Fading Puppy Syndrome Associated with *Toxocara canis* Infection

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Abstract

Aim of this work was studying the role of *Toxocara canis* infection in fading puppy syndrome and evaluating its treatment. Twelve German shepherd puppies suffered from fading puppy syndrome and their dam were presented. The dead puppies (N.=8) were necropsied. On the other hand, living puppies (N.=4) and their dam were examined both clinically and parasitologically. The collected worms from necropsied puppies were parasitologically identified as *Toxocara canis* and their counts ranged from 48 to 75 per puppy. The live cases were treated by Flubendazole. The parasitological examinations of stool of live puppies and dam revealed eggs identified as *Toxocara canis* eggs and their counts ranged from 50 to 350 eggs per gram stool. Post-treatment counts of *Toxocara canis* worms in stool of live puppies and their dam were ranged from 13 to 79 worms per animal. The results of treatment proved the efficacy of Flubendazole for eradication of *Toxocara canis*. *Toxocara canis* considers one of the causes of fading puppy syndrome and could be cured by Flubendazole.

**Keywords:** *Toxocara canis*; death; puppy; diagnosis; treatment

Introduction

Fading puppy syndrome (FPS) is defined as death of puppies within the first two weeks of life and the puppies are born of good average birth weight and gradually lose the suckling reflex, become inactive and die (Goldberg, 2011; Blunden, 2012; Bowman, 2004). Mortality rates of puppies may reach 10-30% by weaning and 15-40% in the first 12 weeks of life (Gill, 2001). Infectious causes of fading puppy syndrome are bacteria, viruses and parasites. Worm burdens can be able to obstruct the small intestine of puppies. Puppy deaths post the fourth day of age can be due to the migrating ascarid *Toxocara canis*. It was reported that as many as 90% of puppies are affected prenatally with *T. canis* (Evans, 1978). The roundworm larvae can cross the placenta and then transmitted in colostrum and milk (Goldberg, 2011). In addition, eating raw cow liver may transmit *Toxocara canis* (Dongil et al., 2012). Adult worms of *T. canis* live in the small intestine of dogs and their puppies. The infested dog may shed 200000 eggs/day, that take two to five weeks to become infective eggs containing larvae (Glicknan and Schantz, 1981). Signs of toxocariasis are variable and may include digestive, respiratory and sometimes nervous symptoms. The signs result from both acute and chronic inflammatory responses that elicited against toxocaral excretory and secretory products (Kerr-Muir, 1994). Toxocariasis is a serious zoonotic disease causes serious problems. (Idalia et al., 2012). In dogs, the prevalence of *Toxocara canis* was reported to be greater in puppies (56.1%) than in mature animals (11.9%) (Malloy and Embil, 1978).

Neonatal deaths may caused by *Toxocara canis* infection as a result of the presence of large worms burden that obstruct gastrointestinal tract or migrate up the biliary tract and cause hepatitis (Ettinger and Feldman, 2012). In this study, *Toxocara canis* infection as a probable cause of fading puppy syndrome was aimed. Therapeutic trial was also achieved.

Materials and methods

Animals

Twelve German shepherd puppies and their dam...
were examined during this work at the teaching hospital of Veterinary Medicine at Department of Medicine and Infectious Diseases, Faculty of Veterinary Medicine, Cairo University. The puppies were 12 days old and suffered from anorexia, emaciation, hypothermia and death. The dead puppies (N.=8) were necropsied. The living puppies (N.=4) and the dam were clinically and parasitologically examined and were treated by Flubendazole at 20 mg/ml (Fluvermal®, Janssen Pharmaceutica) followed by oral administration of paraffin oil. The treatment regime was evaluated by both clinical and parasitological examinations.

**Stool samples**

The stool samples were collected from living puppies before and after treatment and then examined parasitologically using concentration flotation techniques as previously described (Urquhart et al., 2000). Eggs of *Toxocara canis* were counted in the stool samples of live puppies and dam by master technique as previously carried out (Coles, 1986).

**Post-mortem examination of the dead puppies**

Eight dead puppies were examined. Small intestines were necropsied and the worms were collected, identified and counted (Jones et al., 1997).

**Therapy**

The living puppies and their dam were treated using Flubendazole at a dose of 60 mg per puppy and 100 mg for dam once daily for three successive days then repeated after one week (Kerr-muir, 1994). Five ml of Paraffin oil was drenched 24 hours post-treatment. Therapy was evaluated by clinical improvement and stool analysis including eggs counting before and after treatment.

**Results**

**Clinical findings**

The live puppies showed fever (39.4 °C), coughing, dyspnea, pot belly, pale mucous membranes, diarrhea, laziness, emaciation and severe pain, which observed as arched back, tensed abdomen, frequent crying and abnormal gait.

**Postmortem findings**

There were gastroenteritis, pneumonia, liver congestion, enlarged mesenteric lymph node, obstruction of small intestine (Figs. 1 and 2), intestinal rupture, peritonitis and presence of adult worms of *T. canis* in the small intestine (duodenum).

Table 1. Worm burdens and eggs counts in puppies suffered from FBS.

<table>
<thead>
<tr>
<th>Puppies sequence</th>
<th>Status</th>
<th>Worm burdens Identification</th>
<th>Number</th>
<th>Pre-treatment</th>
<th>Post-treatment (two week)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>53</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>48</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>67</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>58</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>75</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>61</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>71</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>Dead</td>
<td><em>T. canis</em></td>
<td>68</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>Live</td>
<td><em>T. canis</em></td>
<td>50</td>
<td>250</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>Live</td>
<td><em>T. canis</em></td>
<td>47</td>
<td>350</td>
<td>0</td>
</tr>
<tr>
<td>11</td>
<td>Live</td>
<td><em>T. canis</em></td>
<td>63</td>
<td>150</td>
<td>0</td>
</tr>
<tr>
<td>12</td>
<td>Live</td>
<td><em>T. canis</em></td>
<td>79</td>
<td>250</td>
<td>0</td>
</tr>
<tr>
<td>Bitch</td>
<td>Live</td>
<td><em>T. canis</em></td>
<td>13</td>
<td>50</td>
<td>0</td>
</tr>
</tbody>
</table>
Worm burden

The worm burdens from the necropsied and treated puppies and dam were counted and illustrated in Table 1 and Fig. 3. The parasitological examinations of the worms revealed that the worms were *Toxocara canis* (Duprey and Schantz, 2003).

Stool analysis

*Toxocara canis* egg was identified based on Conboy (1996).

Therapy

The four treated puppies and dam were clinically improved at four weeks post-treatment that was clear in the disappearance of fever (39.4 °C), coughing, dyspnea, pot belly, pale mucous membranes, diarrhea, laziness, emaciation and pain. The dead *Toxocara canis* worms were passed with the stool after treatment. The *Toxocara canis* eggs were completely disappeared two weeks post-treatment.
**Discussion**

*Toxocara canis* is a serious parasite because of its zoonotic importance as it could infect human via ingestion of food and water contaminated with embryonated eggs and also through eating raw cow liver that contains *Toxocara canis* larvae (Dongil et al., 2012).

Clinical infection of *Toxocara canis* causes variable clinical signs including fever (39.4°C), coughing and dyspnea that could be explained by hepatopulmonary migration from the small intestine to the lung that causes pneumonia. While pot belly is usually observed in *Toxocara canis* infected puppies and it may be caused by intestinal distension with worms burdens and ascities resulted from hypoproteinemia. The pale mucous membranes, laziness and emaciation are usually caused by anemia that resulted from deficiencies of iron, cobalt and other elements consumed by parasite. Diarrhea is a signs to enteritis that is caused by migration of larvae and presence of adult worms in the small intestine.

Pain is clinically observed on the infected puppy as arched back, tensed abdomen, frequent crying and abnormal gait. That could be explained by occurrence of intestinal obstruction, intestinal rupture and peritonitis.

*Toxocara canis* larvae could migrate through the placenta so the puppies are born infected. Also puppies may acquire *Toxocara canis* infection through the dam’s milk or colostrum (Zimmer and Pollock, 1987). Hypobiotic larvae of *Toxocara canis* serve as a reservoir of infection in pregnant bitches. They become reactivated during the last third of pregnancy and many of them enter the uterus or mammary gland, where they infect the fetus or puppy.

Transmission can occur repeatedly to each subsequent litter, without reinfection of the dam. Parasites acquired in utero enter the fetal liver, migrate through the lungs, and develop into adults after approximately 3 weeks. Most of the larvae ingested in the milk do not migrate through the tissues, but complete their development in the intestines. Some bitches develop patent infections during lactation, either from the movement of hypobiotic larvae to the intestines or from the ingestion of larvae from the feces of their puppies (OIE, 2005).

*Toxocara canis* infected puppies may die from obstruction of the gall bladder, bile duct and pancreatic duct, rupture of the intestine and peritonitis, pneumonia, ascites, fatty degeneration of the liver, and may be due to myocarditis. The affected puppies may die with 3 week after birth (OIE, 2005). Clinical toxocariasis in adult dogs is rare (OIE, 2005), which was clear in the dam that showed the lowest counts of eggs and worms.

The therapeutic trial with flubendazole was effective and indicated that both clinical and parasitological improvements of the treated cases. Flubendazole is effective against *Toxocara canis* infection, as it interferes with cellular tubulin formation in the worm, thus disturbing glucose uptake and the normal digestive functions of the worm to such an extent that an autolytic process occurs. Paraffin oil is given to expel the dead *Toxocara canis* worms to avoid hypersensitivity and toxicity caused by autolysis of those worms. The cure of four living pups and dam after treatment is confirmed by improving of clinical signs, killing worms and completely disappearing eggs in stool samples.

**Conclusion**

*Toxocara canis* could infect pups intra-uterine causing digestive and respiratory manifestations. The untreated infected puppy usually dies after birth. *Toxocara canis* burdens considered one of the causes of FPS. The flubendazole was effective to treat *Toxocara canis* infection and save life of the infected puppies. The efficacy of flubendazole to treat *Toxocara canis* infection was evaluated by improving of clinical signs, killing of worms and disappearing eggs in the stool.

**References**


