Canadian Association of Neuropathologists

Abstracts of papers and cases presented at the 43rd Annual Meeting

October 16-18, 2003

Kingston, Ontario

Can. J. Neurol. Sci. 2003; 30: 405-409

The 43rd Annual Meeting of the Canadian Association of Neuropathologists was held from October 16-18, 2003 at the Radisson Hotel Kingston Harbourfront in Kingston, Ontario. The co-ordinator for local arrangements was Dr. John P. Rossiter. The scientific sessions were comprised of 14 platform presentations and 13 diagnostic case presentations. These presentations were organized under the following headings: Tumors (Glial and Non-Glial), Cell Biology, Pediatric and Developmental Neuropathology, Infectious Disorders, and Miscellaneous Disorders.

Several special lectures were given in conjunction with the meeting. The **Royal College of Physicians and Surgeons of Canada Lecture** was delivered by Dr. Michael A. Rudnicki, Senior Scientist and Director of the Molecular Medicine Program at the Ottawa Health Research Institute and Professor in the Department of Medicine at the University of Ottawa. His lecture was entitled "Molecular Mechanisms Regulating Adult Stem Cell Specification in Muscle". An **Invited Member Lecture** was given by Dr. Juan M. Bilbao from the Department of Pathology at Sunnybrook and Women's College Health Sciences Centre, Toronto, on "West Nile Virus Encephalitis: Pathology of Seven Cases from the Year 2002 Zoonosis in Toronto". Dr. David M. Robertson, Professor Emeritus in the Department of Pathology and Molecular Medicine at Queen's University gave a historical vignette on "The Early Days of the Canadian Association of Neuropathologists".

The meeting concluded with a **Symposium on Amyloid and the Brain**, chaired by Dr. Jean Michaud, President. This symposium included the Jerzy Olszewski Lecture presented by Dr. Robert Kisilevsky, Professor Emeritus in the Department of Pathology and Molecular Medicine at Queen's University. Dr. Kisilevsky's address was entitled "Heparan Sulphate During *in vivo* Amyloidogenesis is a Valid Target for Anti-Amyloid Therapy". This lecture was preceded by an address, entitled "Modelling and Treatment of Amyloid Pathology in Alzheimer's Disease", delivered by Dr. Paul E. Fraser, Associate Professor in the Centre for Research in Neurodegenerative Diseases at the University of Toronto. The symposium was completed by a lecture delivered by Dr. Harry V. Vinters, Professor in the Department of Pathology and Laboratory Medicine at the University of California at Los Angeles Medical Center, this talk being entitled "Amyloid in Cerebral Blood Vessels: How Does It Get There and What Does It Do?"

PLATFORM PRESENTATIONS

1. Hepatocellular carcinoma metastatic to skull.

S. Krawitz, J. Megyesi, D. Macdonald, L.C. Ang (Department of Pathology, London Health Sciences Centre, University of Western Ontario).

This 76-year-old man presented with a rapidly growing skull tumour eroding the occipital bone and invading the dura. He first noticed the mass after a "bump on the head" and after localised pain, remained otherwise asymptomatic. He had a remote history of residence in Indonesia and had been previously clinically well with diabetes mellitus. Physical examination was unremarkable with no scalp ulceration. The MRI reported a large lesion originating from the occipital bone, highly vascular, with foci of hemorrhage and apparent infiltration of the superior sagittal sinus. The patient underwent a gross total resection of the lesion, uneventful apart from profuse intraoperative bleeding. Postoperative abdominal imaging showed a liver mass with involvement of the portal venous system. Pathologic examination showed macroscopically, a well defined mass with focal hemorrhage, and microscopically, a malignant neoplasm with trabecular formations, sinusoidal channels, and polygonal cells with focal bile production. The tumour cells were immunopositive for polyclonal CEA (canalicular staining), HepPAR-1, and Hepatitis B core antigen. The patient died within a month postoperatively.

Hepatocellular carcinoma is a slow growing vascular tumour often associated with Hepatitis B infection, usually diagnosed at an advanced stage, with an increasing incidence of skeletal metatasis. Skull metastases are extremely rare. Prognosis of metastatic disease is often poor and treated palliatively, but survival longer than two years has been reported with aggressive treatment. The present case shows the importance of hepatocellular carcinoma in the differential diagnosis of a metastatic skull lesion.

2. Reduced cell proliferation in germinal matrix following periventricular hemorrhage in humans and rats.

M.R. Del Bigio, J. Balasubramaniam, M. Xue (Department of Pathology, University of Manitoba, and Manitoba Institute for Child Health, Winnipeg, Canada)

Periventricular / intraventricular hemorrhage (PVH/IVH) in premature infants can have devastating consequences on

neurological outcome. Imaging studies suggest that the white matter volume is abnormally small in these individuals when they reach childhood. We hypothesized that damage to progenitor cell populations in the subependymal germinal zone might occur after PVH/IVH. Control human brains (n=17) without hemorrhage were studied at 16-44 weeks gestational age using Ki67 immunohistochemistry. In the ganglionic eminence, the labeling index peaked at 50-90% from 20-25 weeks and was negligible by ~35 weeks. Infants born at 24-28 weeks who developed PVH/IVH and survived >12 hours exhibited considerably reduced labeling index. We conclude that extravasated blood can suppress cell proliferation in germinal regions of brain. We developed models of PVH/IVH in mice and rats to test hypotheses in this regard. Rats with unilateral PVH/IVH induced by injection of autologous blood at two days age exhibit contralateral sensorimotor abnormalities that persist into adulthood. They also have reduced brain myelin content ipsilateral to the hemorrhage. Whether this is associated with ultimate failure of cell generation (e.g. of oligodendrocyte precursors whose absence could prevent proper myelin formation) remains to be determined. This has important consequences for understanding some aspects of cerebral palsy.

[Funded by Heart & Stroke Foundation of Manitoba, and supported by Manitoba Institute of Child Health]

3. Mitotically active microglia in human epilepsy.

A.W. Clark, W. Hader. (Departments of Pathology and Laboratory Medicine and Clinical Neurosciences, University of Calgary, Calgary, AB).

Experimental evidence indicates that microglia undergo mitosis in rodent brain in response to a variety of insults (Eliason et al. Dev. Brain Res. 2002; 137:75-79). We have found no reports of microglial mitotic activity in human brain tissue resected for epilepsy. We recently found abundant microglial mitotic activity in a temporal lobe resection from a 26-year-old woman. A subdural grid was used to document a series of seizures in the three days prior to temporal lobe resection. Microscopic examination of routinely processed tissue confirmed hippocampal sclerosis and mild gliosis in the neocortex. Routinely stained sections (six microns thick) revealed numerous isolated mitotic figures in the cortex and subcortical white matter. These mitoses were not associated with lymphocytes or other profiles of hematogenous cells. The mitotic profiles were associated with immunopositivity for CD45 (leukocyte common antigen) but not for GFAP. Counts of Ki67 immunopositive nuclei in ten 1 mm wide fields yielded a mean of 22 (range 16-30) in cortex and 37 (range 15-65) in white matter. There was no evidence of an infiltrating neoplasm either clinically, radiologically or pathologically. The findings indicate that microglia may become mitotically active after seizures in human brain; and that these profiles can be detected in routinely processed surgical material. It is important that this proliferative activity not be mistaken for infiltrating neoplastic cells.

4. Estrogen receptor-α immunoreactivity of the cytoplasm and processes of human reactive astrocytes.

J.P. Rossiter (Department of Pathology and Molecular Medicine, Queen's University and Kingston General Hospital, Kingston, Ontario).

During investigation of a metastatic brain tumour for potential breast carcinoma origin, estrogen receptor-α (ERα) immunolabelling was unexpectedly observed in reactive astrocytes in surrounding brain tissue. Literature review indicated that ERα immunostaining has recently been described in the nuclei, perikarya and cytoplasmic processes of reactive astrocytes following experimental brain injury in the rat and rhesus monkey. This prompted investigation of the pattern of ERα immunoreactivity of human astrocytes in surgical and postmortem tissue in a variety of neuropathological contexts. ERα immunohistochemistry (Novocastra clone 6F11 mouse monoclonal antibody) was performed on paraffin embedded sections, using standard microwave antigen retrieval and ABC techniques. Light microscopic analysis of this material shows strong ERα immunoreactivity of the cell bodies and processes of numerous astrocytes exhibiting classic cytological features of reactive gliosis. Nuclear labelling is frequently less intense than cytoplasmic, or is undetectable, in these cells. ERα immunostaining extends into small diameter distal astrocytic processes. Beading and disintegration of processes (clasmatodendrosis) is dramatically highlighted in some lesions. Focal labelling of histologically unremarkable glia limitans is also seen. The functional significance of astrocytic ERα expression is unclear, but recent literature indicates that it may mediate complex neuroprotective influences of estradiol, including regulation of glial fibrillary acidic protein (GFAP) expression.

5. Axonal regeneration in the filum terminale in adult dysmyelinated rats.

J.M. Kwiecien, R. Avram, J. Tang, C. Hui and J. Bain (Departments of Pathology and Molecular Medicine and of Surgery, McMaster University, Hamilton, ON).

CNS myelin is inhibitory to axonal regeneration. Dysmyelination in the adult CNS, such as in the Long Evans Shaker (LES) rat coincides with spontaneous axonal plasticity observed as abundant sprouting and with regeneration of transected axons in the spinal cord. Injury of the spinal cord in the adult rat, however, results in loss of bladder function and rapidly fatal bacterial hemorrhagic cystitis requiring multiple handling a day not feasible in LES rats. We determined the anatomy and function of the filum terminale (FT), a slender extension of the spinal cord from the conus medullaris (CM) towards the tail. FT is defined by a pia limitans, contains the central canal surrounded by a narrow rim of axons interspersed by oligodendrocytes and astrocytes but not neurons. FTis >3 cm long and narrows from 170 μm at the CM to 50 μm 3 cm caudally. In a study addressing axonal regeneration in the adult dysmyelinated CNS, FTwas crushed 2 mm caudal to the CM and rats allowed survival for 2 weeks. Neurological deficits typical in the spinal cord injury such as urinary bladder dysfunction were absent. In cross sections of FT taken from the site of crush to 3

cm caudal, there was abundant axonal regeneration observed in close association with ependymal cells up to 3 cm distal to the crush. This new model of adult CNS injury allows for detailed studying of axonal regeneration

6. Confirmation of a theoretical model describing the relative contribution of net growth and dispersal in individual infiltrating gliomas.

K.R. Swanson, R. Rostomily, E.C. Alvord, Jr. (Departments of Pathology, Applied Mathematics and Neurosurgery, University of Washington, Seattle, WA USA).

The hypothesis that the behavior of infiltrating gliomas is defined by their net proliferation rate (r) and their net dispersal rate (D) predicts that the "traveling wave" of the detectable edge advances linearly with time (Swanson et al., Can. J. Neurol. Sci., 2002) with a constant velocity: $v^2 = 4Dr$. Following the observations of Kelly et al. (J Neurosurg, 1987, 66:865) and Parisi et al. (Neurosurgery, 1994, 35:1036) that the gadolinium-enhanced T1-weighted MRI corresponds to "solid tumor" and the T2-weighted MRI to "isolated tumor cells", we used the mathematical model to derive a relationship between the radii (RT1 and RT2) and the ratio of the net dispersal rate to the proliferation rate: D/r. We analyzed a set of 70 patients diagnosed with glioblastoma and correlated the differences in their radii (RT1, RT2) with the ratio D/r calculated mathematically. To estimate independent measures for net dispersal (D) and net proliferation (r) still requires a measurement of the velocity (v) which can be estimated from two imaging observations without intervening treatment over an interval of time sufficient to measure significant growth, perhaps as little as one month for the most rapidly growing glioblastoma, perhaps as much as one year for a low-grade glioma.

7. Acute demyelinating myelopathy associated with Sjogren's syndrome/systemic lupus erythematosis (SLE).

B.I. Germin, J.M. Powers*, A. Anandarajah**, and E.K. Richfield* (Departments of Pathology and Laboratory Medicine* and Allergy Immunology Rheumatology Unit**, University of Rochester Medical Center, Rochester, New York).*

The pathology of acute white matter lesions of the CNS in Sjogren's and SLE is poorly understood. Inflammatory vasculitis or a primary demyelinating process are two proposed mechanisms. The paucity of pathological descriptions of acute lesions makes the distinction between these possibilities difficult. We report a case of a cervical spinal cord biopsy from a 46-year-old white woman. At the time of the biopsy she had an acute transverse myelitis felt to be suspicious for a neoplasm. Light microscopy with special stains and electron microscopy revealed this to be an acute demyelinating lesion. We therefore propose that acute demyelination may be an underlying mechanism for white matter lesions in Sjogren's syndrome and SLE.

8. Neuropsychological sequelae and patterns of radiation-induced injury in long-term survivor of grade IV astrocytoma.

E.S. Johnson (Department of Laboratory Medicine and Pathology, University of Alberta, Edmonton, AB).

Leukoencephalopathy with dementia is a recognized complication of radiation therapy. This report, however, documents an unique combination of radiation damage associated over time with different defects in neuropsychological function in a 54-year-old man who survived 19 years after 6,140 cGy CO_{60} radiotherapy for a right parietal Grade IV astrocytoma. Recurrence of tumor was suspected 15 months following irradiation because of onset of difficulties with short-term memory, paralleled by the demonstration on CT imaging and radioisotope scan of a mid-line frontoparietal abnormality. CCNU chemotherapy promptly begot a radiological resolution. After 13 years of neurological stability, the patient's neurological status deteriorated with an increased frequency of falling and, later, dementia. At autopsy the brain contained no residual tumor but showed varied patterns of radiation damage. In addition to radionecrosis of the right parieto-occipital region, there was a diffuse leukoencephalopathy in accord with radiation-induced dementia. Nonetheless, corresponding to the previous radiological abnormality, the most unusual lesion was bilateral atrophy of the corpus callosum and cingulum confined to the field of irradiation. Based upon neuroanatomical correlative studies, this cingulum degeneration accounted for the earlier impairment in short term memory. This case, therefore, illustrates that neuropsychological sequelae of radiation can be due to different patterns of damage, and that long-term survival is never free of risk of these complications.

9. Radiation-induced vasculopathy with fusiform aneurysms: report of a case.

J. Michaud, D. Keene, L. Grimard (Departments of Pathology, Pediatrics and Radio-oncology, University of Ottawa, Ottawa, ON).

This seven year-old male had a past history of a cerebellar vermis subtotal resection of a medulloblastoma. The postoperative treatment included 36 Gy to cranial-spinal axis in twenty fractions with 3D conformal boost of 19.8 Gy in eleven fractions to posterior fossa. A chemotherapy protocol was also administered. In the six months preceding his final admission, he had two episodes of strokes. The last day, he had a generalized seizure, collapsed and died. At autopsy, a recent massive subarachnoid hemorrhage was found in the posterior fossa and at the base of the brain. The distal vertebral and the basilar arteries were thickened and fibrotic. A ruptured fusiform aneurysm of the left anterior inferior cerebellar artery as well as a small non-ruptured one on the opposite side were found. Remote and resorbing infarcts were found in the median and short circumferential basilar artery territories. No residual tumour was found. Histologically, the intima was thickened and fibrotic with variable stenosis of the lumen. The internal elastic lamina was frequently disrupted with segments persisting in the wall of the aneurysms. The media was severely fibrotic. Inflammation was scarce. These changes are consistent with a radiation-induced vasculopathy. A review of eight other cases with radiation-

induced vasculopathy and cerebro-vascular complications indicates various vascular changes varying from large vessel vasculopathy to a moya-moya-like picture.

10. Cortical dysplasia and vascular anomalies: close relationship?

K. Meagher-Villemure[1], J-G. Villemure[2] (Institut Universitaire de Pathologie[1], Neurosurgery[2], Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland).

Focal cortical dysplasia is a localized and well-recognized cortical malformation with a distinctive histological presentation comprising abnormalities of both the architecture and cellular composition of the cerebral cortex.

We report three cases in which cortical dysplasia seemed closely related to some vascular event or vascular malformation. All three children, age at operation 4, 6, 8 years, started having seizures in the first year of life. One patient had an arteriovenous malformation, one patient had a benign neuroglial tumour within a malformation and one patient had within a malformation a significant multicystic component corresponding to remote vascular events. In all three cases, components of cortical dysplasia were found, consisting of disturbance of the cortical architecture, cytomegalic neurons, heterotopic neurons and severe gliosis, occasional balloon cells in one patient, neuroglial heterotopia in the subarachnoid space in one patient and ectopic neuronal nodules in one patient.

An association of cortical dysplasia and a remote vascular event may be underestimated in the spectrum of pathological changes that are encountered in the study of material removed from pediatric epilepsy surgery. The associated vascular changes in most of the cases correspond to an increased vascularity within the dysgenesis, but it may be related to some vascular anomalies like arteriovenous malformation or infarcts complicating an already maldeveloped territory or it may be the primary event leading to the abnormal cortical development.

11. Cortical dysplasia presenting as schizophrenia.

J. Keith, A. Prakash, R. Hammond (Departments of Pathology, Clinical Neurological Sciences and Psychiatry, University of Western Ontario, London, ON).

A 51-year-old male with a lengthy psychiatric history died from an aortic dissection. His past medical history was otherwise unremarkable. He had completed high school without difficulty and had worked in a number of jobs, primarily in a family business. He began to exhibit psychotic behaviour at the age of 31 years when he first reported auditory hallucinations. There were no focal neurological symptoms and specifically no seizures. When medicated, his schizophrenia was under good control and he was judged to be of average intelligence. His family history was significant for a sister and maternal aunt who had both committed suicide. At autopsy, the calvarium and brain were unremarkable in the gross state. Microscopic examination revealed extensive cortical dysplasia with atypical neuronal morphologies, neuronal clusters, disruptions of neuronal polarity and laminar disorganization throughout the neocortex of both hemispheres. A small neocerebellar heterotopia was also present. The case is a rare example of an extensive developmental neuropathology associated with a typical clinical presentation of schizophrenia. It lends support to theories of cortical dysgenesis and abnormal cortical connectivity or signaling as possible underlying etiologies in this illness.

12. Importance for cerebral autopsies remains in CJD.

G.H. Jansen and CJD Surveillance System (Prions Group, PPHB, BSSHCAID, Health Canada, Ottawa, Ontario).

In June 2003 the Canadian CJD surveillance System (CJD-SS) had entered the 421st patient suspected to have CJD in the CJD-database. The CJD-SS has been in existence since 1998. 44% of these patients have proven to be true CJD cases. Of these 186 cases, 161 were confirmed by autopsy (86%). In the non-CJD cases, the autopsy frequency was 51%. Of the 161 autopsy proven CJD cases (the 'definite' cases), 144 are sporadic, 13 have a genetic background, 3 are iatrogenic (all through Lyodura implants), and one case was variant CJD. This last case was briefly mentioned at the CANP-meeting last year.

Clinically the MRI has proven to be of help in identifying variant CJD from other forms of CJD. However MRI is not capable of identifying all forms of CJD when CJD is suspected. The specificity of the 14-3-3 CSF test has proven to be much lower than anticipated in the initial reports in literature. This is a global phenomenon, and is in part due to patient selection bias. Since both of these more recent additions to the clinician's toolbox for identifying CJD are either not sensitive or specific enough, the autopsy still has an invaluable role to play in the diagnosis of CJD.

The CJD-SS is indebted to all neuropathologists who have contributed to the CJD surveillance.

13. An unusual case of rabies with prolonged survival and extreme neuropathology.

I.R. Mackenzie, G. Medvedev and B. Thiessen (Divisions of Neuropathology and Neurology, Vancouver General Hospital, BC).

This 52-year-old man presented with rapidly progressive left arm weakness. History included bipolar affective disorder and renal failure due to lithium toxicity, requiring transplantation and ongoing immunosuppression. Initial examination disclosed a flaccid left arm and mild respiratory muscle weakness. Neuroimaging was normal. CSF showed elevated protein and WBC. Progression was steady and rapid; within a few days he had no voluntary movement or deep tendon reflexes and required intubation. Nerve conduction studies indicated diffuse motor and sensory polyneuropathy. Guillain-Barré syndrome was diagnosed. He was treated with IV Ig and plasmapheresis, without improvement. On day 16 of his illness, EEG suggested severe encephalopathy and gancyclovir was started. MRI of the head showed multifocal lesions in both white and grey matter. CSF PCR was negative for CMV, EBV, and Herpes simplex. He lost all brainstem function, was treated palliatively and died, 24 days after his initial presentation. The postmortem brain was swollen with multifocal areas of tissue softening and discoloration, involving both grey and white matter. Microscopy disclosed meningoencephalitis, rhombencephalitis, myelitis, ganglionitis and radiculitis. In all anatomic regions, a large percentage of the surviving neurons contained eosinophilic

https://doi.org/10.1017/S0317167100003164 Published online by Cambridge University Press

cytoplasmic inclusions. Rabies was confirmed by EM, immunofluorescence, immunohistochemistry and PCR. We believe the artificially prolonged survival and pre-existing immunosuppression may have contributed to the unusually widespread and severe inflammation, tissue destruction and high viral burden.

14. Ascorbate protects human CNS *in vitro* from HIV-1 gp120.

K.A. Walsh, J. Megyesi, J.X. Wilson and R.R. Hammond (Departments of Pathology, Clinical Neurological Sciences, Physiology and Pharmacology, University of Western Ontario, London, ON).

The HIV-1 envelope protein (gp120) is neurotoxic *in vitro* at nanomolar concentrations. This effect can be measured by a reduction in MAP2 and an increase in GFAP. These injuries are associated with an increase in iNOS expression, a source of endogenous oxidation through the production of Nitric Oxide (NO). The latter suggests that oxidative mechanisms of injury may be important and that supporting neuroglial antioxidant reserves may be protective. We have recently shown that ascorbate can reduce iNOS and protect murine astrocytes from exposure to lipopolysaccharide and interferon-gamma (a model of septic encephalopathy).

Human CNS cultures were exposed to recombinant gp120 at 4 weeks *in vitro* and examined for expression of MAP2, GFAP, caspase-3 and iNOS. Ascorbate preincubation protected neurons and astrocytes from the cytotoxic effects of gp120 and this was associated with attenuation of iNOS upregulation. These results suggest that cells of the human nervous system can be protected from HIV-1 gp120 by supplementing neuroglial antioxidant reserves.

(This work was supported by a grant to RH from the Ontario HIV Treatment Network).

TITLES OF DIAGNOSTIC CASE PRESENTATIONS

1. Sinonasal teratocarcinosarcoma

L.C. Ang, S. Krawitz, S. Lownie, J. Yoo, and J.G. Heathcote (Departments of Pathology, Otolaryngology and Clinical Neurological Sciences, London Health Sciences Centre and University of Western Ontario, London, ON)

2. Solitary fibrous tumor

A.S. Easton (Department of Laboratory Medicine and Pathology, University of Alberta, Edmonton, AB)

3. Primary spinal leptomeningeal rhabdomyosarcoma

S. Krawitz, D.A. Ramsay (Department of Pathology, London Health Sciences Centre, University of Western Ontario, London, ON)

4. Papillary glioneuronal tumor

J. Barron (Department of Laboratory Medicine, Memorial University of Newfoundland, St. John's, NF)

5. Astroblastoma

C. Dunham[1], D. George[2], B. Curry[1], G. Sutherland[3] ([1]Foothills Medical Centre, Calgary Laboratory Services/University of Calgary; [2]Foothills Medical Centre, Calgary Laboratory Services; [3]Foothills Medical Centre, Department of Neurosurgery, University of Calgary, Calgary, AB)

6. Diffuse pediatric glioma with extensive oligodendroglia proliferation

Y. Robitaille (Department of Pathology & Cell Biology, University of Montreal, Ste-Justine Hospital, Montreal, QC)

7. Desmin myopathy

L.-N. Hazrati[1], C. Hawkins[1], G. Midroni[2], S. Cohen[3], and J. Bilbao[3] ([1]Department of Pathology, Hospital for Sick Children, Departments of [2]Neurology and [3]Pathology, St Michael's Hospital, University of Toronto, Toronto, ON)

8. Adult-onset orthochromatic leukodystrophy

J.P. Rossiter, A.H. Koeppen (Department of Pathology and Molecular Medicine, Kingston General Hospital & Queen's University, Kingston, ON, and *Neurology Research Service, Stratton VA Medical Center & Albany Medical College, Albany, NY)*

9. Gerstmann-Strassler-Scheinker disease

J. Woulfe[1], C. Bergeron[2], A. Kertesz[3] ([1]Department of Pathology and Laboratory Medicine, The Ottawa Hospital and The University of Ottawa, [2]Department of Laboratory Medicine and Pathobiology and Centre for Neurodegenerative Diseases, University of Toronto and Toronto Western Hospital, [3]Department of Neurology, St. Joseph's Health Care, London, ON)

10. Niemann-Pick's disease, Type C

K. Meagher-Villemure[1], E. Roulet[2] (Institut Universitaire de Pathologie[1], Pediatric Neurologist[2], Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland)

11. Whipple's disease

J. Ferreira (Department of Pathology, Maisonneuve-Rosemont Hospital, University of Montreal, Montreal, QC)

12. HTLV-1 associated myelopathy

I.D. Uphoff (Department of Pathology, Hartford Hospital, Hartford, Connecticut)

13. West Nile viral myeliltis

W. Halliday (Division of Neuropathology, Toronto Western Hospital, UHN, Toronto, ON)