Dramatic oligoclonal paraproteinaemia following a pneumococcal septicaemia

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Summary: We describe a case of paraproteinaemia in which there were high concentrations of four monoclonal bands shown on qualitative, and confirmed by quantitative, techniques. The disorder followed a pneumococcal infection and resolved spontaneously with the complete disappearance of all four bands

Introduction

Paraproteins have been described in association with B cell malignancies and after hyperstimulation of one or more normal B cell clones.¹ The latter leads to one (monoclonal) or several (oligoclonal) bands on electrophoresis.¹ Benign paraproteins are often transient, their concentrations are low and can represent restricted immune response to infection. This report describes a dramatic case of transient paraproteinaemia.

Case report

A 54 year old man was admitted shocked in October, 1988. On examination he had signs of left upper lobe pneumonia and post-tuberculous bronchiectasis. He was resuscitated with intravenous antibiotics and fluids. The recovery was slow and complicated by acute renal failure and sub-endocardial myocardial infarction. Investigations confirmed the presence of left upper lobe pneumonia. Blood cultures grew *Streptococcus pneumoniae*; it was not serotyped. One month after discharge he was readmitted with rigors, due to bronchitis, and although no organism was cultured he responded quickly to oral pivampicillin. Throughout this time the patient had an ESR of greater than 100 mm/h. Bone marrow examination was normal.

Serum electrophoresis was carried out using 1% agarose in barbitone buffer, containing calcium lactate. Ten ml of agarose (Sigma Chemicals, type 111 E.E.O.) was placed onto a 10 cm × 10 cm glass plate. The samples of serum were diluted 50% in the barbitone buffer. Electrophoresis was carried out at 300 volts for 50 minutes using water cooling.

Proteins were fixed in the agarose using saturated picric acid plus glacial acetic acid, then stained with Coomassie blue. Immunofixation was carried out using a commercial kit (Dako Ltd). Total immunoglobulins were assayed using a Laurell 'rocket' technique, the serum being diluted in a barbitone buffer containing formalin.

Qualitative immunological tests revealed four bands, which were also seen on immunofixation for IgG and kappa light chains. Two of the bands were much more dense than the others. This was not due to *in vitro* polymerization as they were seen in fresh samples. Bence Jones protein was not identified in the urine, although small amounts of the IgG paraproteins were found. The peak level of IgG was 32.5 g/l, (normal range 7–19). The level of IgG fell progressively over the subsequent 6 months (Table I). There was slight elevation of IgM which was polyclonal. The ESR has been less than 60 mm/hour since December 1988. Currently the patient is well. He has had no infections since November 1988.

Discussion

Paraproteins may arise in malignant or benign conditions. The majority (90%) of cases are benign

Table I Quantitative measures of immunoglobulin recorded over a 5 month period

Date of sample	IgG	IgM	IgA
(Normal range) g/l	7–19	0.5–2.0	0.9–4.5
03/11/88	32.5	4.9	< 0.9
02/12/88	17.7	5.4	1.1
05/12/88	16.5	5.4	1.1
19/12/88	13.6	6.0	1.0
02/02/89	11.8	6.2	1.3
29/03/89	12.1	6.1	1.0

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Figure 1 Sequential electrophoresis showing gradual fading of four IgG bands; alpha and beta globulins are fading as the acute illness settles; albumin is constant.

and 2% disappear on follow up.² Malignant aetiology is suggested by the presence of Bence Jones protein,^{1,2} monoclonal protein level of greater than 10 g/l,¹ an increase in the level with time^{1,2} and immunoparesis.^{1,2} The paraprotein should be identified by electrophoresis and then typed and confirmed by immunofixation.¹

Danon and Seligmann³ described 14 cases of transient monoclonal gammopathy in which 8 cases of primary or secondary immunodeficiency were identified. One of their patients³ had a monoclonal band of over 20 g/l, although the exact level is not reported. A transient IgA monoclonal gammopathy was reported in a patient with acute lymphoblastic leukaemia, in remission, associated with cytomegalovirus infection; the peak serum

level of IgA was 24.5 g/l.⁴ More recently paraproteinaemias have been recognized in AIDS patients.⁵

The patient we have presented had an enormous, but transient, paraproteinaemia of nearly 20 g/l, in which four monoclonal bands were identified, after pneumococcal infection. He was not immunocompromised. It is probable that the condition represents an aberrant immune response to infection.

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