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Case report

Intravascular malignant lymphomatosis : report of 2 neurological cases

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Abstract

We report two cases of intravascular malignant lymphomatosis (IML) with a clinical expression limited to the central nervous system. The first patient presented with signs of cerebral, cerebellar and spinal cord involvement. The second had an isolated involvement of the spinal cord. In both cases the diagnosis was made at post-mortem examination ; pre-mortem examination of biops tissue from peripheral nerve and muscle in the first case, spleen and liver in the second were unhelpful for the diagnosis of lymphoma. We review the published literature on IML, its ante-mortem diagnosis and treatment.

Key words : Intravascular malignant lymphomatosis ; lymphoma ; chemotherapy ; myelopathy.

Introduction

Intravascular malignant lymphomatosis is an uncommon neoplastic disorder, which often involves the central and peripheral nervous system in the setting of a relentless multisystemic disease : clinical presentations restricted to the nervous system are rare but they represent a difficult diagnostic challenge.

We report two cases of intravascular malignant lymphomatosis presenting with clinical signs restricted to the nervous system. The first patient presented with cerebral , cerebellar and spinal cord signs, the second with an isolated myelopathy. Both cases were diagnosed at post-mortem examination ; pre-mortem examination of neuromuscular biopsy in the first patient and of splenic and hepatic biopsies in the second patient were unhelpful for diagnosing this lymphoma.

Clinical presentation

PATIENT 1

A 54 year-old-man was admitted to the hospital in July 1996 for gait disturbance with asymmetrical lower limb weakness (left > right), urinary retention, and diffuse headache. He had experienced lower limb thrombophlebitis one week earlier.

His previous medical history included severe osteoporosis, prostatic hypertrophy, gastric ulcer, hypertension and Menière's disease. He was treated with bisoprolol, betahistine, terazosine and cimetidine.

Clinical examination revealed a paresis of the left upper limb and lower limbs. Tendon jerks were brisk with a bilateral Babinski and a right Hoffmann's sign. Cerebellar signs predominated on the right side with moderate ataxia, hypermetry and dysdiadocokinesia. Sensory testing showed distal hypoesthesia of the lower limbs. The patient improved initially under methylprednisone 64 mg/d but after a few days he developed asthenia, anorexia, dizziness and confusion.

The only laboratory abnormalities were increased levels of SGPT and gamma-GT. Immunological and serological studies were negative. CSF examination showed increased protein (82 mg/dl on admission ; 114 mg /dl one week later ; 44 mg/dl after 1 month) ; mild lymphocytosis (10 cells/mm^3) and oligoclonal banding on isoelectrofusing in two out of three CSF samples. Brain CT-scan showed a cortico-subcortical parietal low density lesion. Brain MRI showed multiple aspecific lesions (hyperintense on T2- and hypointense on T1-weighted images) of the left middle cerebellar pedunculus, the corpus callosum and the right hemispheric white matter. Thoraco-lumbar MRI was unremarkable. Abdominal CT-scan and ultrasonography were normal. Thoracic CT-scan was suggestive of bilateral segmental emboli. On EMG there was mild denervation in multiple lumbosacral radicular territories suggestive of a cauda equina lesion. Transcranial magnetic stimulation revealed slowing of central motor conduction between C5 and C7 on the left side. A neuromuscular biopsy sampling a distal sensitive branch of the musculocutaneous nerve and peroneal muscle showed mild axonal loss in nerve fascicles and changes of acute denervation in the muscle.

The patient deteriorated gradually and died less than two months after onset of the first symptoms. At autopsy, the fixed brain weighed 1360 gr and showed moderate symmetrical edema of both hemispheres. Coronal sections showed numerous

areas of haemorrhage and yellowish discolouration in the centrum semi-ovale, corpus callosum and cerebellar white matter (figs. 1a & 1b). Histology showed diffuse investment of leptomeningeal and parenchymal vessels by lymphomatous cells of B-phenotype at all levels of the neuraxis and in dorsal root ganglia and vasa nervorum of spinal roots (figs. 1c & 1d). Lymphomatous infiltration was also seen in the kidneys, lungs, myocardium, spleen and liver.

Retrospective study of the neuro-muscular biopsy failed to disclose any tumour cell.

Patient 2

A 73 year-old-man was admitted to hospital in July 1998 for progressive urinary retention and constipation, followed by weakness and dysesthesias of the lower limbs. The latter appeared in a few hours causing gait disturbances and frequent falls. There were no other complaints and no fever.

His previous medical history included right nephrectomy for hypernephroma in 1993, diabetes mellitus type II (treated with gliclazide), and a tuberculous synovitis in the 40's.

Clinical examination showed mild wasting, weakness and hyporeflexia of the lower limbs. Abdominal reflexes were abolished and plantar reflexes were in extension. There were no abnormalities in the upper extremities. There was a Romberg's sign and all sensory modalities were decreased below the dermatomal level T4 bilaterally.

Routine biochemical, immunological and serological studies were unremarkable. CSF analysis showed mildly increased protein levels (73 mg/dl), no oligoclonal bands, a lactic acid level of 23 mg/dl and a glucose level of 106 mg/dl ; cell count was normal (5 mononuclear cells/mm^3). MRI of cervical and thoracic spine was unremarkable, except for a T4 trapezoïdal fracture accompanied by mild disc protrusion but without spinal cord compression. The clinical diagnosis was transverse myelitis of unknown etiology.

After physical therapy, the patient was able to walk alone and resumed with activities of daily life. A few weeks later he had pulmonary embolism and was treated with acenocoumarol. On February 26, 1999, he suddenly presented increased weakness and paresthesias of the right lower limb. This was accompanied by myoclonic jerks spreading up to the right abdominal wall. Sensory testing was unchanged. Laboratory investigations showed increased levels of LDH [333 mUI/ml ; (nl : 120-230)]. Immunological and serological studies remained normal. CSF protein was slightly increased (68 mg/dl) with 4.3 white cells/mm^3 and no oligoclonal bands. Reduction of the central motor conduction velocity bilaterally between C7 and L5 was found on transcranial magnetic stimulation of the motor cortex. Cervico-thoracic MRI was unchanged. Steroïd treatment was instaured two days before discharge.

The patient was admitted for the third time 1 month later because of intermittent fever and chills which appeared three days after the beginning of corticotherapy. There was a slight increase in lower limb weakness and a general deterioration with anorexia, asthenia and weight loss. Clinical examination showed paraparesis with a sensory level at T4. There were signs of inflammation on laboratory testing with increased LDH levels (433 mUI/ml). Chest X-rays were normal. Antibiotics were given for an isolated bacteria but fever persisted. Extensive investigations (cardiac ultrasonography, thoracic, abdominal and brain CT-scans, prostatic ultrasonography, …) were normal except for splenomegaly. Because of persistent fever, splenectomy was performed and accompagnied by hepatic biopsies. On histological examination, the only reported abnormalities were those of chronic spleen and liver congestion. Despite various drug therapies including antibiotics, steroids and an antituberculosis tritherapy, the patient died on May 21, 1999, more than 11 months after appearance of the first symptoms.

At autopsy, macroscopic examination of the brain was unremarkable. Sequellae of spinal cord infarction were seen at the lower cervical and upper thoracic levels (fig. 1e) and they were not associated with inflammatory infiltrates or specific inclusions. However, numerous leptomeningeal and parenchymal small vessels of the brainstem, spinal cord, spinal roots and dorsal root ganglia were filled with large lymphoma cells of -phenotype ($CD20^+$) (fig. 1f). Intravascular lymphomatosis was also seen in the lung, prostate and kidneys.

Retrospective study of the splenectomy specimen revealed a lymphomatous infiltration of the red pulp, which had been previously unnoticed.

Discussion

Intravascular malignant lymphomatosis (IML) was first described in 1959 by Pfleger and Tappeiner, as "angioendotheliomatosis proliferans systemisata". It has been reported in the literature under a variety of names including malignant or systemic angioendotheliomatosis, endothelioma, angiotropic large-cell lymphoma and intravascular lymphoma.

Its histological feature is proliferation of large, atypical lymphoid cells within the lumen of capillaries, small veins and arteries (Wick et Stacey 1991). The histogenesis of IML cells has been variously described as endothelial (Pfleger and Tappeiner 1959) or lymphocytic (Mori *et al*. 1985, Domizio *et al*. 1989). Immunohistochemical and genetic studies indicate that the malignant cells have lymphoid origin and that in most cases, such

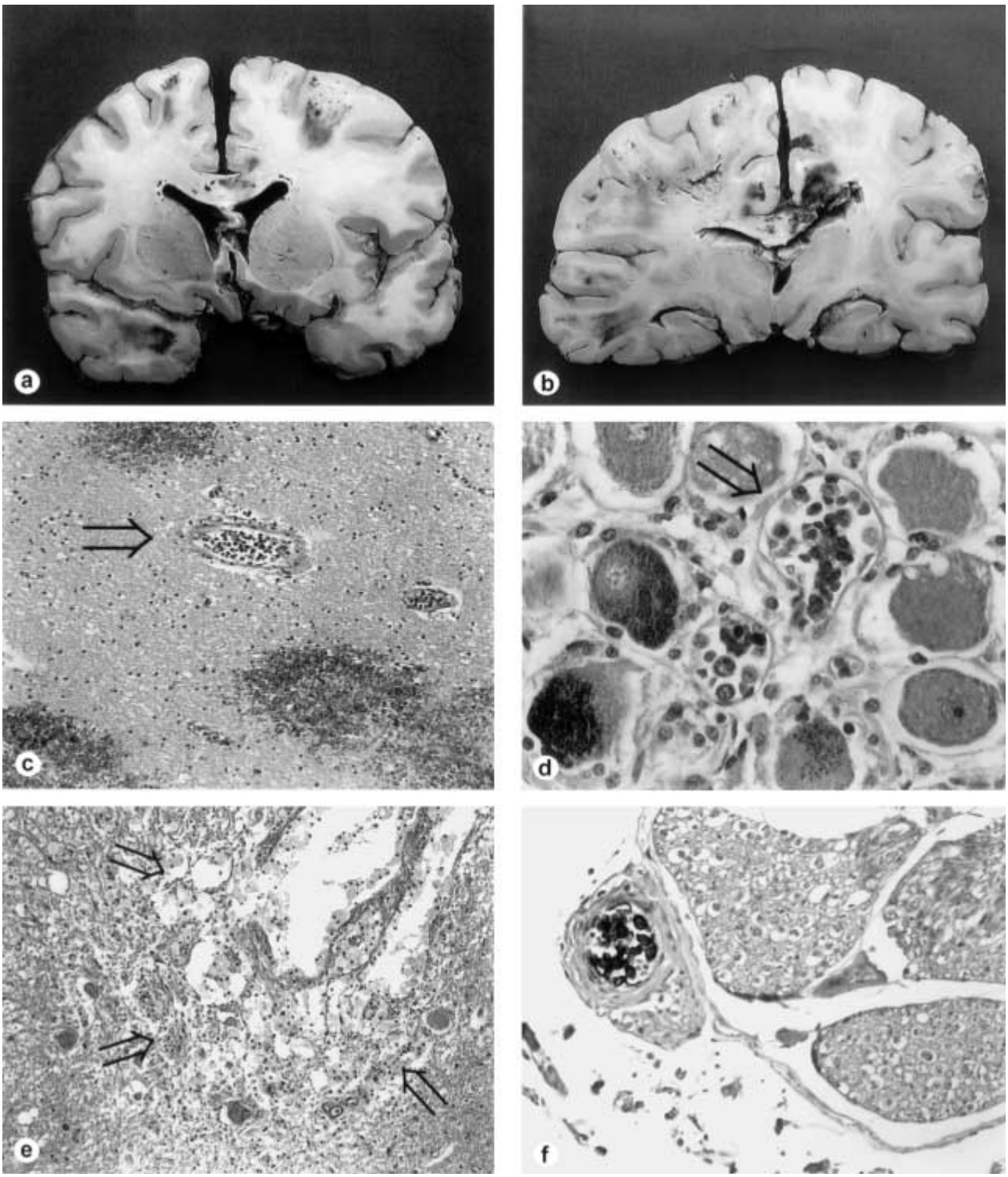

FIG. 1. — Autopsy results :

Case 1 :

(a-b) : numerous ill-defined areas of haemorrhagic and ischaemic changes are present in the centrum semi-ovale and corpus callosum (coronal sections) ;

(c-d) : large lymphoma cells with centroblastic or immunoblastic features (arrows) investing the small vessels in the hemispheric white matter (c : He, 100 X) and in a lumbar dorsal root ganglia (c : HE, 400 X).

Case 2 :

(e-f) : sequellae of spinal cord infarction (arrows) at the cervico-thoracic junction (HE, 100 X) ; $CD20^{+}$ lymphoma cells are seen in the vasa nervorum of a neighbouring dorsal spinal root (immunostaining with anti-CD20, 400 X).

as in ours, they have a B-cell immunophenotype (Otrakji *et al.*1988, Stroup *et al*. 1990, Wick *et al*., 1991, Demirer *et al*., 1994). IML is considered to be a rare, high-grade, extranodal non-Hodgkinian lymphoma with a tropism for the endothelium. The affinity of tumor cells for capillary endothelium may be explained by lymphocyte receptors for endothelial membrane antigens (Demirer *et al*., 1994, Shanks *et al*., 1997).

IML may affect a wide range of age groups with a high incidence in the seventh decade. There is no clear gender difference in incidence. In up to one-half of cases, IML presents clinically with skin lesions characterized by prominent , hyperpigmented, or hypervascular plaques or nodules, located preferentially over the abdomen and the thighs. Up to to two-thirds of patients have neurological symptoms with more frequently diffuse cerebral signs and dementia or focal cerebral signs including stroke-like episodes, partial or generalized seizures (Pellat *et al*. 1993, Liszka *et al*. 1994). Other neurological involvements include peripheral neuropathy, polyradiculopathy, myopathy and myelopathy (Levin et Lutz 1996).

In this report, patient 2 presented with spinal cord symptoms for the first eight months of the disease course, after which he developed diffuse CNS symptoms. Similar cases of isolated myelopathy lasting for many months have been reported in IML (Hamada *et al*. 1991). However, in most reports of IML cases with initial spinal symptoms, the disorder remained limited to the spinal cord for no more than three months (Bots 1974, Dolman *et al*. 1979, Ojeda 1983, Dubas *et al*. 1990). As in our cases, repeated MRI of the spinal cord did not reveal any medullary lesions.

Infiltration by IML of lungs, kidneys, adrenal glands and prostate is frequent. All organs may be involved, individually or in combination, but liver, spleen, lymph nodes and bone marrow are relatively spared until late in the disease course. Patients may also present with general symptoms ("B-symptoms") such as fever of unknown origin (Kuvliev *et al*. 1999), night sweats, chills, weight loss and malaise (Saacson et Norton 1994). Thrombophilia has commonly been reported in IML (Wick *et al*. 1986, Stroup *et al*. 1990, Stahl *et al*. 1991, Curtis *et al*. 1991), which may also have favoured the thrombophlebitis in patient 1 as well as pulmonary embolism in both cases. A more unusual presentation is disseminated intravascular coagulation (Stahl *et al*. 1991).

Common laboratory abnormalities include mild anemia, elevated erythrocyte sedimenation rate and markedly elevated serum lactate dehydrogenase levels (Otrakji *et al*. 1988, Croisile *et al*. 1990). Usually, there are no detectable circulating lymphoma cells in blood and CSF, but the latter is frequently characterized by moderate pleiocytosis and elevated protein levels.

Table 1

Neurological intravascular malignant lymphomatosis (4 groups)

1. Progressive, multifocal cerebrovascular events	78%
2. Spinal cord and nerve root vascular syndromes	38%
3. Subacute encephalopathy	27%
4. Peripheral or cranial neuropathies	21%

(Glass *et al*., Cancer 1993).

Brain MRI may reveal aspecific lesions mostly located in the white matter. However, Liow et al reported two cases with linear, punctate, and patchy enhancement on brain MRI (Liow *et al*. 2000). In these two cases, the pattern of enhancement was correlated with T2 abnormalities (the more pronounced the T2 abnormality, the more patchy/confluent the enhancement). They concluded that careful interpretation of cerebral MRI findings including T2/FLAIR and T1 postgadolinium sequences could allow clinicians and neuroradiologists to make timely diagnosis. Spinal MRI is generally normal even in cases presenting with spinal cord signs (Hamada *et al*. 1991, Nakahara *et al*. 1999, Waring *et al*. 1999, Bequet *et al*. 2000). The diagnosis is made post-mortem in most (> 60%) cases. Sometimes, it may be provided by biopsies of the brain, skin or other involved organs (Liszka *et al*. 1994, Nakahara *et al*. 1999, Vieren *et al*. 1999, Baumann *et al*. 2000, Bequet *et al*. 2000). When there is neurogenic weakness in the legs, muscle biopsy has been diagnostic in 14 out of 19 patients (74%) (Petito *et al*. 1978, Sparling *et al*. 1979, Krieger *et al*. 1982, Shibuya *et al*. 1983, Vital *et al*. 1989, Molina *et al*. 1990, Stroup *et al*. 1990, Stahl *et al*. 1991, Lacomis *et al*. 1992, Glass *et al*. 1993, Harris *et al*. 1994, Sleater *et al*. 1994, Roux *et al*. 1995, Levin et Lutz 1996, Prayson 1996, Butori *et al*. 1997, Suzuki et Koizumi 1997, Kuvliev *et al*. 1999, Nakahara *et al*. 1999). Cerebral biopsies may be falsely negative in neurological cases in which the diagnosis was confirmed post-mortem (Bille *et al*. 1995).

IML with CNS involvement has an extremely poor prognosis. Most patients survive no more than 1 year after the diagnosis has been made (Willemze *et al*. 1987, Levin & Lutz 1996). Transient improvement has been reported with steroids, radiation therapy, plasmapheresis and chemotherapy (Domizio *et al*. 1989, Stroup *et al*. 1990, Beal & Fischer 1982, Nakahara *et al*. 1999). Chemotherapy regimens directed at intermediate and high-grade lymphomas seem to be the most efficient treatments (Di Giuseppe *et al*. 1994, Nakahara *et al*. 1999, Vieren *et al*. 1999). In patients receiving CHOP chemotherapy (cyclophosphamide, doxorubicine hydrochloride, vincristine sulfate and prednisone), 6 (55%) of 11 went into complete remission (Demirer *et al*. 1994). In cases of retractory

IML, a second-line therapy can be performed followed by autologous bone marrow transplantation (Rose *et al*. 1999).

To summarize, IML should be considered in the differential diagnosis of unexplained myelopathy, meningo-encephalitis, acute confusional state, dementia or stroke-like syndromes. Diagnostic work-up should include biopsy of organs known to be frequently involved in IML, such as skin, kidneys, adrenal glands, liver and lungs. Biopsy of peripheral organs may lead to diagnosis avoiding the need for a cerebral biopsy and allowing appropriate treatment to be started. CHOP chemotherapy eventually with G-CSF support is the most effective treatment.

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