A case of Amidostomosis in a racing pigeon (Columba livia) in Greece

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Περιστατικό αμιδοστόμωσης σε ταχυδρομικό περιστέρι (Columba livia) στην Ελλάδα

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ABSTRACT. A dead pigeon (Columba livia) was submitted to the Unit of Avian Medicine, Clinic of Farm Animals, Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece. It derived from a pigeon flock consisted of approximately 100 racing birds of various ages between 4 months to 5 years old. The flock was kept on a terrace indoors in 4 cages of 3 m² each. Near this flock, there were also two other pigeon flocks in a distance of 20 and 30 meters, respectively. The pigeons' health problem had been present in this flock for approximately 3 years before this delivery. Lack of appetite, poor growth of young birds, weakness, depression, vomiting, diarrhea and emaciation were the most important clinical signs of the pigeons in the flock. During necropsy, discoloration and atrophy of liver, spleen and kidneys, edema of gizzard, necrosis in the koilin layer of the gizzard and under this, presence of small parasites, dilatation in the anterior...
INTRODUCTION

Amidostomosis is a chronic parasitic disease of the gizzard in birds (Haralampidis, 2003). The disease is mainly associated with waterfowl (Kavetska et al., 2015), such as geese, ducks and swans (Papazahariadou et al., 1994; Tuggle and Friend, 1999; Papazahariadou et al., 2008). However, the relevant parasites have also been secluded from other species of birds, such as pigeons, chicken (experimental infection) and many other migratory birds (Bowman, 1999; Cole and Friend, 1999; Saif et al., 2008). Mammals are not infected (Bowman, 1999).

The nematodes of the genus Amidostomum spp. are the causative agent of amidostomosis. In Europe, there are six common species of this genus i.e. Amidostomum acatum, A. anseri, A. cygni, A. fulicae, A. henryi, A. spatulatum (Tuggle and Friend, 1999). There are also other species, such as A. boschalis (Haralampidis, 2003). These parasites infect the upper alimentary tract of the birds, particularly, the solid layer of a carbohydrate-protein complex called the koilin layer of the gizzard and less frequently the proventriculus (S. van Riper and C. van Riper, 1985; Taylor et al., 2007).

The life cycle is similar among the Amidostomum species (Saif et al., 2008). More precisely, it is direct, which means that the infective parasitic larva (L3) invades a single host animal and it develops until its reproductive maturity (Tuggle and Friend, 1999). The infection takes place when the bird ingests the L3 larva which is found in the surface of greens and in ponds or when the L3 larva invades the skin of the bird during its swimming. Subsequently, in the first case the infective larva reaches the gizzard through the alimentary tract, whilst in the second case migration of the L3 larva takes place via the lungs and the liver. The larva reaches in reproductive maturity under the gizzard stratum corneum in approximately 15-28 days (in chicks) or in up to 33 days (in adult birds). The parasitic eggs, which are already embryonated are shed through the feces in the outer environment. Subsequently, after its formation,
the L1 larva develops to L3 larva inside the egg in 2-6 days (in 16-27oC) and after that the egg hatches and the L3 larva comes to the outer environment (Haralampidis, 2003; Taylor et al., 2007). The infective larva survives in the environment for 30 days (in 25οC) or even for 3 months (in 2oC) (Haralampidis, 2003). What is more, it can swim actively in water of ponds for up to 30 days (in 5-10oC) and it is able to reach the surface from depths of up to 10cm (Enigk and DeyHazra, 1970).

During the life cycle, the *Amidostomum* spp, attack the koilin layer of the gizzard of their hosts’ (Macklin, 2013). Particularly, the migration and the development of L3 larva as well as the feeding of the blood-sucking adult worm (0.1-0.4 ml blood/adult A. anseris/day) lead to hemorrhage in gizzard and severe erosions of gizzard lining (Beynon et al., 1996; Tuggle and Friend, 1999; Haralampidis, 2003). Large amounts of worms (greater than 35) may denude the whole surface lining of the gizzard, making the edges of the grinding pads degenerate and finally separate from the underlying tissue (Tuggle and Friend, 1999). In addition, enteritis is quite possible to happen, too (Beynon et al., 1996).

There are no pathognomonic signs of amidostomosis in birds, indicating the gizzard parasitism. However, in cases of heavy infection of young birds some symptoms may appear, such as poor growth, loss of appetite, dullness, emaciation, diarrhea, anemia due to blood loss, change of expected liver seize and even considerable mortality (Kobulej, 1983; Beynon et al., 1996; Tuggle and Friend, 1999; Jordan et al., 2001; Saif et al, 2008).

In other words, the heavy parasitism can induce a chronic debilitating disease and the weakness can be so intense that the aquatic birds cannot hold their neck above water (MacNeil, 1970). Furthermore, birds of all ages may be subject to emaciation and general weakness, too (Tuggle and Friend, 1999). However, according to Herman and Wehr (1954), A. anseris itself is not a primary source of loss but rather an important contributing factor (van Riper and van Riper, 1985).

Consequently, amidostomosis may be an important risk factor in some countries and it should not be neglected (Kobulej, 1983).

**CASE HISTORY**

This report describes a case of amidostomosis in a pigeon derived from a pigeon flock, which was located in Neapoli, a region of the western part of Thessaloniki. The pigeon (*Columba livia*) was submitted for necropsy to the Unit of Avian Medicine, Clinic of Farm Animals, Faculty of Veterinary Medicine, Aristotle University of Thessaloniki, Greece. It was a male bird aging 3 years and hatched in this flock. The flock consisted of approximately 100 racing pigeons of various ages from 4 months to 5 years old. These birds were placed on a terrace indoors, in 4 big cages of 3m² each. Near this flock, there were also 2 other pigeon flocks in a distance of 20 meters and 30 meters respectively. An adequate ration special for racing pigeons as well as fresh water were provided to these birds twice per day. Moreover, cleaning of their environment used to take place on a daily basis.

What is more, during autumn 2014 these pigeons participated in racing competitions and the last vaccination against *Paramyxovirus* took place a year before the submission of the dead pigeon to the clinic of Avian Medicine. The pigeons’ health problem had been present in this flock for approximately 3 years before this submission. The symptoms manifested in this flock were not typical of any particular disease. Lack of appetite, poor growth of young birds, weakness, vomiting, diarrhea and emaciation were the most important of them. Furthermore, it should be mentioned that 30 birds had died the previous month.

Post mortem examination of the pigeon revealed discoloration and atrophy of the liver, spleen and kidneys, edema of the gizzard, necrosis in the koilin layer of the gizzard and under this, presence of small parasites. Furthermore, dilatation was also observed in the anterior small intestine (duodenum, jejunum), whilst the second half of small intestine and the rectum were full of hemorrhagic content. Macroscopic lesions were not found in other organs, while the bacteriological examination of liver and spleen was negative. Direct microscopy of smears was negative for *Histomonas* spp.

The parasites and the relative infected tissues were transferred to the Laboratory of Parasitology, Veterinary Faculty of Aristotle University of Thessaloniki for parasite recovery. The nematodes
were collected, cleaned with saline and identified under the stereoscope using morphological identification keys provided by Taylor et al (2007). The worms were identified to be *Amidostomum* spp. According to the findings of the postmortem and laboratory examination, amidostomosis was determined to be the cause of the pigeon’s death.

**DISCUSSION**

Amidostomosis is arguably a common infection in birds, such as geese (Kobulej, 1983), ducks (Borgsteede, 2005) and other aquatic birds (Taylor et al., 2007). In Greece the parasite has been found in swans (Papazahariadou et al., 1994), geese and ducks (Papazahariadou et al. 2008), but not in pigeons, so far. Therefore, taking into account 1) that the infection by *Amidostomum* spp. is more common in aquatic birds and 2) that the pigeon examined derived from a racing pigeons flock in a terrace potentially in contact with aquatic birds which travel to the lakes around the region of Thessaloniki, it is readily understood that this contact maybe a major risk factor of this case of pigeon amidostomosis.

In addition, seasonality is not normally associated with this parasitism in migratory aquatic birds, because these birds become firstly exposed on breeding grounds and then they continue to be exposed throughout their lives (Tuggle and Friend, 1999). However, the problems of this pigeon flock and the case of the pigeon amidostomosis happened during autumn, which is the main period of migration for many aquatic species. In other words, this is also the most possible period for the contact between pigeons and wild aquatic birds. Thus, migration period of aquatic birds could be another possible risk factor of our amidostomosis case.

In order to deal with this case of pigeon amidostomosis, both chemotherapeutics and managing practices should be taken. The treatment of clinical or suspected subclinical cases of amidostomosis can be based on medical substances, such as cambendazole (60 mg/kg) against adult worms and larvae, pyrantel (100 mg/kg) against adult worms, citarin (40mg/kg), mebendazole (10mg/ kg) and fenbendazole. However, toxicity has been reported in pigeons that received fenbendazole per os at the rate of 30 mg/kg for 5 days. Furthermore, flubendazole and ivermectin have been proven to be effective against this parasitic infection as well as albendazole and piperazin (Baker, 2007; Islam et al. 2012; Macklin, 2013).

However, apart from chemotherapeutic treatment, the control of amidostomosis depends on managing practices, as well. Particularly, these practices should focus on the disruption of the parasite’s life cycle. In detail, the possibility of the parasite transmission is greater in crowded and continuously used habitat, because the combination of accumulative fecal contamination and warm ambient temperatures (20-25° C) promote the quick larval development. Additionally, newly hatched birds are least resistant to infection and birds of all ages are susceptible to reinfection. Hence, proper sanitation and good managing practices are of the utmost importance (Tuggle and Friend, 1999; Baker, 2007).

Although, human health considerations have not been reported so far, people who eat gizzards of relative birds (including pigeons) should cook them thoroughly, discard pigeons’ appearing relative clinical signs or lesions and take all the appropriate treating and managing measures to control the prevalence of the parasite and its consequences in their flocks (Tuggle and Friend, 1999).

Although *Amidostomum* spp. has been reported in aquatic birds in Greece, this is the first case of *Amidostomum* spp. in racing pigeons. Therefore, further epidemiological studies are needed, in order to estimate its prevalence in pigeon flocks in different regions of Greece and to elucidate the route of transmission to pigeons.

**CONFLICT OF INTEREST**

The authors of this paper certify that they have no affiliations with or involvement in any organization or entity with any financial interest, or non-financial interest in the subject matter or materials discussed in this manuscript.

**ETHICAL CONSIDERATIONS**

On behalf of all authors it is certified that legal and ethical requirements have been met with regards to the humane treatment of animals described in the study.
Figure 1: Edema of the gizzard, necrosis in the koilin layer and presence of numerous parasites in a racing pigeon (*Columba livia*).

Figure 2: Necrosis and presence of numerous parasites under the koilin layer of the gizzard in a racing pigeon (*Columba livia*).
REFERENCES


