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A man with generalized weakness

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1. Case presentation

A 63-year-old man, without any significant previous medical or surgical history, presented to the Internal Medicine service with a1-month history of generalized weakness, back pain, and repeated episodes of epistaxis. He was a farmer. In physical examination, vital signs were stable and the patient was mildly drowsy. Table 1 shows complete blood count (CBC) analysis and biochemical workup. Due the patient's anemia, a peripheral blood smear (PBS) was made (Fig. 1; Panel A).

What is the diagnosis?

Table 1
Complete blood count and biochemical workup.

Hematology		
Hemoglobin (g/dl)	9.1	13.5–18.0
Mean corpuscular volume (fl)	78.6	81.0–98.0
Red blood cells (cells/Ul)	$3.64 * 10^6$	$4.5 * 10^6$–$6.5 * 10^6$
White blood cells (cells/Ul)	7100	4800–1800
Red cell distribution width (fl)	15.6	10.8–14.9
Platelets (cells/Ul)	126,000	150,000–450,000
INR	1.37	
Biochemical		
ALT (UI/l)	8	2–41
AST (UI/l)	18	2–37
Alkaline phosphatase (UI/l)	292	64–306
Sodium (mEq/l)	138	136–145
Potassium (mEq/l)	3.6	3.8–5.6
Calcium (mg/dl)	13.5	8.4–10.2
Creatinine (mg/dl)	3.4	0.7–1.4
BUN (mg/dl)	36	6–23
Total protein (g/dl)	14.3	6.6–8.8
Albumin (g/dl)	3	3.5–5.3
Globulin (g/dl)	11.3	
Albumin/globulin	0.26	
Erythrocyte sedimentation rate	>120	

2. Diagnosis

2.1. Multiple myeloma (MM)

Para-clinical investigations revealed microcytic hypochromic anemia, an increase in the patient's serum creatinine level, a high erythrocyte sedimentation rate and hypercalcemia. In PBS, erythrocyte rouleaux formation was detected (Fig. 1; Panel A). Because of these findings, we clinically suspected to the diagnosis of multiple myeloma. A skull x-ray was done and showed multiple punched-out lytic lesions (Fig. 1; Panel B). The patient's drowsiness and epistaxis attributed to the marked increase in the concentration of serum protein (hyperviscosity syndrome in MM). Bone marrow study confirmed the diagnosis.

MM is a hematologic disorder characterized by neoplastic proliferation of plasma cells in the bone marrow and production of a monoclonal immunoglobulin, events that resulting in different manifestations of this disease [1]. Plasma cell proliferation can cause skeletal destruction, showing itself by osteolytic lesions, osteopenia, bone pain, and/or pathologic fractures [1,2]. Production of monoclonal protein (M protein) can lead to renal failure (caused by light- chains of Bence Jones proteins) or hyperviscosity (due to the high M-protein concentration in the serum) [1,2]. In cases with a clinical picture consistent with MM, the diagnosis depends on the presence of abnormal clonal bone marrow plasma cells, evidences of end organ damage, and detection of M protein in the serum or urine [1,2].

Conflict of interests

None.

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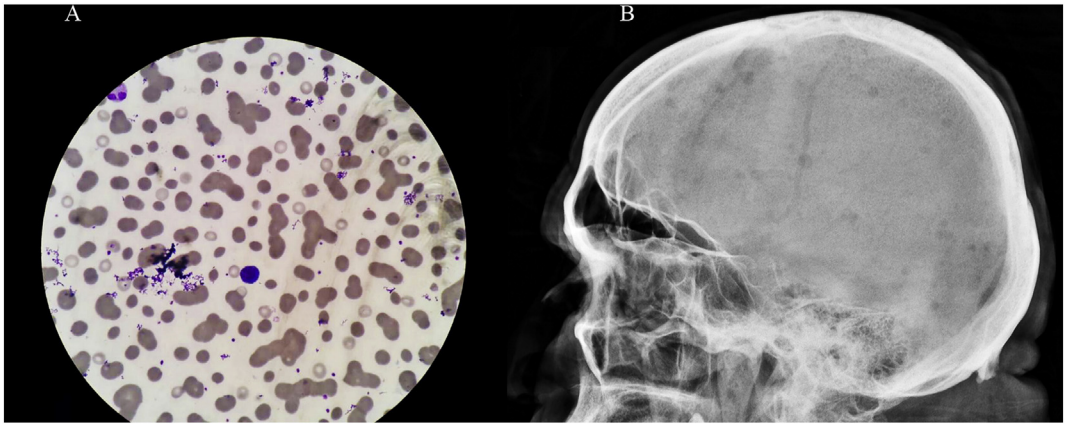

Fig. 1. Panel A: peripheral blood smear showing erythrocyte rouleaux formation; Panel B: skull x-ray (lateral view) showing multiple punched-out lytic lesions.

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