

Secondary Intracranial Tumor Associated with Nasal Adenocarcinoma in a Dog: Clinical, Computed Tomography, Magnetic Resonance Imaging, Histopathologic Findings

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Abstract : A 12-year-old castrated male Miniature Schnauzer dog was presented with an acute seizure. On computed tomography, a mass was observed in the left nasal cavity and the lysis of the left cribriform plate was identified. Post-contrast magnetic resonance imaging showed a hyperintense mass in the left frontal lobe and hyperintense lesions in the left frontal sinus. The mass was tentatively diagnosed with a brain tumor secondary to a nasal tumor. Histopathology revealed that the mass was a nasal adenocarcinoma with invasion into the brain. This case report describes clinical, computed tomography, magnetic resonance imaging and histopathological findings of secondary intracranial tumor caused by extension of nasal adenocarcinoma in a dog. These findings may contribute to enhance the knowledge on secondary intracranial tumors in dogs.

Key words : brain tumor, dog, nasal adenocarcinoma, nasal cavity tumor.

Introduction

Secondary intracranial tumor in the dogs is a malignant disease that includes local extension from nasal tumor and metastasis from other organs (4). According to development of oncologic diagnosis and treatment, the incidence of secondary intracranial tumors is more increasing in veterinary medicine (9). The most common secondary intracranial tumor in dogs has been reported hemangiosarcoma, followed by pituitary tumor, metastatic carcinoma, nasal tumor, histiocytic sarcoma, and melanoma (4). To our knowledge, a case report of secondary intracranial tumor is rare because of progressive clinical deterioration and poor prognosis. This case report describes clinical, computed tomography, magnetic resonance imaging and histopathological findings of secondary intracranial tumor caused by extension of nasal adenocarcinoma in a Miniature Schnauzer dog.

Case

A 12-year-old castrated male Miniature Schnauzer dog was presented to the Konkuk University Veterinary Medical Teaching Hospital with a history of acute seizure. At admission, the dog was alert and all neurologic responses were normal on physical examination. Blood pressure was within normal range and body condition score was 5/5. The results of com-

plete blood count and electrolytes were within normal ranges. Serum chemistry showed elevated concentrations of alkaline phosphatase (ALP) and alanine aminotransferase (206 U/L and 82 U/L respectively; reference range, 15-127 U/L and 19-70 U/L respectively). Radiographic surveys revealed no remarkable findings except narrowing trachea at thoracic inlet on lateral thoracic radiograph. CT (Asteion 4[®], Toshiba, Japan) and MRI (E-scan[®], ESAOTE, Italy) with 2 mm slice thickness were performed. The mass (9.11 × 11.10 × 21.44 mm) was identified at the left nasal cavity and invaded into the left frontal lobe (Fig 1A) Soft tissue materials mixed with bone materials were filled in left frontal sinus (Fig 1B). Post-contrast T1-weighted sagittal image revealed that the hyper-signal intense mass with irregular margin in the left frontal lobe was connected to the nasal cavity (Fig 2A). The mass on the

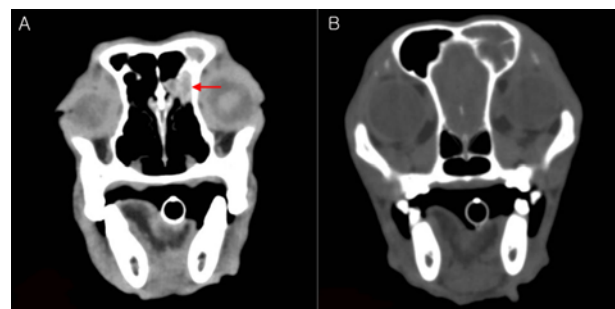


Fig 1. Plain computed tomography (CT) images on transverse view. A: The mass (arrow) is identified at the caudal portion of left nasal cavity. B: Soft tissue materials mixed with bone materials are filled in left frontal sinus.

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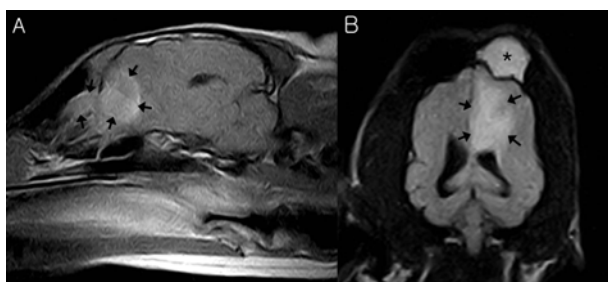


Fig 2. Magnetic resonance images of the brain. A: Post-contrast T1-weighted sagittal image reveals that the hyper-signal intense mass (arrow) in the left frontal lobe connected to the nasal septum and nasal cavity. B: Hyperintense lesions are detected in the left frontal sinus (asterisk) and frontal lobe (arrow) on post-contrast FLAIR dorsal image.

left frontal lobe was iso-signal intense and the lesion on the left frontal sinus was hypo-signal intense on plain T1-weighted images. On post-contrast fluid attenuated inversion recovery (FLAIR) dorsal view, hyperintense mass was detected in the left frontal lobe and hyperintense lesion was identified in the left frontal lobe (Fig 2B). Central fissure was deviated to the right side and the left lateral ventricle was compressed by the mass. Post-contrast T2-weighted images revealed hyper-signal intense in the lesions of both the left frontal lobe and the left frontal sinus. There were no metastatic findings on thorax and abdomen. On cerebrospinal fluid (CSF) analysis, there were no remarkable findings except increased concentration of total CSF protein (51 mg/dl, reference range; 0-30 mg/dl). Based on the clinical and diagnostic images, the mass was tentatively diagnosed as a brain tumor secondary to a nasal tumor.

The dog was medicated with prednisolone (2 mg/kg, PO, BID; Solondo, Yuhan Co, Seoul, Korea) and furosemide (2 mg/kg, PO, BID; Lasix, Handok Co, Korea) for 5 days. However, the dog did not respond with medication and seizure appeared more frequently. Although the dog administered phenobarbital (2.5 mg/kg, PO, BID; Phenobarbital, Hana Pharm, Korea), the symptom progressed worse. The patient was euth-

anized 8 days after diagnosis.

At necropsy of the brain, pinkish purulent discharge was observed in the left frontal sinus. Grossly a white, firm mass was found in the left nasal cavity and the mass infiltrated the frontal lobe in a friable form. Tissues were fixed in 10% neutral buffered formalin, processed routinely, and embedded in paraffin wax for histopathologic examination. 4- μ m sections were stained with hematoxylin and eosin (H &E) and Periodic acid-Schiff (PAS) staining. A couple of foci of tumor presented adherent to the surface and invaded into the parenchyma (Fig 3A). Clumps or mass of tumor had irregular glandular structure and extensively solid pattern of epithelial cells. Cellular pleomorphism, and nuclear atypia were observed. Tumor cells had eosinophilic cytoplasm, predominant nucleoli and high mitoses (2-3 per high-power field). PAS-positive granules in the cytoplasm of tumor cells were observed (Fig 3B). Histological diagnosis was nasal adenocarcinoma with secondary metastasized into the brain.

Discussion

Nasal tumors account for approximately 1% of all canine neoplasia (5,6,11). The mean age of affected dogs is 10 years and male predisposition has been suggested (5,6,11). Medium or large dolichocephalic breeds may be more commonly affected (5,6,11). The frequently occurring breeds are Siberian husky, German shepherd, bulldog, Great Dane, collie, and Shetland sheepdog (1,12). Similar to previous reports, the dog was old age and dolichocephalic breed in this case.

The common clinical signs of nasal tumors are epistaxis, nasal discharge, facial deformity, sneezing, dyspnea, exophthalmos, and ocular discharge (5,6,10,12). Rarely, patients with invasion of nasal tumor into the brain may have neurologic signs such as seizures, behavior change, paresis, and circling (11). In a previous study, 7 animals with nasal cavity tumors showed predominant neurologic signs without respiratory signs unusually and they were a diagnostic challenge (8). Nasal cavity tumor should be considered in the differential diagnosis for animals with neurologic signs. In this case

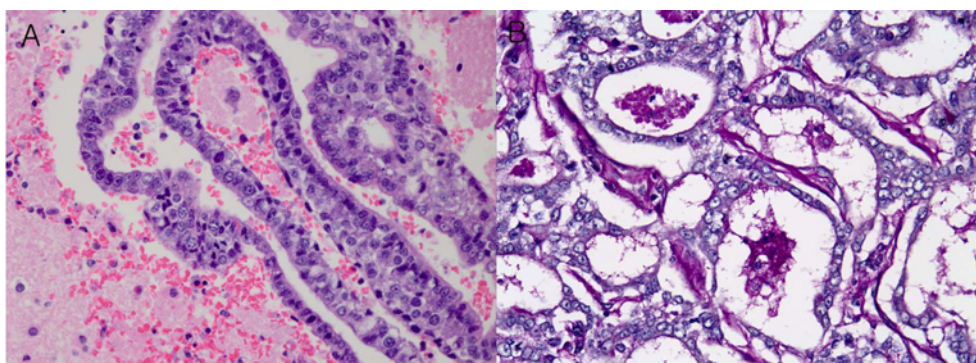


Fig 3. Histopathologic findings of secondary intracranial tumor from nasal adenocarcinoma. A: The tumor consists of epithelial cells forming clumps, masses, and glandular structures (H & E stain, $\times 400$). B: PAS-positive granules in the cytoplasm of tumor cell are observed (PAS stain, $\times 400$).

report, the dog showed only seizure without respiratory signs and deteriorated rapidly.

CT scanning and MRI can be useful in diagnosing nasal tumor or brain tumor (3). CT scanning gives more accuracy for evaluating bony involvement and metastasis than radiography (4,10), while MRI provides detailed information about structures of tumors (4). According to previous reports (3), CT findings of nasal tumors include soft tissue opacity or mineralization in nasal cavity, destruction of ethmoid bone, nasal bone and maxilla, abnormal soft tissue in retrobulbar space, and hyperostosis of the lateral maxilla. In this case, soft tissue mass in nasal cavity, destruction of ethmoid bone, and soft tissue materials in frontal sinus were observed on CT view. On MRI, intracranial tumors are iso-to hypointense in T1-weighted images and iso-to hyperintense in T2-weighted images. Contrast agents plays important role to detect brain tumor because blood barrier is disrupted and allowed penetration of contrast agents into brain tissue in patients with intracranial tumor (7). In this case, the mass was isointense in T1-weighted images and hyperintense in post-contrast T1-weighted and T2-weighted images. Based on CT and MRI images, the present case was tentatively diagnosed with secondary brain tumor caused by invasion of nasal cavity tumor.

The most effective method for definitive diagnosis of secondary brain tumor is histopathologic examination (9). In the present case, H & E stain revealed that brain tumor cells consisted of epithelial cells forming glandular structures and were diagnosed as nasal adenocarcinoma. Furthermore, PAS stain is helpful to identify epithelial mucin produced by adenocarcinoma. The PAS stain was strongly positive for the cytoplasmic granules in the present case.

Treatment of secondary brain tumor in dogs is similar with that of primary brain tumor and consists of surgical removal, irradiation, chemotherapy, and immunotherapy (4,9). However, the major goals of therapy for a brain tumor are to control clinical signs using phenobarbital or bromide and to reduce cerebral edema through glucocorticoids (4). In this case, the patient was treated with prednisolone and phenobarbital but clinical signs were not improved.

Although a few data is present, concerning mean survival times of dogs with secondary brain tumor, prognosis of secondary intracranial tumor is poor (4). In a previous report, the mean survival time of 38 dogs with primary or secondary brain tumors receiving symptomatic treatment was 6 days (2). The dog in the present study was deteriorated progressively as soon as the first symptom appeared and euthanized within 8 days after diagnosis.

Conclusion

The present report describes clinical, computed tomography, magnetic resonance imaging and histopathological findings of secondary intracranial tumor caused by extension of nasal adenocarcinoma in a Miniature Schnauzer dog. To our knowledge, a case report of secondary intracranial tumor is rare because of progressive clinical deterioration and poor prognosis. These findings may contribute to enhance the knowledge on secondary intracranial tumors in dogs.

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개에서 발생한 비강샘암종으로부터 유래한 이차성 두개 내 종양 증례

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요 약 : 12세의 중성화 수술을 한 수컷 미니어처 슈나우저종견이 급성 경련 증상으로 내원하였다. 컴퓨터단층촬영에서 좌측 비강 내 종괴와 시상관의 용해가 관찰되었다. 조영 후 자기공명영상 사진에서 좌측 전엽과 전두동에 고신호 감도의 종괴가 확인되었다. 비강 종양에 의해 이차적으로 발생한 뇌종양으로 진단되었으며 조직검사상에서 비강샘암종이 뇌에 전이된 소견이 관찰되어 확진되었다. 개에서 발생한 비강샘암종이 침습되어 이차적으로 두개 내 종양이 발생한 증례로써 이차성 뇌종양의 이해 증진에 기여할 수 있는 자료를 제공할 수 있을 것으로 기대된다.

주요어 : 뇌종양, 개, 비강샘암종, 비강종양