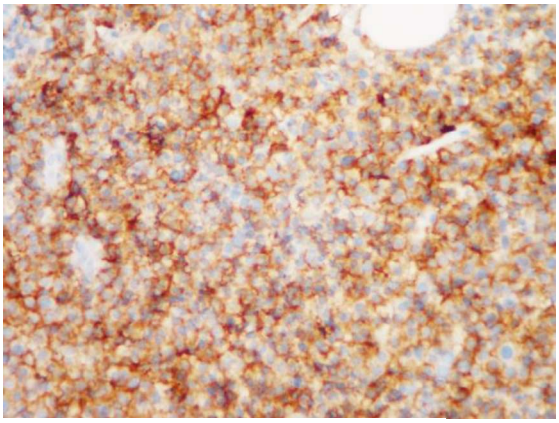

Fig. 3. CD38 immunohistochemical stain (400x)

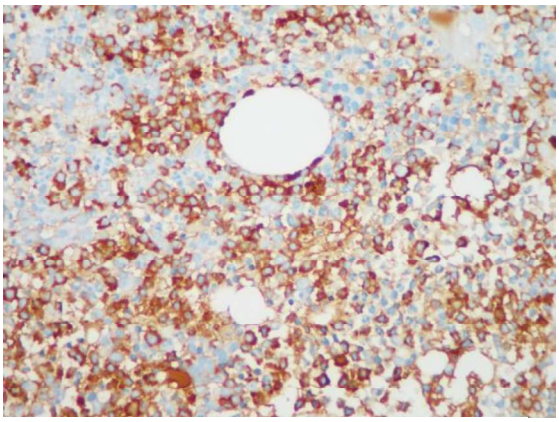

Fig. 4. Kappa immunohistochemical stain (400x)

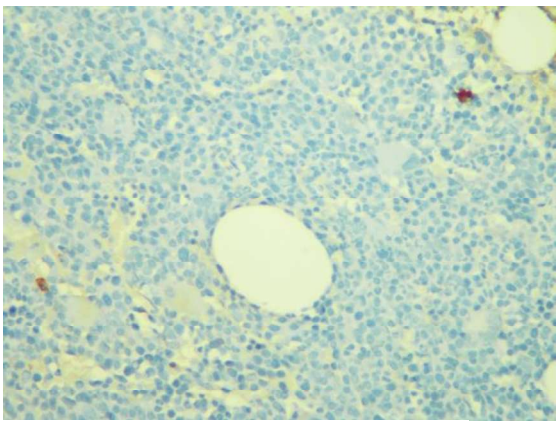

Fig. 5. Lambda immunohistochemical stain (400x)

diagnosis was reported as plasma cell neoplasia. The patient was transferred to the Hematology Clinic with the diagnosis of multiple myeloma and treatment was started with the VCD protocol.

Discussion

Plasma cell neoplasia, metastatic tumors, sarcoidosis and parathyroid diseases were considered among the possible differential diagnoses due to the clinical picture of the patient and presenting with hypercalcemia. The presence of an intracranial mass at the diagnosis stage of our case caused diagnostic difficulties at the beginning, while the presence of hypercalcemia, lytic bone lesions, loss of height suggesting pathological fracture in the vertebrae led to the diagnosis of plasma cell dyscrasia. The patient described here is rare because it was associated with diffuse lytic bone lesions, intracranial mass and hypercalcemia, all thought to be due to multiple plasmocytic infiltration of the skeleton. In our case, myelomatous disease was clinically apparent on initial presentation. For systemic evaluation, in addition to PET-CT imaging, contrasted computed tomography images and bone marrow aspiration and biopsy were performed in our patient.

Intracranial plasma cell tumors have been reported to be extremely rare and may be alone or part of a systemic multiple myeloma [1]. A plasmacytoma involving the pituitary gland has been reported as an extremely rare condition with approximately 22 cases [2]. It is thought that the occurrence of plasma cell neoplasm as a cranial or intracranial mass is a rare condition [3]. It is thought that it is even rarer that myelomatous first appears as a sellar mass and mimics a pituitary adenoma [3]. While there are few reports of plasma cell tumors involving clivus [4-7], cases of plasma cell dyscrasia occurring as a mass in the posterior fossa have been reported less frequently in the literature [8, 9,10]. Tumor cells are believed to originate from the surrounding bone in the sellar region or the mucosa within the petrous or sphenoid bone [11]. In other words, intracranial involvement is usually thought to result from direct spread from adjacent bone lesions in the head dome, skull base, nose, or paranasal sinuses [12].

Intracranial plasmacytoma can be differentiated from the usual meningioma cases, since the skull will often have lytic lesions [10]. Although it is generally known that intracranial plasmacytomas are very sensitive to radiation; definitive treatment for intracranial plasmacytoma complete surgical resection plus radiation therapy have been reported.

The purpose of this case report is to increase the awareness of the intracranial plasmacytomas and multiple myeloma with intracranial growth [12].

In patients presenting with headache, diplopia, intermittent loss of consciousness and accompanying signs and symptoms suggestive of multiple myeloma, plasma cell dyscrasias should also be considered in the differential diagnosis [5]. From a broader perspective, intracranial plasmacytoma and multiple myeloma with intracranial enlargement may be appropriate to be among the neurological aspects of multiple myeloma [13].

In rare cases, intracranial plasmacytoma should be considered in the differential diagnosis in combination with hypercalcemia, diffuse lytic bone lesions and intracranial mass lesion. This report, although rare, reminds us of the value of including a plasma cell tumor in the differential diagnosis of intracranial masses with bone involvement. This case illustrates a rare and interesting presentation of multiple myeloma and highlights that MM can come to the clinic with different faces, as in this case.

Conflict of interest statement. None declared.

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