

26th Annual DSS - 1985

Case 1

*GAN's
Thanks*

The diagnosis was Neurothekeoma. This tumor is of nerve sheath origin but is not associated with von Recklinghausen's disease. The cells are S-100 negative.

1. Gallagher RL, Helwig EB. Neurothekeoma - a benign cutaneous origin. Am J Clin Pathol 1980;759-764.
2. Enzinger FM, Weiss SW. Soft tissue tumors. 1983; p 615, C.V. Mosby Co.

CASE 2

The diagnosis was Pick's Disease, electron probe analysis showed no evidence of mercury.

CASE 3

The diagnosis was transverse myelitis due to Schistosoma mansoni.

1. Case records of the Massachusetts General Hospital. New Eng J Med 1985; 312:1376-1383.
2. Acute Schistosomiasis with transverse myelitis in American students returning from Kenya. JAMA 1984; 252:1116-1123.
3. Marcial-Rojas RA, Fiol RE. Neurologic complications of Schistosomiasis. Ann Int Med 1963; 59:215-230.

CASE 4

The diagnosis was Sparganosis due to Spirometra.

1. Anders K, et al. Intracranial sparganosis: an uncommon infection. J Neurosurg 1984; 60:1282-1286.

CASE 5

The diagnosis was Whipple's disease. At autopsy, the lesions were confined to the brain.

1. Johnson L, Diamond I. Cerebral Whipple's disease: Diagnosis by brain biopsy. Am J Clin Path 1980; 74:486-490.
2. Wilbert SW, et al. Whipple's disease of the central nervous system. Acta Neuropath 1976; 15:31-38.
3. Schochet SS Jr, Lampert PW. Granulomatous encephalitis in Whipple's encephalitis. Electron microscopic observations. Acta Neuropath 1969; 13:1-11.

CASE 6

The diagnosis was free-living amoebic infection. The organism was isolated in tissue culture in which it was cytotoxic. Injection intraventricularly, intraperitoneally, and intranasally in immunosuppressed mice resulted in the death of most animals. There were no histopathologic changes but tissue cultures from these brains were positive. Immunoperoxidase stains with antibodies directed against various genera of amoeba confirmed that the organism belongs to the Acanthamoeba genus; however, the species is yet to be identified. Some observers thought that this was a dual infection.

1. Martinez AJ. Acanthamoebiasis and immunosuppression. J Neuropath Exp Neurol 1982; 41:548-557.
2. Lam AH, et al. Primary amoebic (Naegleria) meningo-encephalitis. J Comput Assist Tomogr 1982; 6:620-623.
3. Visvesvara GS, et al. Isolation, identification and biological characterization of Acanthamoeba polyphaga from a human eye. Am J Trop Med Hyg 1975; 24:784-790.

CASE 7

Because of the absence of inflammatory changes this was not thought to be a form of allergic encephalomyelitis. A diagnosis of nonspecific toxic reaction was made.

CASE 8

There was no general agreement for a diagnosis. Some commentators felt that this was a combination of congenital malformations with a superimposed destructive process which could have been due to an intrauterine infection or ischemia. The presenters favored a congenital infection.

CASE 9

The diagnosis was primary leptomeningeal myxoma.

CASE 10

The diagnosis was ethylene glycol intoxication (oxalosis). The serum calcium fell from 9.5 to 7.9 mg%. A half full can of antifreeze was found in her home.

CASE 11

The diagnosis was methanol intoxication.

1. Sharpe JA, et al. Methanol optic neuropathy. Neurol 1982; 32:1093.
2. McLean DR, et al. Methanol poisoning: A clinical and pathological study. Ann Neurol 1980; 8:161-167.