1970

Horner's Syndrome in a Cat: A Case Report

Monte L. Pauli
Iowa State University

James D. Carter
Iowa State University

Follow this and additional works at: https://lib.dr.iastate.edu/iowastate_veterinarian

Part of the Eye Diseases Commons, Ophthalmology Commons, Small or Companion Animal Medicine Commons, and the Veterinary Pathology and Pathobiology Commons

Recommended Citation
Available at: https://lib.dr.iastate.edu/iowastate_veterinarian/vol32/iss2/2

This Article is brought to you for free and open access by the Journals at Iowa State University Digital Repository. It has been accepted for inclusion in Iowa State University Veterinarian by an authorized editor of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.
Horner's Syndrome in a Cat: A Case Report

By Monte L. Pauli and James D. Carter, M.S., D.V.M.*

Introduction

Horner's Syndrome is a condition caused by lesions affecting the nervous system and manifested by changes in the eye. The syndrome has been described in man* and in animals and is characterized by enophthalmos, ptosis, miosis, and anhidrosis (due to paralysis of the sympathetic innervation of the eye).

The syndrome may result from diseases of or injury to the brain stem, to the lower part of the cervical portion of the spinal cord, to the upper part of the dorsal portion of the spinal cord or to the anterior cervical sympathetic ganglion.

Case History and Clinical Course

A 13-year-old male domestic cat was admitted to the Iowa State University Veterinary Clinic on November 11, 1969, because of anorexia, progressive weight loss, and dyspnea. The patient's history indicated Previous treatment one month earlier for an upper respiratory infection and anorexia. The animal was seen two weeks prior to admission for a mild left conjunctivitis of undetermined etiology.

Rectal temperature on admission was 100°F. Physical examination revealed the patient to be generally debilitated and with a poor hair coat. Unilateral miosis, ptosis, and enophthalmos of the left eye was noted while the right eye appeared normal. Exudate was seen at the nares and there was a palpable, non-moveable mass in the left anterior ventral cervical region. Abdominal examination revealed a partially distended urinary bladder. Lateral and ventrodorsal radiographs of the cervical area revealed the mass to be of water density and to lie above the trachea and larynx. Generalized osteopenia was also observed. A blood specimen was taken; the results of this and subsequent laboratory tests are summarized on Table 1.

Supportive therapy was instituted and consisted of half strength saline with 5% dextrose intravenously and penicillin-dihydrostreptomycin intramuscularly. The urethra was catheterized.

The patient's general condition remained unchanged and on November 14, 1969 a left persistent torticollis developed.

A blood specimen was collected for calcium, phosphorus, and alkaline phosphatase determination. The findings revealed a marked degenerative left shift, high calcium, low phosphorus, and normal alkaline phosphatase (Table 1). A biopsy of the cervical mass was taken and interpreted as a neoplasm.

The client requested euthanasia and permission for autopsy was obtained.

Pathologic Findings

Grossly there was a palpable, non-moveable mass at the left anterior ventral cervical region causing dextrodeviation of the larynx and anterior portion of the trachea due to displacement by the cervical mass. The mass measured approximately 1 x 1.5 x 2.5 cm and extended from the region of the left thyroid gland, anterior to the contralateral tympanic bulla and dorsolaterally to impinge upon the ventrolateral aspect of the transverse processes of the second and third cervical vertebrae. The right parathyroid glands were not grossly visible. The mass was firm, tan, and did not protrude on its exposed surface.

Microscopically a schirrous squamous cell carcinoma of intraglandular salivary duct origin involving the mandibular sa-
The patient's left eyelid is ptosed. The membrane nictitans protrudes over the cornea as a result of enophthalmos. The patient's left pupil is miotic.

Close-up view of the left eye. The miotic pupillary border (arrow) may be seen lateral to the margin of the protruding membrane nictitans.

Table 1: Clinical Pathology Values

<table>
<thead>
<tr>
<th></th>
<th>Nov. 11, 1969</th>
<th>Nov. 13, 1969</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>10.1</td>
<td>11.2</td>
</tr>
<tr>
<td>PCV</td>
<td>26</td>
<td>28</td>
</tr>
<tr>
<td>WBC</td>
<td>12,900</td>
<td>10,450</td>
</tr>
<tr>
<td>Seg.</td>
<td>45</td>
<td>65</td>
</tr>
<tr>
<td>Band.</td>
<td>51</td>
<td>25</td>
</tr>
<tr>
<td>Lympho.</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Mono.</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Platelets</td>
<td>Adequate</td>
<td>Adequate</td>
</tr>
<tr>
<td>RBC Morphology</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Alk. Phos.</td>
<td>—</td>
<td>41.5 I.U.</td>
</tr>
<tr>
<td>Ca</td>
<td>—</td>
<td>11.8 Mg%</td>
</tr>
<tr>
<td>P</td>
<td>—</td>
<td>3.5 Mg%</td>
</tr>
</tbody>
</table>

The ventral laryngeal muscles were diffusely invaded by the carcinoma which was less schirrous than in the cervical mass.

Histopathologically there appeared to be no involvement of the thyroid or parathyroid glands. Lymphatic tissue in the adjacent area appeared normal.

Examination of the vertebral bodies and the proximal tibia revealed no discernable lesions referable to osteopenia.
Discussion

Horner's Syndrome may be caused by cervical tumors, enlarged lymph nodes, cervical rib, aortic aneurysm, mediastinal tumor, esophageal disease and trauma. 1

Although enophthalmos is said to be present in Horner's Syndrom, actual measurements in man have disproved this.1 It is more probably due to ptosis, and the appearance it renders upon the eye. However, in this case report protrusion of the membrane nictitans probably indicated true enophthalmos due to relaxation of the orbital muscles.

The ptosis is caused by paralysis of the smooth muscle (Muller's) of the tarsus innervated by the anterior cervical sympathetic ganglion.2 The ptosis in Horner's Syndrome is neurogenic and may originate from a lesion located in any part of the nerve pathway from the cerebral cortex to the levator palpebrum muscle. The miosis is due to degeneration of the post ganglionic fibers of the anterior cervical sympathetic ganglion, 2 as is anhidrosis of the periorbital area in those animals which sweat.

The initial clinical findings and the high blood calcium, low phosphorus indicated that there might be an existing primary hyperparathyroidism. As a result, primary parathyroid adenoma with secondary Horner's Syndrome must be included in a differential diagnosis.

The histopathology in this case ruled out involvement of the parathyroid glands and indicated a squamous cell carcinoma of salivary gland origin.

BIBLIOGRAPHY