

Oligodendroglioma in a French Bulldog

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Abstract

A 5-year-old, male French bulldog with bradycardia, dyspnea, and decerebrate rigidity was necropsied. Macroscopic findings were restricted to the brain, and a single mass, 1.5×2.0×1.5 cm in size, was observed mainly at the right cingulum with prominently protruding into the dilated right lateral ventricle. The mass was grayish white in color, soft and gelatinous, but not clearly delineated. Microscopically, the mass consisted of diffuse proliferated neoplastic oligodendroglial cells characterized by small, round, and hyperchromatic nuclei with clear cytoplasm and the cells aggressively invaded into the adjacent parenchyma. Immunohistochemistry demonstrated that most of the neoplastic cells were positive for S-100 protein, vimentin, neuron specific enolase (NSE), and neurofilament protein (NFP). From these findings, the mass was diagnosed as oligodendroglioma.

Key words: oligodendroglioma, cerebrum, dog

In animals, neuroectodermal tumors are most common in dogs [6]. Of these neoplasms, astrocytomas comprise the majority of canine glioma in the United State, whereas oligodendrogliomas are most frequently diagnosed in Switzerland [6]. A predilection for glial tumors in brachycephalic dog breeds such as Boxers, Terriers, and Bulldogs has been recognized [1, 5]. Oligodendroglioma is usually noted in middle aged to old dogs [2, 7, 11], and tend to locate in the cerebral white matter, particularly around the lateral ventricle [5, 8]. However, distinguishing between oligodendroglioma and astrocytoma is sometimes difficult [6], because they share several histomorphological features, including high cellularity, necrosis, high mitotic rate, and prominent proliferation of glomeruloid vessels [7]. In addition, the presence of intermingled astrocytic cells, frequently noted in oligodendrogliomas, may further complicate the histology. The purpose of this

short paper is to describe the detailed pathomorphology including immunohistochemistry, and to discuss the characterization of this neoplasm as a differential diagnosis.

A 5-year-old, male French bulldog was referred to an animal hospital because the dog showed depression. During physical examination, arrhythmia and severe bradycardia (heart rates: 40-50 beats per minute) were recognized, and the dog collapsed with severe dyspnea. *Dirofilaria immitis* was not detected in the blood samples, and abdominal and thoracic radiographs showed no abnormalities. Rectal temperature was 38.6°C. The dog had been treated with dexamethasone sodium phosphate to reduce the clinical signs. However, dyspnea and tiring on exercise recurred over the following three weeks. Because the dog demonstrated decerebrate rigidity before death, necropsy was performed to examine the central nervous system. At necropsy, the brain showed moderate edematous swelling, accompanied by flattening of the cerebral cortical gyri and narrowing of the sulci. The posterior portion of the cerebellar vermis was mildly herniated through the foramen magnum. In transverse section through the mamillary bodies, a single mass, measuring 1.5×2.0×1.5 cm, was found at the right cingulum which prominently protruded into the dilated right lateral ventricle (Fig. 1). There was increased cerebrospinal fluid, which was cloudy, pinkish, and mucinous in viscosity. The mass was soft, gelatinous, and grayish white. The brain sample from the neoplastic mass was fixed in 10% formalin, routinely processed, and stained with hematoxylin and eosin (HE) for light microscopic examination. Additional serial sections were prepared for immunohistochemistry. The primary antibodies employed were the following: S-100 protein (Dako, Denmark), mouse anti-swine vimentin (Dako), rabbit anti-neuron specific enolase (NSE) (Dako), rabbit anti-bovine neurofilament protein (NFP) (Dako), rabbit anti-bovine myelin basic protein (MBP) (Dako), and rabbit anti-cow glial fibrillar acidic protein (GFAP) (Dako). These antibodies had been shown to bind in paraffin-embedded sections of canine and human [4, 10]. Positive staining was visualized by streptavidin-biotin complex (SAB) immunoperoxidase method.

Microscopically, the poorly delineated neoplasm was composed of a diffuse proliferation of neoplastic oligodendroglial cells, marked vascular endothelial proliferation, and atypical astroglial cells. The neoplastic oligodendroglial cells were

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arranged in sheets with honeycomb structures, and some of these were infiltrating into the adjacent parenchyma of cortex and perivascular spaces. Neoplastic cells had small, round, basophilic, and hyperchromatic nuclei set in the center of clear cytoplasm (Fig. 2). Mitotic figures are frequently observed specially at the periphery of the neoplasm. The neoplasm has abundant vascular proliferation with an occasional glomeruloid pattern (Fig. 3), and there are several foci of hemorrhages and extensive necrosis in the center of the neoplasm. Occasionally, anaplastic astroglial cells that had oval, large, and eccentric nuclei and abundant eosinophilic cytoplasm were noted around the small blood vessels and sometimes scattered singly within the neoplastic oligodendroglial cells (Fig. 2). A number of the cells were lipidized and rarely mitotic figures were seen.

Immunohistochemically, neoplastic cells reacted moderately to strongly for S-100 protein, vimentin, NSE, and NFP (Fig. 4). Small GFAP-positive astroglial cells were found around the small blood vessels and in areas containing the neoplastic oligodendroglial cells (Fig. 5). However, GFAP positive neoplastic oligodendroglial cells were not observed. All of the neoplastic cells were MBP negative, but myelin sheaths in the white matter were positive. These antibodies have been widely used to suggest a neural origin of neoplastic cells in human, and similar immunostaining patterns as in this case were also reported in oligodendrogliomas [3, 4, 9].

Oligodendrogliomas and astrocytoma sometimes share several histomorphological features as above described. However, oligodendroglial neoplastic cells in this case basically had distinguishing features such as round, hyperchromatic, nuclei surrounded by small amounts of lightly stained cytoplasm, and distinct cell borders. In addition, the astroglial cell component was mainly located around the proliferating blood vessels, and intermediate or transitional cell types were not identified. Therefore, it was concluded that the astroglial cells in the neoplasm were present to be reactive changes. These histologic features combined with results of immunohistochemistry supported the diagnosis of oligodendroglioma in this case.

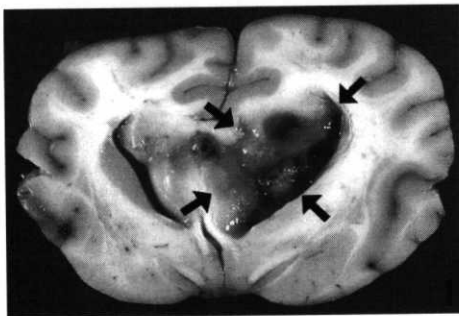


Fig. 1. Transverse section at the level of the mamillary body. The mass was observed at the right cingulum (arrows). The mass protruded into the lateral ventricle causing dilation. On gross finding, the mass was soft, gelatinous and grayish.

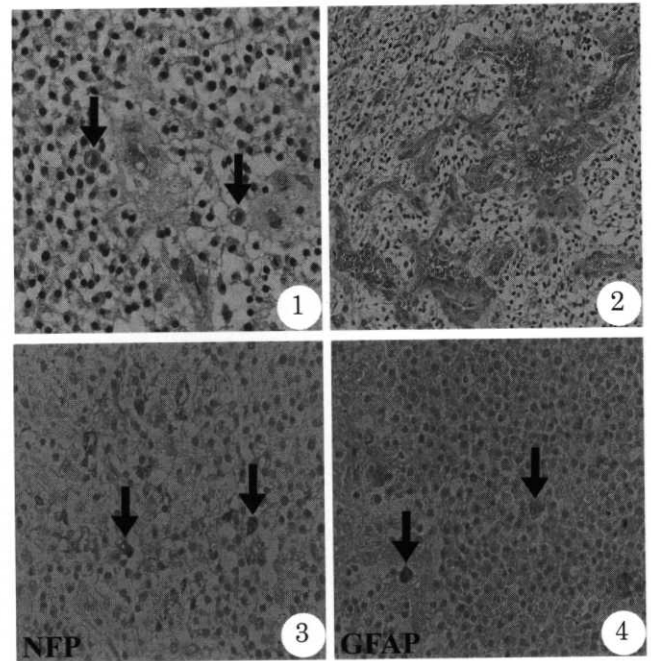


Fig. 2. Neoplastic oligodendroglial cells have small, round, and hyperchromatic nuclei with poorly stained cytoplasm. Occasionally, atypical astrocytes (arrows) were scattered among the neoplastic cells. H&E. $\times 400$ (1). Glomeruloid vascular proliferation is seen in the tumor. H&E. $\times 300$ (2). NFP positive neoplastic cells shows strong immunoreactivity in both of cytoplasm and nuclei (arrows). SAB. $\times 400$ (3). Scattered reactive GFAP positive astrocytes (arrows) are seen among the neoplastic oligodendroglial cells. SAB. $\times 400$ (4).

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