

AANP Slide Session 1971

CASE 7

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This 56-year-old woman was a known alcoholic with documented nutritional cirrhosis and portal hypertension. Recently she had an increase in lethargy, speech difficulty, and a right hemiparesis. On admission the patient was disoriented, lethargic, unable to adduct the left eye, had a droop of the right labial fold, increased flexor tone of the right elbow and knee with right-sided weakness, and a positive Babinski on the right, negative on the left. The blood ammonia was slightly elevated, spinal fluid contained a "few" neutrophils and the protein was (95 mg%). No evidence of AFB or fungi. CSF sugar was 100 mg%, simultaneous blood sugar 124 mg%. There were 175 red cells and 6 white cells. Hematologic Data: Admission hemoglobin was 11.7, Hct, 35.0, RBC 3.28. Reticulocytes 4.6% (range from 2.0-4.6% during hospitalization). The smear showed numerous macrocytes, toxic granulation of lymphocytes, and markedly reduced platelets. Admission platelet count was 22,000 (reaching a maximum of 55,000 during hospitalization then dropping to a minimum of 3,000 just prior to death). WBC- 11,700 on admission (73% PMN's, 8% lymphs, 19% monocytes, no immature forms). This ranged between 8,000 to 15,000 during hospitalization. Bone marrow biopsy was interpreted as hypercellular, somewhat megaloblastic, with numerous megakaryocytes. Coagulation work-up showed, in addition to thrombocytopenia, a prolonged prothrombin time and PTT, increased fibrinogen split products, and numerous "helmetcells" on the smear consistent with a consumptive coagulopathy. The patient was on a hepatic coma regime, her arterial ammonia fell to normal range, the neurologic status continued to deteriorate. She was disoriented with very little response to painful stimuli over the entire body. She had nuchal rigidity, right hemiparesis, greater in the arm than in the leg, and hyperreflexia greater on the right than on the left. The brain scan was interpreted as normal. A left carotid angiogram showed no evidence of a space-occupying intracranial lesion. Hepatic dysfunction was never pronounced; bilirubin never exceeded 2.5 total, enzymes were not elevated, and ascites was minimal or absent. She had periodic evidence of GI bleeding and received several transfusions. Her thrombocytopenia persisted but she was not heparinized, her coma deepened, she developed acidosis and expired.

At autopsy were found bilateral adrenal hemorrhages, multiple cerebral hemorrhages. Spleen was mushy and liver cirrhotic.

Submitted are: 1 stained and 1 unstained section.