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DIAGNOSIS OF MULITPLE MYELOMA: EXPERIENCE OF AN ATYPICAL CASE IN A YOUNG PATIENT

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ABSTRACT

Summary: Since the description of the first case in 1965 by Rowe and Fahey, IgD multiple myeloma (MM) is considered a rare entity within monoclonal gammopathies and represents less than 2% of all MM cases. It is characterized by an aggressive course and a worse prognosis compared to IgA and IgG MMs with a median survival of between one and two years. A reflection of this aggressive course is the presence of complications of the disease at the time of diagnosis such as extramedullary plasmacytomas, myeloma kidney, and amyloidosis. All these findings make MM IgD a unique and complex entity.[1]

We present the case of a young patient, with a history of hyperthyroidism, did not present hypercalcemia or renal failure, the classic manifestation of MM, however, she had plasma cells in the bone marrow, without evidence of extramedullary plasmacytomas, she was given the diagnosis of myeloma multiple by positive immunohistochemistry.

Objective: Learn about Multiple Myeloma through the presentation of a case, in order that readers are trained to identify the key points of the pathology (main epidemiological, clinical and pathological characteristics).

Methodology: This is a retrospective study of Multiple Myeloma, emphasizing its clinical, radiological and histological characteristics.

The information and images obtained belong to the medical personnel in charge of the case whose reinforcements are based on the statistical package Excel, Word and JPG.

Conclusion: Multiple myeloma is a malignant hematologic disease characterized by a proliferation of plasma cells in the bone marrow. The most common age of onset is between 65 and 70 years, however, cases in young people are documented in increasingly severe forms. Diagnosis generally requires demonstrating M protein (which is sometimes present in urine rather than serum, but is rarely totally absent) and / or light chain proteinuria and excess plasma cells in bone marrow.[2]

KEYWORDS : Multiple myeloma, immunohistochemistry, age

INTRODUCTION:

MM is a malignant neoplasm, made up of clones of plasma cells. It is distinguished by plasmacytosis in the bone marrow, production of monoclonal proteins, osteolytic-type bone lesions, kidney disease, anemia, hypercalcemia, and immunodeficiency. The evolution of multiple myeloma is a complex process that consists of various steps that involve early and late genetic changes in the tumor cell, as well as selective conditions that favor an optimal microenvironment in the bone marrow for such changes to occur. tumors induce direct and indirect signaling sequelae in the bone marrow, which[3].

Specifically, multiple myeloma cells alter stromal cell homeostasis and the interaction between stromal cells, the extracellular matrix, and liquid factors (cytokines and growth factors); as a consequence, tumor cells induce direct and indirect signaling sequelae in the bone marrow, which, in turn, promote proliferation, survival, migration, and drug resistance of multiple myeloma cells.[4]

It is classically described as a neoplasm that predominates in the age of 70 years and is reported in 2% in those under 40 years of age; it is even rarer in people under 30 years of age, with an incidence of less than 1%. Regarding its clinical manifestations: bone pain is the most typical and frequent symptom. It appears in 60-80% of patients at the time of diagnosis, usually in the spine, but also in the sternum, ribs, or proximal extremities. Its origin is bone lesions that, by radiology, usually correspond to typical osteolytic images in "punch". Another classic clinical manifestation of MM is anemia. Up to 50% of patients have moderate anemia (approximately 10 g / dl) and 25% of cases severe anemia (<8 g / dl).[5]

Renal failure (RI) appears in 25-30% of patients. Its origin is multifactorial, although the most commonly described cause is the renal elimination of Ig light chains (Bence-Jones proteinuria), whose presence in the tubules is histologically called "myeloma kidney." Other causes are hypercalcemia, hyperuricemia, dehydration, and recurrent urinary tract infections. Infections are the leading cause of death in MM, with an incidence of 0.8-2.2 episodes per patient per year, between 7 and 15 times higher than that observed in patients hospitalized for other reasons6. They are usually bacterial and located in the lung and urinary tract.[6]

When MM is suspected, the following tests may be helpful: Complete blood count: may reveal normochromic, normocytic anemia. High sedimentation rate. Determination of hypercalcemia and renal impairment. Serum electrophoresis: can show a monoclonal protein, which in most cases is IgG or IgA, but can be any kind of Ig. Reduced normal Ig levels are confirmatory. Excess free light chains, either kappa or lambda, can be found in serum. The diagnosis of MM depends on the demonstration of an increase in plasma cells

(> 10%) in the bone marrow. Bone radiographs may show lytic lesions.[7]

CASE PRESENTATION

A 22-year-old female patient with a personal medical history of primary hyperthyroidism under treatment of 10 mg of thiamazole and 20 mg of propranolol, presented with a clinical picture characterized by weight loss, anorexia, watery diarrhea and anemic syndrome (paleness, asthenia, adynamia) with a one-year evolution, having as an apparent cause the abandonment of antithyroid treatment. In addition, he presented repetitive symptoms of pain in the pelvic limbs without radiation and colicky abdominal pain located in the right upper quadrant; with this symptomatology his admission is decided.

Hematic biometry extension tests were requested: hemoglobin 6.6 g / L, leukocytes 3.480 / mm3, erythrocyte sedimentation rate (ESR) in 113 mm / h, DHL 179 IU / L, total bilirubin 2.8 mg / L, globulin of 6.5 g / dL, Coomb's direct negative, ANA's 1:51 positive.

During hospitalization, the patient had clinical deterioration, with worsening of the anemic syndrome, non-painful splenomegaly and hepatomegaly, globulin levels 9.4 g / dL, albumin 2 g / L, this figure was permanently high, without hypercalcemia, it was also requested Protein electrophoresis that reported a monoclonal IgG peak (Photo 1) with B2 microglobulin values of 8.13 mg / dl.

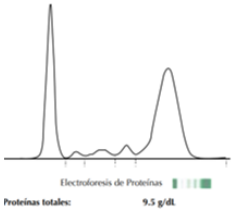

Photo 1: Protein electrophoresis, monoclonal peak

Due to hyperviscosity syndrome, three sessions of plasmapheresis were performed, biopsy of periumbilical fat with negative Congo red stain, without lytic lesions in metastatic bone series, in the BODYTAC there was loculated right pleural effusion, hepatomegaly and splenomegaly, without kidney disease (Photo two).

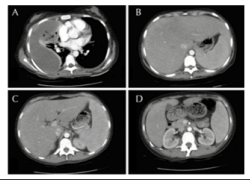

Photo 2: Contrast tomography of the chest and abdomen showing: loculated empyema in the right hemithorax (A), hepatomegaly and splenomegaly (B), without enhancement with contrast medium (C) or kidney disease (D).

Given the suspicion of monoclonal gammopathies, it was decided to take a bone marrow aspirate, obtaining 10% plasma cells, bone biopsy with hypercellularity and infiltration by plasma cell neoplasia with restriction of positive kappas light chains, negative lambdas (Photo 3) with These findings established the diagnosis of symptomatic multiple myeloma, stage III (ISS).

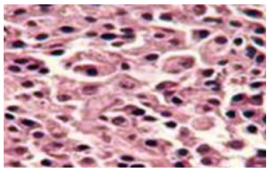

Photo 3: Biospy of bone marrow: infiltration of plasma cells

DISCUSSION

Multiple myeloma is the second hematologic neoplasm in order of frequency. It is defined by the presence of monoclonal plasma cells with the capacity to produce a monoclonal paraprotein and cause clinical alterations in the form of anemia, kidney failure, hypercalcemia, or bone lesions. [8]

It is described with a peak incidence in the seventh decade of life, approximately 98% of cases, thus 2% affect those under 40 years of age and only 0.3% under 30 years[9]

For all that has been said, when analyzing our clinical case, a young patient with a history of primary hyperthyroidism under controlled treatment, the same one who presented with nonspecific symptoms (weight loss, diarrheal stools and anemia), and after conducting extension tests, about All positive immunohistochemistry led to the diagnosis of MM.

With which we can say that the classic clinical presentation of MM: more frequent in older adults; bone pain, kidney failure, hypercalcemia; Our case was not fulfilled, making it an atypical presentation, which is why we documented it.

CONCLUSION

The presentation of MM is generally in the sixth or seventh decade of life, but the casuistry of presentation in our case has been in the second decade of life, making this case exceptional, it should be noted that cases in young people are associated with forms each time more serious. It has been seen that renal involvement with hypercalcemia is typical, not being found in our case, in addition the presence of monoclonal proteins, anemic syndrome and bone pain is associated with MM, however, the plasmoblastic variety only designates immature plasma cells greater than 10% , the same ones that conferred the histological diagnosis.[10,11]

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