CASE 3

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This 47 year old female was in good health until the past year when she developed a bright red rash over the extremities and trunk associated with chills and fever. A diagnosis of scarletina was made but she failed to improve on antibiotic therapy. She was hospitalized at another hospital with fever, enlargement of the spleen and severe anemia (hematocrit 11%). Both direct and indirect Coombs' tests were positive. There was inversion of the A/G ratio and the urine was positive for Bence-Jones protein. Bone marrow showed erythroid hyperplasia. The blood could not be typed for transfusion. On one attempt at transfusion she suffered a severe reaction. She developed enlarged lymph nodes but no specific diagnosis was established on biopsy. Two months after admission to the first hospital splenectomy was performed. Study of lymph nodes and spleen suggested Hodgekin's disease. Her mental status deteriorated, with light stupor and hallucinations. Prior to her admission to the University Hospital, the skin rash again appeared and she was febrile. At this time there was enlargement of axillary and cervical lymph nodes. Laboratory studies showed Hgb 9.7 gms; hematocrit 34%; reticulocyte count 9%, 2.8%. Platelets 904,000. Serum glob. 3.8%, alb. 3.2%. Serum electrophoresis albumin 41.5%. Alpha$_1$ globulin 5.3%, alpha$_2$ globulin 18.1%, beta globulin 16.9%, gamma globulin 18.2%. Coombs test was negative on one occasion but later the indirect reaction was 3 +. L.E. preparations were negative. Physical examination on admission showed a thin female with pallor of skin and mucus membranes and a temperature of 100.4°. There were slight telangectasis, ecchymoses and petechiae of the arms and lower legs. The liver was not enlarged. She was lethargic, emotionally labile, depressed, with memory loss. Neurological examination showed no cranial nerve abnormality. There was generalized proximal and distal atrophy of the extremities with bilateral symmetrical weakness. The patient developed seizures shortly after admission, became more lethargic and developed cog-wheel rigidity. She deteriorated mentally and developed hallucinations. She became comatose and died 14 days after admission.

Significant laboratory studies during this admission: Negative test for cryoglobulins. P.P.D. - negative for histoplasmin and blastomycin. Spinal fluid studies: Pressure normal; protein 96-238; glucose 53-70; cells 0. EEG: Diffuse encephalopathy.

Pertinent general pathology: Widespread, scattered acute necrotic foci in liver, heart, lung, esophagus and adrenal. Toxoplasma present in these foci in all organs except liver. Candida also present in lung sections. Increased plasma cells in lymph nodes and bone marrow.

Brain: Diffuse, irregular areas of hemorrhagic softening most with greatest involvement in the cerebellum.

Sections: H & E

Diagnosis: Dysproteinemic disorder. Disseminated toxoplasmosis. Cerebral toxoplasmosis, angitis, vascular necrosis, progressive reactive encephalitis.

Points for discussion: Is incidence of adult toxoplasmosis increasing? Why? Explanation for more extensive involvement of brain than viscera? Does presence of vascular necrosis suggest sensitization factor? Is the reaction of the brain related to release of organisms from encystment? Significance of absence of meningeal reaction