Journal of the Egyptian Society of Parasitology, Vol. 51, No. 3, December 2021
J. Egypt. Soc. Parasitol. (JESP), 51(3), 2021: 603 – 616
(Online: 2090-2549)

SOME MISCELLENOUS ZOONOTIC NEMATODES: A REVIEW

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Abstract

The miscellaneous nematodes are *Angiostrongylus costaricensis,* anisakiasis, *Baylisascaris procyonis*, capillariasis, dirofilariasis, dracunculiasis, and trichostrongyliasis. Man usually acquire infection by ingesting these eggs via contaminated food or water, which hatch in the small intestine and release larvae that penetrate intestine and migrate to lungs a few days later. But, thelaziasis is an Arthropod-born disease by non-blood sucking flies or filthy flies.

With increasing globalization, parasitic infection of the central nervous system (CNS), once considered a "tropical" infection, is becoming increasingly more prevalent in all parts of the worldwide. The immunosuppression increases susceptibility to opportunistic parasitic infection. Although infected individuals may remain asymptomatic for many years, a higher parasite burden is correlated with greater morbidity and mortality.

Key words: Miscellaneous nematodes, Distribution, Pathogenicity, Diagnosis, Treatment.

Introduction

The nematodes (roundworms) reviewed included 1- Angiostrongyliasis, 2- Anisakiasis, 3- *Baylisascaris procyonis,* 4- Capillariasis, 5- Dirofilariasis, 6- Dracunculiasis, and 7-Trichostrongyliasis (Wu *et al,* 1997).

The eosinophilic meningitis is a rare clinical entity that can be useful in narrowing the differential diagnosis of CNS disease, and is the presence of 10 or more eosinophils/microl in the cerebrospinal fluid (CSF) or a CSF eosinophilia of at least 10% (Lo Re and Gluckman, 2003). The commonest helminthic parasites that invade CNS are *Angiostrongylus cantonensis* and *Gnathostoma spinigerum* (also, *Baylisascaris procyonis*), but other infections and noninfectious conditions may also be associated. Both, *A. cantonensis* and *G. spinigerum* caused severe CNS compromise (Ramirez-Avila *et al,* 2009).

Review, Discussion and Conclusion

1- *Angiostrongylus cantonensis*: *Angiostrongylus costaricensis* is a filariform (Nematoda: Angiostrongylidae) or called rat lungworm causes eosinophilic enterocolitis, a granulomatous inflammatory reaction within intestinal wall. Appendix, distal small bowel, or right colon may be involved. Eosinophilic gastroenteritis is a rare digestive disease characterized by triad of eosinophilic infiltration of segments of gastrointestinal tract, gastrointestinal abnormalities varied frodyspepsia, obstruction to diarrhea and ascites (Gonsalves, 2019). These disorders can be concomitant-esophageal eosinophilia associated with increased risk of gastric, duodenal, and colonic eosinophilia, in many patients, more than 1 gastrointestinal region contained eosinophilia (Dellon *et al,* 2020). Besides, *A. cantonensis* is the most common cause of human eosinophilic meningitis and meningoencephalitis (Baheti *et al,* 2009).

Life cycle of *A. costaricensis* begins with eggs laid by adult worms in the mesenteric arterioles of rats, the definitive hosts. The first-stage larvae passed in the stool and ingested by a snail or slug (intermediate host). Life cycle is completed with mollusk ingestion by the rat, and the third-stage larvae (infective larvae) migrate to the ileocecal area (Baird *et al,* 1987). This nematode worm may cause of

Humans can acquire the infection by eating raw or undercooked snails or slugs infected with the parasite; infection may also be transmitted by raw produce contaminated with larva-containing slug secretions. Alternatively, infection can be transmitted by ing-

estion of infected paratenic animals, such as crab or freshwater shrimp. In humans, worms migrate to the mesenteric arteries and release eggs into the intestinal tissues. Intense endothelial damage with subsequent arteritis and thrombosis can ensue, with necrosis of adjacent enteric tissue. The parasite usually dies in the gastrointestinal tract; eggs are not shed in stool (Kramer *et al,* 1998). *A. costaricensis* was reported most frequently from Central and South America, especially Costa Rica, the United States and elsewhere (Loria- Cortés and Lobo-Sanahuja, 1980).

Clinical manifestations: Many infections are asymptomatic. Depending upon the severity of the inflammatory reaction within the bowel wall, fever, abdominal pain, anorexia, vomiting, constipation, bowel obstruction, mesenteric ischemia, or perforation can develop. Presentation with recurrent gastrointestinal bleeding has also been described (Waisberg *et al,* 1999). If the eosinophilic infiltrate occurs in the ileocecal region, the clinical symptoms can mimic appendicitis. However, the presentation is often more indolent than is typical for acute appendicitis, and episodes may recur over several months.

A tumor-like mass is usually palpable on abdominal examination (Silvera *et al,* 1989). Rarely, eggs and larvae can be carried to extraintestinal sites (Rodriguez *et al,* 2008). When occurred, mesenteric lymph node enlargement, testicular involvement with pain and erythema, or hepatic lesions with tender hepatomegaly (Vázquez *et al,* 1994).

Diagnosis: A definitive diagnosis may be established by identifying parasite on histopathological examination of biopsies or surgical resections. Adults measure 2 to 3.5cm. Stool examinations were not helpful since neither eggs nor larvae appeared in stool, but stool examination may be useful to exclude other parasitic causes, particularly *E. vermicularis* (Graeff-Teixeira *et al,* 1997). ELISA and PCR assays were developed (Geiger *et al,* 2001). High grade peripheral eosinophilia (up to 20 to 50%) occurred and may help to differentiate this infection from acute appendicitis (da Silva *et al,* 2003). Other causes of eosinophilic enteritis include anisakiasis, enterobiasis, toxocariasis, and ancylostomiasis *caninum* (Liu *et al,* 1995). Eamsobhana *et al*. (2006) in Bangkok reported that a multi-dot ELISA was developed as rapid & simple differential diagnosis of eosinophilic meningitis due to helminthic infections. Ultrafiltered, purified antigens of *A.* (= *Parastrongylus*) *cantonensis, Gnathostoma spinigerum* and *Taenia solium* metacestodes, the commonest parasites to invade CNS and cause eosinophilic pleocytosis were dotted onto a single nitrocellulose membrane strip.

Treatment: Most patients have a self-limited course and usually can be observed with supportive care in the absence of specific medical or surgical therapy. In some cases, surgery was pursued to exclude appendicitis or other causes of pathology. Antiparasitic agents have not proven efficacious in any clinical trial. It may be best to avoid anthelminthic administration if diagnosis was clear, as therapy might lead to erratic migration followed by worsening of the disease (Pérez *et al,* 2009).

In Egypt, Yousif and Lämmler (1975) evaluated the role of 16 species of aquatic snails of four families were by quantitative technique under standardized conditions for their suitability as intermediate hosts for *Angiostrongylus cantonensis*. They reported that all snail species proved to be susceptible to infection with *A. cantonensis*, and first stage larvae reached the infective third stage in all of them. El Shazly *et al*. (2002) In Dakhalia Governorate found *Bulinus truncatus, Lymnaea natalensis, L. alexandrina, and Cleopatra cyclostomoides* were found naturally infected with the larvae of *Parastrongylus cantonensis*. Abo-Madyan *et al.* (2005) in Fayoum Governorate detected the immature stages of the nematode parasite *Angiostrongylus* (=*Parastrongylus*) *cantonensis* in the fresh water snail *Physa acuta.* Ibrahim (2007) at Al-Salam irrigation Canal and Al-Abtal village (North Sinai G.) found that *L. carinatus, Cleoptra bulimoides, C. cyclostomoides, Bu-*

linus alexandrina, L. natalensis and *Melania tuberculta* were naturally infected with *A. cantonensis* larvae in prevalence that ranged from 0.63 to 2.24%.

2- Anisakiasis: Anisakiasis is a zoonotic roundworm infection (Anisakidae) caused by *Anisakis simplex* sensu stricto, *A. physeteris*, *A. pegreffii, A. berlandi* (=*A. simplex* C), the *Pseudoterranova decipiens* complex (*P. decipiens* sensu stricto, *P. azarasi, P. cattani,* and others), and *Contracecum osculatum* complex, or *Pseudoterranova* species.

Marine mammals (whales, sea lions, seals, dolphins, porpoises, and walruses) are natural hosts; humans are incidental hosts. Human anisakiasis equivalent is ascariasis (Audicana and Kennedy, 2008). Anisakiasis was described in many regions involved in fish and marine mammals, but commonly in Japan, likely as frequent ingestion of raw fish (Berger and, Marr, 2006), also, known as sushi worm or herring worm (*Anisakis* sp.), or cod worm (Pseudoterranova sp.).

Anisakiasis life cycle of begins with passage of unembryonated eggs in the stool of marine mammals. In the water, first- and second-stage larvae are formed; subsequently, these are ingested by crustaceans and migrate to muscle tissues. The larvae are transferred to fish and squid via predation, which maintain third-stage larvae that are infective to humans and marine mammals. Upon ingestion by marine mammals, the larvae develop into adult worms, which become embedded in the stomach mucosa and produce eggs shed in the stool (CDC, 2019).

Humans become infected by eating undercooked or raw infected fish. Salmon, herring, cod, mackerel, and squid transmit *Anisakis* species; halibut, cod, and red snapper transmit *Pseudoterranova* species. Larvae are grossly visible in the fish; properly trained sushi chefs can detect them. After ingestion, the larva (usually one or two) penetrates the human gastric and intestinal mucosa. Maturation begins, but the parasite dies because it is not in its natural host. Dying organism induces an inflammatory reaction and a tissue abscess develops with a pre-dominance of eosinophils. In some cases, larvae perforate the intestinal wall and form an abscess within the peritoneal cavity, but thorough cooking to 70ºC or adequate freezing -20ºC for a minimum of 72hrs are the best preventive measures (Hochberg and Hamer, 2010).

Clinical manifestations: Most symptoms associated with anisakiasis were due to direct tissue damage or due to an allergic reaction (Caramello *et al,* 2003). Immediately after ingestion of infected raw fish, some individuals develop pruritus and tingling of the posterior oropharynx. Gastric anisakiasis usually develops one to eight hours after ingestion of raw fish and is characterized by acute epigastric pain, nausea, and vomiting. Intestinal anisakiasis usually develops up to a few days following ingestion of raw fish and may be associated with severe abdominal pain, abdominal distension, and a palpable inflammatory mass that causes intestinal obstruction (Park *et al,* 2008). Diarrhea with blood or mucus may also develop.

Eosinophilic gastroenteritis or enterocolitis can also occur. A syndrome mimicking appendicitis may occur if ileocecal region was involved (López-Serrano *et al,* 2000). Symptoms are vague and the illness can be misdiagnosed as appendicitis, acute abdomen, stomach ulcer, or ileitis. *Anisakis* larvae occasionally penetrate into the peritoneal cavity or other visceral organs (extraintestinal anisakiasis) and cause eosinophilic granuloma confused with neoplasm (Nawa *et al*, 2005).

Allergic reactions ranging from mild urticaria to anaphylactic shock can occur (Lorenzo *et al,* 1999). Associated fever and peri-pheral eosinophilia are common. Symptoms usually arise acutely; a chronic relapsing course can also occur, which may mimic seafood allergy (Daschner *et al,* 1998). Seafood allergy is also common in patients allergic to shellfish to react clinically to more than one type of shellfish. Extensive cross-reactivity among shellfish is explained by the fact that invertebrate tropomyosin is the pan allergen with significant sequence homology throug-

hout many invertebrate species, including crustaceans, mollusks, squid, octopus, parasites, and non-marine insects; such as cockroach (Kang *et al,* 1979), bedbugs (Abou-Gamra *et al*, 1991), fleas (El Okbi *et al,* 1991), ants (Sanad *et al,* 2002), grasshoppers (Taylor, 2008), dust mites and biting lice (Morsy *et al,* 2001) and mosquito Skeeter syndrome (Abdel Motagaly *et al,* 2017). However, there are clearly other factors that influence cross-reactivity, as homology cannot be used to predict an individual patient's sensitivity patterns. Invertebrate tropomyosins demonstrate less homology with vertebrate tropomyosins (Ayuso *et al,* 1999).

Differential diagnosis of small bowel obstruction due to anisakiasis includes tumor, Crohn's disease, primary eosinophilic gastroenteritis, other parasitic infections (*Strongyloides, Ascaris, Toxocara,* & *Ancylostoma*), bacterial infections (*Yersinia*, TB), intussusception, and ischemia (Ramos *et al,* 2005).

Diagnosis: The diagnosis can be made via visualization of worm recovered from emesis or by endoscopy (approximately 2 to 2.5 cm x 1 to 2mm). These larvae are grossly visible to the naked eye of the endoscopist. Endoscopic examination may demonstrate an ulcerated bleeding lesion in the stomach or duodenum with a worm at the center. Barium studies may demonstrate narrowing of the intestinal lumen in areas with mucosal inflammation. A thread-like filling defect suggesting a worm is visualized in some cases (Matsui *et al,* 1985). Total and *Anisakis*-specific IgE levels were elevated, particularly in patients who develop an allergic reaction following infection (Daschner *et al,* 1999). ELISA and immunoblot tests were developed for anisakiasis diagnosis (Iglesias *et al,* 1997). An antigen-capture ELISA reported sensitivity and specificity near 100% (Lorenzo *et al,* 2000). PCR tests were developed (Chen *et al,* 2008).

Treatment: Physical removal of the parasite (by regurgitation, endoscopy, or surgery) is curative. Symptomatic therapy is usually adequate if the worm is in the distal bowel and cannot be retrieved by endoscopy, since *Anisakis* larvae can only survive for a few days in the human intestinal tract. Surgery may be necessary for worms that have penetrated the intestine, omentum, liver, or pancreas (Sakanari and McKerrow, 1989). Successful treatment was reported by Albendazole 400mg orally twice daily for 3 to 5 days, but diagnosis was presumptive (Pacios *et al,* 2005).

In Egypt, Mostafa *et al*. (2020) found that the Mediterranean horse mackerel had the high larval mean intensity of 20 larvae/fish.

3- *Baylisascaris procyonis*: Zagers and Boersema, 1998) in Germany wrote that *B. procyonis* is an ascarid that parasitizes the small intestine of raccoons, which was not so pathogenic in the raccoon as larvae don't migrate in this host. They added that in other animals, larvae migrate through the body, but do not develop into adults in the intestine, only become encysted in granulomas with a preference in the brain. In man the larvae cause different larva migrans syndromes or neural larva migrans syndrome with severe brain symptoms, which sometimes fatal. Sorvillo *et al.* (2002) in USA reported that *B. procyonis*, a roundworm infection of raccoons (*Procyon lotor*), is emerging as an important helminthic zoonosis, principally affecting young children. Raccoons have increasingly become peri-domestic animals in close proximity to human residences. When *B. procyonis* eggs are ingested by a host other than a raccoon, migration of larvae via tissues (larval migrans), invade the brain and eye, causing severe disease and death. The prevalence of *B. procyonis* infection in raccoons is often high, and infected animals can shed enormous numbers of eggs in their feces. Eggs can survive for extended times in the environment and the infectious dose of *B. procyonis* is relatively low. Timm *et al,* (2016) in the United Kingdom reported that the raccoon (*Procyon lotor*), or common raccoon, is a medium-sized mammal native to North America, Europe, the Caucasus, Japan, and Russia, having a body length of 40

to 70cm, and a body weight of 5 to 26kg. Its grayish coat consists of dense under-fur that insulates it against cold weather. Three of the raccoon's most distinctive features are its extremely dexterous front paws, its facial mask, and its ringed tail, which are themes in mythologies of indigenous Americans related to animal. They added that it is nocturnal omnivorous, eating 40% invertebrates, 33% plants, & 27% vertebrates. CDC (2018) reported that *Baylisascaris* in raccoons can infect people as well as a variety of other animals, including dogs. Human infections are rare, but can be severe if the parasites invade the eye (ocular larva migrans), organs (visceral larva migrans) or brain (neural larva migrans). French *et al.* (2019) in Canada reported that the *B. procyonis*, the roundworm of raccoons (cities and forests) is an emerging helminthic zoonosis in North America. Since the larval form is capable of causing neurological dise-ase in more than 150 species of birds and mammals including man. They added that by understanding factors that influence carriage of the parasite by raccoons is important for mitigating risk.

4- Capillariasis: There are two major clinical syndromes of capillariasis, an intestinal disea- se (*Capillaria philippinensis*) and hepatic disease (*C. hepatica*). Intestinal capillariasis occurs most frequently in the Philippines and Thailand, but was observed in other countries. Rare cases of human infections with *C. hepatica* were reported worldwide.

Intestinal capillariasis: Intestinal capillariasis is caused by *C. philippinensis*, a parasite of fish-eating birds (which seem to be the natural definitive host). Adult worms of *C. philippinensis* reside in the bird and human intestinal tracts. Unembryonated eggs are passed in the stool and become embryonated in fresh water where they are ingested by fish. Subsequently larvae hatch, penetrate the fish intestine, and migrate to muscle tissues. Ingestion of raw or under-cooked fish results in bird and/or human infection.

Unembryonated eggs can also become embryonated in the human intestine; released larvae can cause autoinfection, leading to hyperinfection; a massive number of adults (Grencis and Cooper, 1996). Thus, exposure to even a small parasite load can result in massive infection.

Clinical manifestations: Symptoms associated with intestinal capillariasis include chronic watery diarrhea, mal-absorption, and wasting. Fever, abdominal pain, and peripheral eosinophilia may also be present. As parasite burden increases, mal-absorption can become very severe. Electrolyte abnormalities and protein loss result, led to marked cachexia and cardiomyopathy. Untreated infection led to death within a few months (Cross *et al,* 1972).

Diagnosis and treatment: Diagnosis of intestinal capillariasis is by detecting characteristic eggs in stool specimens. Eggs measure 45 by 20microns with plugs at each end. Larvae may also in stool specimens. Treatment for intestinal capillariasis consists of Albendazole® (400mg once daily for 10 days) or mebendazole (200mg twice daily for 20 days).

Supportive therapy with fluids and nutritional supplements may also be required. Relapse is common, particularly if patients do not complete the full course of therapy; in such cases, re-treatment may be necessary (Cross, 1992). Optimal management of relapse is uncertain; based on case reports retreatment with course of Albendazole® for 20 days or Mebendazole® for 30 days was reasonable (Belizario *et al,* 2010).

Hepatic capillariasis: Hepatic capillariasis is caused by *C. hepatica*, a parasite of rodents, dogs, pigs, and other mammals. Hepatic capillariasis was described worldwide. Human infection is rare or misdiagnosed; infection is most common among children <3 years of age (Pampiglione and Gustinelli, 2008). Infection is acquired by ingesting eggs in contaminated food, water, or soil. The life cycle begins with ingestion of embryonated eggs by a suitable mammalian host. Larvae are released in the intestine and migrate via the portal vein to the liver, where they

mature into adults and lay eggs in the parenchyma after about four weeks. Occasionally, larvae migrate to the lungs, kidneys, or other organs (Klenzak *et al,* 2005).

Eggs didn't pass in the stool of the host; as they remain in the liver until the animal dies or is eaten by a predator. Eggs ingested by scavengers or predators are passed in the stools of these animals and embryonate in the environment after about 30 days.

Clinical manifestations: Symptoms include acute or subacute hepatitis associated with fever, hepatomegaly, and marked eosinophilia (Sawamura *et al,* 1999). Inflammation and/or fibrosis of liver can result, which clinically may resemble toxocariasis visceral or ocular larva migrans (Morsy, 2020).

Diagnosis and treatment: The diagnosis of capillariasis in humans is usually made by finding adults and eggs in biopsy or autopsy specimens. Eggs are not passed in the stool of the host. Identification of *C. hepatica* eggs in stool reflects spurious passage of ingested eggs and is not diagnostic of clinical infection. Optimal treatment was unknown. However, Albendazole® & Thiabendazole® were successfully used in a few cases (Choe *et al,* 1993).

In Egypt, *C. yamaguti* was identified as a new species from the Egyptian Nile fish *bagrus bayad* (Tadros and Mahmoud, 1964). *C. hepatica* was recovered in *R. norvegicus* trapped in Dakahlia Governorate (El Shazly *et al,* 1994), and Ahmed *et al.* (1999) reported *C. philippinensis* in 4 cases a male & 3 females aged 12-45 years presented with severe diarrhea of 1-7 months duration associated with vomiting and central abdominal colics. El-Dib and Doss (2002) reported 44 cases (37 females & 7 males), with ages ranged from 10 to 65 years mainly in Bani-Suif and El-Menia Governorates. El Karaksy *et al.* (2004) in El Menia G. reported *C. philippinensis* in 2 young sisters of a fisherman with persistent profuse watery diarrhea of 1 year duration. Both sisters were hypoalbuminemic, hypokalemia and hyponatremia but, only the younger had pedal edema and treated successfully with albendazole 200mg twice daily. El Shazly *et al.* (2008) in Dakahlia and Menofia Governorates reported *C. hepatica* in domestic rodents mainly *Rattus norvegicus.* Khalifa *et al.* (2020) detected *Capillaria* spp. in stray cats with 3% prevalence and added that Copro-DNA proved a satisfactory sensitive and specific test for clinically suspected patients. They concluded that intestinal capillariasis must be considered in chronic diarrhea and hypoalbuminemia patients since if they were misdiagnosed and untreated, they would suffer fatal complications.

5- Dirofilariasis: Dirofilariasis is caused by a zoonotic filarial nematode. The life cycle of dirofilariasis begins when an infected mosquito species (*Aedes, Culex, Anopheles,* & *Mansonia*) takes a blood meal, introducing third-stage (L_3) filarial larvae. Usually a domestic dog or coyote is infected although a wide variety of other animals can be infected including cats, weasels, aquatic mammals, beaver, horses, and humans. The L_3 larvae molt into L4 larvae and then adults that reside in the subcutaneous tissues (*D. repens*) or the heart of the definitive host (*D. immitis*). In humans, *D. immitis* filaria lodge in pulmonary arteries and never mature into fully gravid worms (Jelinek *et al*, 1996). In definitive hosts, adult worms can live for 5 to 10 years. The female worms are capable of producing microfilariae which circulate in the peripheral blood and are ingested by a mosquito during a blood meal. In the mosquito's abdomen the microfilariae develop into L_1 and subsequently into L_3 larvae that can infect another host when the mosquito takes a blood meal (Fleck *et al,* 2009).

Dirofilariasis is particularly common in the Mediterranean region, but was described in many regions, including the United States, Europe, and Asia (Kim *et al,* 2002).

Clinical manifestations: There are two major clinical syndromes: pulmonary dirofilariasis (caused by *D. immitis*) and subcutaneous dirofilariasis (caused by a few different dirofilarial species). In addition to these syn-

dromes, there have been a few reports of human dirofilariasis in other sites, such as the peritoneal cavity, male genital tract, liver, or central nervous system (Poppert *et al,* 2009). A peripheral eosinophilia of about 10% was seen (Flieder and Moran, 1999).

Pulmonary dirofilariasis: Pulmonary dirofilariasis is caused by *D. immitis* (also known as the dog heart worm since it commonly cause of congestive heart failure in dogs). In definitive hosts, adult worms of *D. immitis* live in the heart. In humans, larvae lodge in small caliber pulmonary arteries and never mature into fully gravid worms. The organisms can cause infarcts or granulomas that may result in the appearance of nodules or cavities on chest radiography (Hiroshima *et al,* 1999). The radiographic appearance is often described as a "coin lesion" that is usually 1 to 3cm in diameter and can be confused with the lung tumor. Most human infections are asymptomatic; often infection is discovered when chest imaging is performed for some other reason. Some patients may develop chest pain, cough, hemoptysis, fever, and malaise (Solaini *et al,* 2008).

Subcutaneous dirofilariasis: Subcutaneous dirofilariasis is caused by a few different dirofilarial species, including *D. repens, D. tenuis*, and others. These species are parasites of dogs and cats (*D. repens*), raccoons (*D. tenuis*), or other mammals. Adults can develop in humans, but sexual maturity and production of microfilariae do not occur since humans are an incidental host.

Skin lesions consist of a coiled, degenerating worm in subcutaneous tissues, typically around the eye or on the genitalia or limbs (Khoramnia and Wegner, 2010). Usually, the worm is encased in dense fibrous tissue. The nodule can be erythematous and tender, and may be associated with an abscess. Concomitant allergic symptoms including urticaria and fever may also develop (Arvanitis *et al,* 1997).

Diagnosis: Definitive diagnosis of dirofilariasis requires biopsy of the involved tissue for histopathologic identification. *Dirofilaria* are 400 to 500microns in diameter and characterized by a thick multilayered cuticle with longitudinal ridges, lateral cords, and internal lateral thickening of the cuticle (Tzanetou *et al,* 2009). In one series of 60 patients with solitary pulmonary nodules presumed caused by *D. immitis*, 90% contained 1 worm; occasionally 2 or 3 worms were present in the same nodule (Ciferri, 1982). Serology using either ELISA or IHAT is not well standardized or widely used (Glickman *et al*, 1986). PCR is used for diagnosis (Rivasi *et al,* 2006)

Treatment: Treatment consists of a simple extraction or complete surgical excision of the worm. No specific medical therapy for dirofilariasis is required. Usually the nematode lesions calcify without treatment (Khurana *et al*, 2010). Some suggested the administration of Ivermectin® (150mcg/kg PO as a single dose) followed by diethylcarbamazine (2mg 3 times daily for four weeks) to destroy any other worms that might be clinically silent (Marusi *et al,* 2008). But, the efficacy was questionable and this treatment was usually not necessary.

In Egypt, Abdel-Rahman *et al.* (2008) in Assiut Governorate reported 3 *D. repens* including living worms from the left lung of pulmonary human cases. Antibodies to *D. immitis* antigens were observed in sera of 6/174 (3.4%) feral cats (*Felis catus*) collected from Cairo (Al-Kappany *et al,* 2011). Elsayad *et al.* (2012) in Alexandria reported two locally acquired human cases of dirofilariasis *repens.* They were 36 years old male presented to the surgical outpatient clinic complaining of a painful nodule in his right arm. The second case was a 23 years old woman presented to the dermatologist with a nodule on the right side of the chest. Diagnosis was done by one worm was extracted from each nodule and identified parasitological, histological, and by SEM. They concluded that *D. repens* human infections have become increasingly recognized worldwide as inadvertent human pathogens. Meanwhile Mikhail *et al.* (2009) in Egypt reported the abundan-

ce of eight species of C*ulex,* three species of *Aedes,* 12 species of *Anopheles* and one species of *Culiseta.* Besides, Shoukry and Morsy (2011) and El Bahnasawy *et al.* (2011) reported the introduction of mosquito-borne diseases to Toshka District and Aswan Governorate respectively from South Sudan.

6- Dracunculiasis: *Dracunculus medinensis* (Linnaeus, 1758), which was considered by Gallandant, 1773; as an important human parasite for most of recorded history, and one of the best known human pathogens since antiquity (Muller, 1971). Dracunculiasis (also known as Guinea worm) is caused by *Dracunculus medinensis.* D. *medinensis* larvae were found in stationary water bodies such as ponds, large, open wells (with stairs), or rain-filled cisterns (Muller, 1979), where they were ingested by copepods (small crustaceans) of genus *Cyclops* in which *D. medinensis* larvae develop to an infective comma-shaped stage within 14 days (WHO, 2015). Animal reservoirs were domestic dogs in Chad, Ethiopia, and Mali, and domestic cats in Chad and Ethiopia, as well as wildcats and baboons that were a potential problem for dracunculiasis eradication program (Durrant *et al,* 2020).

Humans are the only known host of *D. medinensis*; infection is transmitted by consumption of unfiltered water containing copepods (small crustaceans) infected with larvae of *D. medinensis*. Following ingestion, the copepods die and release larvae penetrate host stomach and intestinal wall; and then they enter abdominal cavity and retroperitoneal space (Greenaway, 2004). After maturation into adult worms, males die and females (70 to 120cm in length) migrate in the subcutaneous tissues. Approximately a year after infection, the fertilized female worm migrates to the skin surface and induces a painful papule (usually on the distal lower extremity, but may occur on the genitalia, buttocks, or trunk). Sebai and Morsy (1975) in Saudi Arabia extracted *D. medinensis* from the supra-sternal notch from an immigrant youth Yemeni. One or more worms emerges and the patient experiences a burning sensation. When the patient soaks leg in fresh water to relieve the discomfort, and the worm releases larvae into the water. Larvae are ingested by a copepod and become infective after two weeks (and two molts). Human ingestion of the copepods completes the cycle. In some cases, the worms die before they can emerge through the skin. In such cases, the worms eventually calcify and may be detected incidentally on radiographs or may be palpable beneath the skin.

Clinical manifestations and diagnosis: Just prior to the formation of the skin papule, systemic symptoms can develop including fever, urticaria, pruritus, dizziness, nausea, vomiting, and diarrhea. The papule measures 2 to 7cm and pain is severe as the worm emerges. This clinical manifestation is the basis for diagnosis. A peripheral eosinophilia may be present.

Rarely, worm can migrate to ectopic sites, such as the lung, eye, pericardium, or spinal cord, and can produce abscesses at these locations. Secondary infections can le-ad to systemic sepsis. Chronic arthritis or paraplegia and contractures can develop, particularly if the worm migrates via joint or finds its way into central nervous tissue, where aseptic abscesses and cystic swelling occur when worms rupture before emerging, causing an acute inflammatory response-from the host's immune system (Roberts *et al,* 2012).

Treatment: Treatment consists of slow extraction of the worm combined with wound care and pain management (CDC, 2018). The worm should be wound around a stick, extracting a few centimeters each day. It may take many weeks or months for the entire worm to be removed. If the worm is broken or not fully extracted, an intense, painful inflammatory reaction with swelling along the worm tract can develop (Sutton and Canyon, 2015). Whitty (2014) mentioned that the ecological changes occurred in Guinea worm may be the equivalent of the evolution of drug resistance in chemotherapy-lead campaigns. He concluded that pos-

sibility for one generation to eradicate a disease is very motivating, and very difficult. The many failed eradication attempts outnumber the one current success (smallpox), although two eradication campaigns for polio and Guinea worm are tantalizingly close to their goals. Early stages of a well-planned eradication campaign generally go well; it is the last stage where technical, biological, social and political problems occur.

Epidemiology & prevention: Watts (1998) reported that Sudan, the country which has the largest number of cases today, is Egypt's southern neighbor. Because of the nature of the disease (in endemic areas it is most common among poor, rural people), it may not have come to attention of urban-based health personnel. In the period before the details of the transmission cycle were known, the attitudes and mindsets of physicians and travellers also have to be taken into account in interpreting written reports of the disease. An examination of documentary sources from the nineteenth and twentieth centuries in European languages does not show any clear evidence for dracunculiasis transmission in Egypt during that period. Cases noted in Egypt, especially by the quoted Dr. Clot Bey (1820s), most likely originated beyond the borders of the country, in Sudan and, to a lesser extent, from endemic areas in the Middle East. However, many later commentators merely repeated what Clot Bey had written. A further difficulty is that some published reports which apparently concern dracunculiasis in Egypt, actually refer to cases in animals rather than humans.

Dracunculiasis occurs commonly among adults in rural settings. This parasite has caused substantial morbidity in many parts of Africa, Yemen, and India. Infection was completely eradicated from Asia (Hopkins *et al,* 2000), and an intense eradication program between 1986 and 2010 reduced the number of countries with endemic dracunculiasis from 20 to 3 in Ethiopia, Mali, and South Sudan (Barry, 2007). Niger and Nigeria reported zero indigenous cases for the first time in 2009. Few infections are reported outside an endemic focus in war-torn Sudan, where control programs have been hampered CDC, 2011a).

Safe drinking water supplies can prevent infection (Hopkins, 1983). Community surveillance and education regarding the mode of transmission is important for control. Other strategies include using of nylon filters for drinking water to remove copepods, use of insecticides in drinking water sources to kill copepods, covering papules with occlusive dressings, and covering drinking water sources so that infected individuals do not immerse infectious papules to propagate infection. CDC (2001) reported that Guinea worm eradication campaign showed a great success, but only 74,258 cases of GWD in 15 countries in sub-Saharan Africa that had fallen to just 15 cases in each of Chad and Ethiopia as of 2017 (Hopkins *et al,* 2018). However, only Angola, Chad and South Sudan reported human dracunculiasis cases (CDC, 2020). Sutton and Canyon (2015) reported that the success of these eradication programs was contingent upon many factors. Nothing is assured, and progress remains fragile and vulnerable to setbacks. Security must be ensured in Guinea worm transmission areas in Africa and polio transmission areas in Pakistan and Afghanistan. Technical solutions alone cannot guarantee eradication. Grassly and Orenstein (2018) reported that either Guinea worm disease or polio would soon become the second human disease to be eradicated, and this nematode worm is on track to be the first to be wiped out without a vaccine, and probably the first animal species to be deliberately made extinct.

7- Trichostrongylosis is caused by several nematodes of the *Trichostrongylus* species which infect sheep, cattle, and other herbivorous mammals worldwide; humans are incidental hosts. Eggs are passed in the stool of the definitive host (usually an herbivorous mammal), and rhabditiform larvae hatch within several days; they become infective

filariform (third-stage) larvae after 5 to 10 days (and two molts). Infection is transmitted to humans by ingestion of these larvae, which mature into adults in the small intestine. Trichostrongylosis is typically transmitted by ingestion of unwashed vegetables fertilized with contaminated manure (CDC, 2011b).

Occasionally, infection can occur via larval penetration of the skin. Larvae mature to adults in the small intestine, where they embed in the mucosa and cause inflammation.
Clinical manifestations: Most infections are asymptomatic. In the setting of heavy infection, abdominal pain, diarrhea, and anemia can develop. Mal-absorption and wasting can ensue if mucosal damage is severe. A peripheral eosinophilia may be observed.

Diagnosis: Trichostrongylosis diagnosis is generally established by identifying characteristic eggs in the stool. Stool concentration techniques may be needed, particularly in the setting of light infections. The diagnosis can also by identification of characteristic Trichostrongylus larvae or adult worms on endoscopic evaluation of the duodenum.

Treatment of trichostrongylosis consists of mebendazole (100mg twice daily for 3 days) or albendazole (400mg orally once). Also, Pyrantel pamoate (11mg/kg up orally once; maximum dose of 1g) was successfully used (Boreham *et al,* 1995).

Conclusion and Recommendations

Angiostrongylus costaricensis causes eosinophilic enterocolitis. Life cycle is transmission between rodents and snails or slugs; humans are incidental host. Diagnosis may be established by identifying the organism on histopathological examination of biopsies or surgical resections. Most patients have a self-limited course and usually observed with suportive care in absence of specific medical or surgical therapy.

Anisakiasis causes gastroenteritis or enterocolitis. Life cycle is transmission between marine mammals, crustaceans, fish, and squid; humans are incidental hosts. Diagnosis by visualization of the worm recovered from emesis or by endoscopy. Physical removal of parasite (by regurgitation, endoscopy, or surgery) is curative.

C. philippinensis causes intestinal capillariasis. Life cycle is transmission between birds and fish; humans are incidental hosts. Diagnosis was by characteristic eggs in stools. Treatment with albendazole or mebendazole recommended.

Dirofilariasis consists of two major clinical syndromes: pulmonary dirofilariasis (*D. immitis*) and subcutaneous dirofilariasis (few different species). Life cycle is trans-mission between dogs and mosquitoes; humans are incidental hosts. Histopathologic diagnosis was by biopsy of involved tissue. No specific therapy for dirofilariasis is required.

Dracunculiasis causes Guinea worm infection, with man the only host; transmitted by consumption of unfiltered water containing copepods (small crustaceans) infected with larvae of *D. medinensis*. After ingestion, larvae penetrate the gastrointestinal tract and adult worms migrate to subcutaneous tissues causing painful papules. Treatment consists of slow extraction of worm combined with wound care and pain management.

Trichostrongylosis causes asymptomatic infection or gastrointestinal illness. Life cycle is transmission between herbivorous animals, with man as an incidental host. Diagnosis by identifying characteristic eggs in stool. Treatment with mebendazole or albendazole (Grade 2C) recommended

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