JAMC, Vol. 8, No. 2, 1996

CASE REPORT

MONOCLONAL GAMMOPATHY

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CASE 1

A 63 years old male patient with known ischaemic heart disease presented with incontinence and weakness in the legs which was progressively increasing over the past 4 months and recently he noticed mild weakness in the upper limbs as well. Neurological examination revealed a fully conscious and intelligent gentleman with normal speech, a supple neck and intact cranial nerves. Lower limb examination revealed mild wasting, no fasciculation, a grade 3 power, absent knee jerks, and plantars could not be elicited. The position and vibration senses were absent and sense of pain and temperature were impaired, the perineal sensations were intact and the anal reflex was present. The upper limb examination revealed 4 weakness with sluggish tendon jerks. The CSF examination showed a total protein of 120 mg/100ml with normal cell count. His plasma protein electrophoresis showed monoclonal gammopathy. The urine did not show any light chains and the bone marrow examination was normal. His nerve conduction studies and electromyography confirmed a polyneuropathy. The patient received five sessions of plasmapheresis after which there was some increase in power in both the upper and lower limbs. The patient was then started on immunoglobulin at the rate of 400 mg/kg/day which came out to 25 gm per day. After five courses of Gamma Immune-N his lower limb power increased to 4 and he was able to walk with support.

Diagnosis: Chronic inflammatory demyelinating polyneuropathy secondary to monoclonal gammopathy.

CASE 2

A 56 years old male patient presented with two months history of malaise, weight loss and episodic blurring of vision. On examination he looked thin but healthy. His temperature was normal. No lymph nodes were palpable and he was not anaemic or jaundiced. His BP was 150/85 and his cardiovascular system was normal. He had no carotid bruit. Fundoscopy revealed a small haemorrhage in the left eye and soft exudate in the right eye. Neurological examination and examination of the respiratory system was normal. The abdomen was soft and there was no visceromegaly.

Investigations:

Hb 13.5 gm/l00ml. ESR 80 mm in 1st hour.

TLC 8000/cu mm with polymorphs 67%, lymphocytes 31 % and eosinophils 2%.

Blood sugar and blood urea was normal.

X-ray chest and X-ray skull was normal, the ECG was normal.

Serum proteins 80 gm/1. Albumin 40 gm/1. Globulin 40 gm/1.

Plasma protein electrophoresis showed a monoclonal gammopathy.

Bone marrow examination showed an increase in the number of plasma cells and many of these plasma cells were larger in size with pale cytoplasm.

Urine examination was normal and did not show light chains.

Diagnosis: Monoclonal gammopathy due to multiple myeloma.

CASE 3

A 66 years old female presented with 2 weeks history of fever and left sided pleuritic chest pain. She had been feeling generally unwell over the past six months and had been complaining of tiredness, anorexia and weight loss. On examination she was frail looking, anaemic and was running a temperature of 101 F. Her BP was 140/70 and her cardiovascular system was normal. She had signs of consolidation in the left lung base. She was slightly drowsy but the neurological examination was otherwise normal. The abdomen was soft, the spleen was just palpable, the liver was just palpable, there were no abdominal masses and no signs of ascites.

Investigations:

Hb 10 gm/100 ml. ESR 130 mm at one hour.

TLC 14,000/cu nun. Polymorphs 85 %, Lymphocytes 15%.

X-ray chest showed consolidation in the left lower lobe.

X-ray skull showed multiple punched out lesions. The ECG was normal.

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Plasma protein electrophoresis showed monoclonal gammopathy of the IgG type.
Bone marrow showed increased numbers of giant plasma cells confirming the diagnosis of multiple myeloma.

Diagnosis: Monoclonal gammopathy due to multiple myeloma complicated by left lower lobe pneumonia.

DISCUSSION

Monoclonal gammopathy is the hallmark of symptomatic paraproteinaemias. In this condition only one type of immunoglobulin or its light chains increase in the circulation because a single chain of B cells is triggered to produce immunoglobulin. Sometimes such proliferation of B cells is relatively benign, asymptomatic and slowly progressive and not associated with impaired immunity '. However, two important pathological conditions are a cause of serious illness - multiple myeloma and Waldenstrom's macroglobuhnaemia. In multiple myeloma die B cell differentiates into plasma cells and may result in formation of a monoclonal production of any one type of immuno-globulin, whereas in Waldenstrom's macroglobulinaenua the resulting monoclonal antibody is only of the IgM type.
I am reporting three cases with different clinical presentations. The first patient is still under my care and is fully mobile and independent 18 weeks after all medication was stopped. He has still not developed any features of myeloma or Waldenstrom's macrolobulinaemia or any other haematological malignancy and may be a rare example of a benign monoclonal gammopathy which has caused symptoms.

Case 2 is a good example of a patient with the hyperviscosity syndrome. These patients present with varied neurological features but most commonly visual symptoms due to poor retinal circulation.

The presence of retinal haemorrhages and exudates is common [2]. Case 3 presented with an acute infection which is a very common mode of presentation of myeloma patients due to diminished immunity.

It has been reported that patients with corneal opacities due to deposits of immunoglobulins and associated asymptomatic monoclonal gammopathy may later develop multiple myeloma 3.

It has been suggested that family members of patients with gammopathies may have a higher incidence of immunological dysfunctions [4].

REFERENCES

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