

9-1-2012

Horner's Syndrome Associated with Escherichia Coli Infection in a Raccoon (*Procyon lotor*) - A Case Report

Fernando Nájera Muñoz

Alfredo Carlos Suárez Saavedra

Follow this and additional works at: <https://digital.car.chula.ac.th/tjvm>



Part of the [Veterinary Medicine Commons](#)

Recommended Citation

Muñoz, Fernando Nájera and Suárez Saavedra, Alfredo Carlos (2012) "Horner's Syndrome Associated with Escherichia Coli Infection in a Raccoon (*Procyon lotor*) - A Case Report," *The Thai Journal of Veterinary Medicine*: Vol. 42: Iss. 3, Article 16.

Available at: <https://digital.car.chula.ac.th/tjvm/vol42/iss3/16>

This Short Communication is brought to you for free and open access by the Chulalongkorn Journal Online (CUJO) at Chula Digital Collections. It has been accepted for inclusion in The Thai Journal of Veterinary Medicine by an authorized editor of Chula Digital Collections. For more information, please contact ChulaDC@car.chula.ac.th.

Horner's Syndrome Associated with *Escherichia Coli* Infection in a Raccoon (*Procyon lotor*) - A Case Report

Fernando Nájera Muñoz^{1*} Alfredo Carlos Suárez Saavedra²

Abstract

This clinical case describes a three-year-old male raccoon (*Procyon lotor*), castrated, from La Lajita Oasis Park Zoological Park (Spain) which showed lack of appetite, state of mental depression and mild dehydration. During clinical examination an abscess was detected in the left ventrolateral area of the neck as well as clinical signs compatible with Horner's Syndrome in the left eye. A hypersensitivity test was carried out by sympathetic innervation with topical epinephrine 0.0001% and results suggested the presence of second-order Horner's Syndrome formed by pressure from the abscess on the sympathetic innervation of the left eye. A radiologic study of the neck was performed and blood and samples of the abscess exudates were obtained. A culture of the exudates revealed the presence of *Escherichia coli*. Initial treatment was introduced according to indications resulting from the previously obtained antibiogram. Symptoms disappeared six days after the initial treatment. While Horner's Syndrome is a common clinical finding in small animal clinics, it is more complicated to observe in wild carnivores, yet diagnostic and therapeutic techniques applicable to domestic animals can be used as a reference for small carnivores of the Procyonidae family.

Keywords: bite wounds, *Escherichia coli*, Horner's syndrome, *Procyon lotor*, raccoon

¹Zoo Veterinarian La Lajita Oasis Park Fuerteventura. Spain.

Project Manager Thailand Clouded Leopard Consortium. Smithsonian Institute. Washington DC USA. Zoological Park Organization of Thailand.

²Senior Veterinary Student, Veterinary School, University of Las Palmas de Gran Canaria, Trasmontaña, s/n, 35416, Arucas, Las Palmas, Spain

*Corresponding author: E-mail: nanonaj@hotmail.com

บทคัดย่อ

กลุ่มอาการ Horner's ที่สัมพันธ์กับการติดเชื้อ *Escherichia Coli* ในแรคคูน

Fernando Nájera Muñoz^{1*} Alfredo Carlos Suárez Saavedra²

กรณีศึกษาทางคลินิกรายนี้แสดงให้เห็นว่า แรคคูน (*Procyon lotor*) เพศผู้ตอน อายุ 3 ปี จาก La Lajita Oasis Park Zoological Park ประเทศสเปน มีอาการเบื่ออาหาร ซึม และมีภาวะขาดน้ำปานกลาง จากการตรวจร่างกายพบผิวหนังบริเวณคอด้านหลังซ้าย (ventrolateral) และแสดงอาการที่มีลักษณะสอดคล้องกับกลุ่มอาการ Horner's ที่ดวงตาข้างซ้าย ทำการตรวจสอบการทำงานระบบประสาทซิมพาเทติกด้วยการหยอดอิพิเนพรีน ขนาด 0.0001% พบว่าลักษณะสอดคล้องกับกลุ่มอาการ Horner's แบบที่ 2 โดยมีสาเหตุมาจากการที่เส้นประสาทซิมพาเทติกของตาข้างซ้าย เกิดจากแรงดันการกดทับ ของผิวหนัง จากการถ่ายภาพรังสีที่บริเวณคอ การเก็บเลือดและตัวอย่างจากผิวหนัง เมื่อนำตัวอย่างผิวหนังไปเพาะเชื้อในอาหารเลี้ยงเชื้อ พบว่า มีเชื้อ *Escherichia coli* เมื่อทำการรักษาในเบื้องต้นโดยอาศัยผลการเพาะเชื้อ พบว่าสัตว์มีอาการดีขึ้น 6 หลังจากการรักษา ในขณะที่กลุ่มอาการ Horner's เป็นอาการทางคลินิกที่พบได้ในโรงพยาบาลสัตว์เล็ก แต่จะสังเกตได้ยากในสัตว์ป่ากินเนื้อ อย่างไรก็ตามข้อมูลที่มาจากการวินิจฉัยและการรักษาโรคในสัตว์เลี้ยงสามารถนำมาประยุกต์ใช้กับสัตว์กินเนื้อขนาดเล็กในกลุ่ม Procyonidae ได้เช่นกัน

คำสำคัญ: แผลถูกกัด *Escherichia coli* กลุ่มอาการ Horner's *Procyon lotor* แรคคูน

¹Zoo Veterinarian La Lajita Oasis Park Fuerteventura. Spain. Project Manager Thailand Clouded Leopard Consortium. Smithsonian Institute. Washington DC USA. Zoological Park Organization of Thailand.

²Senior Veterinary Student, Veterinary School, University of Las Palmas de Gran Canaria, Trasmontaña, s/n, 35416, Arucas, Las Palmas, Spain

*ผู้รับผิดชอบบทความ E-mail: nanonaj@hotmail.com

Introduction

Horner's syndrome consists of a blockage of the sympathetic innervation of the eye and its connections in some points of the trineuronal pathway which is divided in three neuroanatomic parts: central, preganglionic and postganglionic (Boydell, 2000; Ofri, 2008; De Lahunta, 2009). This produces a series of ophthalmologic clinical signs which, in domestic carnivores, emphasize in the form of miosis, ptosis, enophthalmia, protrusion of the third eyelid, anisocoria and reduced palpebral fissure (van den Broek, 1987; Kern et al., 1989; Morgan and Zanotti 1989; Boydell, 2000). When the first-order neuron is affected, Horner's syndrome is associated with other additional neurological alterations, but when the second or the third-orders are affected, only the typical clinical signs of Horner's syndrome are detectable (De Risio, 2009). Generally, the main causes of Horner's syndrome in dogs and cats are congenital alterations, cranial, cervical or thoracic traumas, vertebral disc diseases, otitis media, neoplasia, brachial plexus root avulsion, thoracic masses, central nervous system infections, ischemic and idiopathic myelopathy (Griffiths et al., 1974; De Lahunta and Alexander, 1976; Brightman et al., 1977; van den Broek 1987; Kern et al., 1989; Morgan and Zanotti

1989; Obradovich et al., 1992; Melián et al., 1996; Herrera et al., 1998; Boydell, 2000; Baines, 2001). Severe cervical spinal injuries or thoracic facial spinal injuries have been suggested as possible causes of first-order Horner's syndrome and brachial plexus root injuries, neck soft tissue injuries such as incised wounds, infections or of iatrogenic origin, as possible causes of second-order Horner's syndrome (Montiani and Petersen-Jones, 2002). Raccoon (*Procyon lotor*) is a carnivorous mammal of the Procyonidae family found in captivity in zoological parks. Although Horner's syndrome is commonly found in small animal clinics, it is more complicated to be observed in wild carnivores. The objective of this paper was to confirm if the diagnosis and treatment of Horner's syndrome of domestic carnivores were valid for wild carnivores.

Materials and Methods

A castrated male raccoon, three years of age and eight kg in weight, was located in an enclosure in the La Lajita Oasis Park, a zoological park in Fuerteventura, Spain, with seven other castrated males. It was presented with loss of appetite, mild dehydration and a state of mental depression for various days. Consequently, it was transferred to a

cage in the veterinary clinic in the zoological park. No other raccoons showed clinical signs. For clinical examination, the animal was immobilized inside a carry cage. During the examination an abscess of the size of a walnut was detected in the left ventrolateral area of the neck and clinical signs were apparent in the left eye compatible with Horner's syndrome: miosis, ptosis, enophthalmia, protrusion of the third eyelid, anisocoria and reduced palpebral fissure (Fig. 1 & 2).

For the hypersensitivity test by sympathetic innervations, epinephrine 0.0001% (Adrenaline™ 1 mg/ml injectable solution, B. Braun Medical, Spain) was instilled in the conjunctival sack of each eye to determine the approximate location of possible blockage in the trineuronal pathway due to the great sensitivity of the iris sphincter muscle when presented to such drug when deprived of the specific neurotransmitter (Shamir and Ofri, 2007; Ofri, 2008). The non-affected eye was used as control. When the postganglion neuron is affected, mydriasis is produced within 20 minutes following application. If the preganglion or central neuron is affected, mydriasis is expected to appear within 20 to 45 min following administration (Kern et al., 1989; Boydell et al., 2000; De Lahunta, 2009). In the described case, mydriasis was produced in the left eye more than 45 min after application. Therefore, and due to the lack of additional neurologic clinical signs suggesting a first-order Horner's syndrome, the presence of a second-order Horner's Syndrome was considered. Moreover, the exact location of the injury could not be determined due to the non-existence of pharmacologic tests which differentiate the injury from the central to the pregangliar neuron, except for cocaine and hydroxylamphetamine tests that are used in humans (Walton, 2003; Mughal, 2009) which were not carried out.

A detailed clinical examination of the animal sedation was performed by using 60 mg of intramuscular Ketamine (Ketaset™, Fort Dodge Veterinaria, Spain) and 0.4 mg Medetomidine (Domitor™, Orion Corporation, Finland) intramuscular and an intravenous infusion of glucose solution (Glucosado™ 10%, B. Braun VetCare, Spain) and a polyelectrolyte solution (Lactato Ringer™, B. Braun VetCare, Spain) to enhance the hydroelectrolytic state of the animal. The fur on the cervical mass area was cut and sterilized with povidone-iodine (Betadine™ 10% dermic solution, Meda Pharma, Spain). After, a 21-gauge needle (Sterican™ 21G, B. Braun Medical, Spain) was introduced in the center of the mass and placed on a 10 ml syringe (Injekt™ 10 ml, B. Braun Medical, Spain) sucking in slowly until obtaining 8 ml. of purulent exudates which revealed the presence of an abscess. For aperture, an incision was practiced on the ventral border of the mass following the skin tension lines (Fig. 3). The sides of the incision were opened to their fullest using Kocher tweezers and, with a sterile swab (Port-A-Cul™ tube, Becton Dickinson, Spain) a sample of the exudates were obtained for culture and antibiogram. To drain the abscess a 20 ml syringe (Injekt™ 20 ml, B. Braun Medical, Spain) and sterile

gausses were used, with which the exudates were removed using pressure to ease their way out. When the amount of exudate was small and difficult to extract, saline solution was applied inside the cavity with pressure, allowing it to pour out. It was decided not to close the practiced incision in order to facilitate the exit of exudate that might remain, scarring by second intention healing.

Blood sample was obtained to carry out a hemogram and blood biochemistry, and a radiographic study of the neck was performed, detecting no other accentuated alterations in the rest of the clinical examination. According to the established reference intervals in cats, provided by the laboratory service of the Veterinary Hospital of the Veterinary Department of the Las Palmas de Gran Canaria University, no abnormal values were obtained in the hemogram and blood biochemistry.



Figure 1 Rostral view of the patient



Figure 2 Left latero rostral view



Figure 3 Left ventro lateral view of the neck. Notice incision and exudate (white arrow)

The radiographic study of the neck revealed inflammation of the soft tissue in the cervical area, suspectedly caused by a bite from another animal.

Immediately after the surgery and once the animal recovered from anesthesia, an initial therapeutic plan was introduced prior to obtaining the microbiological results, including 5 mg/kg SC enrofloxacin every 24 hours for 21 days (Baytril™ 5% injectable solution, Bayer Sanidad Animal, Spain), and 12.5 mg/kg SC amoxicillin and clavulanic acid every 24 hours for 6 days (Synulox™ 40 ml injectable solution, Pfizer Salud Animal, Spain). Additionally, eye drops of Polymyxin B, Neomycin and Gramicidin (Oftalmowell™ 5 ml eye drops, UCB Pharma, Spain) were applied as a preventive measure to avoid secondary bacterial overgrowth in the affected eye. As the animal was restricted from receiving food during the initial 48 hours, a vitamin complex with electrolytes and amino acids was administered (Duphalyte™, Fort Dodge Veterinaria, Spain).

The microbiological culture of the exudate revealed the presence of *Escherichia coli* and the antibiogram determined that the pathogen was sensitive to enrofloxacin, amoxicillin and clavulanic acid, cefovecin, chloramphenicol and gentamicin. Therefore, it was decided to continue the treatment with enrofloxacin, and amoxicillin and clavulanic acid. Clinical signs disappeared six days after starting the treatment and the animal recovered without any apparent side-effects. Once the incision performed on the raccoon's neck closed by second intention healing, the animal was reintroduced to the rest of the raccoon group.

Results and Discussion

Horner's syndrome is described in human beings (Walton, 2003), dogs and cats in numerous studies (van den Broek 1987; Kern et al., 1989; Morgan and Zanotti., 1989). It has also been described in other domestic mammals such as horses, cows and other ruminants (Smith and Mayhew, 1977; Simoens et al., 1990; Peace et al., 1997; Hahn, 2003; Firshman et al., 2003; Nahn, 2006), and in wild carnivores (Lambrechts and Berry, 2000; Adaska and Lynch, 2004) and birds (Williams and Cooper, 1994; Gancz et al., 2005). In this case Horner's syndrome was produced as a consequence of a bite wound in the lateroventral region of the neck caused by another raccoon and causing the development of an abscess, which, as it was growing in size, was displacing adjacent structures until compressing the sympathetic enervation of the left eye, disabling the transmission of impulses, and causing the clinical signs described above.

In general, bite wounds can be contaminated with bacteria from the oral cavity of the aggressor animal, bacteria found on the victim's skin or other microorganisms found in the environment, due to the fact that the devitalized tissue, ischemia and the generated clearance provide an ideal environment for the development of bacteria (Goldstein, 1992; Waldron and Zimmerman-Pope, 2002), allowing the production of local or systematic infections (Shamir,

2002). In human beings the majority of wounds from animal bites contain a mix of aerobic and anaerobic microorganisms which is thought to reflect the diversity of flora from the aggressor animal's mouth, and in less quantity from the victim's skin (Goldstein, 1992; Talan et al., 1996). The presence of *Escherichia coli* has been described as part of the oral microbiota of domestic dogs and wild raccoons (Shapir and Carter, 1975; Bailie et al., 1978; Barrat et al., 1982; Kamata et al., 1988). In dogs, its presence is associated with bacterial infections in wounds produced by other congeners although infections produced by *Staphylococcus* spp, *Streptococcus* spp and *Pasteurella* spp, amongst others, (Kelly et al., 1992; Griffin and Holt, 2001; Meyers et al., 2008; Mouro and Vilela, 2010) are more common. Additionally, in raccoons the presence of *Escherichia coli* has been described as part of their regular conjunctiva flora (Pinard et al., 2002), however no bibliographic references exist regarding its relation with cervical area infections. Hence, the presence of *Escherichia coli* in bitten wounds produced among domestic carnivores suggests the possibility of these kinds of wounds in raccoons, despite the lack of knowledge regarding microbiota present in bite wounds on raccoons or in their oral cavity.

Regarding the test based on the hypersensitivity principle by sympathetic innervation with epinephrine 0.0001%, it seems to be functional on raccoons even though in other domestic mammals other adrenergic agonists, such as phenylephrine between 1 and 10%, are used (Shamir and Ofri, 2007; Ofri, 2008). Nevertheless, bear in mind that even with these species this test should only be practiced to guide, as results vary in relation to how much time has passed since the injury was produced, how serious the damage is, and its distance from the iris. Regarding the results obtained from the treatment against the infectious process, we can suggest that the common drugs chosen for use on domestic carnivores are functional on raccoons, bearing in mind that it is necessary to carry out an antibiogram for indications on which antibiotics the pathogen is sensitive to.

References

- Adaska, J.M. and Lynch, S. 2004. Fibrocartilaginous embolic myelopathy in a Sumatran tiger (*Panthera tigris sumatrae*). J Zoo Wildl Med. 35(2): 242-244.
- Bailie, W.E., Stowe, E.C. and Schmitt, A.M. 1978. Aerobic bacterial flora of oral and nasal fluids of canines with reference to bacteria associated with bites. J Clin Microbiol. 7(2): 223-231.
- Baines, S.J. and Langley-Hobbs, S. 2001. Horner's syndrome associated with a mandibular symphyseal fracture and bilateral temporomandibular luxation. J Small Anim Pract. 42(12): 607-610.
- Barrat, J., Blancou, J. and Gerard, Y. 1982. Buccal microflora of wild carnivores (particularly foxes) in Lorraine, France. Preliminary results. Revue Française de la Santé Publique. 19: 25-29.
- Boydell, P. 2000. Idiopathic Horner's syndrome in the golden retriever. J. Neuroophthalmol. 20(4): 288-

- 290.
- Boydell, P. and Brogan, N. 2000. Horner's syndrome associated with *Neospora* infection. *J Small Anim Pract.* 41(12): 571-572.
- Brightman, A.H., Helper, L.C. and Parker, A.J. 1977. Congenital Horner's syndrome. *Canine Pract.* 4(5): 19-20.
- De Lahunta, A. 2009. Lower motor neuron-general visceral efferent system. In: *Veterinary Neuroanatomy and clinical neurology.* 3rd ed. A.E.G. De Lahunta (ed). Pennsylvania: WB Saunders Company: 168-191.
- De Lahunta, A. and Alexander, J.W. 1976. Ischemic myelopathy secondary to presumed fibrocartilaginous embolism in 9 dogs. *J Am Anim Hosp Assoc.* 12(1): 37-48.
- De Risio, L. and McConnell, J.F. 2009. Second order Horner's syndrome in a cat. *J Feline Med Surg.* 11(8): 714-716.
- Firshman, A.M., Hayden, D.W., Valberg, S.J. and McKenzie, E.C. 2003. Horner's syndrome associated with fungal mediastinitis in a horse. *Equine Vet Educ.* 15(2): 82-85.
- Gancz, A.Y., Malka, S., Sandmeyer, L., Cannon, M., Smith, D.A. and Taylor, M. 2005. Horner's Syndrome in a Red-bellied Parrot (*Poicephalus rufiventris*). *J Avian Med Surg.* 19(1): 30-34.
- Goldstein, E.J.C. 1992. Bite wounds and infection. *Clin Infect Dis.* 14(3): 633-638.
- Griffin, G.M. and Holt, D.E. 2001. Dog-bite wounds: Bacteriology and treatment outcome in 37 cases. *J Am Anim Hosp Assoc.* 37(5): 453-460.
- Griffiths, I.R., Duncan, I.D. and Lawson, D.D. 1974. Avulsion of the brachial plexus-2: clinical aspects. *J Small Anim Pract.* 15(3): 177-183.
- Hahn, C.N. 2003. Horner's syndrome in horses. *Equine Vet Educ.* 15(2): 86-90.
- Herrera, H.D., Suranit, A.P. and Kojusner, N.F. 1998. Idiopathic Horner's syndrome in collie dogs. *Vet Ophthalmol.* 1(1): 17-20.
- Kamata, H., Yanai, Y., Imaizumi, K., Fukuda, Y. and Murasugi, E. 1988. Oral flora of the dog. *J Jpn Vet Med Assoc.* 41(8): 549-554.
- Kelly, P.J., Mason, P.R., Els, J. and Matthewman, L.A. 1992. Pathogens in dog bite wounds in Harare, Zimbabwe. *Vet Rec.* 131(20): 464-466.
- Kern, T.J., Aromando, M.C. and Erb, H.N. 1989. Horner's syndrome in dogs and cats: 100 cases (1975-1985). *J Am Vet Med Assoc.* 195(3): 369-373.
- Lambrechts, N.E. and Berry, W.L. 2000. Caudal Cervical Disc Protrusion in a Bengal Tiger (*Panthera tigris tigris*). *J Zoo Wildl Med.* 31(3): 404-407.
- Melián, C., Morales, M., Espinosa de los Monteros, A. and Peterson, M.E. 1996. Horner's syndrome associated with a functional thyroid carcinoma in a dog. *J Small Anim Pract.* 37(12): 591-593.
- Meyers, B., Schoeman, J.P., Goddard, A. and Picard, J. 2008. The bacteriology and antimicrobial susceptibility of infected and non-infected dog bite wounds: Fifty cases. *Vet Microbiol.* 127(3-4): 360-368.
- Montiani Ferreira, F. and Peterson-Jones, S.M. 2002. Neuro-ophthalmology. In: *BSAVA Manual of Small Animal Ophthalmology.* 2nd ed. S.M. Petersen-Jones and S.M. Crispin (eds). England: British Small Animal Veterinary Association. 257-275.
- Morgan, R.V. and Zanotti, W. Horner's syndrome in dogs and cats: 49 cases (1980-1986). *J Am Vet Med Assoc.* 194(8): 1096-1099.
- Mouro, S. and Vilela, C.L. 2010. Clinical and bacteriological assessment of dog-to-dog bite wounds. *Vet Microbiol.* 144(1-2): 127-132.
- Mughal, M. and Longmuir, R. 2009. Current pharmacologic testing for Horner syndrome. *Curr Neurol Neurosci.* 9(5): 384-389.
- Nahn CN. 2006. Miscellaneous disorders of the equine nervous system: Horner's syndrome and polyneuritis. *Equine Clin Tech Equine Pract.* 5(1): 43-48.
- Obradovich, J.E., Withrow, S.J., Powers, B.E. and Walshaw, R. 1992. Carotid body tumors in the dog: Eleven cases (1978-1988). *J Vet Intern Med.* 6(2): 96-101.
- Ofri, R. 2008. Neuroophthalmology. In: *Slatter's Fundamentals of Veterinary Ophthalmology.* 4th ed. D. Maggs, P. Miller and R. Ofri (eds). Pennsylvania: WB Saunders Company. 318-351.
- Peace, L.W., Wallace, L., Rosenfeld, C.S. and Sansone, J. 1997. Intracranial squamous cell carcinoma causing Horner's syndrome in a cow. *J Vet Diagn Invest.* 9(1): 106-108.
- Pinard, C.L., Brightman, A.H., Yeary, T.J., Everson, T.D., Cox, L.K., Chengappa, M.M. and Davidson, H.J. 2002. Normal Conjunctival Flora in the North American Opossum (*Didelphis virginiana*) and Raccoon (*Procyon lotor*). *J Wildl Dis.* 38(4): 851-855.
- Saphir, D.A. and Carter, G.R. 1976. Gingival flora of the dog with special reference to bacteria associated with bites. *J Clin Microbiol.* 3(3): 344-349.
- Shamir, M.H. and Ofri, R. 2007. Comparative neuro-ophthalmology. In: *Veterinary Ophthalmology.* 4th ed. K.N. Gelatt (ed). Ames: Blackwell Publishing. 1406-1469.
- Shamir, M.H., Leisner, S., Klement, E., Gonen, E. and Jonston, D.E. 2002. Dog bite wounds in dogs and cats, a retrospective study of 196 cases. *J Vet Med A.* 49(2): 107-112.
- Simoens, P., Lauwers, H., Muelenaere, C., de Muylle, E. and Steenhaut, M. 1990. Horner's syndrome in the horse: a clinical, experimental and morphological study. *Equine Vet J.* 22(10): 62-65.
- Smith, J.S., Mayhew, I.G. 1977. Horner's syndrome in large animals. *Cornell Vet.* 67(4): 529-542.
- Talan, D.A., Citron, D.M., Abrahamian, F.M., Moran, G.J. and Goldstein, E.J.C. 1999. Bacteriologic analysis of infected dog and cat bites. *N Engl J Med.* 340(2): 85-92.
- Van den Broek, A.H.M. 1987. Horner's syndrome in cats and dogs - a review. *J Small Anim Pract.* 28(10): 929-940.
- Waldron, D.R. and Zimmerman-Pope, N. 2002. Superficial skin wounds. In: *Textbook of small animal surgery.* 2nd ed. D. Slatter (ed). Pennsylvania: W.B. Saunders Company. 259-

273.

Walton, K.A. and Buono, L.M. 2003. Horner's syndrome. *Curr Opin Ophthalmol.* 14(6): 357-363.

Williams, D.L. and Cooper, J.E. 1994. Horner's syndrome in an African spotted eagle owl (*Bubo africanus*). *Vet Rec.* 134(3): 64-66.