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Gastric Adenocarcinoma Mimicking Plasmacytoma During the Course of Multiple Myeloma(MM) in a Geriatric Patient

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ABSTRACT

Multiple myeloma is the neoplastic proliferation of monoclonal plasma cells, usually bone marrow originated. It may cause various organ dysfunctions. It is not so rare to detect secondary malignancies associated with MM but this co-existence between MM and gastric cancer has been not very frequently reported. Secondary malignancy risk should be kept in mind during the follow-up of patients with MM.

Key words: multiple myeloma, plasmocytoma, gastric cancer

MULTİPLE MYELOMLU YAŞLI HASTADA PLAZMASİTOMU TAKLİT EDEN MİDE ADENOKARSİNOMU

ÖZET

Multiple myeloma genellikle kemik iliği kökenli monoklonal plazma hücrelerinin neoplastik proliferasyonudur. Birçok organda fonksiyon bozukluklarına yol açabilir. Multiple myelomlu hastalarda ikincil kanserler nadir olmamakla birlikte mide kanseri ve multiple myelom birlikteliğine literatürde az rastlanılmaktadır. İkincil kanser riski multiple myelomlu hastaların izleminde akılda tutulmalıdır.

Anahtar sözcükler: multipl myelom, plazmasitom, mide kanseri

Multiple myeloma is the neoplastic proliferation of monoclonal plasma cells-usually bone marrow originated (1). It may cause varying organ dysfunctions, pain in the bones or fractures, acute renal injuries, anemia, infections, neurological disorders, hypercalcemia and hyperviscosity syndromes (2). It is not so rare to detect secondary malignancies associated with MM (3) but this co-existence between MM and gastric cancer has been not very frequently reported (4).

Case report

A 68 years-old-woman complained of fatigue, weakness and dyspnea lasting for a month. The biochemical tests showed anemia, high sedimentation rate and hyperglobulinemia (Figure 1). Her past medical history was negative for any chronic diseases and medications. Physical examination was not contributory. Laboratory tests were as follows; urea 38 mg/dl, creatinine:0,8 mg/dL, ESR:135 mm/h, sodium: 133

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Table 1. The biochemical tests at admission.

Urea	38 mg/dl	WBC	8600
Creatinine	0.8 mg/dl	RBC	3.22 M/Ul
Sodium	133 mmol/L	Hemoglobin	7.2 gr/dl
Potassium	5.4 meq/l	Hematocrit	23.9 %
Total protein	8.9 gr/dl	Platelet	365000
Albumin	3.8 gr/dl	IgE	19.5 mg/dl
Globulin	5.1 gr/dl	IgM	63.7 mg/dl
Calcium	11.9 mg/dl	IgG	3720 mg/dl
Sedimentation	135 mm/h	IgA	398 mg/dl
LDH	492 U/L		

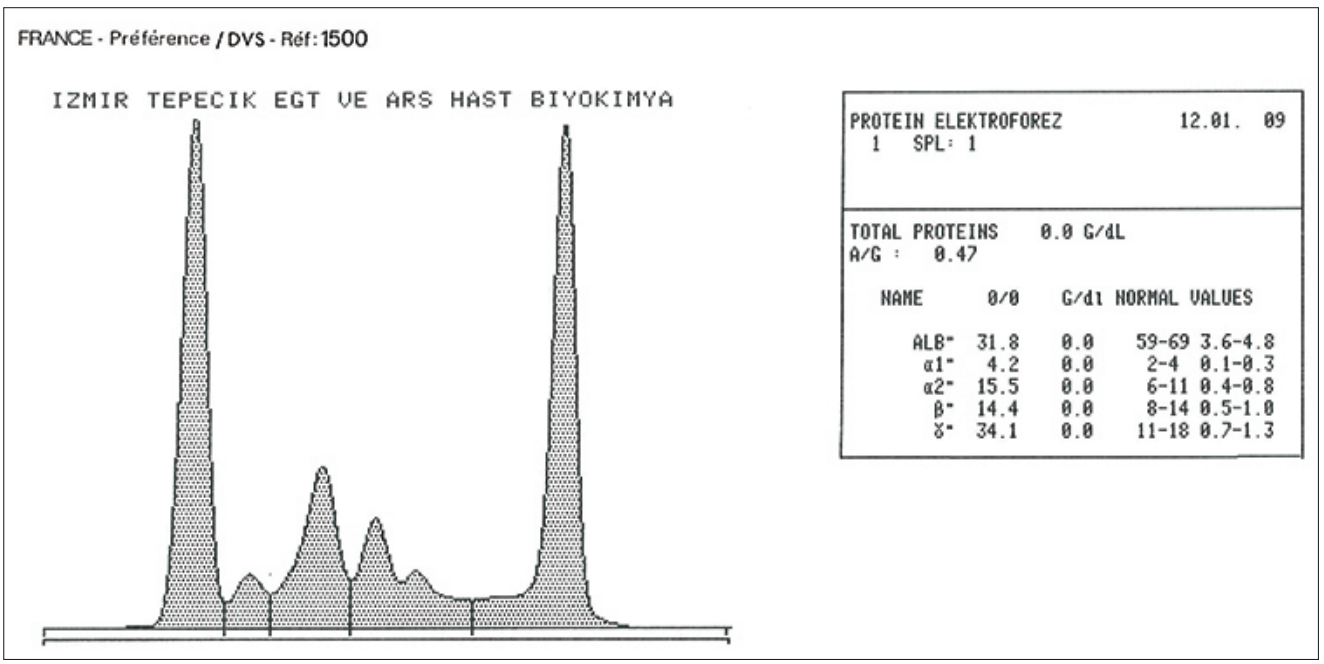


Figure 1.

mmol/L, total protein:8,9 g/dL, albumin:3,8 g/dl, globulin:5,1 g/dl, potassium:5,4 mmol/L, calcium:11,9 mg/dL, RBC: 3,22 M/Ul, hemoglobin:7,2 gr/dL, hematocrit:23,9%, IgG:3720 mg/dL, IgA:398 mg/dL, IgE:19,5 mg/dL, IgM:63,7 mg/dL, lactat dehydrogenase:492 U/L. The peripheral blood smear was remarkable for rouleaux formation, microcytic and hypochromic erythrocytes. The cranial X-ray graphics revealed lytic bone lesions in the skull. The bone marrow aspiration was performed and aspirate showed nearly 30% plasma cells (Figure 2). MM was the diagnose and vincristine, doxorubicin, dexamethasone (VAD) chemotherapy was planned as an initial start. During the third cycle as she had been complaining about dyspepsia and vomiting, abdominal ultrasound imaging was performed and an epigastric mass was determined. A mass

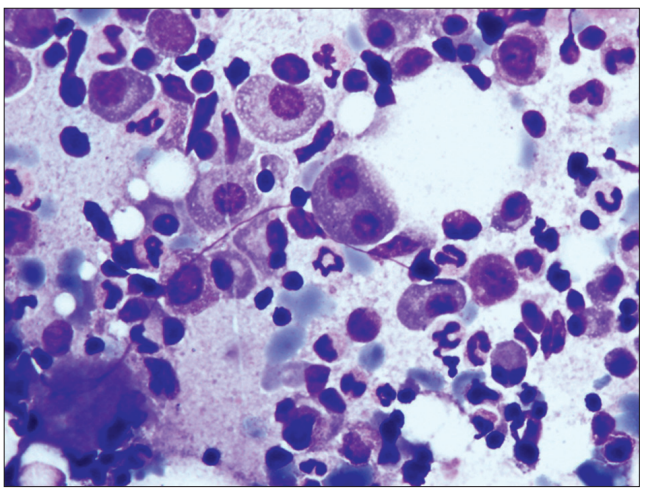


Figure 2.

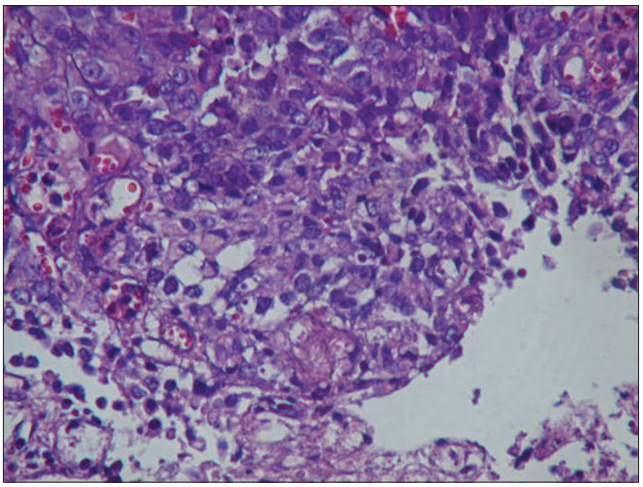


Figure 3.

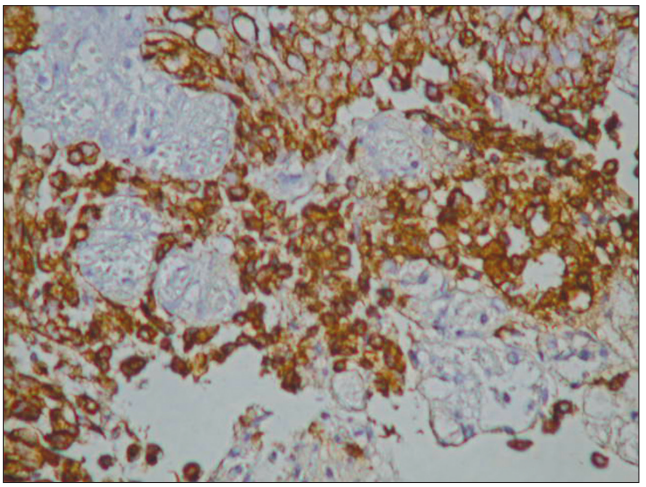


Figure 4.

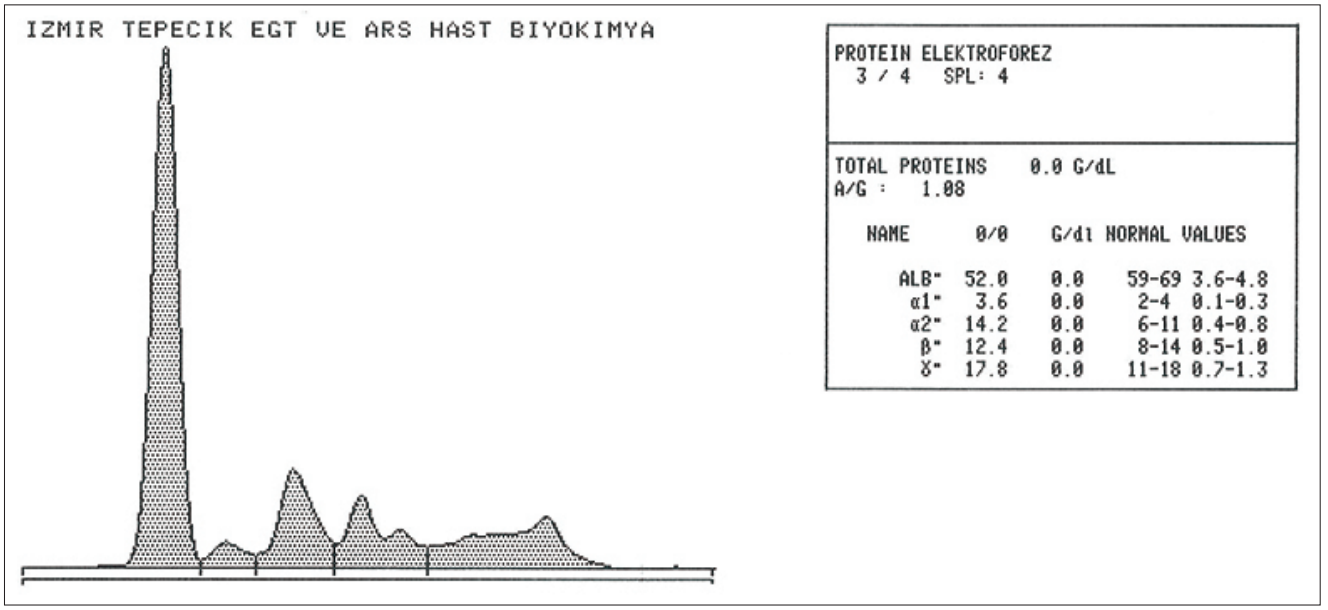


Figure 5.

of 5*6 cm on the corpus of the stomach was determined throught an abdominal CT scan. The esophagogastroduodenoscopy and biopsy were performed (Figure 3). Low differentiated adenocarcinoma was reported (Figure 4,5). Meanwhile, the chemotherapy regimen was regularly applied. After the fourth cycle was applied successfully biochemical tests revealed; sedimentation rate: 53 mm/h, Ig G:678 mg/dL, hemoglobin: 11 gr/dl, hematocrit: 31,5%, albumin: 4,3 gr/dl and globulin 2,3 gr/dl. Protein electrophoresis was without the monoclonal peak (M-spike) and control bone marrow aspirate was almost normal (Figure 6). She was accepted to achieve complete remission. After the careful medical oncology evaluation, a general surgery was planned for the patient. A total gastrectomy was performed. The pathological result was also low differentiated tubular adenocarcinoma. She was under regular follow-up in our out-patient clinic. After one year, there were no significant lesions or lymphadenopathies at the whole body Computered tomography scans. Biochemical tests revealed; sedimentation rate: 31 mm/h, total protein:7 g/dL, albumin: 4,2 g/dl, globulin 2,8 g/dl, red blood cells:4,01 M/Ul, hemoglobin: 12,6 gr/dL, hematocrit:37,2%, IgG:1270 mg/dL, IgA:280 mg/dL, IgM:62,9 mg/dL, calcium:8,9 mg/dL, creatinine:0,6 mg/dL, lactate dehydrogenase:571 U/L.

Discussion

The growing number of malignant hematological diseases associated with secondary malignancies seems to be

one of the most important entities in clinical hematology and oncology (5). Is MM behaving as a risk factor for the co-existence of secondary solid neoplasms? It was speculated that alkylating agents, radiotherapy, environmental factors and immunologic tolerance may cause development of a secondary primary tumor in patients with MM (6). Plasmocytomas clinically may present as gastric involvement by extramedullary plasmocytoma and they should be considered in the differential diagnosis of a patient with a history of MM especially if haematemesis, melaena or dyspeptic symptoms occur. Although plasmocytomas seem to be more frequent as a gastric lesion among MM patients secondary malignancies as gastric cancers should not be underdiagnosed (7). In a retrospective study analyzing secondary malignancies among patients with MM it was shown that 26% of the patients were associated with gastric cancer (8). Like in our case multiple myeloma may precede a secondary malignancy or a solid tumor may precede MM (9).

Conclusion

Here we reported a very rare combination of synchronous gastric adenocarcinoma and MM. The time of occurrence of second malignancy and the type of presentation was unusual. Secondary malignancy risk should be kept in mind during the follow-up of patients with MM.

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