Primary tumors in the neurohypophysis should be differentiated both from tumors that occur in adjacent structures and infiltrate into the neurohypophysis and from metastases. The small size of the tumor and its confinement to the neurohypophysis ruled out tumor origination in surrounding tissues. Meningioma was ruled out by positive staining for GFAP and S100. No other tumors were observed in the animal, making a metastatic tumor unlikely.

Two varieties of primary neurohypophyseal tumors are recognized: astrocytomas and granular cell tumors. Astrocytomas arise from pituicytes, the neurohypophyseal glial cells, which are considered astrocytes by most investigators. Astrocytomas from the neurohypophysis are also known as pituicytomas. Granular cell tumors are characterized by plump oval cells with periodic acid–Schiff-positive cytoplasmic granules. These cells may be modified astrocytes, but their exact lineage is still a matter of debate.

This tumor was diagnosed as an astrocytoma, based on histologic findings and positive staining for GFAP. Positive staining for GFAP, vimentin, and S100, as seen in this tumor, is commonly seen in human astrocytomas. The variation in staining intensity among individual tumor cells indicates differences in the quantity of intermediate filaments and S100 protein and may reflect the degree of differentiation of the tumor cells. An inverse relationship between the malignancy of glial cell tumors and the level of GFAP expression has been suggested.

Well-documented cases of primary astrocytomas in the neurohypophysis have been reported in human beings, rats, and a Siamese cat. These tumors may cause clinical symptoms by compression or invasion of adjacent tissues. No evidence of clinical disease related to the tumor was observed in this rhesus monkey. This is the first report of such a tumor in a nonhuman primate.

Acknowledgement

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References


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Brief Communications and Case Reports

Fig. 1. Ventral midbrain. Note the prominent perivascular cuffs, microglial nodules, and a diffuse increase in microglia. HE. Bar = 100 μm.

Fig. 2. Ventral midbrain. There is a small perivascular cuff (lower right), an ill-defined microglial nodule (arrow), and a varied, diffuse increase in microglia. HE. Bar = 25 μm.

trophils, 3,978 lymphocytes, 234 monocytes, and no eosinophils or basophils per microliter. Elevated serum aspartate aminotransferase (1,414 IU/liter) with normal serum alkaline phosphatase and serum gamma glutamyl transferase was attributed to recumbency. Other values from a 16-chemistry panel were within normal limits. Blood lead levels were not detectable.

The cow was euthanatized and necropsied. No gross lesions were found. Histologic lesions were limited to the central nervous system. Sections examined included temporal cerebral cortex, hippocampus, occipital cerebral cortex, ventral midbrain at the level of the rostral colliculus, left trigeminal ganglion, pons and cerebellar hemisphere at the level of the cerebellar peduncles, and spinal cord at the level of the second cervical vertebra. Lesions were similar in nature but most severe in the ventral midbrain and pons, mild to moderate in the cerebral cortex and hippocampus, and minimal in the spinal cord.

Lesions in the ventral midbrain (Figs. 1, 2) and pons included severe lymphohistiocytic perivascular cuffing, moderate regionally diffuse rarefaction of the neuropil, scattered microglial nodules, and rare neuronophagia. Mild focal accumulations of lymphocytes in the meninges were noted adjacent to areas with the most severe involvement. Neutrophils were present in very low numbers and were only found within the most prominent perivascular cuffs. The hippocampus contained scattered microglial nodules and neuronophagia, mild diffuse microglia, and variable lymphohistiocytic perivascular cuffs. Sections of temporal and occipital cerebral cortex contained scattered microglial nodules, mild diffuse microglia, and rare neuronophagia. Perivascular cuffing was moderate in the temporal cortex and mild in the occipital cortex and adjacent white matter. The cervical spinal cord changes were limited to mild perivascular cuffing and minimal microgliosis of the gray matter. Lesions were not found in the cerebellar hemisphere or in the trigeminal ganglion.

Serologic tests for antibodies to pseudorabies, infectious bovine rhinotracheitis (IBR), and bovine virus diarrhea (BVD) were negative. Direct fluorescent antibody staining of frozen sections from the ventral midbrain, pons, and temporal and occipital cortices with conjugates for IBR, BVD, and rabies viral antigens yielded negative results. After 16 hours of incubation, homogenates of combined nervous tissues produced small foci of cytopathic effect in Vero cell monolayers, but not in bovine turbinate cell monolayers. Direct fluorescent antibody staining of Vero cell cultures was negative for IBR, BVD, and rabies viral antigens but was positive with the indirect fluorescent antibody technique for eastern equine encephalomyelitis (EEE) viral antigen. Duplicate tissues and homogenates referred to the National Veterinary Services Laboratory (NVSL, Ames, IA) confirmed these results by the identification of a togavirus interpreted to be EEE virus based on electron microscopy and complement fixation testing of the homogenate (Dr. D. Pederson, NVSL, Ames, IA, personal communication).
Differential diagnoses for nonsuppurative encephalitis in bovine species include rabies, pseudorabies, malignant catarrhal fever, sporadic bovine encephalomyelitis, IBR, and BVD. Neither gross nor histologic lesions characterized these diseases were found in the appropriate tissues. IBR as a cause of encephalitis has been found in neonates but is most common in calves 4 to 7 months of age. Occasional lymphohistiocytic perivascular infiltrates were seen in cattle 5 to 34 months of age with persistent BVD infection, although BVD antigen was not found in those infiltrates. Focal satellitosis around degenerating neurons was described but is of uncertain significance.

The isolation of EEE virus has been reported from pigeons, monkeys, human beings, rodents, insects, Pharo and bob-white quail, ring-necked pheasants, dogs, goats, pigs, and horses. The lesions in quail, pheasants, dogs, goats, pigs, and horses are described as neuronal degeneration and necrosis, small glial nodules around injured neurons, and a prominent neutrophilic infiltration. The neutrophilic infiltrate was considered to be a consistent feature of EEE infection in all of these species and was especially severe in the cerebral cortex, as is seen in horses. EEE virus was isolated from two naturally infected calves, 1 and 6 weeks of age, with a prominent neutrophilic infiltration of the neopil. These reports emphasize the young age of the affected species, the acute clinical disease leading to death (12 hours to 4 days), and a prominent neutrophilic infiltration. Differences in this case include the mature age of the cow and the lack of a significant neutrophilic infiltrate. The progress of the clinical illness in this case was unrecognized prior to recumbency, which may have provided adequate time for the perivascular cuffs to shift from predominantly neutrophilic infiltration to predominantly lymphohistiocytic, with only a few remaining neutrophils. Alternatively, the antigenic stimulus may have primarily elicited a lymphohistiocytic response rather than a neutrophilic response, perhaps because of differences in antigenic modulation or processing.

EEE virus should be considered as a sporadic cause of encephalitis with lymphohistiocytic perivascular cuffing in adult cattle.

References

Request reprints from Dr. E. D. McGee, Mississippi Board of Animal Health, Veterinary Diagnostic Laboratory, PO Box 4389, Jackson, MS 37216 (USA).

Cutaneous Mastocytosis in a Dog

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Key words: Cutaneous mastocytosis; urticaria pigmen	os	a; dog.

Mastocytosis refers to a disorder of unknown origin characterized by an abnormal proliferation of systemic or cutaneous mast cells. A cutaneous form has been described in a foal,1 kittens,1,2 and a pig.3 Cutaneous mastocytosis in children is referred to as urticaria pigmentosa.2 In the foal and the kittens, and in children, the cutaneous lesions regressed spontaneously.

A male Jack Russell Terrier developed multiple cutaneous masses at 3 weeks of age and was pruritic and lethargic. Biopsies taken from two nodules when the puppy was 10 weeks old contained moderate numbers of round cells. The cells formed a nonencapsulated, poorly circumscribed mass that blended with adjacent tissues. A large amount of edema separated these cells from each other. The cells formed rows as they dissected between collagen fibers or they coalesced into sheets. The round cells had abundant amphophilic to eosinophilic cytoplasm in sections stained with hematoxylin and eosin and had numerous metachromatic cytoplasmic granules when stained with toluidine blue. Nuclei were usually central and were oval or indented. Chromatin was clumped, and some nuclei contained faint nucleoli. Eosinophils were scattered throughout both nodules. One section contained an extensive ulcer and numerous neutrophils in the subjacent dermis. The diagnosis was mastocytosis.

At 13 weeks of age, the dog was still lethargic and pruritic and had approximately ten discrete cutaneous masses ranging from 1 to 5 cm in diameter on its head, neck, legs, perineum, and trunk. Masses varied considerably in their gross ap-