Case Report

Autoimmune hemolytic anemia and hyperglobulinemia leading to the diagnosis of multiple myeloma

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Introduction

Autoimmune hemolytic anemia (AIHA) is a condition in which self-antibodies bound to antigens on the membranes of red blood cells initiate their destruction (hemolysis) via the complement and reticuloendothelial systems. Multiple myeloma (MM), on the other hand, is characterized by a clonal expansion of plasma cells in bone marrow, causing bone tissue destruction, renal failure and hematopoietic suppression. Although the association of MM and anemia is common, AIHA as the anemic manifestation of MM is rare. 1

Case report

Herein we report on the case of a 69-year-old, black patient under treatment with prednisone and methotrexate for rheumatoid arthritis over the ten years leading up to this report. Six years ago, the patient was investigated by the hematology department due to an unstable leukopenia (2.86 × 10^3/μL), without the involvement of any other series. Since the beginning of treatment for rheumatoid arthritis, the leukocyte count of the patient was controlled, with variations being attributed to the rheumatoid arthritis itself.

However, other alterations were found during a routine appointment in the Rheumatology Department including anemia (hemoglobin: 7.2 g/dL), low hematocrit (22.6%), high ferritin levels (732 mg/dL), positive direct coombs test, and elevated total and indirect bilirubin. By protein electrophoresis, the total protein was elevated (11.8 g/dL), the albumin/globulin ratio was low (0.50), the albumin was low (3.94 g/dL) and the gamma globulin was high (6.25 g/dL) with a monoclonal component. The patient was referred back to the hematology department and diagnosed with hypergammaglobulinemia accessory to MM. After a clinical and laboratory evaluation, the diagnosis of MM was confirmed by electrophoresis and bone marrow biopsy. A bone marrow biopsy typically shows a clonal population of plasma cells, which can be detected by immunohistochemistry and flow cytometry. The diagnosis of MM is made when at least five percent of the bone marrow is occupied by plasma cells. The patient was treated with bortezomib and dexamethasone, with improvement in the anemia, hematocrit and total bilirubin levels. Since then, the patient has been under treatment with a stable response.
Moreover, despite with disease, Waldenstrom’s MM has been shown to be a monoclonal protein, of which very high levels are seen in MM, is the antibody responsible for AHAI and the pathogenesis of this progression is still unclear. Moreover, due to the lack of concrete data, the possibility of this pathogenesis, that is, AHAI as the cause of MM cannot be ruled out. The involvement of immunosuppressants, such as interferon-alpha, which are used in the treatment of MM and other lymphoproliferative disorders, may also play an important role in the development of AHAI, an autoimmune phenomenon similar to Evans-like syndrome. The number of reported cases is extremely low, which restricts the progress of research.

Conclusion

This case describes a rare condition, AIHA associated with MM. The low number of reported cases and consequently the few studies on this association hinders our understanding. However, ongoing studies should clarify both the pathogenesis and the intimacy of the relationship between the two entities in the future, thereby allowing earlier diagnosis and treatment that is more effective. The medical community should consider the diagnosis of AHAI in the presence of MM as well as vice versa, in order to make earlier diagnoses and treatments that are more effective. In addition, new cases need to be reported to support research related to these two pathologies, so that through a larger analysis, science can reach a coherent and effective conclusion.

Conflicts of interest

The authors declare no conflicts of interest.

References

