

33rd ANNUAL B.C. NEUROSCIENCE ACADEMIC DAY
Friday March 19, 2010
Paetzold Centre, Vancouver General Hospital

Registration:

8:30 – 9:00 (coffee)

Morning Session: Experts in their Field

Moderator: Chris Honey

1. 9:00 – 9:30 “Advances in developmental neurobiology”
Tim O’Conner,
UBC, Brain Research Centre

2. 9:30 – 10:00 “Advances in the molecular diagnostics of brain tumours”
Steven Yip, MD, PhD, FRCP
Consultant Pathologist, BCCA

3. 10:00 – 10:30 “Advances in our understanding of brain neuronal growth
and Autism” Kurt Haas, PhD
UBC, Brain Research Centre

- 10:30 – 10:45 Coffee

4. 10:45 – 11:45 **Dr. P.D. MOYES LECTURE**
“Minocycline for Acute Spinal Cord Injury:
A Pilot Study”
Professor John Hurlbert
University of Calgary

5. 11:45– 11:55 Update on Korle-bu Neuroscience Centre initiative
Dr. J.S. Lapointe

- 12:00 – 13:15 **BUFFET LUNCHEON**
at Medical Student Alumnae Centre with
B.C. Neurological and Neurosurgical Nurses Association.

5. 13:15 – 14:15 **5th annual Ken Berry Clinical Pathological Conference**
Presenter: Dr. M. McKeown
Moderator: Dr. K. Dorovini-Zis

*****see case history below*****

Afternoon Session: “Resident Presentations”

6. 14:30 – 14:45 “The Learning Curve for Endoscopic Transnasal Transsphenoidal Resection of Pituitary Tumours” Dr. Tamir Ailon
7. 14:45 – 15:00 “Subtotal Resection of Vestibular Schwannomas:
A retrospective review of one centre's experience” Dr. Albert Tu
8. 15:00 – 15:15 “Acoustic Neuroma Resection after Stereotactic Radiotherapy”
Dr. Chris Gillis
9. 15:15 – 15:30 “A Descriptive Analysis of Prognostic Indicators in Patients with
Non-Convulsive Status Epilepticus in a Tertiary Hospital
Population.” Dr. Chantelle Hrazdil
10. 15:30 – 15:45 “A Validation Study of the Diagnostic Accuracy of the
Revised 2008 Consensus Criteria for the Diagnosis of
MSA-C in British Columbia
Dr. Kristin Jack
11. 15:45 – 16:00 “Electrocorticography and Seizure Outcomes in Children with
Lesional Epilepsy.” Dr. Jennifer Gelinias

16:00 ADJOURNMENT

BC NEUROSCIENCE DAY
March 19, 2010

KEN BERRY CLINICOPATHOLOGICAL CONFERENCE

Moderator: Dr. K. Dorovini-Zis. Presenter: Dr. M. McKeown

A 67-year-old man was admitted to the hospital for further evaluation of rapidly progressive cognitive decline.

The patient was in a stable state of health until approximately 3.5 months earlier, when he began to experience gradual decline in his memory. Prior to that he was fully independent and had been functioning at a perfectly normal level. According to his wife, he was initially becoming forgetful, mixing up and unable to recall events or conversations that had happened earlier in the day, had problems recalling the names of his grandchildren and was forgetting important appointments, such as doctor's appointments. His memory problems became gradually worse to the extent that he could not be left unattended for fear that he might have an accident or get lost, since he was getting unfamiliar with his surroundings. He developed problems performing daily tasks, such as dressing himself, buttoning, shaving or combing his hair. He often appeared tired, confused and lethargic. His wife noted problems with his language, such as mixing words and having problems explaining or describing the things that he usually did, to the extent that his sentences were not understood properly by his family. He lost his interest in doing crossword puzzles or playing games that he usually did with his family and he eventually quit reading newspapers and doing his hobbies. He became unable to do his finances and his wife had to take over. In addition, his wife noted that for the last month he had weakness and numbness in the left hand and she also reported that he had transient left leg weakness of 4 days duration 3 months earlier. His sleep was poor at times. He seemed depressed, had occasional crying episodes and was getting frustrated with his condition. His behavior was not disinhibited or aggressive. He had no headaches, fever, seizures, vomiting, decreased appetite, weight loss, skin rash, joint pains or ulcers.

He entered another hospital, where examination showed that he was markedly inattentive, confused and drowsy. He did not appear particularly ill or in distress. He knew he was in a hospital, but did not know the day, month or year. He could not remember any medical tests that were performed earlier in the day. He had trouble following commands and he could not cooperate with the visual field examination. He was unable to spell the word "world" or follow simple one step commands and could not remember from one minute to the next. He had no difficulty naming objects shown to him. His speech was mildly dysarthric, but not dysphasic. He had a mild left pronator drift and mild asterixis. The left arm and left knee reflexes were mildly increased. There was a suggestion of mildly increased tone in the left arm that tended to be slightly flexed when he held it out. The gait was satisfactory and plantar responses were downgoing. Sensory examination and formal assessment of strength could not be performed since the patient was not cooperating. An EEG showed normal background activity with an alpha rhythm of 9 cycles/sec, but there was much superimposed episodic slowing. Frequent runs of very

high amplitude 1.5-2 cycles/sec delta activity were present. These bursts did show good symmetry and cycling between hemispheres. A cranial CT scan showed mild atrophic changes and no other abnormalities. An MRI showed extensive confluent signal abnormalities within the left greater than right centrum semiovale extending to the subcortical U fibers in both frontal lobes, left parietal lobe, left posterior temporal/occipital region and inferior cerebellar hemispheric white matter extending on the right to the brachium pontis. A 6mm high T2/PD signal intensity lesion was present in the right thalamus. There was mild generalized cortical atrophy with concordant ventricular enlargement. CSF examination showed protein of 0.38 mg/L and glucose 4.4mg/L. He was referred to this hospital.

The patient worked as a truck driver and had retired two years before his present illness. He is married with 3 healthy children. He has 4 siblings. One sister died of lung cancer and another one has multiple sclerosis. One brother and another sister are well. There was no family history of a similar neurological condition. The patient smoked 1 pack of cigarettes per day. Past medical history included hypertension, dyslipidemia, ischemic heart disease, mild myocardial infarction, chronic obstructive lung disease, chronic renal insufficiency and hypothyroidism. There was no HIV risk. His medications included lasix, hydralazine, metoprolol, eltroxin, ramipril, lipitor, lopressor, thyroxin, pantoprazole and aspirin.

The patient was afebrile, the pulse was 55 and the blood pressure 190/95mm Hg. His chest, cardiovascular and abdominal examinations were normal. On neurological examination he seemed drowsy, but rousable, inattentive and in no distress. He was aware of being in a hospital and was oriented to the year, but not to province, month or day. Language was fluent with intact naming and writing simple sentences. He was unable to read his own writing. Comprehension was intact for one step commands and repetition was normal. He had left-to-right confusion and acalculia, but not finger agnosia. Memory examination showed a 3/3 recall, but it was short. He had only 1/3 long-term memory. There was no apraxia, agnosia or neglect. He had visual spatial difficulties on drawing a clock and copying a cube. The pupils were equal and reactive to light bilaterally and extraocular movements were full. Fundoscopy was normal and there were no field defects. There was no facial asymmetry and sensation over the face was normal. The tongue was not deviated. Lower cranial nerve examination was normal. Motor examination showed a spastic catch on the left arm, decreased tone in the left upper and lower extremities and asterixis on the left side, but no drift. Power was full (5/5) in all extremities. There was no neck rigidity. Reflexes were symmetrical and brisk. A positive Babinski sign was present on the left. Sensory examination was normal with the exception of decreased vibration at the toes bilaterally. Gait and coordination were within normal limits.

Laboratory investigations showed normal hemoglobin, hematocrit, WBC and platelet counts. The urea was 8.6mmol/L (n=2-8.2), Creatinine 161 μ mol/L (n=60-115). PTT, INR, Na, K, serum folate and B12 levels and liver function tests were normal. Antithyroid peroxidase Ab was slightly elevated to 81U/ml (n=0-60). Vasculitis studies (ANCA, ANA, ENA) were negative. CSF examination showed glucose 3.3 mg/L,

protein 465 mg/L, 3 WBC (mostly lymphocytes) and an IgG synthesis rate of 10.4. Serology and CSF analysis for *Borrelia Burgdorferi*, as well as CSF cytology were negative.

An MRI showed prominent areas of high density in the cerebellar white matter bilaterally and in the deep white matter of both hemispheres, particularly the frontal lobes with a subcortical distribution. A small focus was present in the right thalamus. The gradient sequence demonstrated some low intensity in the putamen and external capsule bilaterally. These lesions did not show findings of acute stroke on the diffusion sequence. On post-gadolinium sequences these areas enhanced irregularly. An EEG was attempted, but was not completed as the patient became agitated in between lucid periods. A CT of the abdomen and pelvis showed no evidence of intra-abdominal malignancy. A chest CT showed multiple non-calcified bilateral pulmonary nodules measuring up to 9mm in diameter, which were indeterminate and left apical pleural parenchymal calcifications consistent with previous granulomatous exposure (TB). There was no evidence of primary neoplasm. On the 10th hospital day a brain biopsy was performed. The patient remained disoriented. He was put on a trial of high dose prednisone (60mg qd) and was transferred to the referring hospital 9 days later.

Seven months later he was transferred to an extended care unit. His prednisone was tapered to 30mg o.d. His condition remained stable until 5 months later when he was admitted to hospital with cardiac failure, increasing shortness of breath and lethargy. On examination he had a diffuse macular erythematous rash on his trunk and extremities, which blanched on pressure. A CT scan of the chest showed findings consistent with emphysema, interstitial lung disease and bronchiectasis. An echocardiogram showed an ejection fraction in the range of 40-50%. His condition continued to deteriorate despite medical treatment and he died 3 weeks later, 17 months from the onset of his illness.