Clinics in Oncology

Case Report
Published: 02 Jul, 2016

A Case Report of Non-Secretorymyeloma with Renal Failure

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Abstract

Non-secretory myeloma (NSM) is a rare form of multiple myeloma with the feature of absence of monoclonal gammopathy in serum and urine by immunofixation electrophoresis. It accounts for 1-5% of all cases of multiple myeloma, and is supposed to be associated with longer survival. But NSM usually presented with more advanced disease at initial stage than classic myeloma. Here we present a case of NSM complicated with progressive renal failure and anemia.

Keywords: Non-secretory myeloma; Renal failure; Anemia; Osteolytic lesion

Introduction

Multiple myeloma, a cancer of plasma cells, accounts for about 1% of all tumors and 13% of hematological malignancy cases [1]. Of them, secretory type accounts for approximately 95-97% of all cases, where non-secretory type accounts for the other 1-5% [2]. Non-secretory myeloma (NSM) was first described by Serre in 1958 and is characterized by the absence of M protein band on serum or urine immunofixation electrophoresis [3]. The pathogenetic mechanisms underlying the incapability of neoplastic plasma cells to synthesize M proteins have been reported to differ from case to case. Moreover, with serum free light chain (FLC) assays, about three-fourths of non-secretory cases were found to actually have elevated FLC levels and/or abnormal FLC ratios [4], and were considered as minimally secretory, hyposecretory, or oligosecretory cases [3]. NSM cases often fail to exhibit classical symptoms ofanaemia, neutropenia, thrombocytopenia, recurrent bacterial infection, and end organ damage [5], leading to the delaying of diagnosis. Here we reported a case of NSM with renal failure and anaemia.

Case Report

A 69-year-old man presented with a history of consistent chest pain lasting for at least 2 months. Before coming to visit our hospital, the patient has already received routine blood and biochemical tests as well as radiographic imaging studies one month ago in another hospital. Routine blood test indicated slightly increased white blood cells of $10.8*10^9$/L with leukocyte count of $0.9*10^9$/L, no anaemiaandthrombocytopenia were found. Serum lactate dehydrogenase (LDH) was significantly increased (1319U/L). Haematuria and proteinuria were also seen in the patient, and blood test revealed increased creatinine of 148.2μmol/L, which all indicated renal damage. Magnetic Resonance Imaging (MRI) study showed diffuse degenerative changes in cervical spine, and bone lesions in thoracic and cervical spine. Ultrasonographic findings were within normal limits and endoscopic examination indicated esophagitis and erosive gastritis.

After admitted into our hospital, routine blood test was performed again, which indicated a normal white blood cell count of $8.91*10^9$/L, a normal leukocyte count of $1.60*10^9$/L, and normocytic nomochromic anaemia (9.70g/dl) which fell to 8.7g/dl four days later. His erythrocyte sedimentation rate (ESR) was 21mm/h. Serum albumin (3.60g/dl) and total globulin (2.11g/dl) were within the normal range, butthe levels of IgA, IgG, and IgM were significantly decreased. Additionally, FLC assays showed that Kappa and Lambda were both in normal limits with their ratio significantly decreased to 0.68. However, serum protein electrophoresis didn't show any monoclonal protein band and gamma globulin levels were normal. Urine bence-Jones protein was undetectable in this patient. Bone SPECT-CT scan showed multiple increased intake, which suggested bone metastasis or multiple myeloma. Yet no hypercalcemia was observed by biochemical blood test. Bone marrow aspiration was performed from the posterior iliac spines of the patient, and the aspirates showed

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Received Date: *08 Jun 2016*
Accepted Date: *28 Jun 2016*
Published Date: *02 Jul 2016*

Citation:
Zhu Y, Yan F, He B, Shen J, Xiao Z. A Case Report of Non-Secretorymyeloma with Renal Failure. Clin Oncol. 2016; 1: 1056.

numerous plasma cells of 65%. Binucleated, multinucleated forms and occasional mitotic figures were also observed in these plasma cells. The immunophenotyping results for bone marrow aspirates revealed cells positive for CD38, CD138, Lambda, and HLA-DR. Cytogenetics were performed with FISH showing the 1q21 amplification (38.7%), RB1 deletion (35.0%), D13S319 deletion (39.0%), p53 normal, and virtual karyotype showing 2 deletion, 13 deletion, and 5q amplification.

The patient had already developed intorenal failure when admitted into our hospital with serum creatinine increased to a high level of 692.0μmol/L and serum blood urea nitrogen at a high level of 25.70mmol/L, and haematuria and proteinuria persistently existed. Acute dialysis was then arranged. The patient was diagnosed as non-secretorymyeloma (NSM)in clinical stage IIIB according to Durie/Salmon staging system and stage III according to the International Staging System (ISS), and had a significantly high level of β2-microglobin at 39.29mg/L (range 1.0-3.0mg/L), which indicated a poor prognosis. However, the patient refused to accept further chemotherapeutic regimen in our hospital.

Discussion

NSM was reported to have clinical features of elevated hemoglobin levels, lower ESR values, higher neurological presentation incidence, less aggressive osteolysis, low risk of renal myeloma, lower hypercalcemia incidence, and elevated immunoglobulin levels [3]. Our patient here, however, presented anaemia, normal ESR values, severe osteolytic lesions, renal failure, normal blood calcium, and decreased IgA, IgG, IgM levels, whose complaint of bone pain was extremely obvious. Therefore, the paradoxically clinical presentation of the NSM case prompted us to discuss it here.

Anaemia is commonly presented in the majority of myeloma patients, but anaemia in NSM cases was reported to be less frequent than that in secretory myeloma cases. A case of megaloblastic anaemia (MA) in NSM was described in 2013, which suggested the case of MA was due to the increased cobalamin uptake and consumption and paraprotein synthesis [6]. However, treatment with folic acid and cobalamin significantly accelerated disease progression, and some postulations were raised as cobalamin acted as a paracrine growth factor to increase plasma cell growth and osteolytic activity [6]. Our NSM case here presented normocytic normochromic anaemia with hemaglobulin continuously decreasing. The bone marrow aspiration confirmed the erythroid hypoplasia. And that, we thought, was due to the occupation of neoplastic plasma cells into bone marrow and caused the marrow hematopoietic cell replacement.

In multiple myeloma patients, renal damage commonly occurs due to increased serum free light chain and/or hypercalcemia [7]. Our patient here presented a progressed renal damage to renal failure, but no increased serum light chain and hypercalcemia were detected. Someone reported a rare case of NSM associated with acute renal failure due to massive renal infiltration by neoplastic plasma cells, and increased renal size was observed in the reported case [8]. However, ultrosonographic examination for this patient did not show swollen kidney. And no ongoing renal infection and hypotensive episodes were presented in this case. Before admitting to our hospital, the patient had a short history of using unknowing herbs, which may have a risk leading to renal damage. Renal biopsy was not performed in the patient, so we here can't provide solid evidences to figure out the association between NSM and renal failure and the actual cause of renal failure in the patient.

In conclusion, paraprotein absence does not exclude the diagnosis of multiple myeloma, especially in the case of NSM. Histologic, serologic, and radiographic features should be carefully evaluated.

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