Short communication

Clinical case report of oesophagostomiasis in captive Hamadryas Baboon (*Papio Hamadryas*) in Ethiopia

Rea Tschopp 1, 2* and Jakob Schneider 3

¹Armauer Hansen Research Institute, Jimma Road, PO Box 1005, Addis Ababa, Ethiopia

² Swiss Tropical and Public Health Institute, Socinsstr 57, 4002 Basel, Switzerland

³Addis Ababa University, Medical Faculty, Department of Pathology, Ethiopia

*Corresponding author: Armauer Hansen Research Institute, Jimma Road, P.O. Box 1005, Addis Ababa, Ethiopia, Email: rea.tschopp@unibas.ch

http://dx.doi.org/10.4314/evj.v20i1.10

Abstract

Oesophagostomum spp is a nodule causing nematode found in livestock and primate species in Sub-Saharan Africa. The L3 infectious larvae invade the colonic wall, forming nodules in which the larvae continue their development. It can be a serious zoonotic risk in some areas in Africa. Primates are natural hosts for the parasite and do in general not show any symptoms. We present here a clinical case of Oesophagostomiasis in a captive hamadryas baboon (Papiohamadryas) in Ethiopia who died of acute peritonitis as a result of the infection with the parasite. Post-mortem was performed and colon tissue analyzed by cytology. This case highlights the importance of Oesophagostomiasis in captive baboons that undergo stress full events and the need for adequate management.

Keywords: PapioHamadryas, Oesophagostomiasis, Ethiopia

Introduction

Oesophagostomum spp are nodule-causing namatodes from the Strongylidae family, shown to frequently infect intestines of livestock such as pigs and ruminants as well as non-human primates who are believed to be natural hosts (Ghai et al., 2014). They occur in Brazil, China, Indonesia and Philippines.In Africa, the disease is mainly found in northern Togo and Ghana (West Africa) in an endemic state with severe zoonotic implications (Polderman et al., 1995;

Gasser et al., 2005). However, the agent was also isolated from other countries in people and/orprimates such as Uganda, Togo, Congo and Gabon (Gigase et al., 1987; Krief et al., 2010; Ghai et al., 2014) suggesting that Oesophagostomiasis is probably more common in Sub-Saharan Africa than previously thought. Oesophagostomum spp has been isolated in feces from cercopithecinae, colobinae and hominidae (Mengistu Legesseand and Erko, 2004; van Lieshout et al., 2005; Krief et al., 2008; Makouloutou et al., 2014; Ota et al., 2015). Eight species of Oesophagostomum have so far been recorded in wild primates, with the 3 most common being O. bifurcum, O. stephanostomum and O. oculeatum. All three are zoonotic but O. bifurcum seems to be the more prevalent species to regularly infect humans (Yelifari et al., 2005; Gigase et al., 1987; Gasser et al., 2005).

The infection in animal and people occur with the ingestion of L3, the infective larvae that contaminates water, food, soil and dust and is very resistant outside the host (Krepel, 1994). After ingestion, L3 enter the intestinal mucosa and form tiny nodules in which it develop into L4. L4 remains either in the nodules or goes back into the lumen to develop into adults that copulate and shed eggs via feces (Gasser *et al.*, 2005). In people it can cause serious intestinal disease (Storey *et al.*, 2000; Ziem *et al.*, 2005). There are two distinct clinical forms: the "dapaong tumor", which is a uninodular disease with a single large painful mass formed around encapsulated worms, leading to complications such as abscessing or intestinal occlusion (Polderman *et al.*, 1995; Poldermann *et al.*, 1998). The less common multinodular disease within the colonic wall and other intra-abdominal structures causes pain, weight loss and chronic diarrhea (Storey *et al.*, 2000).

In primates, the disease is most often asymptomatic. However, it has been shown to be pathogenic in situations of stress (captivity, transportation) (Steward and Gasbarre, 1989). We present here a clinical case of oesophagostomiasis in a captive hamadryas baboon (*P. hamadryas*) in Ethiopia.

Material and Methods

Signalment and history

The patient was a sub-adult captive male Hamadryas baboon (*P. hamadryas*), from unknown origin within Ethiopia. He has been kept in captivity in Addis

Ababa, as a single pet animal for an unknown time; he was then confiscated and relocated into another captive facility 30 km outside Addis Ababa (rescue and rehabilitation center) before a planned release into the wild. The animal was aggressive and stressed upon arrival and could not be mixed with other Hamadryas baboons. After approximately 6 months, he was moved into an enclosure with 5 other Hamadryas baboon of similar age. He showed indifference to the others and remained mostly by himself, cohabiting in friendly terms. The other baboons then started bullying him with increasing frequency leading to a physical separation 2 weeks later into an enclosure adjacent to the previous one. He stayed there for a couple of weeks not showing any clinical signs. He was suddenly found very weak and in hypothermia one morning, 3 months after having joined the group. After treatment was initiated (glucose *per os*, warm blankets) he recovered and was eating by early afternoon, and behaving normally. By late afternoon, however, the animal started becoming drowsy and was found dead an hour later.

Post-mortem examination

External examination

The animal's body condition was fair and there were no visible signs of discharges from anus, mouth or nose or of enlarged superficial lymph nodes. Tail and foot pads showed several small ulcerative injuries. The animal was still in rigor mortis at the time of the autopsy however, there was a striking rictus, eyes were wide open and both hands in tight claw position.

Internal examination

In-situ examination of the abdominal cavity showed an accumulation of approximately 150 ml of tea-colored liquid within the abdomen. Abdominal organs did not show any visible abnormalities with the exception of the colon. The entire length of the colon showed blue-ish discoloration with a striking pattern of dark-blue. Miliary pea-size (3-5 mm) dark-blue nodules could be seen on the serosa, embedded within the colon wall or protruding from the serosal (Figure 1).



Figure 1: In-situ post-mortem examination. Note the multiple dark nodules (thick black arrows), the ascites and the hypperaemic mesenterium

Some were also found protruding from the mucosa side and were of pale grey color. Mesenterium was partly hyperaemic, partly blue-ish colored. Mesenterial lymph nodes were enlarged and visible as garlands covering the whole colon but also spleen and pancreas. Distal ileum and colon lumen contained only few numbers of nematodes. The thoracic cavity did not show any abnormalities. The diagnosis upon post-mortem examination was acute peritonitis with associated lymphadenomegaly, ascites, and multi-nodular colonic changes.

Laboratory analysis

A segment of 4 cm of the ascending colon was fixed in 10% formalin, embedded in paraffin, cut by microtome (5 micrometer) and stained with hematoxylin-eosin (HE).

Results

Gross pathological examination showed slightly elevated bluish lesions visible on both the mucosal and serosal surface. Nodules varied in diameter between 3 and 5 mm and did not reach the mucosa. Most were well delimited blackish nodules, filled with coagulated blood (Figure 2).



Figure 2: Slices of parts of the formalin fixed colon tissue. Well delimited dark nodules between 3 and 5 mm in diameter, sometimes protruding on the serosa and lumen side $\frac{1}{2}$

Upon microscopy of the colon segment, lesions were well delimited consisting of abscess like cavities with varying amount of erythrocytes. The surrounding tissue was edematous and dense with inflammatory infiltrates such as neutrophils, lymphocytes and macrophages with abundant eosinophilic cytoplasm (Figure 3A).



Figure 3A: Cross-section of the colon of a hamadryas baboon (P. hamadryas) showing two well delimited but not capsulated helminthomas. The abcess-like cavities are filled with erytrocytes and contain parts of a helminth (arrows). Helinthomas are surrounded with dense inflammatory infiltrates and oedema. The mucosa was intact.

Few of them reached the serosa, perforating into the peritoneal cavity. Most of these cavities included cross-sections of a helminth, which diameter ranged from 200 to 300 microns. The helminths's outer cuticle and internal spaces were often poorly preserved. However, in some slides the large platymyrian muscle cells, the thick muscled esophagus and the intestines with brushed borders could be seen clearly (Figure 3B).

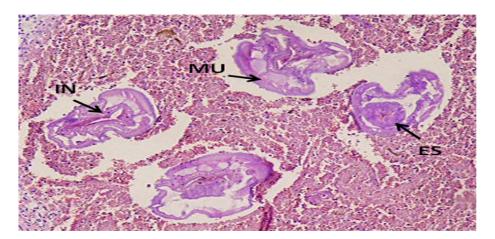


Figure 3B: Numerous cross-section of a nematod larvae within the colon wall. Note the large muscled esophagus (ES), the large platymyrian muscle cells (MU) and the intestine with brush borders (IN)

Discussion

The post-mortem, histological pictures and morphological characteristics were consistent with helminthomas caused by *Oesophagostums*pp, a typical nodular disease in livestock and primates. To our knowledge this is the first reported case of clinical Oesophagostomiasis in a captive Hamadryas baboon (*P. hamadryas*) in Ethiopia. Several studies have though isolated *Oesophagostomum*eggs and/ or larvae from feces from sheep and goat in the country (Regassa Fikru *et al.*, 2006; Bersissa Kumsa *et al.*, 2011). Likewise, the parasite was isolated from feces in one study from 10.2% of olive baboons (*P. Anubis*) and 4.9% of vervets (*C. aethiops*) (Mengistu Legesse and Erko, 2004). However, the true prevalence of *Oesophagostomum*spp in Ethiopia is largely unknown because the parasite is difficult to diagnose from feces alone since its eggs look identical to eggs of hookworms, *trichostrongylus* spp and *ternidensdeminutus*.

Coproculture and allowing the egg to develop through all development stages to L3 increases diagnostic accuracy but is time consuming, unreliable and needs skilled laboratory staff. Recently, new PCR techniques have been giving new insights in the taxonomy and epidemiology and host-specific variants of the parasite (Verweij et al., 2000; Ota et al., 2015; Gasser et al., 2005; van Lieshout et al., 2005). The epidemiology of the disease however, remains still largely unknown in Sub-Saharan Africa and Ethiopia in particular and further in-depth studies need to be carried out to assess parasite variants and zoonotic threat. In the present study we could not identify with certainty the species of *Oesophagostomum* involved.

Oesophagostumum spp is highly prevalent amongst primates and remains usually asymptomatic and hosts tolerate well the presence of nodules. Most nematodes leave the nodules before the onset of clinical disease and develop in the lumen into adult worms who produce eggs that then appear in feces (Krief et al., 2008). However, in stressful events such as capture, transportation or mixing with new animals and/or captive situations, the disease can swiftly progress into clinical manifestation with diarrhea, weight loss and mortality. For instance, newly obtained primates for laboratory purpose accounted for the highest mortality within days of arrival due to Oesophagostomumspp as described in the 80s (Steward and Gasbarre, 1989).

In our case, the animal underwent various stressful events within several months (restrains, various transportations, new environments, mixing with new animals). It is interesting that he never showed any diarrhea even in the final stages of the disease and he has not lost weight. This is also supported by the fact that nematode burden in the intestinal lumen was minimal. Clinical manifestations are likely due to the inability of the parasites to leave the nodule and continue their normal development in the colonic lumen (Ziem et al., 2005). The exact pathological mechanism leading to this scenario is still unknown but it seems evident that stress factors have a huge contribution. In our case, nodules perforated the intestinal serosa and lead to a fatal acute peritonitis.

This clinical case highlights the importance of stress management in captive primates and that Oesophagostomiasis and its potential lethal consequences should be taken serious consideration when dealing with primate rescues, captive management, rehabilitation and transportations in Ethiopia since all these events are linked with some level of stress. Also changes in environment may play a role for instance when an animal from lowland arid habitat such as for example Hamadry as baboons being moved into a highland colder habitat. These environmental shifts can affect the immunity of the animal and/or lead to contacts with other parasite species found in the new environment for which the animal is immuno-naive.

Conclusion

Finally, this case also highlights potential Zoonotic risks of *Oesophagostomum* spp infection. More research needs to be carried on that topic in Ethiopia. Staff dealing directly with captive primates is at risk but also communities living in close proximity with free-ranging primates and sharing their habitats as it is often the case in many places in Ethiopia.

Acknowledgments

We thank the Born Free Foundation in Ethiopia and the staff of Ensesekoteh, particularly Zelealem Tefera, Bereket Girma and Tilahun Bayena

References

- Fikru, R., Teshale, S., Reta, D. and Yoseph, K., 2006. Epidemiology of gastrointestinal parasites of ruminants in Western Oromia. *Int. J. Applied Res. Vet. Med.*, 4, 51-57.
- Gasser, R.B., De Gruijter, J.M. and Polderman, A.M., 2005. Insights into the epidemiology and genetic makeup of Oesophagostomum bifurcum from human and non-human primates using molecular tools. *Parasitol.*, 132, 453–460.
- Ghai, R.R., Chapman, C.A., Omeja, P.A., Davies, T.J. and Goldberg, T.L., 2014. Nodule worm infection in humans and wild primates in Uganda: Cryptic species in a newly identified region of human transmission. *PLoS Negl. Trop. Dis.*, 8(1), e2641.
- Gigase, P., Baeta, S., Kumar, V. and Brandt, J., 1987. Frequency of symptomatic human oesophagostomiasis (helminthoma) in northern Togo. In: Geerts, S., Kumar, V. and Brandt, J. (Eds.), Helminth zoonoses. Martinus Nijh off Publishers, Dordrecht/Boston/Lancaster. pp. 228-236.
- Krepel, H.P., 1994. *Oesophagostomumbifurcum* infectionin man: a study on the taxonomy, diagnosis, epidemiology and drug treatment of *Oesophagostomumbifurcum* in northern Togo and Ghana. PhD Thesis Leiden University, The Netherlands.

- Krief, S., Jamart, A., Mahe, S., Leendertz, F.H., Matz-Rensing, K., Crespeau, F., Bain, O. and Guillot, J., 2008. Great apes: does self-medication in wild apes influence disease progression. J. Med. Primatol., 37(4), 188-195
- Krief, S., Vermeulen, B., Lafosse, S., Kasenene, J.M., Nieguitsila, A., et al., 2010. Nodular worm infection in wild chimpanzees in Western Uganda: A risk for human health. *PLoS Negl. Trop. Dis.*, 4(3), e630.
- Kumsa, B., Tadesse, T., Sori, T., Duguma, R. and Hussen, B., 2011. Helminths of sheep and goats in Central Oromia (Ethiopia) during the dry season. *J. Ani. Vet. Adv.*, 10(4), 1845-1849.
- Makouloutou, P., Mbehang Nguema, P.P., Fujita, S., Takenoshita, Y., Hasegawa, H., Yanagida, T. and Sato, H., 2014. Prevalence and genetic diversity of Oesophagostomum stephanostomum in wild lowland gorillas at Moukalaba-Doudou National Park, Gabon. Helminthol., 51(2), 83-93.
- Legesse, M. and Erko, B., 2004. Zoonotic intestinal parasites in *Papioanubis* (baboon) and *Cercopithecus aethiops* (vervet) from four localities in Ethiopia. *Acta. Trop.*, 90, 231–236.
- Ota, N., Hasegawa, H., McLennan, M.R., Kooriyama, T., Sato, H., Pebsworth, P.A. and Huffman, M.A., 2015. Molecular identification of Oesophagostomumspp. From village chimpanzees in Uganda and their phylogenetic relationship with those of other primates. *R. Soc. Open Sci.*, 2, 150-471.
- Polderman, A.M. and Blotkamp, J., 1995. Oesophagostomum infections in humans. *Parasitol. Today.*, 11(12), 451-456.
- Polderman, A.M., Anemana, S.D. and Asigri, V., 1999. Human Oesophagostomiasis: A regional public health problem in Africa. *Parasitol. Today.*, 15(4), 129-130.
- Stewart, T.B. and Gasbarre, L.C., 1989. The veterinary importance of nodular worms (*Oesophugostomumspp*). *Parasitol. Today.*, 5(7), 209-213.
- Storey, P.A., Faile, G., Hewitt, E., Yelifari, L., Polderman, A.M. and Magnussen, P., 2000. Clinical epidemiology and classification of human oesophagostomiasis. *Trans. R. Soc. Trop. Med. Hyg.*, 94, 177–182.
- Van-Lieshout, L., de-Gruijter, J.M., Adu-Nsiah, M., Haizel, M., Verweij, J.J., Brienen, E.A.T., Gasser, R.B. and Polderman, A.M., 2005. *Oesophagostomumbifurcum* in non-human primates is not a potential reservoir for human infection in Ghana. *Trop. Med. Int. Health.*, 10(12), 1315-1320.
- Verweij, J.J., Polderman, A.M., Wimmenhove, M.C. and Gasser, R.B., 2000. PCR assay for the specific amplification of *Oesophagostomum bifurcum* DNA from human faeces. *Int. J. Parasitol.*, 30, 137-142

- Yelifari, L., Bloch, P., Magnussen, P., Van Lieshout, L., Dery, G., Anemana, S., Agongo, E. and Polderman, A.M., 2005. Distribution of human *Oesophagostomum bifurcum* hookworm and *Strongyloides stercolaris* infections in northern Ghana. *Trans. R. Soc. Trop. Med. Hyg.*, 99, 32–38.
- Ziem, J.B., Spannbrucker, N., Magnussen, P., Olsen, A., Nii Amon-Kotey, D., Frenzel, K., Nang-Beifubah, A., Westendorp, R.G.J. and Polderman, A.M., 2005. Oesophagostomumbifurcum-induced nodular pathology in a highly endemic area of northern Ghana. Trans. R. Soc. Trop. Med. Hyg., 99, 417–422.