Case History

HPI: A 73 year old white man with a history of chronic lymphocytic leukemia and Parkinson's disease presented from an outside hospital for further evaluation of mental status decline for the past few months. Per his family, he had been having worsening right sided weakness, difficulty with coordination, word finding, and overall thinking.

Four months prior to his presentation, he was in his usual state of health including playing golf frequently. Two months ago, he became lethargic and had difficulty performing his independent activity of daily living. He experienced memory lapses and episodes of confusion, such as not knowing how to use his computer and forgetting his girlfriend's phone number. He was evaluated by his Neurologist for Parkinson's disease and had some medications adjusted. Around this time, he developed blurry vision.

A week later, his daughter noted that he had a new right facial droop. He was hospitalized and was found to have evidence of prior strokes but no recent stroke. He was readmitted for dizziness and confusion. His symptoms were thought to be due to his Parkinson's medications, which were stopped. Two weeks later, he experienced progressive right sided weakness, mental slowing, and slurred speech. On presentation to VCU, he was somnolent and history was obtained from his family. He had a 15 pounds weight loss over a 2 week period and difficulty walking. He had no fevers, chills, night sweats, seizures, headaches, other focal weakness, urinary symptoms, shortness of breath, or dysphagia.

Past Medical/Surgical History:

- Chronic lymphocytic leukemia received 6 cycles of Rituxan and Fludara. Most recent bone marrow biopsy three months ago revealed hypercellularity and increase from 38% to 46% abnormal cells
- Prostate cancer diagnosed 25 years ago s/p prostatectomy
- Basal cell cancer underwent excision
- Coronary artery disease with a stent to right coronary artery in 2003
- Parkinson's disease diagnosed 1 year prior
- Hypertension
- Hypothyroidism

Family History:

Unable to obtain due to altered mental status

Social History:

- Lives in Chester, Virginia
- Does not have pets. Has not travelled recently
- Retired from mining work on soda ash
- No exposures to ill contacts
- No history of alcohol, tobacco, or drugs

Medications:

Allergies:

Sulfa drugs

❖ Metoprolol 12.5 mg every 12 hours

Levothyroxine 50 mcg daily

Plavix 75 mg daily

❖ Aspirin 81 mg daily

❖ Valsartan 80 mg daily

Review of Systems:

A complete ROS was performed and was negative except as above.

Physical Exam

Vital Signs: BP 138/76, HR 89/min, RR 18/min, Temp 36.1,

Pox 100% on RA

General: Overweight man in no apparent distress. Lethargic but

arousable. Follows simple commands. Mumbles

unintelligible words

HEENT: PERRLA, right visual field neglect, no nystagmus, sclera

anicteric, moist mucous membranes, oropharynx clear and without lesions, right face flattening. Fundoscopic exam with clear optic disc margins and no AV nicking, cotton wool

spots, or Roth spots.

Neck: Supple, no carotid bruits, no JVD, no lymphadenopathy

Lymph: No cervical, axillary, or inguinal lymphadenopathy

CV: Regular rhythm, constant S1/S2; II/VI systolic murmur at the

left lower sternal border. PMI non-displaced, normal in size

Lungs: Full and symmetric excursion, clear breath sounds bilaterally Abdomen: Soft, nondistended, normoactive bowel sounds, nontender.

Ext: No edema, 2+ distal pulses bilaterally

Neuro: Lethargic, oriented to person. CN III-XII with right facial

flattening and right visual field neglect but otherwise intact. Strength is 5/5 in LUE, 4/5 in LLE, and 1/5 in RUE and RLE. DTRs symmetric but 1+ in all extremities. Left extremities with response to painful stimuli. Unable to perform cerebellar testing. Downgoing babinski on the left and

upgoing on the right. No clonus. Cog wheel rigidity in LUE. Skin: No rashes, erythema, ecchymoses, purpurae, or petechiae

Laboratory Data and Imaging

Sodium 139 mmol/L, Potassium 4.6 mmol/L, Chloride 111 mmol/L, Bicarbonate 24 mmol/L, BUN 25 mg/dL, Cr 0.96 mg/dL, Glucose 121 mg/dL, Calcium 9.1 mg/dL

AST 32 units/L, ALT 32 units/L, ALP 86 units/L, Total bilirubin 0.5 mg/dL, Conjugated bilirubin 0.2 mg/dL, Albumin 3.8 gm/dL

WBC 8.1 x10 9 /L , Hemoglobin 11.3 g/dL, Mean Corpuscular Volume 91.0 fL, Platelets 76 x 10 9 /L

Differential: 23% Neutrophils, 73% Lymphocytes, 0.5% Monocytes, 1.3%

Eosinophils, 2.1% Basophils

PT: 10.8, PTT: 28, INR: 1.1

TSH: 1.39 uIU/mL Ammonia: 23 umol/L Vitamin B12: 1136 pg/mL ESR: 29 mm in 1 hr CRP: <0.3 mg/dL HIV: negative RPR: Nonreactive

Crypto Ag: negative

Histo Ag: negative CSF studies from outside hospital: 2 WBC, 3 RBC, no malignant cells

Blood cultures: no growth Fungus cultures: no growth AFB culture: no growth

CXR: Trace interstitial edema. No consolidation, lymphadenopathy, or

masses

CT Head without contrast: Multifocal edema in the right parietal and occipital lobes, splenium of the corpus callosum, and most prominent region in the left frontoparietal lobe. No midline shift or evidence of herniation. Focal hypodensities in the left thalamus and left vertebral peduncle. Mucosal thickening in the right maxillary sinus.

MRI Head with and without contrast:

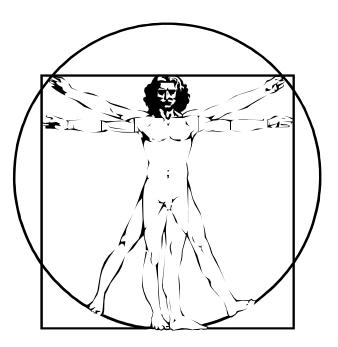
- ❖ Large area of T2 hyperintensity in the left frontoparietal lobe, mainly involving the white matter. It extends and involves the posterior aspect of the corpus callosum mainly the splenium. There is mild diffusion restriction and minimal leptomeningeal enhancement in the frontoparietal convexity.
- ❖ Other areas of abnormal T2 hyperintensity in the right parietal and occipital cortex with cortical thinning,
- T2 hyperintense focus in the left thalamus and left cerebral peduncle.

EEG: Moderate generalized slowing with intermittent delta activity suggestive of bihemispheric dysfunction. Mild asymmetry with better developed rhythms of the right, indicating left hemispheric structural abnormality.

A definitive test was performed that revealed the diagnosis.

Department of Internal Medicine Clinicopathologic Conference

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73 year old man with progressive mental status decline and white matter brain lesions.