CASE REPORT

Intravascular Lymphoma with Conus Medullaris Syndrome Followed by Encephalopathy

Martin Savard, Steve Verreault, Peter V. Gould, Vincent Bernier, Jean-Pierre Bouchard

ABSTRACT: ***Background:*** Intravascular large cell lymphoma (ILCL) is a diagnostic challenge, with neurological, cutaneous and constitutional symptoms. The natural history is usually an evolution to a comatose state. As invasive procedures are usually required for diagnosis, recognizing the typical clinical pattern is critical since an effective treatment is available. ***Method:*** After an extensive literature review of the subject, we report a case of ILCL, analyzing clinical, laboratory, radiological and pathological data. We will also give a special attention to the clinical picture of a conus medullaris (CM) lesion with subsequent encephalopathy in the same patient. ***Results:*** We report here a 61-year-old woman with a paraplegia caused by a CM lesion, evolving about one year latter to encephalopathy and eventual coma, with the diagnosis of ILCL confirmed by autopsy. The present case is similar to eight other cases in literature who had CM lesion associated with ILCL, knowing that 80-90% of these patients will eventually evolve to encephalopathy without treatment. ***Conclusions:*** ILCL is a recognized but rare cause of coma. Diagnosing it is tremendously important since it is fatal if left untreated. We propose that this specific picture (conus medullaris lesion, eventually evolving to encephalopathy) is quite characteristic and will directly result in better outcome if recognized.

RÉSUMÉ: Lymphome intravasculaire avec syndrome du cône médullaire suivi d'une encéphalopathie. Le lymphome intravasculaire à grandes cellules (LIGC) représente un défi diagnostique. Il s'accompagne de symptômes neurologiques, cutanés et généraux. La maladie évolue habituellement vers un état comateux. Comme on doit avoir recours à une procédure invasive pour établir le diagnostic, il est très important de reconnaître son tableau clinique typique parce qu'il existe un traitement efficace. ***Méthode :*** Nous avons procédé à une revue de littérature exhaustive et nous rapportons un cas de LIGC avec analyse des données cliniques, biochimiques, radiologiques et anatomopathologiques. Nous portons une attention particulière au tableau clinique de la lésion du cône médullaire (CM) avec encéphalopathie subséquente chez le patient. ***Résultats :*** Nous rapportons le cas d'une femme de 61 ans présentant une paraplégie causée par une lésion du CM, qui a évolué un an plus tard vers une encéphalopathie et éventuellement un coma. Le diagnostic de LIGC a été confirmé à l'autopsie. Ce cas est similaire à huit autres cas rapportés dans la littérature de lésions du CM associées à un LIGC et il est connu que 80 à 90% de ces patients évoluent vers une encéphalopathie s'ils ne sont pas traités. ***Conclusions :*** Le LIGC est une cause connue mais rare de coma. Il est extrêmement important de poser le diagnostic parce que cette maladie est fatale si elle n'est pas traitée. Ce tableau clinique (lésion du cône médullaire évoluant éventuellement vers une encéphalopathie) est caractéristique et l'issue sera meilleure si cette pathologie est identifiée.

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Intravascular large cell lymphoma (ILCL) is an extremely rare disorder, with a little more than one hundred cases reported in the literature, which remains difficult to diagnose. Cardinal manifestations are neurological (encephalopathy, stroke and myelopathy), cutaneous and constitutional. It was first described by Pfleger and Tappeiner in 1959 under the name *angio-endotheliomatosis systemisata proliferans* because the authors thought the lesion was endothelial in origin.[1] However, it is now recognized that the tumor is rather an intravascular proliferation of lymphomatous cells, obstructing small vessels.[2,3] Unfortunately, the vast majority of neurological cases lack other manifestations, in particular cutaneous lesions.[4] Moreover, there is no easy reliable paraclinical diagnostic test available, so invasive procedures are often required.[5,6] Consequently, the diagnosis of ILCL is based on clinical grounds requiring highly

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skilled physicians. Recognizing a typical clinical pattern evoking ILCL could help physicians to be more aware of this condition, thus resulting in earlier diagnosis. This is critical because an effective treatment is available.[7,8]

Case report

A 61-year-old woman presented in December 2002 with recent headache and scotomas mimicking migraine, superimposed on a four month history of progressive paraparesis and bowel and bladder incontinence. She also reported a 20 pound weight loss in the last few months. The initial physical exam showed bilateral leg paresis (with strength about 3/5), requiring the use of a walker. Anal tone was diminished and sensory exam of lower limbs showed symmetrical diminution of all sensation (touch, cold, pinprick, vibration). There were no cognitive or upper motor neuron deficits. Spinal magnetic resonance imaging (MRI) showed a gadolinium-enhancing lesion in the conus medullaris (CM) compatible with myelitis, with another lesion at T11-T12 (Figure 1). A complete work-up, including brain MRI, visual evoked potentials and lumbar puncture for cerebrospinal fluid analysis (CSF), was done and showed no relevant abnormalities except for high CSF protein at 0.56 g/L (0.15 – 0,40) and isolated elevated serum lactate dehydrogenase (LDH) at 381 U/L (90 – 200). The CSF oligoclonal banding was absent.

Following dexamethasone treatment she improved greatly and was able again to walk without help. She was discharged with a weaning regime of dexamethasone. Her blood LDH remained elevated afterward (1923 U/L, normal 313 – 618 in another laboratory) and the sedimentation rate (ESR) was 55 mm/h (normal < 10). She relapsed in July 2003 and consulted in another hospital where they initiated a mitoxantrone treatment (8 mg/m^2 IV at each four weeks for three times, thereafter 12 mg/m^2 IV every three months with a maximum of 140 mg/m^2) although she did not fill the criteria for multiple sclerosis. No noteworthy improvement came from that treatment. She also presented deep venous thrombosis of the legs during this period and was treated with warfarin.

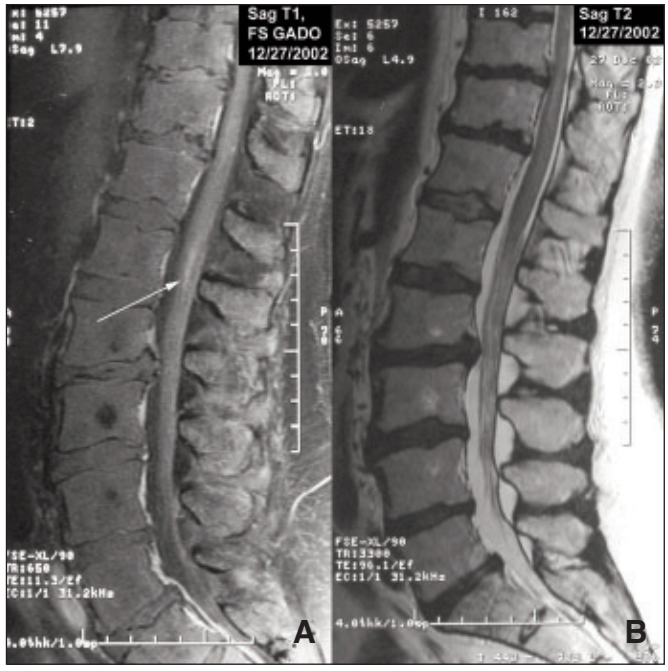

Figure 1: *(A) Sag T1 FS gadolinium-enhanced MRI showing an enhancing conus medullaris lesion (arrow) in December 2002. (B) The same lesion in Sag T2.*

She came back to our center in May 2004 because of a newly appearing confusional state (paranoid remarks and lack of words) combined with hyperthermia, neutropenia (1.41 X 10^9/L, normal >2) and significant weight loss (80 pounds in the last year). This febrile neutropenia did not respond to antibiotics. The new laboratory results showed no meaningful data with the exception of an evolving thrombocytopenia (from 113 X 10^9/L to

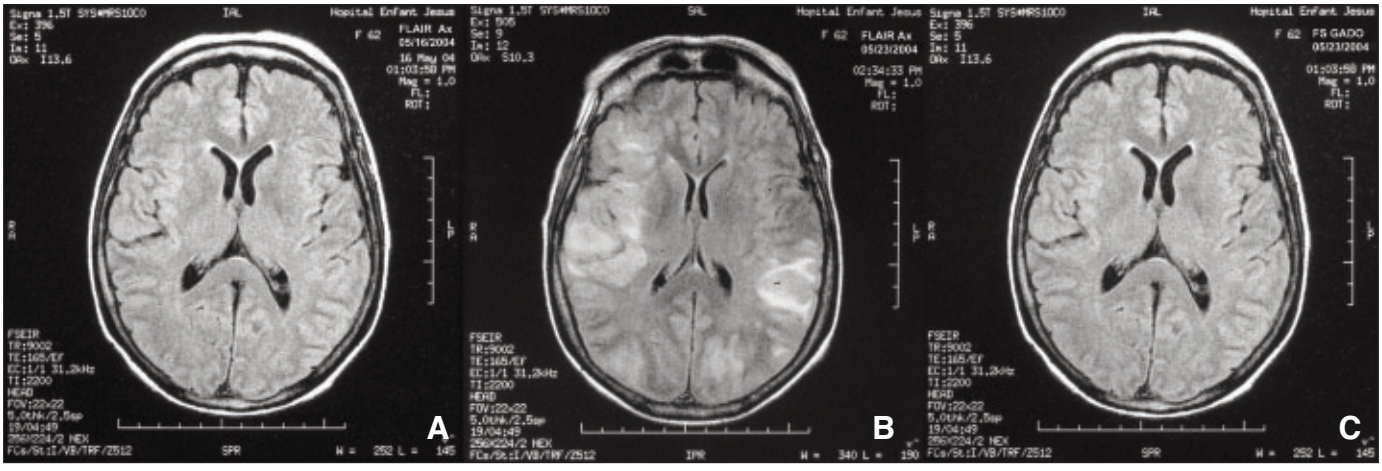

Figure 2: *(A) Normal axial FLAIR brain MRI at the time of presentation with encephalopathy on May 16, 2004. (B) Axial FLAIR brain MRI showing multiple hyperintense lesions when the patient became comatose on May 23, 2004. (C) Ax T1 FS gadolinium-enhanced brain MRI without significant enhancement the same day.*

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Table 1: Cases of ILCL reported in literature with conus medullaris involvement, both clinically and paraclinically (MRI and/or autopsy)

Age	Presentation	Evolution	Treatment	ILCL diagnosis	Ref.
78	Paraparesis Pain/hypoesthesia of sacral dermatome	Death from cardiogenic shock 1 month later	None reported	Autopsy	16*
43	Legs pain Double incont. Headache	**Confusion** Death	1st : Steroids 2nd : Methotrexate, Dexamethasone	Brain biopsy	17
63	Paraparesis Urinary incont.	Death from gastrointestinal bleeding	1st : Immunoglobulins, steroids 2nd : Foscarvir	Autopsy	18
71	Backache Urinary retention	**Confusion** Then death from respiratory distress	?	Autopsy	19**
64	Fever + weight loss Lower limb weakness Urinary retention	Death from abdominal sepsis	1st : Steroids 2nd : Cyclophosphamide + ACVB 3rd : Methotrexate + Holoxan-VPr6	Muscle + telangiectasia biopsy	20
41	Urinary incont. Impotence Genital sens. Loss	Paraplegia, Seizure, **Confusion** Death	1st : Steroid 2nd : Cyclophosphamide	Autopsy	21
71	Legs dysesthesia Paraplegia Urinary incont.	TIA Seizure	1st : Steroid 2nd : MBVP	Brain biopsy	22
63	Paraparesis	Double incont. **Confusion** Death	Immunoglobulins Steroids Azathioprine	Autopsy	23

Legend: ACVB = adriamycine, cyclophosphamide, vindesine, bleomycin, Incont. = Incontinence, MBVP = methotrexate etoposide BICNU solumedrol, Ref = Reference. * No spinal cord MRI was done for this cases. Others had positive CM lesion on MRI. ** Informations taken from the abstract (article in Japanese). Note: Another article reported a patient with CM syndrome[27], but here with no paraclinical confirmation (MRI or autopsy).

43 X 10^9/L). She continued to deteriorate until she became very drowsy one week after her admission and developed seizures on May 22. Brain MRI, negative a few days before, revealed numerous multifocal subcortical lesions (Figure 2). Her level of consciousness deteriorated, prompting intubation and transfer to the ICU on May 23. ESR climbed to 94 mm/h and CSF protein to 1.58 g/L. The patient empirically received methylprednisolone.

A cerebral stereotaxic biopsy was planned, but she died the morning surgery was scheduled on May 27. Autopsy showed a widespread intravascular large B-cell ($CD20^+$, $CD79A^+$, $CD3^-$, $CD30^-$, $CD34^-$, $CD117^-$, Alk^-) lymphoma involving all organs including the spinal cord and the brain (Figure 3) where secondary multiple ischemic lesions were seen, causing fatal cerebral oedema.

Comment

Intravascular large cell lymphoma is a rare lymphoma, usually of B-cell lineage (85% of cases,[9] though some of T-cell have been described),[7,8,10] characterized by a proliferation of large, blastic, neoplastic lymphocytes within the lumen of small blood vessels, obstructing them and causing haemorragic, ischemic or necrotic lesions in the organs involved. Men are more often affected than women (1.2-2 to one)[4,6] and the mean age at disease presentation is approximately 60 years[4,5,8,14] (with a range typically between 40 to 80, even a stillbirth[15] having been described).

The biggest challenge concerning ILCL is the early diagnosis. Neurological signs are seen in two third of the patients,[14] including subacute encephalopathy (80 – 90%),[4,6] multiple strokes (approximately 80%),[14] myelopathy (40%, with half of these as the presenting manifestation),[4,6,14] seizure (25%)[5] and cranial neuropathy (21%),[6] particularly of VII and VIII. Peripheral neuropathy, radiculopathy and myopathy are rarely seen.[14] With the latest progress in neuroimaging techniques, the most recent ILCL cases with myelopathy often show a CM involvement (in at least eight cases to our knowledge, see Table 1),[16-23] which is a unusual site for a myelitis.[24] Thus, ILCL should

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be considered among causes of CM lesion (Table 2). We propose that the eventual outcome will be better if therapy is started before the occurrence of cognitive dysfunction. Also, because encephalopathy is usually a late manifestation (37% at diagnosis and 82% before death),[4] this kind of evolution (CM involvement evolving to an encephalopathy), as in our case, seems characteristic of ILCL.

After central nervous system dysfunction, the most common features are constitutional symptoms (fever, weight loss, asthenia), found in 50% of patients and which herald, as in our case, lymphoproliferative disorder. Cutaneous manifestations (nodules, indurate plaques, purpura, telangiectasia)[2,5,20] are found in one out of three cases.[4] There are conflicting reports about the possible association of neurological and cutaneous symptoms in the same patient. Chapin[4] reported only two cases with skin manifestations among his 64 neurological ILCL cases, but other series revealed a 20% association.[5,14] Nevertheless, searching for these kind of abnormalities is important because a biopsy of these lesions can often give the diagnosis.[14] Other organ dysfunction has been described (lungs,[8] adrenals,[2] prostate,[7] spleen[10]) but they are extremely rare, despite the very high incidence of involvement of these organs at autopsy. Notably lymphadenopathy was absent.[5,8,25]

Paraclinical findings[4-6] usually encountered in this condition are elevated blood LDH without alteration of the remaining liver function tests (85 – 90%), elevated ESR (80%) and CSF protein with presence of oligoclonal bands[4] (only reported in a nine patients series with seven of them positive). In our case, blood LDH and CSF protein were increased on the first investigation. Anemia (45%) and thrombocytopenia (23%, as in our case) are often found.[6] The relative lack of malignant cells in CSF cytology (3%) and blood smears (5%) probably occurs because the abnormal cells cling to the vascular endothelium rather than passing through the vessel wall.[4-6] Bone marrow biopsy may be very useful according to some,[26] but is seldom positive according to another series,[7] with only one out of eight patients positive. Cerebral ischemic damage is usually well shown on neuroimaging studies and cerebral angiography can give exactly the same appearance as seen in vasculitis.[4] Finally, an increase of adrenal gland volume is another finding that raises the possibility of ILCL.[14,27]

Considering all these pitfalls, ILCL antemortem diagnosis is rarely made, as in our case. In this regards, even if cerebral or meningeal biopsy near a documented lesion (which is considered sufficient by some authors)[6] has a sensitivity of 33 to 100%,[5,6] each should be considered since with a tissue diagnosis an effective treatment is available. Of course, any other organ lesion found during the systemic imagery should be biopsied (especially the skin) first if possible since, as in our case, this is a disseminated disease.

Given the hematopoietic malignancy nature of this disease, corticosteroids, unfortunately often given with the idea of treating a simple myelitis, generate a dramatic but transient improvement.[5,6,18] A CHOP treatment (cyclophosphamide, doxorubicine, vincristine and prednisone), as in other high-grade lymphoma, is often used with success, remission exceeding 50% in some series.[7,27-29] Methotrexate has been suggested but with less evidence[30] and radiotherapy is recognized as ineffective.[7] Of course, no controlled data is available because of the disease rarity.

Table 2: Suggested differential diagnosis of non traumatic conus medullaris lesion

Intravascular lymphoma[16,17,18,19,20,21,22,23]
Multiple sclerosis[31, 32]
Tuberculosis[33,34,35,36,37,38, 39]
Sarcoidosis[40,41,42]
Endometriosis[43,44]
Schistosomiasis[45,46,47]
Cysticercosis[48,49,50,51]
Toxoplasmosis[52,53]
Postvaccination[54,55]
Lung metastasis[56]
Primary nervous system neoplasia (as myxopapillary ependymoma)

Note: Included in this table are etiology with, to our knowledge, at least two patients reported in the literature. Differential diagnosis of cauda equina lesion is not included (ex.: HSV-2, CMV…)

Finally, we want to emphasize that when one faces the rare association of myelitis, and particularly CM syndrome, followed by encephalopathy, ILCL is potentially one of the most important entities to exclude, and should probably be considered as soon as the patient presents with a CM lesion. In the light of an effective treatment this is vital, as an early diagnosis of this otherwise lethal condition will improve the outcome.

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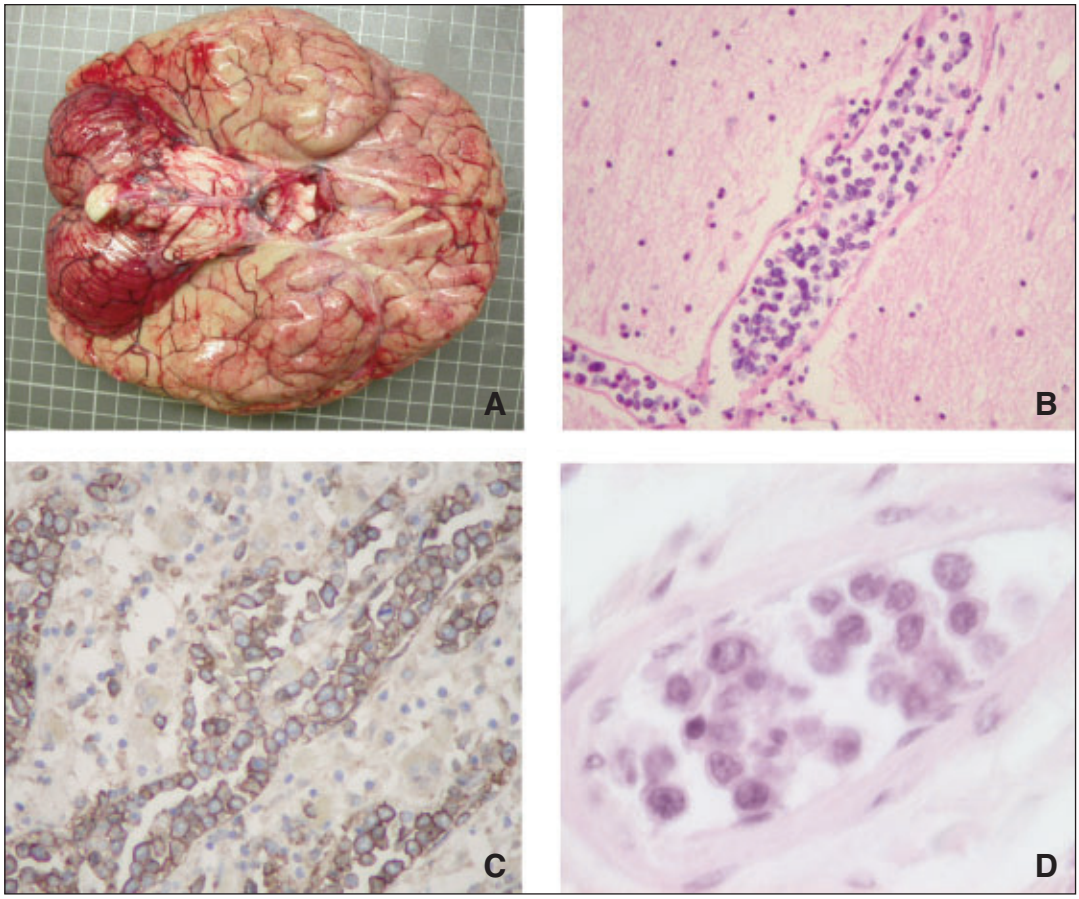

Figure 3: *(A) Gross photograph of the inferior side of the brain, showing oedema of cerebral hemisphere and cerebellar congestion. (B) Large blastic lymphocytes proliferate in cerebral vessels at autopsy (H&E 20X). (C) Adrenal vessels contain the same blastic lymphocytes (CD20 40X). (D) Spinal cord vessels with the same blastic lymphocytes at high magnification (H&E 60X).*

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