Am Association Neuropath
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31ST ANNUAL DIAGNOSTIC SLIDE SESSION ← File

CASE 1

The PAS and silver methenamine stains revealed a fungal organism intermediate in morphology between Aspergillus and Candida. The diagnosis was granulomatous meningitis due to Trichosporon.

Ness MJ, Markin RS, Wood RP, et al. Disseminated Trichosporon beigellii infection after orthotopic liver transplatation. Am J Clin Pathol 1989; 92:119-123

Oto T, Ishikawa Y, Fujii R, et al. Disseminated Trichosporon capitatum infection in a patient with acute leukemia. Cancer 1988; 61:585-588

Watson KC, Kallichurum S. Brain abscess due to Trichosporon cutaneum. J Med Microbiol 1970; 3:191-193

CASE 2

The intravascular tumor cells stained postively for leukocyte common antigen and B cell lymphocyte markers. It was classified as a large cell lymphoma. The diagnosis was systemic angiotropic lymphoma.

Kayano H, Katayama I. Primary hepatic lymphoma presenting as intravascular lymphomatosis. Arch Pathol Lab Med 1990; 114:580-584

Vital C, Heraud A, Vital A, et al. Acute mononeuropathy with angiotropic lymphoma. Acta Neuropathol 1989; 78:105-107

CASE 3

The tumor cells were negative for factor 8, the T cell marker Leu 4, and kappa and lambda light chains. They stained positively with B-cell markers and the adhesion molecule LFA-1. The disease was confined to the brain. The diagnosis was cerebral angiotropic lymphoma.

Russien FF, de Rijk D, Bikker A, Roos E. Involvment of LFA-1 in lymphoma invasion and metastasis demonstrated with LFA-1 deficient mutants. J. Cell Biol 1989; 108:1979-85

Ferry JA, Harris HL, Picker LJ, et al. Intravascular lymphomatosis (malignant angioendothelialiomatosis). A B-cell neoplasm expressing surface homing receptors. Mod Pathol 1988; 1:444-452

Doman CL. Immunohistochemistry of so called "neoplastic angioendotheliosis". Acta Neuropathol 1986;72:197-199

CASE 4

The bone marrow was histologically identical to the dural lesion and lesions were also found in the liver and ureter. The large cells stained postively for factor 8 and the platelet specific glycoprotein 3A and are megakariocytes. The diagnosis was meningeal myeloid metaplasia. The gross photograph was not included.

Landolfi R, Colosimo C Jr, DeCandia E, et al. Meningeal hematopoiesis causing exophthalmus and hemiparesis in myelofibrosis: effect of radiotherapy. Cancer 1988; 62:2346-9

Brown JA, Gomez-Leon G. Subdural hemorrhage secondary to extramedullary hematopoiesis in post polycythemic myeloid metaplasia. Neurosurgery 1984;14:588-591

Ligumski M, Polliack A, Benbasset J. Myeloid metaplasia of the CNS in patients with myelofibrosis and agnogenic myeloid metaplasia: a report of 3 cases and review of the literature. Am J Med Sci 1978; 275:99-103

CASE 5

Terminally the WBC was 380,000 with 40% blast forms. Leukemic infiltrates were found in lymph nodes, liver, spleen, and retroperitoneal fat. Chloracetate esterase stained the blastic precursers of the myeloid series in the brain. The large cells were negative but stained positively with factor 8. Groups of smaller cells showed surface staining with ulex. These were thought to be foci of erythroid differentiation in the tumor. The diagnosis was atypical myeloblastic syndrome with blastic transformation.

Adachi M, Ryo R, Yoshida A, et al. Refractory anemia terminating in acute megakaryoblastic leukemia (M7). Acta Haematol 1989;81:104-108

San Miguel J, Gonzalez M, Canizo M, et al. Leukemias with megakaryoblastic involvement: clinical, hematological, and immunologic characteristics. Blood 1988; 72:402-407

Dekker A, Elderson A, Pont K, Sixam J. Menigeal involvement in patients with acute nonlymphocytic leukemia. Incidence, management, and predictive factors. Cancer 1985; 56:2078-82

CASE 6

Focal areas in the tumor stained positively for reticulum. The tumor was strongly glial fibrillary protein positive and cytokeritin negative. The diagnosis was epitheloid glioblastoma multiforme.

Mork SJ, Rubinstein LJ, Kepes JJ, et al. Patterns of epithelial metaplasia in maliganant gliomas II. Squamous differentiation of epithelial-like formations in gliosarcomas and glioblastomas. J Neuropathol Exper Neurol 1988;47:101-118.

Galloway PG, Roessmann U. Anaplastic astrocytoma mimicking metastatic carcinoma. Am J Surg Pathol 1986;10:728-732.

Kepes JJ, Fulling KH, Garcia JH. The clinical significance of "adenoid" formations of neoplastic astrocytes, imitating metastatic carsinoma in gliosarcoma. A review of five cases. Clin Neuropathol 1982;1:139-150.

CASE 7

The hypertropic neurons resembled those seen in Pick's disease and were strongly argentophilic. Ultrastructural and marker studies showed that these neurons contained excessive accumulations of cytoskeletal intermediate filaments. The diagnosis was radiation encephalopathy with neuronal giantism. This case has been published.

Caccamo D, Herman MM, Rich H, Rubinstein LJ. Focal neuronal giantism and cerebral cortical thickening after therapeutic irradiation of the CNS. Arch Pathol Lab Med 1989; 113:880-885.

CASE 8

A MRI 14 months prior to death showed a normal basilar artery. Two weeks prior to death an aneurysm was seen. At autopsy the systemic vessels were grossly normal. It was concluded that the rupture of the aneurysm was the result of congenital weakness of the vessel wall. The diagnosis was rupture of radiation-induced aneurysm in the setting of Ehlers-Danlos Syndrome, type 3.

Benson PJ, Sung JH. Cerebral aneurysms following radiotherapy for medulloblastoma. J Neurosurg 1989;70:545-550

Gomori JM, Levy P, Weshler Z. Radiation-induced aneurysm of the basilar artery. Angiology 1987;38:147-150

McCready RA, Hyde GL, Bivins BA, et al. Radiation-induced arterial injuries. Surgery 1983;93:306-312

CASE 9

No histologic changes were demonstrated in voluntary muscle. Electron microscopy showed mitochondrial hypertrophy in neurons. The diangosis was Leigh's disease.

Kretzschmar HA, DeArmond SJ, Koch TK. Pyruvate dehydrogenase complex deficiency as a cause of subacute necrotizing encephalopathy (Leigh disease). Pediatr 1987;79:370-373

DiMauro S, Serenella S, Zeviani M, et al. Cytochrome c oxidase deficiency in Leigh syndrome. Ann Neurol 1987;498-506

Leigh D. Schacute necrotizing encephalomyelopathy in an infant. J Neurol Neurosurg Psychiat 1951;14:216-221

CASE 10

The microvesicular hepatic fatty metamorphesis associated with gluteric acidemia was not found, nor were myelin vacuolar changes seen. The tail of the caudate was present but showed gliosis. The cerebellum was normal. The diagnosis was infantile bilateral striatal necrosis.

Friede RL. Developmental Neuropathology. New York:Springer Verlag, pp 110, 111, 490-509, 1989

Bergman I, Finegold D, Gartner Jr. JC, et al. Acute profound dystonia in patients with glutaric acidemia. Pediatrics 1989;83:228-234.

Chow CW, Haan EA, Goodman SI, et al. Neuropathology in glutaric acidemia type I. Acta Neuropathol 1988;590-594

Mito T, Tanaka T, Becker LE, et al. Infantile bilateral striatal necrosis. Clinicopathological classification. Arch Neurol 1986; 43:677-680

CASE 11

No paracrystaline inclusions were seen on electron microscopy. The atropic fibers were type I. The PAS was normal. Biochemical analysis of the muscle revealed a low carnitine level. The diagnosis was lipid storage myopathy with carnitine deficiency secondary to valproic acid therapy.

Cotariu D, Zaidman JL. Valproic acid and the liver. Clin Chem 1988;34:890-897.

Engel AG. Carnitine deficiency syndromes and lipid storage myopathies. In: Engel AG, Banker BQ, eds. Myology, Basic and Clinical. New York: McGraw-Hill, 1986:chapter 57.

Murphy JV, Marquardt KM, Shung AO. Valproic acid asociated abnormalities of carnitine metabolism. Lancet 1985;1:820-821.

Ohtani Y, Endo F, Matsuda I. Carnitine deficiency and hyperammonemia associated with valproic acid therapy. J Ped 1982;101:782-785