CHAPTER 8

COELOMIC DISORDERS

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COELOMIC DISTENSION

Dropsy

Overview: Dropsy is a commonly applied term for coelomic (i.e., abdominal) distention due to ascites, or the effusion and collection of fluid freely throughout the coelomic cavity. It is a nonspecific syndrome and a clinical presentation, as opposed to a defined disease. Dropsy, or ascites, is generally a sign of another ongoing disease process, oftentimes one that is multisystemic and impacting coelomic organs and tissues.

Etiology: Dropsy may be caused by a variety of potential etiological agents, both infectious and noninfectious. Generally, dropsy is associated with infectious disease processes associated with viral, mycotic or bacterial infection. In particular, pathogenic aeromonad, pseudomonad and vibrio bacteria have all been associated with free serous fluid accumulation in the coelomic cavity among various species of fishes.[1] Stressors such as rapid change in water temperature may predispose fish to bacterial diseases associated with dropsy.[2] *Rhabdovirus carpio*, the causative agent of spring viremia of carp (SVC), is another of the more recognized causative agents associated with dropsy among some cyprinids and other fishes.[3] Dropsy associated with SVC has also been more specifically called “infectious dropsy.”[4] Parasitism of the coelomic cavity or coelomic organs and infections of mixed etiologies have also been associated with dropsy.[2]

Clinical presentation: Clinically affected fish display coelomic distension of varying degrees of severity (**Figures 8.1** and **8.2**). Severely affected scaled fish may have protrusion of the scales that causes them to stand erect from the body surface, causing lepidorthosis or a “pine cone” appearance (**Figure 8.3**). Although dropsy is not unique to any taxonomic group of fishes, it is especially well recognized among pond cultured and hobbyist cyprinids.

Differential diagnosis: As dropsy is more accurately defined as a clinical presentation of disease rather than a disease entity, the differential diagnoses are numerous, including not only the potential etiologies that lead to free coelomic fluid accumulation but also other various causes of coelomic distension: neoplasia, organomegaly, gastrointestinal obstruction, egg-binding.

Diagnosis: Diagnostic imaging may be useful in determining if coelomic distention is related to fluid accumulation or some other cause. If free coelomic fluid is suspected or observed, coeliocentesis and evaluation of the coelomic fluid through cytology and microbial (i.e., bacterial, viral and fungal) culture may often be diagnostically useful.

Management/control: Treatment and control options depend upon the underlying etiology.

Cichlid bloat, Malawi bloat

Overview: Generally considered a disease syndrome similar to dropsy, affecting specifically Malawi cichlids, a popular group of ornamental fish among aquarium hobbyists.

Etiology: Causative agents are varied and underlying infectious diseases, parasitism, and nutritional imbalances have been associated with cichlid bloat.[5,6] Among potential bacterial etiologies, both clostridiosis (*Clostridium difficile*) and francisellosis (*Francisella noatunensis subsp. orientalis*) have been reported in association with Malawi bloat.[6,7]

Clinical presentation: Clinically affected fish may display coelomic distension characterized internally by ascites, hemorrhage into the coelomic cavity, organomegaly and granulomatous inflammation of organs particularly the digestive tract.[5,7] Additional signs may include anorexia, dyspnea, lethargic behavior and mortality.

Differential diagnosis: Like dropsy, Cichlid or Malawi bloat is a clinical syndrome rather than a

Figure 8.1 **Severe coelomic distention in a female tilapia (*Oreochromis* sp.) due to cystic ovaries. (Image courtesy of S.A. Smith.)**

Figure 8.2 **Severe coelomic distention in a female madtom (*Noturus* sp.) due to cystic ovaries. (Image courtesy of S. Boylan.)**

Figure 8.3 **Lepidorthosis (i.e., "pine-cone" appearance) in tilapia (*Oreochromis* sp.) due to coelomic distension. (Image courtesy of S.A. Smith.)**

specific disease. Differential diagnoses include the potential etiologies described for dropsy, as well as other various causes of coelomic distension that can impact Malawi cichlids, including neoplasia, organomegaly and gastrointestinal obstruction.

Diagnosis: Diagnostic imaging may be helpful in determining if coelomic distention is related to fluid accumulation or some other cause. If free coelomic fluid is suspected or observed, coeliocentesis and evaluation of the coelomic fluid through cytology and microbial culture may often be diagnostically useful.

Management/control: Treatment and control options vary and depend upon determination of the underlying etiology.

Salmonid water belly

Overview: Water belly occurs among salt water pen-reared salmonid fish, including Atlantic salmon (*Salmo salar*), Chinook salmon (*Oncorhynchus tshawytscha*) and rainbow trout (*Oncorhynchus mykiss*). This condition is idiopathic and presents as marked coelomic distension related largely to the accumulation of seawater in the stomach (**Figure 8.4**). Although it may clinically resemble dropsy, the fluid accumulation occurs within the gastrointestinal tract and not free within the coelomic cavity.

Etiology: The etiology has not been determined, although it is associated with dietary changes such as intensive feeding regimes and food composition, particularly related to fat and carbohydrates.[8]

Clinical presentation: Water belly has been reported to occur in sea-reared salmon and trout in both the Pacific Northwest (United States and Canada) and Europe.[9] Fish show severe distension of the coelomic cavity and mortality may also result. On necropsy, the stomach is fluid (seawater) filled, with stomach contents accounting for up to 40% of whole body weight.[10] Development of this condition is likely to

Figure 8.4 **Coelomic distension in a captive salmonid due to "water belly," an idiopathic condition where there is an accumulation of sea water in the stomach. (Image courtesy S. Boylan.)**

be chronic and as a result the stomach may be thin-walled and the liver may be atrophied.

Differential diagnosis: Differentials include ascites (i.e., dropsy), organomegaly, neoplasia and gastrointestinal obstruction.

Diagnosis: Gross external observation of marked coelomic distension coupled with the signalment (e.g., farmed salmonid) may provide a presumptive diagnosis. Verification that the coelomic distension is related solely to an enlarged seawater-filled stomach may be documented on necropsy.

Management/control: In some instances, dietary changes such as reduction of the feeding rate have been reported to improve this condition among affected fish.[11]

Miscellaneous etiologies (noneffusive)

Overview: In addition to the accumulation of free fluid within the coelomic cavity, many other factors affecting the size of coelomic organs or contents of the coelomic cavity may potentially result in apparent gross distension of the fish. Coelomic distension is a clinical sign of disease related to one or more coelomic organs or tissues, and further diagnosis is usually required to identify the true underlying etiology and appropriate management options.

Etiology: Distension of the coelomic cavity that is not related to effusion and/or accumulation of free fluid may be attributable to various underlying causes such as organomegaly, overinflation of the swim bladder, egg retention in ripe females, parasitism, and neoplasia associated with coelomic tissues or organs.

Clinical presentation: Grossly apparent distention of the body cavity may present to varying degrees. In subtle cases, comparing the body profile of an individual fish to another presumably normal specimen of the same species and life stage may help to identify any distension and whether it is general or localized. For laterally compressed fish, observing them from above in dorso-ventral orientation is most likely helpful. In more severe cases, protrusion of scales (i.e., lepidorthosis) over the lateral body walls may be evident, as seen in severe cases of dropsy. Coelomic distension may be generalized or restricted to a specific quadrant or region of the body cavity. Depending on the etiology and severity, other nonspecific clinical signs may present, such as anorexia, lethargy, ataxia or abnormal swimming behavior, and hyperpigmentation.

Differential diagnosis: Dropsy and peritonitis should also be considered in the differential diagnosis list along with the potential underlying etiologies previously mentioned.

Diagnosis: Identification of the underlying cause is potentially aided by imaging techniques such as radiography, ultrasound, or computerized tomography, if available. Coeliocentesis to check for and evaluate coelomic fluid content may help rule out dropsy and its potential infectious etiologies. Exploratory surgery or laparoscopy for a nonlethal diagnosis of an individual fish, or euthanasia and necropsy of an isolated specimen to evaluate disease within multiple individuals or a population of fish may provide a more definitive diagnosis.

Management/control: Management options are dependent on the underlying etiology.

COELOMIC INFLAMMATION

Steatitis

Overview: Also known as "yellow fat disease," steatitis is an inflammatory condition affecting adipose tissue. Usually, steatitis is described affecting coelomic adipose stores that are associated with the mesentery and sometimes associated with pancreatic tissue among fishes. Still, the condition has been reported to affect other fat stores in fish as well, such as pericardial adipose tissue, fins or skin.[12] The condition is most frequently reported in cultured fish in association with unsuitable diets, but may also occur in wild fish.

Etiology: Steatitis is generally nutritional in origin and commonly associated with lipid peroxidation. It occurs in fish fed rancid feed or other feed items with unsuitable lipid components in the diet. For example, steatitis has been reported to occur among hatchery fish that were fed liver as a starter feed.[13]

Clinical presentation: Clinical signs may vary, but inflammation of the visceral adipose tissue may present grossly as discolored (cream to yellow) masses in the coelomic cavity where fat stores are normally found (**Figure 8.5**). Histologically, steatitis may appear as granulomatous inflammation with lymphocytic, eosinophilic granular and giant

Figure 8.5 **Steatitis in a cultured yellow perch (*Perca flavescens*). (Image courtesy of S.A. Smith.)**

cells, as well as fibrosis. Inflammation may produce adhesions that involve coelomic organs. Additional clinical signs not involving the coelomic cavity may occur if the steatitis involves other body tissues. Fin loss and ulcerative skin lesions have been described in an atypical case in channel catfish.[12] In addition, muscle involvement and necrosis has been reported in conjunction with steatitis. Lethargy, hyperpigmentation and increased mortality in a population of farmed Atlantic bluefin tuna (*Thunnus thynnus*) has also been associated with steatitis.[14]

Differential diagnosis: Differentials include infectious or noninfectious peritonitis, liver lipidosis, multisystemic diseases associated with granulomatous inflammation (e.g., mycobacteriosis and norcardiosis), and pancreatitis.

Diagnosis: Diagnosis is based on gross and histological findings and ruling out potential differential causes. Identification of the underlying (i.e., nutritional) etiology and a positive response to correction of the dietary anomaly is confirmatory.

Management/control: Management is dependent on identifying and correcting the underlying nutritional etiology. An appropriate change of feed is often corrective for the population of fish.

Infectious peritonitis

Overview: Infectious peritonitis is a generalized inflammation of the peritoneal lining of the coelomic cavity caused by an infectious agent. It is often associated with multisystemic diseases, accompanied by infection and inflammation of coelomic organs. Numerous etiological agents are possible.

Etiology: There are multiple microbial agents associated with peritonitis in fish. The majority of reports of peritonitis in fish describe bacterial etiologies, and the list of potential causative agents is extensive. *Streptococcus* spp. (Gram-positive cocci) has produced multisystemic disease in fish species including tilapia.[15] Mycobacteriosis in fish (*Mycobacterium* spp., acid-fast positive short bacilli) often involves multiple coelomic organs and can be associated with peritonitis as well.[16] *Vagococcus salmoninarum* (Gram-positive bacillus) has been associated with an infectious peritonitis among Atlantic salmon (*Salmo salar*), rainbow trout (*Oncorhynchus mykiss*) and brown trout (*Salmo trutta*).[17] Aeromonad and pseudomonad bacteria are causative agents of various multisystemic and hemorrhagic diseases among fish species and are also associated with peritonitis. For example, *Pseudomonas fluorescens*, the agent of a hemorrhagic septicemia among tropical aquarium fish, marine fish, and pond fish, is often associated with fibrinous peritonitis.[18] Besides bacterial agents, the rickettsial pathogen *Piscirickettsia salmonis* has also been shown to produce peritonitis lesions in association with multisystemic disease among cultured salmonid fish.[19] Systemic diseases caused by fungal pathogens may also include peritonitis. Examples include phaeohyphomycosis (*Veronaea botryosa*) in cultured sturgeon, and granulomatous peritonitis caused by *Fusarium solani* in a desert pupfish (*Cyprinodon macularius*) and in a pink-tailed triggerfish (*Melichhthys vidua*).[20,21]

Traumatic injury may also be an underlying cause of peritonitis, and it may be resultant from predation, fighting or various fishing activity. For instance, retained fish hooks penetrating through the gastrointestinal tract may produce an infectious peritonitis associated with secondary bacterial infection in species that are fished commercially or recreationally.[22] Venting, the process of inserting a hollow needle through the abdominal wall to release gasses from barotraumatized fish, can also lead to an infectious peritonitis.[23] Spawning stress has also been identified as a precursor to bacterial peritonitis among fish.[24]

Clinical presentation: Clinical signs will vary with the etiological agent and species affected but generally include nonspecific indicators of disease such as anorexia and lethargy as well as increased mortality in the population. Coelomic distension may be noted with serous to serosanguinous exudate. Inflammatory change is also generally evident on necropsy, appearing as focal to diffuse discolored lesions or hemorrhage along the peritoneum and affecting coelomic organs. Various causative agents such as *Mycobacterium* spp. or intracoelomic protozoans may also produce a granulomatous inflammatory response apparent as nodules on coelomic organs.[16,25] A pseudomembrane covering coelomic organs may also form in association with inflammatory change, as is noted in a case of peritonitis associated with pasteurellosis among farmed Atlantic salmon.[26] Intracoelomic adhesions may also be present (**Figure 8.6**).

Differential diagnosis: Infectious peritonitis may be associated with a variety of microbial agents of infectious disease among fishes, including bacteria, rickettsia, fungi and fungal-like organisms, and protozoans. Intracoelomic parasitism by helminths, such as larval cestodes, may also produce similar lesions. Noninfectious causes of peritonitis such as aseptic vaccine reactions in the peritoneum, and other causes of ascites and abdominal distension can also be differentials.

Diagnosis: Diagnosis is likely to be based on microbial culture or molecular-based detection of the causative organism. Peritoneal effusion may be a readily available source of diagnostic material for cytology and microbial culture, obtained either by coeliocentesis in a live fish or necropsy in a recently perished or sacrificed specimen. Histopathological examination of the peritoneum as well as other coelomic organs involved may also be useful in the identification of the infectious agent as well as characterization of the inflammatory response. If circumstances warrant, predisposing factors such as traumatic injury to the body wall, peritoneum, or coelomic organs should also be ruled out.

Management/control: Management is based on the nature of the infectious agent identified and circumstances unique to the case, but cases of bacterial infection would generally involve antimicrobial therapy as warranted.

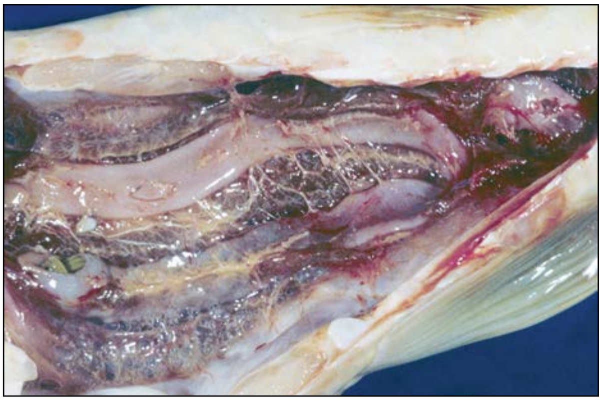

Figure 8.6 Numerous coelomic adhesions in a common carp (*Cyprinus carpio*) as a result of a systemic infection of *Aeromonas hydrophila*. (Image courtesy of S.A. Smith.)

Vaccine-associated peritonitis

Overview: Intracoelomic vaccination with oil-adjuvanted vaccines may produce a sterile granulomatous peritonitis at or near the injection site. A phenomenon observed primarily among farmed salmonids, particularly Atlantic salmon, vaccine-associated peritonitis has also been noted in Atlantic cod (*Gadus morhua*).[27,28] Although this peritonitis is generally not fatal and often only noted incidentally at slaughter or necropsy, it is of concern from the perspectives of both poor animal husbandry practices and reduced quality of fillets in food fish.[29]

Etiology: Mineral oils that are not readily metabolized and are used as adjuvants for intraperitoneal vaccination of fish can produce strong localized inflammatory reactions at or near the site of injection.

Clinical presentation: This disorder may not be clinically apparent until an internal examination of the fish is performed. Nonspecific clinical signs such as anorexia or lethargy have not typically been reported, although poor feed uptake and reduced growth have been reported in association with oil-adjuvanted vaccination among salmonids.[30] Internally, a granulomatous peritonitis may be accompanied by fibrous adhesions between coelomic organs and the peritoneum, as well as melanization of affected tissues (**Figure 8.7**). Hyperemia due to hemorrhage and fibrinous exudate may sometimes be apparent.[31] Pigmented foci in the musculature of the body wall may also be associated with the peritonitis and appear microscopically as granulomatous inflammatory lesions in the white muscle tissue.[29] Extracoelomic effects are also possible such

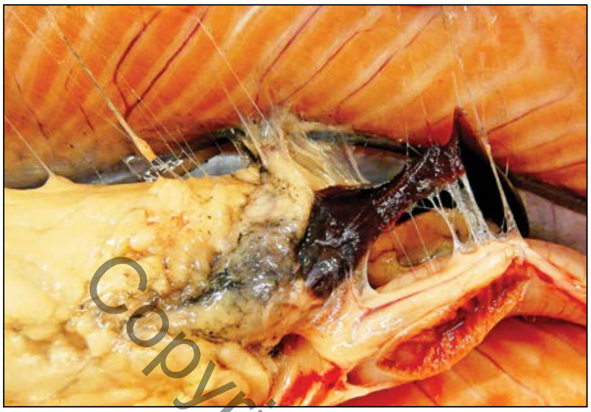

Figure 8.7 Post-vaccination adhesions with melanization comprising the spleen and pyloric caeca in an Atlantic salmon (*Salmo salar*). (Image courtesy of T. Poppe.)

as granulomatous uveitis as reported among Atlantic salmon (*Salmo salar*) or systemic autoimmunity manifested as multisystemic inflammation and production of autoantibodies.[32–34] Uncomplicated cases of vaccine-associated peritonitis may be self-limiting and not overly problematic, and lesions may regress over months to years.[35]

Differential diagnosis: Differential diagnoses will include other causes of peritonitis that produce similar lesions or masses within the coelomic cavity, such as parasitism with encysted helminths, diseases associated with focal granulomatous inflammation such as mycobacteriosis, and neoplasia.

Diagnosis: Diagnosis is based upon a combination of signalment (e.g., fish previously vaccinated with an oil adjuvant vaccine), clinical and histological presentation, and ruling out other differential diagnoses (i.e., lack of presence of infectious agents in association with the peritonitis).

Management/control: There are no management options after the peritonitis has been noted.

Coelomic parasitism (helminths)

Overview: Parasitism by helminths is frequently observed among fish, and the coelomic cavity is a common location for their occurrence. As a general rule, the presence and severity of lesions associated with helminths in the coelomic cavity is dependent upon both the life stage and pathogenic nature of the parasite species as well as upon the number of organisms present. For developmental stages of helminths that rapidly encyst within the coelomic cavity, host lesions and associated impacts may be minimal, whereas those parasites that migrate extensively in the coelomic cavity are more likely to produce significant lesions and even mortality.

Numerous fish species serve as intermediate, paratenic, or definitive hosts for helminth parasites. Both larval and adult stages of nematodes can parasitize the coelomic cavity of fish. Larval stages of cestodes (i.e., pleurocercoids) are generally among the most harmful metazoan parasites of fish, and may heavily damage coelomic organs and tissues. This is especially true for those species that migrate for a length of time within the host rather than encysting. For many species of trematodes, fish serve as an intermediate host for development from the cercarial stages through the encysted metacercarial stage in target organs or tissues. This development often involves tissue migration through the coelomic cavity and its organs. Lesions and organ dysfunction may result from large numbers of metacercarial cysts displacing normal tissue. Acanthocephalan are generally gastrointestinal adult parasites of fish that embed in the lumen wall of the host's gut. Effects of an infestation are mostly contained to the gastrointestinal tract, however, in severe cases perforation of the gut wall by the parasite may cause peritonitis and associated lesions in the coelomic cavity.

Etiology: Larvae are generally the most pathogenic stages of nematodes among fish. Larval ascarid nematodes such as *Anasakis* spp. may be problematic among marine fishes, whereas larval *Eustronylides* spp. may affect freshwater fishes. *Contracaecum* species utilize a variety of fresh water and marine fishes as intermediate hosts. Adult life stages generally inhabit the gastrointestinal tract of fish, although adults of some dracunculoidean species may occupy the coelomic cavity.[36] Examples include *Philomena* spp. among salmonid fishes, and *Philometra* spp. that impact striped bass and other species. The cestode *Ligula* sp. has a global distribution and is particularly harmful among catfish, suckers and minnows in freshwater environments. They may grow from the larval pleurocercoid to the adult stage within the body of the fish, reaching over 20 cm in length and a body weight that may exceed that of its fish host.[37]

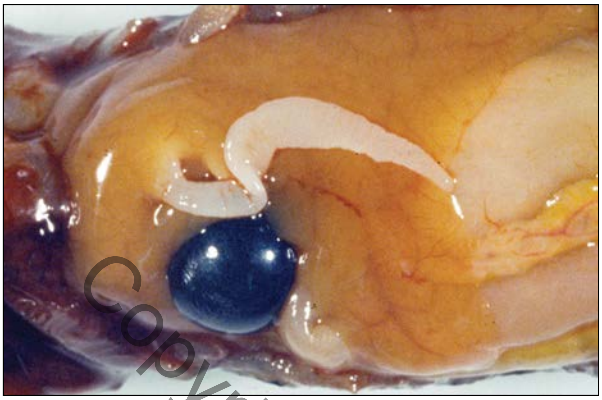

Figure 8.8 **Larval *Diphyllobothrium* sp. in coelomic cavity of an Alaska blackfish (*Dallia pectorali*). (Image courtesy of S.A. Smith.)**

Diphyllobothrium spp. affect many types of fishes, including salmonids in particular. These cestodes may be long lived and therefore may be most pathogenic among older fish (Figure 8.8). *Proteocephalus ambloptis*, the bass tapeworm, is likewise problematic among centrarchid species including largemouth bass (*Micropterus salmoides*) and smallmouth bass (*Micropterus dolomieu*). Multiple species of trematodes may produce coelomic disease among fishes. *Posthodiplostomum* spp., the so-called white grub, is a common complex of various trematodes species affecting freshwater fishes to the point of lethality, particularly among centrarchids and cyprinids. *Clinostomum marginatum*, the yellow grub, is another example of a trematode that affects many freshwater fish species and may be damaging to viscera due to its coelomic migration. Acanthocephalans are also known to cause infestations of many types of freshwater and marine fishes. Important genera in fishes include *Acanthocephalus*, *Paragorgorhynchus*, *Termisentic* and *Neoechinorhynchus*.[38]

Clinical presentation: Clinical signs will vary considerably in coelomic nematode infestations. Oftentimes fish are completely asymptomatic. Signs may be as benign as the observed presence of roundworms either encysted or free in the coelomic cavity or within the coelomic viscera with no apparent host tissue reactions associated. At the other end of the spectrum, a clinical presentation of fulminant peritonitis with fibrosis and adhesions is also possible. Appearance varies with the nematode species and life stage. For instance, *Eustrongyloides* spp. are generally deep red in color, whereas *Anisakis* spp. usually appear whitish. Larval stages of *Eustrongyloides* may be quite large and damaging to coelomic organs, especially the gonads, of freshwater fish. Larval *Anisakis spp.* may encyst in various coelomic organs or tissues, including muscle, of marine fishes and there is zoonotic potential when undercooked fish containing the parasite is consumed by humans. Philometrids may be highly pathogenic to a variety of fish in both fresh and salt water. *Philometra* species that affect the gonads are particularly damaging.[39] Adult stages of *Philometra* may also encapsulate in the coelom and produce an associated peritonitis.[37] A mild infestation of cestode pleurocercoids in the coelomic cavity may not present with clinical signs, especially if the parasites are encysted. In greater numbers, encystment in viscera may be associated with inflammation, generalized peritonitis with adhesions, and even death. For instance, *Diphyllobothrium* pleurocercoids may produce a chronic granulomatous peritonitis among salmonid fishes with fibrous coelomic adhesions and nodular lesions throughout the viscera.[40] Severe infestations of *Ligula* sp. may be accompanied by peritonitis, gross coelomic distension, and organ compression or distortion, potentially resulting in multiple organ dysfunction and death of the host. In smaller fish, rupture of the body wall may also be a potential sequelae of *Ligula* sp. infestation.[41] Clinical indications of trematode-associated coelomic disease depend upon the specificity of the target organs. In many cases, encysted trematodes and associated host tissue reactions are apparent as small pinpoint or larger multifocal discolorations either externally, internally, or both. Within the coelomic cavity, these visceral cysts may be apparent in the muscle of the body wall or on organs and may also be associated with inflammation and fibrosis.[42] Visceral migration of the cercarial stage prior to encystment may also produce associated inflammatory or degenerative changes in tissues characteristic of peritonitis. While mild cases of larval trematodes may be virtually asymptomatic, severe infestations with highly pathogenic species may be fatal. Acanthocephalans parasitizing the intestines of fish are sometimes visible or palpable within the intestine upon examination at surgery or necropsy. Secondary impacts to the coelomic cavity generally only occur in severe infestations due to perforation of the gut wall and secondary fibrous peritonitis.[43] Leakage of gastrointestinal contents into the coelomic cavity may produce additional complications of peritonitis such as hemorrhage, inflammation, effusion, or necrosis of serosal surfaces of the intestine or other coelomic organs.

Differential diagnosis: Differentials include the various forms of coelomic parasitism as well as other causes of peritonitis.

Diagnosis: Diagnosis is based on observation and identification of the parasite, generally upon surgery or necropsy. Perforation of the gut wall may be evident grossly with acanthocephalan parasites and clinical signs of peritonitis may also be apparent. Parasite identification is based on taxonomical identification using morphological features and/or molecular identification to genus/species.

Management/control: Management options are often limited for control of helminths within the coelomic cavity. Oftentimes in the case of few to no accompanying symptoms, no treatment or control is warranted. Anthelminthic treatment may not be effective or advisable for control of parasites that are free or encysted within the body cavity, as opposed to parasites found within the gastrointestinal tract.[44] Control of alternative host populations may help to break the parasite's life cycle in some cases. Culling of affected fish within a population may also be necessary if significant disease is noted.

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