A length dependent sensori-motor neuropathy revealing a multiple myeloma

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Neurological manifestations are common in myeloma patients; most are due to nervous or vascular compressions. Peripheral neuropathies are rare: 1-5% in osteolytic myeloma,[1,2,3] and 30-50% in osteosclerotic myeloma (POEMS syndrome). Osteolytic multiple myeloma (MM) is mainly accompanied by peripheral sensorimotor (PNP) or sensory polyneuropathy, and more rarely by acute extensive PNP, Denny Brown PNP or by chronic progressive or with relapse polyradiculoneuritis.[4] Often, the PNP is the first symptom leading to the discovery of myeloma.[4] We report one case of length dependent PNP revealing MM.

A 67-year-old, male, non-drinker, was treated 4 years ago by chemotherapy (Epirubicin, Vincristine, cyclophosphamide and prednisone) followed by 2/3 gastrectomy for a centroblastic non-Hodgkin's malignant lymphoma of MALT type (mucosa associated lymphoma tissue) of the stomach without extra gastric localization (IE stage of Muschoff). At the end of the treatment, the patient was in complete remission and did not have any secondary iatrogenic complication with chemotherapy. Eighteen months later, he presented with basi-thoracic girdle pain. The neurological examination showed an abolition of the Achilles' tendon reflex, and of the medioplantar reflex, a hypoesthesia of the hands, the feet and the periumbilical area. However, there were no cranial nerves attack, and no motor deficiency. The remainder of the somatic examination showed no organomegaly or peripheral adenopathy. The diagnosis of length dependent PNP was suspected and confirmed by the electromyogram. Various examinations were then carried out in the search of a causal affection: the complete blood count and the renal function were normal, glycemia was 4.8 mmol/l, sedimentation rate was raised to 125 at the first hour, calcium was 2.89 mmol/l, protidemia was 78 g/l. Protein electrophoresis showed a gamma peak at 21.5 g/l, the serum immunoelectrophoresis, showed a monoclonal gammopathy IgA type. Bence Jones proteinuria was negative. Myelogram showed a medullary infiltration to 50% by atypical plasma cell. The cranium radiography revealed multiple lytic lesions (**Figure 1**). The thoraco-abdominal scanography showed multiple lytic costal and dorsal rachis lesions without visceral or ganglion attack. The medullary MRI eliminated a radicular compression. The remainder of the etiologic investigation eliminated a relapse from the lymphoma (normal gastric fibroscopy), of toxic metabolic, infectious, or iatrogenic origin. The patient received polychemotherapy by vincristine, melphalan and prednisone, the evolution was carried out towards aggravation and the patient died 7 months later.

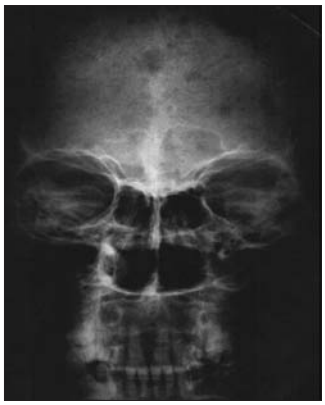

Figure 1 - Cranium radiography revealed multiple lytic lesions.

Although PNP related to MM are well described, they remain rare and are rarely revealed at the disease onset. The average age of occurrence is around 50 years, and the strong preponderance in males is clear. The diagnosis of an advanced length dependant sensorimotor polyneuropathy was made on the abolition of Achilles' tendon reflexes, the distribution of the sensory deficit and the decrease of both sensory and motor conduction velocity on the electromyogram. In addition, the patient presented an osteolytic MM of IgG lambda type on the existence of the oligoclonal peak and the medullary plasmocytes cells infiltration to 50%: stage III of SALMON and DURIE MM. The association between myeloma and neuropathy is well known, mainly in osteosclerotic myeloma, but its physiopathology remains unclear and widely discussed.[5] The neuropathy is probably due to an infiltration of peripheral nerves by plasmocytes and amyloid deposits. Clinical signs of neuropathy are observed in 2-8% of the lymphomas, and the electro physiological abnormalities are those of a multi neuritis in 35% of the cases.[4] In our patient, the lymphomatosis origin was drawn aside by the clinical examination, the negativity of the gastric fibroscopy and the absence of ganglionic or visceral localization on the scanner. For neuropathies related to lymphomas, it is necessary to add those induced by chemo and radiotherapy, in particular Vincristine, which is at the origin of a cumulative dose dependent neurotoxicity, partially reversible with stopping

treatment. However, occurrence of the neurological signs after the treatment (18 months), eliminates neurological toxicity by Vincristine. The metabolic causes are easily drawn aside by the biological check-up and the absence of macroglossia and renal or cardiac involvement. Occurrence of MM among already treated patients for a non-Hodgkin's malignant lymphoma was reported, but the clinical features were typical repetitive infections with osseous pain.

In conclusion, it is justified in the presence of a polyneuropathy, even in the absence of the suggestive clinical signs to seek a monoclonal plasma cell proliferation by an immunochemical study of the serum and urine, and by skeleton radiography.

Received 23rd October 2005. Accepted for publication in final form 25th April 2006.

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