CASE 1

The diagnosis was Hereditary Adult-Onset Leukodystrophy. The condition is considered by Seitelberger as a form of adult onset Pelizaeus-Merzbacher disease. Histologic similarities to Canavan's disease were mentioned by several observers.


CASE 2

There was no definite diagnosis. The material in the astrocytes showed some similarity to Rosenthal fibers but there were significant differences. A relationship to Alexander's disease was postulated.


CASE 3

The diagnosis was melanotic schwannoma. The tumor is of uncertain malignant potential.

CASE 4

The diagnosis was malignant schwannoma with rhabdomyosarcomatous differentiation. The patient has neurofibromatosis.


CASE 5

The inclusions observed in this case of metastatic adenocarcinoma are identical to the inclusions originally described by Russell as "organisms" which he thought were fungi. The term "Russell bodies" was subsequently used to describe gamma globulin deposits in the cytoplasm of plasma cells. By EM, true Russell bodies are heterogeneous consisting of intracytoplasmic lumina, cytoplasmic nuclear inclusions, apoptotic bodies, and large nucleoli.


CASE 6

The biopsy was obtained in 1982. The patient was given only antiepileptic medications. Her seizures are under control and 7 years later she is functioning in a managerial capacity. The diagnosis was chronic encephalitis. The plasma cells were polyclonal and contained "Russell bodies." No viral particles were demonstrated on EM.

CASE 7

The diagnosis was primary granulomatous angiitis with multiple small infarcts, and secondary acute zygomycotic vasculitis with ruptured mycotic aneurysm.


CASE 8

The diagnosis was systemic lymphoproliferative disorder with focal herpes encephalitis due to varicella zoster.


CASE 9

The diagnosis was rhabdomyositis and cardiomyositis. Although structures resembling myxovirus were observed on EM, the viral culture was negative and a definite viral etiology could not be established. The possibility of this being an autoimmune disease was raised.


CASE 10

The diagnosis of the presenter was postictal cerebral and cerebellar hemiatrophy: developing as a result of the release of cytotoxic neurotransmitters from the epileptogenic tissue. The CT scans clearly demonstrated that the hemiatrophy was acquired not congenital. The presenter further stated that the sparing of the corpus striatum argued against an ischemic or vascular basis for the destructive lesions in the right hemisphere.
