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A Case of IgE Myeloma: Methodology and Review of the Literature

Pat A. Allevato, MD,* Michael J. Deegan, MD,* Jau-Wen Chu, PhD,* Mary B. Perry, MS,* and Carolyn L. Barth, PhD*

A 56-year-old man presented with a one-year history of progressive weakness predominantly affecting his extremities and persistent low back pain. Ouchterlony immunodiffusion of the concentrated urine detected a marked increase in lambda light chains. A sternal bone marrow documented a diagnosis of multiple myeloma. Screening high resolution agarose gel electrophoresis revealed diffuse hypogammaglobulinemia and, retrospectively, an equivocal, faint band which migrated in the fast gamma region. By using a combination of immunoelectrophoresis and immunofixation electrophoresis, this questionable band was determined to represent an IgE lambda monoclonal protein. Radioimmunoassay for IgE documented a serum concentration of 50.6 mg/dl. No intact IgE was found in the urine. Following chemotherapy, the patient’s serum IgE level decreased significantly, and he is presently asymptomatic.

Features of special interest in this case include the low serum IgE level on presentation, which was difficult to detect on the screening electrophoretogram, and the use of immunofixation electrophoresis in the detection and characterization of these “difficult” gammopathies.

IgE myeloma is the rarest and most recently described among the plasma cell dyscrasias. This rarity undoubtedly reflects the low level of IgE normally found in serum (1) and thus the small number of IgE producing cells present (2). Since the initial observation by Johansson and Bennich in 1967, only 19 cases have been reported (3-21), including a case of benign monoclonal IgE gammopathy (22) and several other reports where monoclonal IgE was associated with other disorders (23-26).

The purpose of this paper is to describe the clinical and immunochemical features of this new case of IgE myeloma and to compare them with the previously reported cases. In addition, the methodology used to resolve serum paraproteins present in low concentration will be briefly discussed.

Case Report

A 56-year-old Caucasian man presented in March, 1983 complaining of a one-year history of progressive weakness and tiredness with persistent low back pain, pain in both legs and arms, and difficulty in chewing due to extreme jaw weakness.

His past medical history was remarkable for bilateral carpal tunnel syndrome in 1981. There was no history of asthma, hay fever, or food allergy. The patient was not taking any medication.
The patient was started on chemotherapy including vincristine, 1,3-bis-(2-chloroethyl)-1-nitrosourea (BCNU), Adriamycin, cyclophosphamide, melphalan, and prednisone with gradual improvement. Two months following treatment, his urine protein and serum IgE level had decreased significantly (Table I). The patient is presently asymptomatic and is being followed on an outpatient basis.

**Materials, Methods, and Results**

**Protein studies**

Rate nephelometric quantitation of serum immunoglobulins using a Beckman nephelometer documented diffuse hypogammaglobulinemia with an IgG level of 586 mg/dl, IgM 34 mg/dl, and IgA 29 mg/dl.

High resolution agarose gel electrophoresis (HRAGE) of serum and concentrated urine was performed on Panagel slides (Worthington) consisting of 1% agarose on plastic supports in barbital buffer (pH 8.6) at 200 volts for 45 minutes. After electrophoresis was completed, the agarose gel was fixed in picric acid solution for ten minutes, rinsed in 95% ethanol, and stained in Amido Black. Serum protein electrophoresis showed a total protein level of 6.4 g/dl, albumin 4.2 g/dl, alpha-1 globulin 0.3 g/dl, alpha-2 globulin 0.68 g/dl, beta globulin 0.79 g/dl, and gamma globulin 0.48 g/dl. Retrospectively, a faint, equivocal band was present in the fast gamma region (Fig. 1a).

Double immunodiffusion in agar was performed according to the method of Ouchterlony (27). The concentrated urine revealed markedly increased free lambda light chains with a trace of IgG and negative IgM and IgA. Double immunodiffusion of the serum for IgE was positive up to a dilution of 1:256.

Immunofixation electrophoresis (IFE) was performed by a modified method of Ritchie and Smith (28). The patient's serum and pooled human control sera were diluted with barbital buffer (pH 8.6) to obtain a final protein concentration of 5 µg/10 µl. Three to five microliters of the diluted sample were applied to the Panagel slides, and the proteins were separated as described above by HRAGE (Panagel Electrophoresis Reagent Kit and Migration Unit, Worthington DR0047601 and DR0047602). After the electrophoresis run was completed, the gel was removed, and one Whatman #1 filter paper strip was laid over the cathodal portion of each sample. Approximately 100-150 µl of antiserum against IgG, IgA, IgM, IgD, IgE, lambda, and kappa was evenly applied along the center length of each strip, and the gel was incubated with the antiserum in a humidity chamber for one hour at room temperature. The gel was removed, rinsed with saline, and pressed for 10 minutes using drying blotter pads. Following further washes with saline and deionized water and repressing between wash cycles, the slide was dried at 50°C for 10 to 15 minutes and stained in Amido Black for 10 minutes. The slide was then destained in reagent grade water until the background was clear, blotted, and air dried. The serum demonstrated a broad monoclonal IgE band in the fast gamma region with a lambda light chain band which had the same migration. A second, darker staining lambda band, which migrated in the beta region and was obscured by the C-3 component of complement in the HRAGE, represented free monoclonal lambda light chains (Fig. 1b).

Immunoelectrophoresis (IEP) of the serum and urine was carried out on agarose plates (Paragon, Beckman) by a modification of Scheidegger’s method (29). The urine IEP demonstrated a heavy arc corresponding to markedly elevated lambda light chains (Fig. 2a). The serum displayed two abnormal arcs joined by a line of

**TABLE I**

<table>
<thead>
<tr>
<th></th>
<th>Total Urine Protein (g/vol)</th>
<th>Total Serum Protein (g/dl)</th>
<th>Serum IgE (mg/dl)</th>
<th>Immunoglobulins (mg/dl)</th>
<th>Bone Marrow Plasmacytosis (%)</th>
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</thead>
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<td>Pretherapy</td>
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<td>6.1</td>
<td>50.6</td>
<td>586 34 29</td>
<td>23.5</td>
</tr>
<tr>
<td>2 Months Post-Therapy</td>
<td>0.6</td>
<td>6.3</td>
<td>19.8</td>
<td>457 30 23</td>
<td>9</td>
</tr>
<tr>
<td>Normal Range</td>
<td>0-0.1</td>
<td>6.8-8.5</td>
<td>0-0.05</td>
<td>600-50-250 60-300</td>
<td>0-1</td>
</tr>
</tbody>
</table>

135
identity; one arc signified free lambda light chains, and the other possibly indicated monoclonal lambda chains associated with the IgE moiety or polyclonal lambda chains associated with the IgG immunoglobulin (Fig. 2b). The serum also documented an abnormal arc precipitating with anti-IgE antiserum (Fig. 2b); the normal serum failed to form a precipitin line with this antiserum because of the normally low level of IgE. No intact immunoglobulins were present in the urine by either IEP or fractional separation by Sephadex G-75 chromatography (Pharmacia Fine Chemicals). The total serum IgE level as measured by solid-phase radioimmunoassay (30) was 230,000 U/m,l (50.6 mg/dl).

**Immunohistochemistry**

Immunohistochemical localization of IgE, kappa, or lambda in lymphocytes was accomplished using a direct immunoperoxidase staining technique. Cytospin slide preparations of Ficoll/Hypaque mononuclear cell suspensions prepared from bone marrow aspirate were used. A negative reagent control (primary antibody replaced with a 1:20 dilution of Horseradish-peroxidase-T5 mg/10 ml PBS) and a positive tissue control slide were included in the evaluation.

The bone marrow plasma cells displayed intracytoplasmic positivity for both IgE and lambda light chains (Figs. 3a,b, and c).
Discussion

The clinical and immunochemical features of 20 cases of IgE myeloma are summarized in Tables II and III.

The first two cases (3,4) were characterized by plasma cell leukemia, monoclonal light chains of the lambda type, and absence of bone lesions; however, subsequent reports showed a much greater heterogeneity associated with this entity. The mean age at presentation in this study was 61.7 years with a range of 48 to 77. No significant differences in age were present between men and women. Male to female ratio was almost equal (11:9) approximating that of IgG and IgA myeloma (31).

The clinical features were similar to other types of multiple myeloma except for a higher frequency of hepatosplenomegaly (35%), which is fairly uncommon in myeloma (32). Bone lesions were predominantly lytic (50%); however, two cases (10%) showed osteosclerosis, a feature which, when unrelated to therapy or pathological fracture, occurs in no more than 3% of
patients with non-IgE type of myeloma (33-35). In this respect, one of the reported cases (5) remains questionable (20) because of the appearance of the sclerotic lesions following melphalan therapy (36). Plasma cell leukemia was reported in five patients (25%), four of whom were men. This feature has been cited in only 1.5 to 2% of reviewed myeloma cases (37,38) and to date appears to be the most characteristic feature of IgE myeloma (20,21). Anemia, with a hemoglobin level of less than 12 g/dl, was detected in 18 patients (90%) and appears to be more common and severe than in other myeloma types (32).

Severe renal failure (BUN greater than 80 mg/dl) was found in three cases (15%). This compares to a similar frequency in IgG and IgA myeloma but is less than half as common as in light chain disease (39). Hypercalcemia

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr) at Diagnosis and Sex</th>
<th>Clinical Features</th>
<th>Hepatosplenomegaly</th>
<th>Lytic or Sclerotic Bone Lesions</th>
<th>Severe Renal Failure</th>
<th>Plasma Cell Leukemia (% Plasma Cells)</th>
<th>Survival from Diagnosis (months)</th>
<th>Reference</th>
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<tbody>
<tr>
<td>1</td>
<td>50,M</td>
<td>chest and low back pain</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>+ (61)</td>
<td>42</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>60,M</td>
<td>fatigability, poor vision</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>+ (65)</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>65,F</td>
<td>weakness, fatigue, nosebleed</td>
<td>+</td>
<td>LS</td>
<td>—</td>
<td>&gt; 2.5</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>51,M</td>
<td>bone pain</td>
<td>+</td>
<td>L</td>
<td>—</td>
<td>&gt; 6</td>
<td>7</td>
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<td>5</td>
<td>48,F</td>
<td>bone pain</td>
<td>—</td>
<td>L</td>
<td>—</td>
<td>&gt; 16</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>59,F</td>
<td>back pain</td>
<td>—</td>
<td>L</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>57, M</td>
<td>weakness, headaches, blurred vision</td>
<td>+</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>69,F</td>
<td>hip and back pain</td>
<td>—</td>
<td>L</td>
<td>—</td>
<td>—</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>9</td>
<td>54, M</td>
<td>pathological fracture, weakness, weight loss, pain</td>
<td>—</td>
<td>L</td>
<td>+</td>
<td>+(27)</td>
<td>17</td>
<td>11</td>
</tr>
<tr>
<td>10</td>
<td>69, M</td>
<td>anorexia, nausea/vomiting, weight loss</td>
<td>—</td>
<td>LS</td>
<td>—</td>
<td>—</td>
<td>6</td>
<td>12</td>
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<tr>
<td>11</td>
<td>64, F</td>
<td>back and rib pain, weight loss</td>
<td>—</td>
<td>L</td>
<td>+</td>
<td>—</td>
<td>7</td>
<td>13</td>
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<tr>
<td>12</td>
<td>65, F</td>
<td>weakness, weight loss</td>
<td>—</td>
<td>L</td>
<td>—</td>
<td>—</td>
<td>NR</td>
<td>14</td>
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<tr>
<td>13</td>
<td>55, F</td>
<td>epistaxis, pollinosis</td>
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<td>L</td>
<td>—</td>
<td>—</td>
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<td>—</td>
<td>—</td>
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<td>+(12)</td>
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<td>15</td>
<td>77, M</td>
<td>chest and back pain, weight loss, weakness, fatigability, recurrent infections</td>
<td>—</td>
<td>L</td>
<td>—</td>
<td>—</td>
<td>6</td>
<td>16</td>
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<td>16</td>
<td>68, M</td>
<td>&quot;pyonephritis&quot;</td>
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<td>—</td>
<td>—</td>
<td>50</td>
<td>17</td>
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<tr>
<td>17</td>
<td>71, F</td>
<td>back pain, weight loss</td>
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<td>—</td>
<td>—</td>
<td>—</td>
<td>&gt;24</td>
<td>18</td>
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<td>18</td>
<td>69, F</td>
<td>Herpes Zoster infection</td>
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<td>—</td>
<td>—</td>
<td>—</td>
<td>&gt;11</td>
<td>19</td>
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<tr>
<td>19</td>
<td>67, M</td>
<td>right shoulder pain</td>
<td>—</td>
<td>L</td>
<td>—</td>
<td>—</td>
<td>&gt; 4</td>
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<tr>
<td>20</td>
<td>56, M</td>
<td>low back pain, weight loss, anorexia, nausea/vomiting</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>9</td>
<td>21</td>
</tr>
<tr>
<td>21</td>
<td>6, M</td>
<td>weakness, weight loss, confusion</td>
<td>—</td>
<td>—</td>
<td>+</td>
<td>+(20)</td>
<td>present case</td>
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</table>
IgE Myeloma

TABLE III
Biochemical and Immunochemical Features of IgE Myeloma (20 Cases)

<table>
<thead>
<tr>
<th>Case Number</th>
<th>Hemoglobin (g/dl)</th>
<th>Total Serum Protein (g/dl)</th>
<th>Urine Protein (g/24 hrs)</th>
<th>Light Chain Type</th>
<th>Serum IgE (mg/dl)</th>
<th>M-Protein Electrophoresis Mobility</th>
<th>Serum Calcium (mg/dl)</th>
<th>Serum Creatinine (mg/dl)</th>
<th>Percent Plasma Cells in Bone Marrow at Diagnosis</th>
<th>Reference</th>
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<td>1</td>
<td>10.2</td>
<td>9.5</td>
<td>NR</td>
<td>L</td>
<td>NR</td>
<td>fast-γ</td>
<td>4500</td>
<td>NR</td>
<td>1.1</td>
<td>3</td>
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<td>2</td>
<td>4.0</td>
<td>12.5</td>
<td>3.18</td>
<td>L</td>
<td>7500</td>
<td>γ</td>
<td>6.5</td>
<td>9.0</td>
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<td>K</td>
<td>2700</td>
<td>mid-γ</td>
<td>12.9</td>
<td>10.1</td>
<td>1.2</td>
<td>5</td>
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<td>4</td>
<td>7.0</td>
<td>10.1</td>
<td>NL</td>
<td>K</td>
<td>180</td>
<td>x</td>
<td>10.5</td>
<td>NR</td>
<td>NR</td>
<td>6</td>
</tr>
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<td>5</td>
<td>6.5</td>
<td>11.3</td>
<td>NR</td>
<td>K</td>
<td>6300</td>
<td>γ</td>
<td>12</td>
<td>1.9</td>
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<td>6</td>
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<td>8.4</td>
<td>0.05 (g/L)</td>
<td>K</td>
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<td>γ</td>
<td>9.5</td>
<td>NR</td>
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<td>8</td>
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<td>9.5</td>
<td>NR</td>
<td>K</td>
<td>2100</td>
<td>β-2</td>
<td>9.9</td>
<td>1.2</td>
<td></td>
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<td>9</td>
<td>6.3</td>
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<td>16 (g/L)</td>
<td>K</td>
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<td>fast-γ</td>
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<td>β</td>
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<td>(g/L)</td>
<td>K</td>
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<td>NR</td>
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<td>NR</td>
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<td>γ</td>
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<td>0.9</td>
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<td>L</td>
<td>50.6</td>
<td>fast-γ</td>
<td>9.8</td>
<td>1.1</td>
<td>23.5</td>
<td>present case</td>
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</tbody>
</table>

*Defined as light chains present in urine detected by pH indicator colorimetric screening methods, heat test, and sulfosalicylic acid test. NR = not reported, NL = normal.

(sera calcium greater than 11 mg/dl) was present in only two (14.3%) of 14 patients and is less frequent than in other myeloma types (40). Interestingly, of the nine women with IgE myeloma, five (55.6%) had a history of ovarian lesions which included Brenner tumor (5), cysts (15,20), serous papillary cystadenocarcinoma (10), and one case (14) in which the type of ovarian lesion was not reported. The significance of this is presently not known.

None of the reported cases showed any evidence of amyloidosis, although our case presented with symptomatology suggestive of this disorder. The light chains were typed as kappa in almost three quarters of patients with IgE myeloma as compared to two thirds of patients with IgG and IgA myeloma and only one tenth of those with IgD myeloma (18,39,40). The M-protein migrated predominantly in the fast gamma region on zone electrophoresis. The serum IgE concentration ranged from 50.6 to 7500 mg/dl with a mean of 3056 mg/dl. Marrow plasmacytosis exceeded 50% in 9 of 15 cases (60%) where such information was documented.

The mean survival time from diagnosis in 11 patients who died was 15.4 months with a range of 1.3 to 30 months. Survival time from diagnosis was less than 24 months in 9 patients. Despite a reported favorable initial response to chemotherapy in many patients, the average survival time is shorter than for IgG and IgA myeloma but longer than for IgD myeloma and light chain disease of the lambda type (31,32). Well-recognized features denoting a relatively poor prognosis are similar to those of other types of myeloma (41,42) and include renal failure, severe anemia, extensive skeletal involvement, and Bence Jones proteinuria (16).
The present case is of interest because of the low level of M-protein in the serum (50.6 mg/dl) which was not initially appreciated as an M-spike on the screening electrophoregram. This, coupled with the markedly elevated lambda light chains found in the urine, led to an initial consideration of light chain disease even though features commonly present in this disorder, such as hypercalcemia, Bence Jones proteinuria, renal failure, amyloidosis, and lytic bone lesions were absent (43). It is quite conceivable that some patients with IgE myeloma have been typed as light chain disease in the past (16).

This leads to a discussion on the evaluation of monoclonal proteins when present in low concentration. Traditionally, monoclonal proteins (MPs) in serum or urine have been studied by a combination of zone electrophoresis and classical IEP (44,45). Recent improvement in the average survival of patients with multiple myeloma has been dependent on earlier diagnosis and thus less tumor cell burden and decreased serum MP concentration (46). It may be difficult, on the basis of IEP analysis alone, to detect a small serum MP due to the limited resolving power of this technique (47). Other paraprotein isolation and purification methods are time consuming and laborious. We use HRAGE with IFE in conjunction with IEP when studying difficult monoclonal gammopathies. IFE consistently appears to be superior to both IEP and routine protein electrophoresis for the detection and characterization of MPs, particularly at concentrations of less than 1000 mg/dl (48-55). IFE offers several advantages over other techniques including decreased performance time, so that diffusion leading to band broadening is kept to a minimum with resulting increased resolution, reduced reagent consumption, and its easy interpretation and comparison to the agarose electrophoretic pattern (56). Closely apposed bands are easily resolved by IFE but may be completely lost in IEP (55-57).

In conclusion, we have presented the clinical and immunoochemical features of a new case of IgE myeloma with a review of the literature. This entity appears to be associated with an increased frequency of severe anemia, less hypercalcemia and amyloidosis, a tendency to plasma cell leukemia and gammapathy of the kappa light chain type, and a shorter survival. Additional information awaits further documented cases. We have suggested the use of the more sensitive technique of immunofixation electrophoresis to study these monoclonal gammopathies in hope that earlier diagnosis and increased survival rates will be attained.

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