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Secondary Brain Tumor Caused by Infiltration of Nasal Neuroendocrine Carcinoma in a Chihuahua Dog: Clinical, Magnetic Resonance Imaging and Histopathological Findings

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Abstract : A 14-year-old neutered female Chihuahua was presented because of seizure episodes and circling to the left side. Based on neurological examination, the lesion was localized on left forebrain. The mass in the left nasal cavity and breaching of the nasal septum were seen magnetic resonance images. And there was a presence of contrast enhanced mass involving the rostral left brain. Based on diagnostic image analysis, this lesion strongly suggested secondary brain tumor infiltrated by nasal cavity. The patient's symptoms were well controlled by a combination therapy of prednisolone and lomustine (CCNU), and survived for two months after diagnosis. This case was definitively diagnosed as a nasal neuroendocrine carcinoma based on histopathological findings. This report describes the clinical findings, imaging characteristics, and pathologic features of secondary brain tumor which caused by infiltration of nasal neuroendocrine carcinoma in a dog.

Key words **:** brain tumor, dog, neuroendocrine carcinoma.

Introduction

Secondary brain tumors include both metastatic neoplasias and tumors that affect the brain by local extension. The frequency of occurrence of secondary brain tumors in veterinary patients is increasing (5). This increase can be attributed to the fact that veterinary patients with systemic malignant neoplasms are being treated with various chemotherapeutic agents that prolong the lifespan of the patient, thereby allowing more time for the development of central nervous system (CNS) metastasis (2). The common secondary brain tumors in dogs include local extension of nasal tumors and metastases from mammary gland, prostate, or pulmonary tumors (1,5). In dogs, carcinomas, followed by sarcomas, account for the majority of the reported brain metastases (5).

In this report, we describe a case of secondary brain tumor caused by infiltration of nasal neuroendocrine carcinoma in a Chihuahua dog.

Case

A 14-year-old neutered female Chihuahua presented with intermittent seizure episodes and circling to the left side. Physical examination revealed lethargy and mild exophthalmos of the left eyeball. Neurological examinations revealed continuous circling to the left side and delayed postural reactions in the right limbs. The results of the neurological examination suggested that the clinical signs were caused by a lesion in the left forebrain. The results of complete blood count (CBC) and serum chemical analysis were within normal ranges.

We performed brain magnetic resonance imaging (MRI) (E-scan; ESAOTE, Italy) using a 0.2 Tesla unit and obtained T1- and T2-weighted images and postcontrast T1-weighted images. MRI confirmed the presence of a mass in the left forebrain; the mass was isointense on the T1-weighted images and hyperintense on the T2-weighted images (Fig 1). The lesion was enhanced after intravenous (IV) administration of gadolinium-diethylenetriamine pentaacetic acid (0.1 mmol/kg body weight) (Omniscan; Nycomed, Inc., Princeton, NJ) (Fig 1). The mass in the left nasal cavity and invading of the nasal septum were readily seen in post-contrast T1-weighted dorsal plane images (Fig 2A). Post-contrast T1-weighted transverse images revealed the left sphenopalatine sinus was destructed by the mass. This lesion showed the presence of breaching of the cribriform plate (Fig 2B and C). The results of CSF analysis were normal. On the basis of the results of MRI and CSF analysis, the patient was tentatively diagnosed with a secondary brain tumor caused by infiltration from the nasal cavity.

We administered prednisolone (1 mg/kg body weight, per

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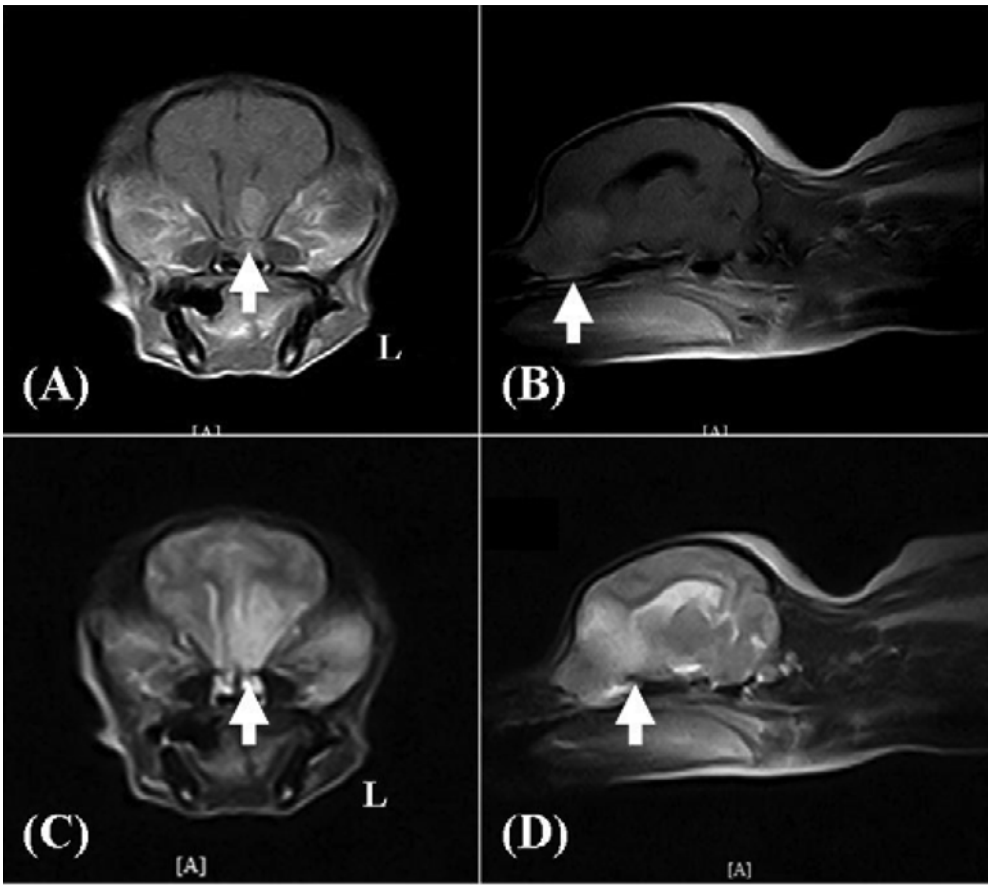

Fig 1. MR images of the present case. A: Post-contrast transverse T1-weighted image. Enhanced lesion is showed at the left cerebral parenchyma (arrow). B: Post-contrast sagittal T1-weighed image. Enhanced lesion is located cranial part of forebrain (arrow). C: Transverse T2-weighed image of same level with the panel A, a well defined hyperintense lesion is seen (arrow). D: Sagittal T2-weighed image of same level with the panel B.

os [PO] q 12 hours) (Prednisolone; Korea Pharma Co., Ltd., Korea) and lomustine (CCNU; 60 mg/m^2, PO q 6 weeks) (Lomustine; medac Gmbh, Hamburg, Germany) to the patient, and 1 week later, the circling and postural-reaction deficits disappeared. However, 2 months later, the clinical signs relapsed and the patient was euthanized.

The necropsy revealed a mass in the left forebrain (Fig 3). Most tumor cells had pale eosinophilic granular cytoplasm, hyperchromatic nuclei, and a number of mitotic figures. These tumor-cell arrangements were separated by fibrovascular septa, and some tumor cells formed pseudotubular patterns (endocrine-like packing) because of their basement membrane (Fig 4).

On the basis of the histopathological findings, the tumor was diagnosed as a neuroendocrine carcinoma.

Discussion

In dogs and cats, neuroendocrine carcinomas have been reported to occur in the intestine, liver, bile duct, lungs, gall bladder, esophagus, skin, and nasal cavity (3,4). However, there have been very few reports of neuroendocrine carcinomas in the nasal cavity, and these tumors have been reported only in dogs and horses (3,4,6). Most neuroendocrine carcinomas are extremely aggressive (3).

According to a previous report (5), nasal neoplasia was responsible for tumor distribution in only 6.2% of 177 dogs with secondary intracranial neoplasia (11/177): these tumors included nasal carcinoma (9/11), nasal sarcoma (1/11), and neuroendocrine carcinoma (1/11). Furthermore, the cerebrum was the most common tumor location (10/11) in the brains of dogs showing secondary intracranial neoplasia caused by nasal tumors. The present case was definitively diagnosed as secondary brain tumor caused by infiltration of nasal neuroendocrine carcinoma, and the tumor location was the left forebrain.

Similar to primary brain tumors, secondary brain tumors produce clinical signs of neurological dysfunction both by invading brain tissue and by causing secondary effects such as hemorrhage, inflammation, and obstructive hydrocephalus (1). The clinical signs of neurological dysfunction reflect the tumor locations within the brain as well as the degree of secondary effects of the tumor. Unlike primary brain tumors, secondary brain tumors are often associated with rapid development of neurological dysfunction. A previous report (5) showed that seizures and changes in mentation were the most common

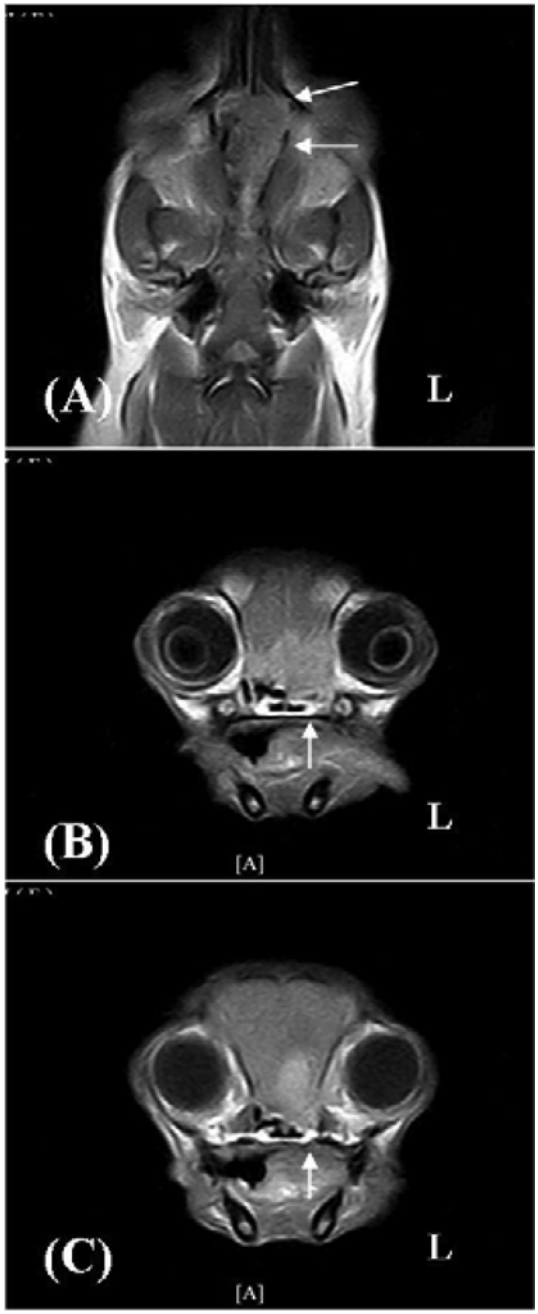

Fig 2. Post-contrast T1-weighted MR images. Post-contrast T1-weighted dorsal plane image (A) and post-contrast T1-weighted transverse images (B and C). A: The mass in the left nasal cavity and invading of the nasal septum are readily seen in this image (arrow). However, there is no evidence to encroach onto the cribriform plate in this image. B and C: There is a presence of contrast enhanced mass involving the rostral left brain. The left sphenopalatine sinus is destructed by the mass. This lesion shows the presence of invading of the cribriform plate.

signs in dogs with secondary brain tumors caused by nasal tumors, and the patient in the present study showed lethargy with intermittent episodes of seizures. Generally, dogs with nasal tumors show clinical epistaxis or have a history of this

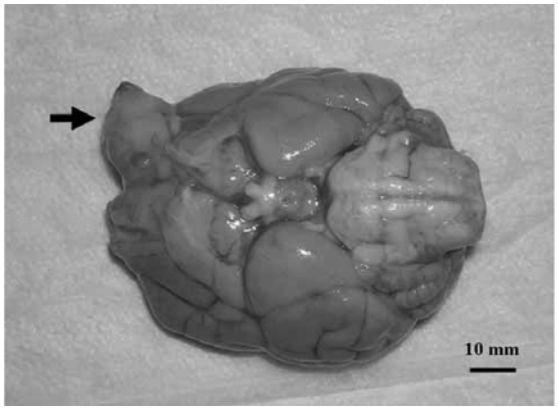

Fig 3. Necropsy findings of the present case. A mass is located at the left cranial area of forebrain (arrow).

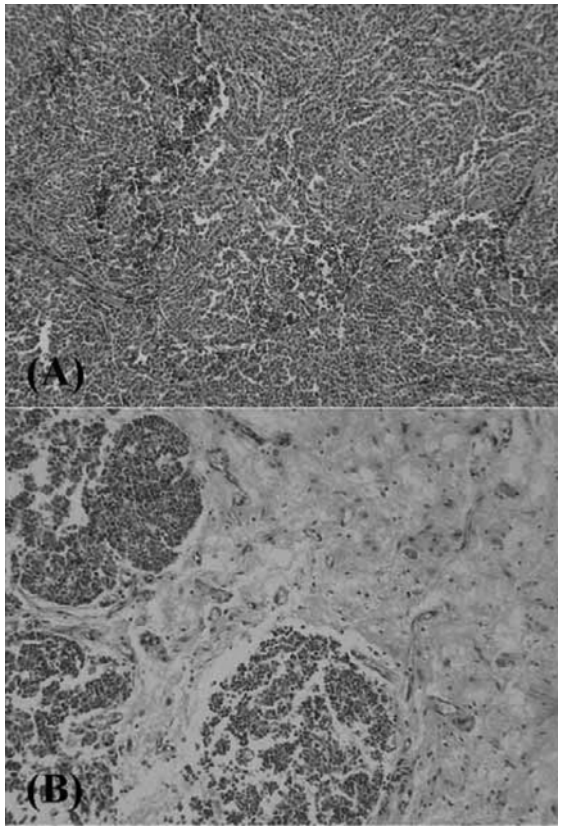

Fig 4. Histopathological findings of this case. A: Neuroendocrine carcinoma. (× 100, Hematoxylin and eosin stain). B: Tumor cells extended into the cerebral tissues (× 100, Hematoxylin and eosin stain).

symptom. However, epistaxis or nasal discharge was not observed in the patient in this study.

Similar to primary brain tumors, secondary brain tumors are usually observed in middle-aged to older dogs (1). A previous report demonstrated that the mean age at diagnosis of the secondary intracranial neoplasm caused by nasal tumors was 10.4 ± 1.2 years, and the median duration of the clinical signs was 27 days (5).

Definitive diagnosis of secondary tumors depends on the histopatholgical identification of the specific brain tumor. Computed tomography (CT) and MRI are commonly used for the diagnosis of brain tumors. Furthermore, the appearance of multiple intracranial masses on the CT or MR images of a patient suspected to be suffering from metastatic disease is a strong confirmatory evidence for secondary brain neoplasia (1). In the case of nasal tumors, CT or MRI can provide confirmatory evidence of invasion into the cranial cavity. Although CT is valuable in identifying bony destruction and soft tissue involvement, MRI provides less artifacts and higher resolution soft tissue images compared to CT in regions of the skull and brain (7). In the present case, we diagnosed an infiltration of the nasal tumor into the cerebral parenchyma on the basis of the MRI findings. The extracranial mass in the left nasal cavity and breaching of the nasal septum were seen in MR images. On T2 weighted images, the mass showed hypersignal intensity and enhanced after contrast study. Furthermore, destruction of the left sphenopalatine sinus and breaching of the cribriform plate by the mass were seen in MR images. Based on MRI findings, we could diagnose the present case with a secondary brain tumor caused by infiltration from the nasal cavity.

The supportive therapy for secondary brain tumors in dogs is the same as that administered for primary brain tumors, and definitive therapy may consist of surgical removal, chemotherapy, radiation therapy, or a combination of 2 or more methods. However, metastatic secondary brain tumors or nasal tumors showing invasion into the cranial cavity are rarely treated due to their poor prognosis (1). The present patient was treated with prednisolone and lomustine chemotherapy, and the survival time was 2 months.

In conclusion, this report describes the clinical findings, imaging characteristics, and histopathological features of a secondary brain tumor that was caused by infiltration of nasal neuroendocrine carcinoma.

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치와와견에서 발생한 비강 신경내분비암종의 침윤에 의한 이차적인 뇌종양 증례; 자기공명영상과 조직학적 특성

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요 약 : 14년 령의 암컷 치와와견이 발작증세 및 좌측으로의 선회운동 증상을 보여 내원하였다. 신경검사결과 좌측 대뇌 병변이 의심되어 자기공명영상 촬영을 실시하여 좌측 비강에서 좌측 대뇌로 파급된 병변을 확인하였다. 영상 분석상에서 비강 내에서 유래된 2차적인 뇌종양이 강력하게 의심되었다. 환자의 증상은 프레드니솔론과 로무스틴의 혼합치료 후 상당히 개선되었고 진단 후 2개월간 생존하였다. 조직 검사 결과 비강에서 유래한 신경내분비암종으로 확진되었다. 본 증례보고는 개에서 발생한 비강유래 신경내분비암종의 침윤에 의한 2차적인 뇌종양의 임상적인 특징, 영상진단 결과, 그리고 조직학적인 특성을 잘 보여주고 있다.

주요어 : 뇌종양, 개, 신경내분비암종.