

Aggressive Light Chain Myeloma Originating a Double Peak on Serum Electrophoresis: What's Underneath?

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Dear Editor,

Free light chain myeloma is commonly characterized by hypogammaglobulinemia at presentation. For this reason, in this setting of patients, the observation of a double M-band on serum electrophoresis in the absence of complete immunoglobulins at immunoelectrophoresis is a rare phenomenon.

We report the case of a 57-year-old diabetic Caucasian male whose recent medical history began with the admission to the emergency department with metabolic acidosis and bone pain. Blood samples showed severe kidney failure, mild anemia, and hypercalcemia, as follows: hemoglobin 9.4 g/dL, creatinine 9.54 mg/dL, total serum protein 7.6 g/dL, albumin 3.6 g/dL, and calcium 12.9 mg/dL. Beta-2 microglobulin was 34.6 mcg/mL, and NTproBNP was 1,154 pg/mL, with no sign of heart failure. Radiologic examinations showed several vertebral osteolytic lesions. A bone marrow biopsy was performed revealing massive plasmacellular infiltration for more than 90%. Cytofluorimetric essay performed on a bone marrow sample showed 99% of the CD138/CD38/CD33/CD56 positive plasma cells expressing cytoplasmic lambda chains.

At electrophoresis, a protein migration in the gamma region of 16.6% (1.3 g/dL)

with a double M-band of 7.4% (0.59 g/dL) and 8.5% (0.65 g/dL), respectively, was present (Fig. 1), while immunoelectrophoresis on serum and urine uniquely detected the presence of lambda free light chains, and the immunoglobulin dosage was consistent with global hypogammaglobulinemia. The free light chain dosage was as follows: lambda 44,171.5 mg/L, kappa 6.7 mg/L. The patient was initially treated with bortezomib-thalidomide-dexamethasone but decided to continue treatment elsewhere, due to logistical reasons. As far as we know, he was still alive after 1 year.

The literature is really poor regarding this phenomenon, which seems to be rarely reported. To sum up what is commonly known of double gammopathies, it is possible to outline the following settings: simultaneous antigenic expansion of two B-cell clones, second malignant clone, single malignant clone with subclone selection, light chains exceeding heavy-chain production in a pathological monoclonal gammopathy, or dimerization of IgAs [1, 2]. We were not able to classify our patient into any of these categories, since immunoelectrophoresis only detected lambda light chains in serum samples.

In 1976, Sölling [3] described the presence of dimeric lambda chains in serum of

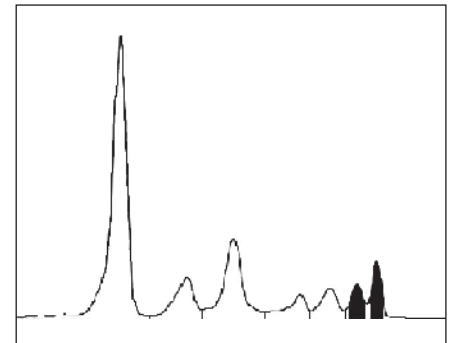


Fig. 1. Densitometric tracing of serum protein electrophoresis revealing a double M-band in a patient affected by light chain myeloma.

both normal and anephric individuals. Differently from kappa chains, which were mostly noncovalently linked and existed as predominant monomeric forms, lambda chains generated covalent links and were detectable as stable dimers. Moreover, the presence of serum concentration of light chains was found to be correlated to renal dysfunction.

In 2015, Yang et al. [4] described a cohort of 26 patients affected by multiple my-

eloma who had a clonal M-band made of intact immunoglobulins with two different types of lambda light chains, which migrated differently on immunofixation electrophoresis. The authors concluded that “the two λ light chains may be different” [4].

In conclusion, we can hypothesize that our patient had a biologic condition similar to that described in the Chinese cohort [4], with the difference of being a light chain myeloma. The double peak we report could have been originating from both an ex-

ceeding production of two different types of lambda free light chains, each determining an M-band or an extraordinary quantity of identical lambda light chain, emphasized by renal insufficiency, which partially underwent dimerization.

Statement of Ethics

The patient provided written informed consent to the publication of the clinical data and laboratory image.

Disclosure Statement

The authors have no conflicts of interest to declare and received no funding for this paper.

Author Contributions

All authors contributed to the clinical management of the patient and critically reviewed the paper. E.G. drafted the paper. U.B. provided the laboratory image.

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