

TABLE I, B. Results of testing latex positive sera for inhibition by concentrated banana or latex extract in a latex RAST

Patient	Percent inhibition produced by banana extract	Percent inhibition produced by latex extract
1	46.0	85.0
2	56.0	58.2
3	26.9	67.7
4	29.5	ND
5	25.0	ND
6	-7.0	ND
7	22.0	ND
8	7.7	ND
9	2.0	42.0

ND, Not done.

the concentrated banana extract produced inhibition of 22% to 56% with six of the nine sera (Table I, B).

CONCLUSIONS

Our study confirms in 16 of 24 patients the single case report of M'Raihi et al. of cross-reactivity between latex and banana allergens. The 16 sera were 10 banana-ELISA positive sera which were also latex-ELISA positive and six latex-RAST positive sera which were inhibited by banana extract. It is inter-

esting that all our banana positive sera had detectable IgE specific to latex, whereas none of our latex positive sera had detectable banana-specific IgE in the same assay. This suggests there is only partial cross-reactivity between these two allergens. Limited cross-reactivity is also suggested by the fact that even after 10-fold concentration, banana extract could only partially inhibit the binding of six of nine latex positive sera to latex in a RAST. The results of the inhibiting banana positive sera with latex might have been less impressive if sera had been available from individuals with clinical histories of anaphylaxis from banana contact. In light of the increasing concern about latex-induced anaphylaxis, the cross-reactivity between banana and latex allergens may be clinically important. Physicians evaluating patients should keep the possibility of banana and latex cross-reactivity in mind.

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Acute nonlymphocytic leukemia in systemic mastocytosis with biclonal gammopathy

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Systemic mastocytosis is a hematologic disease characterized by widespread infiltration of mast cells into multiple organ sites.¹ This disease has been found to be associated with abnormal prebeta-lipoproteins, oligoclonal immunoglobulins, and malignancy, especially in adults.^{1,2} We presented a case in "Allergy Grand Rounds" concerned with lymphadenopathic mastocytosis in this patient with biclonal gammopa-

TABLE I. Proportions of phenotypes in typical and atypical cell population

CD markers	Typical lymphocyte population (%)	Atypical lymphocyte population (%)
Pan leukocyte		
CD45 Bright	91	1
CD45 Dim+ CD14 -	7	44
CD45 Dim+ CD14 +	0	55
T-cells		
CD3 (Leu4)	85	2
CD5 (Leu1)	64	1
CD4 (Leu3a)	45	1
CD8 (Leu2a)	34	1
CD3 + DR + (activated)	4	1
CD7 (Leu9)	37	0
B cells		
CD19 (Leu12)	7	1
CD19+ CD5 +	1	0
CD10 (Calla)	0	0
Myeloid cells		
CD14 (LeuM3)	0	55
CD13 (My7)	1	37
CD33 (My9)	2	99
Nonlineage		
HLA DR	15	43

thy.³ We present this brief communication to further summarize the patient's progression leading to a malignant process.

CASE REPORT

The patient was a 56-year-old black male retired farmer without a significant past medical history, who was admitted to the Nassau County Medical Center with generalized fatigue, severe weight loss, postprandial nausea, and diarrhea for a period of 1 year. Physical examination was remarkable for hepatomegaly, splenomegaly, right axillary lymphadenopathy, and hyperpigmented lesions on the trunk, with a positive Darier's sign.

Complete blood cell count revealed 4400 white blood cells with 43% neutrophils, 39% lymphocytes, 14% monocytes, and 3% eosinophils. Hemoglobin was 11.8 gm/dl. Sedimentation rate was 65 mm/dl. The bilirubin was slightly elevated at 1.3 mg/dl, and the alkaline phosphatase was also elevated at 180 u/L. Although the plasma histamine level was normal, a 24-hour urine histamine was elevated at 98 µg.

Chest radiograph, electrocardiogram, upper gastrointestinal examination, barium enema, and CT scan of the chest were all unremarkable. CT scanning of the abdomen demonstrated moderate hepatosplenomegaly without focal lesions as well as extensive and marked mesenteric and retroperitoneal adenopathy.³

A diagnostic laparotomy was performed. Lymph node and liver biopsy specimens both showed mast cell infiltration.

Serum protein immunoelectrophoresis demonstrated

two beta/gamma spikes, and immunofixation revealed IgG/kappa, IgA/lambda, biclonal gammopathy.³ IgG was 1870 mg/dl, IgA was 2000 mg/dl, and IgM was 147 gm/dl. Urine immunofixation demonstrated a monoclonal free kappa light chain. Bone marrow biopsies revealed pancytopenia and paratrabecular fibrosis without plasmacytosis. A skeletal survey showed diffusely increased bony density.

The patient was treated with a combination of H1 and H2 antagonists for 3 years, and then ascites, pedal edema, progressive weight loss, postprandial diarrhea, generalized weakness, and malaise developed. He was admitted and treated with oral sodium cromolyn, which was successful in ameliorating gastrointestinal complaints.⁴ A repeat bone marrow biopsy showed moderate megakaryocytic and erythrocytic hypoplasia, paratrabecular fibrosis, and mast cell infiltration. In addition, marked granulocytic hyperplasia with increased myeloid immaturity was observed with a markedly increased white blood cell count. The peripheral blood smear showed blast forms, and immunophenotyping by flow cytometry revealed typical lymphocytes and an atypical large cell population. The large atypical cells expressed markers typical of myeloid lineage (Table I). Almost all atypical cells were CD45 dim (suggesting myeloid lineage, or an immature stage) and CD33 (MY9) positive. Some proportions of the atypical cells also expressed CD14 (Leu M3), CD13 (MY7), and HLA DR markers. The presence of these markers were consistent with a diagnosis of acute nonlymphocytic leukemia of the myelomonocytic type. The patient declined a trial of chemotherapy and died of his malignancy a month later.

DISCUSSION

This patient had multiple diagnoses, including lymphadenopathic mastocytosis with involvement of the spleen, liver, abdominal lymph nodes, skeleton, bone marrow, skin, and gastrointestinal tract. He was also diagnosed with acute nonlymphocytic leukemia predated by the discovery of a biclonal gammopathy.³ Oligoclonal immunoglobulins have been observed in systemic mastocytosis, but this is the first patient with systemic mastocytosis to our knowledge with a biclonal gammopathy.² Oligoclonal immunoglobulins were found to be associated with those patients who had a secondary malignancy as well as a more malignant form of mastocytosis.² Biclinal gammopathy most commonly occurs as an IgG/IgA combination, and a significant portion (16%) of these patients are diagnosed with lymphoproliferative disorders such as lymphoma and lymphocytic leukemia.⁵

As a group, patients with systemic mastocytosis have an abnormally increased incidence of hematologic disorders and malignancies including dysmyelopoietic syndromes, myeloproliferative disorders, acute leukemia, and malignant lymphoma. Patients with systemic mastocytosis and coincident hematologic disorders have a significantly decreased survival time compared with the patients with systemic mastocytosis alone.⁶

We have presented this brief communication to provide a follow up of our "Allergy Grand Rounds" presentation on a patient with lymphadenopathic mastocytosis and biclonal gammopathy.³ Mastocytosis has

been suggested to be due to an innate oncogenic aberration in a hematopoietic stem cell resulting in abnormal mast cell proliferation.² The development of leukemia could correspond to an increased proliferation of cellular forms that possess this aberration.² Perhaps, T-cell derived mast cell interleukin growth factors (IL-3, IL-4, IL-9, IL-10) are partly involved in regulating the expression of neoplasia in a subset of patients with systemic mastocytosis.³ Finally, this case integrates biclonal gammopathy with systemic mastocytosis and the likelihood of developing malignancy.

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C1 esterase inhibitor in pregnancy

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We present a case of a pregnant woman who was admitted with repeated episodes of angioedema. Serum C1 esterase inhibitor level (C1INH) was found to be low, although C4 levels were normal. The inhibitor was reassayed in the postpartum period and was found to be within normal limits. Similar labo-

ratory findings were found in 2 of 22 asymptomatic women during and after pregnancy.

CASE REPORT

A 26-year-old woman who was in excellent health until 4 years before consultation had episodic periorbital edema and itchy eyes that lasted 4 to 5 hours and recurred initially at 2-week intervals, then recurred with increasing frequency, so that after 5 months the symptoms were present daily. Nine months after onset she began working at Wrigley's Gum Factory where she was exposed to several sugars, gum resin, and aspartame, and she noted that her symptoms occurred more frequently in the morning and during the daytime at work. One time on exposure to aspartame dust

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