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REVIEW

Capillaria hepatica in man—an overview of hepatic capillariosis and spurious infections

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Abstract *Capillaria hepatica* (syn. for *Calodium hepaticum*) is a zoonotic nematode parasitizing in the livers of rodents as main hosts and in numerous other mammals including humans. It is the causative agent of the rare conditions of hepatic capillariosis and spurious *C. hepatica* infections in humans. In this review, 163 reported cases of infestations with this parasite (72 reports of hepatic capillariosis, 13 serologically confirmed infestations and 78 observations of spurious infections) are summarized with an overview on the distribution, symptoms, pathology, diagnosis, serology and therapy of this rare human pathogen.

Introduction

Capillaria hepatica (syn. for *Calodium hepaticum*) is a parasitic nematode of wild rodents and numerous other mammals with a worldwide distribution. Adults of this zoonotic helminth parasitize the liver; females lay ova into the liver parenchyma, and eggs are released into the environment only by the death of its host. Fifty-nine cases of the hepatic capillariosis were documented in humans by 2001, and children below the age of 8 years were the most affected group (Schmidt 2001). Hepatic capillariosis causes a variety of symptoms, but the triad of persistent fever, hepatomegaly and leucocytosis with eosinophilia is usually present. Until now, the main diagnostic tool to determine infestations with *C. hepatica* is a histopathological examination of samples taken at liver biopsy. Spurious infections occur when unembryonated eggs of *C. hepatica* are ingested, pass through the intestine and are shed with faeces.

Based on the detection of *C. hepatica* in Arvicolinae hosts in western Austria on one hand and the medical relevance of capillariosis in Central Europe on the other hand, we started to summarize reported cases of human capillariosis, serologically confirmed samples and reports of spurious infections (Führer et al. 2010).

Taxonomy, morphology and biology

C. hepatica (Bancroft, 1893) is a nematode parasite of the suborder Trichinellina and the family Capillaridae (Fauna Europaea 2011). Although Moravec (1982) categorized *C. hepatica* to the genus *Calodium*, the name *C. hepaticum* is rarely used, and most authors have retained the name *C. hepatica*. Further synonyms are *Trichocephalus hepaticus* Bancroft, 1893 and *Hepaticola hepatica* Hall, 1916. The family Capillaridae contains three species of zoonotic importance, namely: *Capillaria philippinensis*, *Capillaria aerophila* and *C. hepatica*.

Adult *C. hepatica* are long, slender-shaped nematodes with a narrow anterior body part (0.007–0.01 mm), whereas the posterior part is gradually thicker. Sexual dimorphism is

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present; females have a length of 27–100 mm and a width of 0.1–0.89 mm whereas males are smaller with 15–50× 0.04–0.1 mm (reviewed in Schmidt 2001). The eggs are barrel-shaped with polar plugs, striation and numerous minipores in the outer shell. The eggs resemble those of *Trichuris trichiura* but differ in size (40–67×27–35 μM). Larval stages differ in size (I, 0.14–0.25 mm; II, 0.16–0.4 mm; III, 0.2–1.56 mm; IV, 1.1–3.6 mm) (reviewed in Schmidt 2001).

The life cycle of *C. hepatica* is a direct one, with an extremely high affinity to the liver. After the ingestion of embryonated eggs, larvae hatch in the intestine and enter the liver via the portal vein system. In the liver parenchyma, they mature to adults, mate, and about 28 days later, females lay unembryonated eggs which become encapsulated by the host's liver parenchyma. Those eggs are not shed by the host and develop only to the eight-cell stage, until the host's death. The eggs reach the environment only through the decay of the host's carcass or through the shedding of eggs in the faeces (predator, cannibalism and scavenger). Within 5 to 8 weeks, eggs embryonate under optimal conditions (temperature, moisture and exposure to air). The life cycle is completed by the ingestion of embryonated eggs by a new host, whereas the ingestion of unembryonated eggs leads to spurious infections only (reviewed in Juncker-Voss et al. 2000; Li et al. 2010; Schmidt 2001).

Epidemiology

C. hepatica is a zoonotic parasite with worldwide distribution. The main hosts are rodents, but there are documented cases where this parasite colonizes the livers of more than 140 mammal species in over 50 countries around the globe (Fig. 1) (Schmidt 2001; Führer et al. 2010). This nematode has been reported not only in more than 80 Muridae species, but also in marsupials, carnivores, hominids and other mammals of the families: Didelphidae, Macropodidae, Potoroidae, Erinaceidae, Soricidae, Leporidae, Antilocapridae, Equidae, Erethizontidae, Procaviidae, Tayassuidae, Canidae, Felidae, Mustelidae, Sciuridae, Castoridae, Caviidae, Geomyidae, Myocastoridae, Dipodidae, Callitrichidae, Cebidae, Cercopithecidae, and Hominidae (reviewed in Schmidt 2001).

In humans, overall, 163 cases of infections with *C. hepatica* have been found in literature during research for this review article—72 cases of hepatic capillariosis, 13 serologically confirmed reports and 78 documentations of spurious infections.

Unsanitary and poor hygienic conditions (e.g. drinking water from rivers) and the presence of rodents (e.g. rats) and domestic animals increase the risk of infestations with *C. hepatica*. Frequent contact with animals and a surrounding rat population was reported by several authors (e.g. Camargo et al. 2010; Choe et al. 1993; Silverman et al. 1973). Cochrane et al. (1957) reported a heavy infestation in a rodent (*Mastomys natalensis*) caught at a hepatic capillariasis patient's home, and Juncker-Voss et al. (2000) found this nematode in the liver of mice caught in the zoological garden of Vienna, where two employees were tested positive for antibodies against *C. hepatica*. Pica (especially geophagia) is a special risk for children. Sand-, earth- and dirt-eating habits were documented by several authors in children with hepatic capillariasis (e.g. Silverman et al. 1973; Cochrane et al. 1957; Calle 1961).

Hepatic capillariosis—geographic distribution

Human cases of hepatic capillariosis have been reported in Europe (Germany, Switzerland, Italy, England, Greece, former Czechoslovakia, former Yugoslavia and Turkey), North and South America (USA, Canada, Mexico and Brazil), Asia (India, Korea, Japan and Thailand), Africa (South Africa, Ivory Coast and Nigeria) and Oceania (New Zealand).

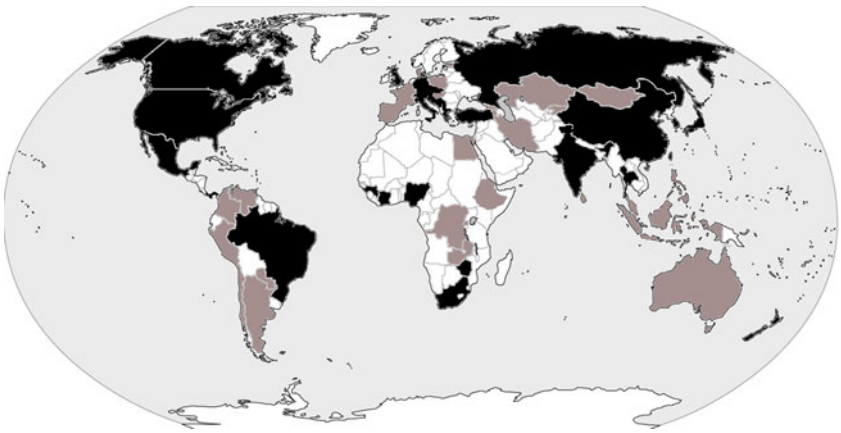

Fig. 1 Distribution of *C. hepatica*. *Black*, reported cases in humans including spurious infections; *gray*, reported cases in animals. Revised and updated after Schmidt (2001)

Sixty per cent of all reports were in children below the age of 8 years, and in 59% of all infestations, females were affected. This disease was mainly diagnosed at autopsy or coincidentally, but if a correct diagnosis is made in time, the chance to survive increased from a mortality rate of 53% to 3% (Table 1).

Within this review, we include two reports with first diagnoses other than *C. hepatica* infestations. Odunjo (1970) conducted 1,498 autopsies of children in Nigeria and described a helminthic anaphylactic shock syndrome in 84 children. Eosinophilia in the bone marrow, clusters of eosinophils in the liver and plasma cell hypertrophy were present. In 18 cases, yellowish granulomata with worms and/or eggs were examined in the liver and diagnosed as *Trichuris trichiura*. Those 18 cases were classified as infestations with *C. hepatica* by Schmidt (2001). Furthermore, Sumner and Tinsley's (1967) case of a visceralis larva migrans in the liver of a female patient in England was classified as hepatic capillariosis by Slais (1974). Table 2 gives an overview of all documented hepatic capillariasis cases found during our research activity for this review.

Clinical symptoms

Liver capillariosis is primarily associated with a triad of symptoms—persistent fever, hepatomegaly and leucocytosis with eosinophilia. Hypergammaglobulinaemia and increased levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST) and lactic dehydrogenase (LDH) are commonly observed. Tables 3, 4 and 5 give an overview of the frequency of symptoms, patients' haematology/blood chemistry reports and clinical chemical parameters documented in cases with hepatic capillariosis.

Rarely, chronic hepatic injuries caused by *C. hepatica* infestations induce kidney or lung calcifications (Ewing and Tilden 1956). In one case, the induction of an IgA nephropathy was suspected (Choe et al. 1993).

Diagnosis

Imaging techniques

Computed tomography enables the diagnosis of a hepatomegaly and mass or cystic lesions in the liver. To date, this technique was used in seven cases of hepatic capillariosis. A low-density area with unclear margin was observed by Kohatsu et al. (1995). Kim et al. (2002) described a round nodule with peripheral enhancement on hepatic arterial phase (CTHA) and a well-demarcated nodule with homogenous low density on aortoportogram phase (CTAP).

Ultrasonography can be used for the diagnosis of a hepatomegaly caused by *C. hepatica*. It enables the visualization of hypoechoic, space-occupying lesions and the observation of cystic or subcapsular nodules (6 mm–2 cm) in the liver.

In a case described by Kohatsu et al. (1995), magnetic resonance tomography visualized a granuloma caused by *C. hepatica* as a tumour of low intensity. A liver artery angiography revealed no compression, proliferation or enhancement around the tumour in the artery phase, but the surrounding tissue stained densely in the parenchyma phase. A hepatic scintigraphy showed a defect of a liver segment (Kohatsu et al. 1995).

Liver biopsy

Liver biopsy is still the cornerstone of diagnosis. *C. hepatica* larvae primarily invade the sinus hepaticus where maturation and egg laying is accomplished (Li et al. 2010).

Table 1 Cases of infestations with *C. hepatica* in humans

	Overall	Children (<8 years)	Adults	Female	Male
Hepatic capillariosis	72	44 (61%)	28 (39%)	29[a] (59%)	20[a] (41%)
Survived	28 (39%)	18	10	14	13
Died (incl. diagnosis at autopsy)	38 (53%)	24	14	13	7
Died (excl. diagnosis at autopsy)	2 (3%)	2	None	2	None
Outcome unknown	6 (8%)	2	4	2	None
Eggs only	17 (24%)	12	5	10	6
Worms/-fragments only	13 (18%)	None	13	3	3
Worms + eggs	27[b] (37%)	25[b]	2	6	3
Granuloma only	4 (6%)	2	2	2	None
Unknown	11 (15%)	5	6	8[c]	8[c]

[a] According to data availability

[b] Including Odunjo (1970)

[c] Including the nine cases reported by Šlais and Stěrba (1972) and Šlais (1973, 1974)

Table 2 Human cases of hepatic infestations with *C. hepatica*; ND: no data

Author (year)	Country	Sex	Age	Parasitic form	Diagnosis	Outcome (follow-up)
MacArthur (1924); Dive et al. (1924)	India	M	20 years	Eggs	Autopsy	Died
McQuown (1950)	USA (New Orleans)	F	17 months	Eggs	Autopsy	Died 26 h after admission
Otto et al. (1954)	USA (Maryland)	F	7 years	Eggs + worms	Liver biopsy/ autopsy	Died after 2 years
Turhan et al. (1954)	Turkey	M	60 years	ND	Autopsy	Died
Ewing and Tilden (1956)	USA (Hawaii)	F	15 months	Eggs + worms	Liver biopsy	Died after 4 months
Cochrane et al. (1957); Cochrane and Skinstad (1960)	South Africa (Transvaal)	F	15 months	Eggs + worms	Liver biopsy	Survived (3.5 years)
Ward and Dent (1959)	USA (Louisiana)	F	2 years	Eggs	Autopsy	Died
Kallichurum and Elsdon-Dew (1961)	South Africa (Durban)	F	5 years	Granuloma in liver	Autopsy	Died
Calle (1961)	USA (Illinois)	M	20 months	Eggs + worm fragments	Liver biopsy	Survived (10 months)
Romero Garcia et al. (1962)	Mexico (Guadalajara)	F	22 months	Granuloma in liver	Liver biopsy	Survived
Piazza et al. (1963)	Brazil (Sao Paulo)	F	25 years	Eggs	Autopsy	Died
Camain et al. (1965)	Ivory Coast	M	11 months	ND	Autopsy	Died
Sumner and Tinsley (1967)	England	W	57 years	Worms	Liver biopsy	Survived
Odunjo (1970)	Nigeria—18 cases		2–4 years	Worms and/or eggs	Autopsy	Dead
Cislaghi and Radice (1970), (1971); Pampiglione and Conconi (1970)	Italy (Brindisi)	F	3 years	Eggs	Liver biopsy	Unknown
Šlais and Stěrba (1972); Šlais (1973)	Czechoslovakia—9 Patients	4 m 5 w	19–83 years	Worm Granuloma	Autopsy	Died
		F	78 years	Worm	Autopsy	Died
		M	48 years	ND	Autopsy	Died
		F	83 years	ND	Autopsy	Died
		M	67 years	ND	Autopsy	Died
		F	55 years	ND	Autopsy	Died
		F	76 years	ND	Autopsy	Died
		M	78 years	ND	Autopsy	Died
Eaton (1972)	Canada (Saskatoon)	F	ND	ND	Autopsy	Died
Silverman et al. (1973)	South Africa (Bethal)	F	17 months	Eggs	Liver biopsy	Survived (1 year)
Xu and Li (1979)	China (Guangdong Province)	ND	ND	ND	Liver biopsy	ND
Vargas Carreto et al. (1979)	Mexico (Puebla)	M	2 years	ND	ND	Survived (1 month)
Pereira and França (1981); Pereira and Mattosinho Franca (1983)	Brazil (Sao Paulo)	M	39 years	Eggs + worms	Liver biopsy	Survived (8 years)
Attah et al. (1983)	Nigeria	F	27 years	Eggs	Liver biopsy	Unknown
Berger et al. (1990)	Switzerland (Luzern)	F	1 year	Eggs (at 2nd biopsy)	Liver biopsy	Survived (5 months)
Pannenbecker et al. (1990); Müller et al. (1990)	Germany	F	18 months	Eggs	Liver biopsy	Survived
Kokai et al. (1990)	Yugoslavia	F	2 years	Granuloma	Liver biopsy	Survived
Sekikawa et al. (1991)	Japan	M	26 years	Worms	Liver biopsy	Survived
Choe et al. (1993)	Korea	F	14 months	Eggs + fragments	Liver biopsy	Survived (2 years)
Anonymous (1993)	Mexico (Acapulco)	ND	5 years	ND	ND	ND
Kohatsu et al. (1995)	Japan (Okinawa)	F	32 years	Worm fragments	Liver biopsy	Survived (8 months)
Yfanti et al. (1996)	Greece (Peloponnesos)	F	60 years	Worms + eggs + larvae	Liver biopsy	Survived (8 months)

Table 2 (continued)

Author (year)	Country	Sex	Age	Parasitic form	Diagnosis	Outcome (follow-up)
Govil and Desai (1996)	India	M	6 years	Eggs	Liver biopsy	Survived
Bustamente-Sarabia et al. (1996)	Mexico	M	19 months	Eggs + worms	Liver biopsy	Survived
González Barranco et al. (1996)	Mexico	F	54 years	ND	Autopsy	Died
Zlatkovic et al. (1998)	Yugoslavia	M	20 months	ND	Liver biopsy	Survived
Terrier et al. (1999)	Switzerland (Lausanne)	M	2 years	Eggs	Liver biopsy	Survived (3 years)
Sawamura et al. (1999)	Brazil (State of Sao Paulo)	M	2 years	Eggs	Liver biopsy	Survived (24 years)
		F	35 months	Eggs	Liver biopsy	Survived (2 years, 4 months)
		F	18 months	Worms + eggs	Liver biopsy	Survived
Keven et al. (2001)	Turkey	ND	54 years	Eggs	Liver biopsy	Survived (9 months)
Kim et al. (2002)	Korea	M	39 years	Worm	Liver biopsy	Survived (12 months)
Lin et al. (2004)	China (Tangzhuang)	ND	ND	Worm	Liver biopsy	ND
Huang and Lin (2004)	China (Fujian Province)	ND	ND	Eggs	Liver biopsy	ND
Klenzak et al. (2005)	USA (New England)	M	54 years	Granuloma	Liver biopsy	Survived
Orihuela-Chávez et al. (2006)	Mexico (Vecaruz)	M	19 months	Eggs	Liver biopsy	Survived (6 months)
Nabi et al. (2007)	India (Nasik)	M	14 months	Eggs	Liver biopsy	Survived
Tesana et al. (2007)0	Thailand (Chiang Mai Province)	F	33 years	Eggs	Right hepatectomy	Survived
Koea and Smith (2008)	New Zealand	F	84 years	Necrotic parasites	Lobectomy	Survived

Granulomata can be found in various degrees of activity. They have been described as yellowish nodules with sizes between 1 mm and 3.5×2 cm, containing eggs and/or worms (or necrotic material) surrounded by a rim of diverse inflammatory cells, including eosinophils, neutrophils, lymphocytes, plasma cells, foreign-body giant cells, macrophages, multinuclear giant cells and histiocytes. Rarely, Charcot–Leyden crystals and epitheloid cells have been

Table 3 Symptoms of infestations with *C. hepatica* in humans (including only cases with provided information); ND: no data

	Hepatic capillariosis				
	All cases	Children <8 years	Adults	Worms only	Eggs only
Eosinophilia	87% (n=30)	95% (n=19)	60% (n=10)	60% (n=5)	86% (n=14)
Leucocytosis	90% (n=30)	100% (n=19)	90% (n=10)	100% (n=4)	100% (n=14)
Fever	83% (n=30)	95% (n=19)	50% (n=10)	29% (n=7)	79% (n=14)
Hepatomegaly	87% (n=30)	95% (n=20)	50% (n=10)	14% (n=7)	93% (n=15)
Anaemia	50% (n=30)	60% (n=20)	20% (n=10)	14% (n=7)	43% (n=14)
AST	80% (n=15)	90% (n=10)	60% (n=5)	100% (n=2)	80% (n=10)
ALT	79% (n=14)	77% (n=9)	60% (n=5)	100% (n=2)	56% (n=9)
Hyper-γ-globulin	88% (n=8)	83% (n=6)	ND	ND	100% (n=3)
Splenomegaly	27% (n=30)	35% (n=20)	10% (n=10)	None	36% (n=14)
Renal calcification	7% (n=30)	10% (n=20)	None	None	None
Respiratory disease	23% (n=30)	30% (n=20)	10% (n=10)	None	29% (n=14)
Weight loss/Poor appetite	23% (n=30)	10% (n=20)	30% (n=10)	14% (n=7)	21% (n=14)
Night sweat	10% (n=30)	5% (n=20)	20% (n=10)	14% (n=7)	14% (n=14)
Lymphatic symptoms	7% (n=30)	10% (n=20)	None	None	14% (n=14)
Intestinal problems	33% (n=30)	45% (n=20)	10% (n=10)	None	50% (n=14)
Other parasites	20% (n=30)	30% (n=20)	None	None	36% (n=14)

Table 4 Haematology and blood chemistry parameters at infestations with *C. hepatica*; ND: no data

	All cases	Worms only	Eggs only	Children (<8 years)	Adults	Reference range[b]
Leukocyte count (/µl)	7,600–66,000 (*n*=28)	7,600–14,300 (*n*=4)	12,900–64,900 (*n*=14)	13,000–66,000 (*n*=19)	7,600–64,000 (*n*=9)	3,540–9,060
Eosinophils (%)	1–94 (*n*=23, *x*=45.6)	1–22 (*n*=3, *x*=8.2)	1–94 (*n*=11, *x*=47)	14–85 (*n*=16, *x*=55)	1–94 (*n*=7, *x*=24,7)	0–8
Total eosinophils (/mm^3)	3,693–32,526 (*n*=4)	ND	19,800–24,200 (*n*=2)	3,693–32,526 (*n*=4)	ND	70–140
Neutrophils (%)	11–95 (*n*=5)	94 (*n*=1)[a]	11–95 (*n*=4)	11–19.9 (*n*=3)	94–95 (*n*=2)[a]	40–70
Polymorph. neutro. /Bands (%)	7–75 (*n*=5)	ND	18–75 (*n*=2)	7–45 (*n*=4)	75 (*n*=1)	Bands, 0–10
Lymphocytes (%)	2–43 (*n*=9)	ND	2–26 (*n*=6)	13–43 (*n*=7)	2–5 (*n*=2)	20–50
Monocytes (%)	2–7 (*n*=6)	ND	2–7 (*n*=6)	2–7 (*n*=4)	2–4 (*n*=2)	4–8
Platelet count (/µl)	81,000–789,000 (*n*=10)	159,000 (*n*=1)	191,000–789,000 (*n*=5)	81,000–789,000 (*n*=7)	159,000–256,000 (*n*=3)	165,000–415,000
ESR (mm/h)	76–120 (*n*=4)	ND	76–105 (*n*=2)	76–100 (*n*=2)	105–120 (*n*=2)	F, 1–25; M, 0–17
Haemoglobin (g/dl)	5.7–16 (*n*=11)	16 (*n*=1)	6.9–13 (*n*=5)	5.7–9.7 (*n*=9)	13–16 (*n*=2)	M, 13.3–16.2; F, 12.0–15.8
MCHC (g/l)	304 (*n*=1)	ND	304 (*n*=1)	304 (*n*=1)	ND	310–370
MCV (fl)	76 (*n*=1)	ND	76 (*n*=1)	76 (*n*=1)	ND	79–93.3
Haematocrit (%)	21–45.5 (*n*=6)	45.4 (*n*=1)	22.7–31.6 (*n*=2)	21–24 (*n*=3)	31.6–45.4 (*n*=3)	M, 38.8–46.4; F, 35.4–44.4

[a] Cases with 1% eosinophils

[b] Reference range according to Kasper et al. (2005)

Table 5 Clinical chemistry parameters at infestations with *C. hepatica*; ND: no data

	All cases	Worms only	Eggs only	Children (<8 years)	Adults	Reference range[a]
Total protein (g/L)	62–100 (*n*=7)	62 (*n*=1)	71–98.5 (*n*=3)	71–100 (*n*=5)	62–93 (*n*=2)	67–86
Albumin (g/L)	21–47 (*n*=8)	ND	21–47 (*n*=5)	21–47 (*n*=7)	25 (*n*=1)	M, 40–50; F, 41–53
SGOT (U/L)	18–800 (*n*=16, *x*=181)	62–100 (*n*=2, *x*=81)	18–800 (*n*=10, *x*=152)	36–800 (*n*=10, *x*=247)	18–100 (*n*=6, *x*=152)	7–41
SGPT (U/L)	11–550 (*n*=15, *x*=136)	127–138 (*n*=2, *x*=132)	11–174.8 (*n*=9, *x*=62)	11–550 (*n*=9, *x*=169)	25–138 (*n*=6, *x*=75)	12–38
Lactic dehydrogenase (U/L)	211–835 (*n*=5)	ND	211–590 (*n*=4)	428–835 (*n*=4)	211 (*n*=1)	100–190
Alkaline phosphatase (U/L)	86–887 (*n*=11)	86–327 (*n*=3)	87–887 (*n*=6)	241–887 (*n*=4)	86–327 (*n*=7)	30–120
γ-Glutamyltranspeptidase (U/L)	20–8,322 (*n*=6)	84 (*n*=1)	20–8,322 (*n*=3)	20–8,322 (*n*=3)	84–112 (*n*=3)	1–94
Globulin (g/dl)	4.3–6.8 (*n*=3)	ND	4.3–6.8 (*n*=3)	4.3–7.6 (*n*=2)	6.8 (*n*=1)	2–3.5 (40–50%)
γ-Globulin (g/dl)	1.5–5 (*n*=8)	ND	1.76–4.4 (*n*=3)	1.5–5 (*n*=7)	>1.7 (*n*=1)	0.7–1.7 (13–23%)
Total bilirubin (mg/dl)	0.3–3.15 (*n*=9)	0.5–1.19 (*n*=2)	0.3–3.15 (*n*=3)	0.3–3.15 (*n*=5)	0.5–1.19 (*n*=4)	0.3–1.3
CRP (mg/dl)	0.59–11.2 (*n*=3)	0.59 (*n*=1)	11.2 (*n*=1)	6+ (*n*=1)	0.59–11.2 (*n*=2)	0.08–3.1
IgA (mg/dl)	787 (*n*=1)	ND	787 (*n*=1)	787 (*n*=1)	ND	60–309
IgM (mg/dl)	213 (*n*=1)	ND	213 (*n*=1)	213 (*n*=1)	ND	53–334
IgG (mg/dl)	3,240 (*n*=1)	ND	3,240 (*n*=1)	3,240 (*n*=1)	ND	614–1,295
IgE (IU/L)	1,450 (*n*=1)	ND	1,450 (*n*=1)	1,450 (*n*=1)	ND	10–179
CA19-9 (U/ml)	383 (*n*=1)	ND	383 (*n*=1)	383 (*n*=1)	ND	0–37
CEA (ng/dl)	0.8 (*n*=1)	ND	0.8 (*n*=1)	0.8 (*n*=1)	ND	0–3.4

[a] Reference range according to Kasper et al. (2005)

observed. Some degenerated ova have been found in (or were associated with) foreign-body giant cells (Cochrane et al. 1957; Sawamura et al. 1999). Granuloma can be clearly divided from the surrounding liver tissue, and liver cells outside of the granuloma appear healthy with a normal amount of glycogen. Granuloma can also be free of eggs or worms, containing necrotic material and nucleic debris, while others contain fragments of worms surrounded by giant cells (Calle 1961). Inflammatory infiltration proceeds until the end of the encapsulation of eggs/parasites, and septal fibrosis may occur (Li et al. 2010).

Care should be taken to exclude mix-ups with other helminths parasitizing (occasionally) in the liver and causing eosinophilia, granuloma and/or eosinophilic lesions such as *Toxocara canis* or *Toxocara cati* (visceral larve migrans), *Baylisascaris procyonis*, *Clonorchis sinensis*, *Ascaris suum*, *Ancylostoma* sp. and *Fasciola hepatica* (Lim 2008).

Serology

An indirect immunofluorescent test (IIFT) basing on *C. hepatica* eggs of liver sections of massively infested mice and human serum samples was established by Juncker-Voss and others (2000). This sensitive assay shows bright fluorescence in all sections of the parasite eggs, including the inner structure of the eggs. However, 10% of all human serum samples were reported to present low non-specific fluorescence at dilutions below 1:20, and thus, it is recommended to use dilutions above 1:40. At titres of 1:20 and 1:40, no cross-reactivity against *Echinococcus granulosus*, *Echinococcus multilocularis*, *F. hepatica*, *Schistosoma mansoni*, *Toxocara canis*, *Trichuris trichiura* nor *Trichinella spiralis* was observed.

An IIFT and an enzyme-linked immunosorbent assay (ELISA) based on goat anti-rat IgG, antigen from eggs and worms of *C. hepatica* and human sera were developed by Assis and others (2004). Specific apple-green fluorescence of the shell and egg content was observed in positive samples in conducting the IIFT; which was based on the sera of infested rats against eggs and worms of *C. hepatica*. At ELISAs, old infestations showed stronger positive reactions in comparison to new ones, whereas IIFTs reveal positive results 15 days–3 months post-infection and turn negative 4 months after infection. Spurious infections give negative or non-specific results, disappearing at higher dilutions.

Serology cannot only be used for the confirmation of severe cases of hepatic capillariosis; it can also be chosen as a tool for the diagnosis of early stages of infestation, cases with no or milder symptoms or in epidemiological studies. Those specific and sensitive tests are useful techniques for the diagnosis of *C. hepatica* infestations, but to the best of our knowledge, none are available commercially, and so they are limited to the availability of worm antigen. Accordingly, no severe case of hepatic capillariosis has yet been diagnosed by serology.

Serology—cross-reactivity

In general, care should be taken in the interpretation of serological results. One patient with suspected systemic ornithobilharziosis (a subsequently detected *C. hepatica* infestation) gave a strong positive result at an ELISA for *Schistosoma* spp. which was excluded by the fact that this 1-year-old child had never been in an endemic country (Berger et al. 1990). A case of serological cross-reactivity led to the misdiagnosis of a true *C. hepatica* infestation, which was wrongly diagnosed as the first case of a liver dirofillariosis in Korea. An ELISA with crude extracts of an isolated worm from the patient's liver biopsy gave a positive result for *Dirofilaria immitis* (Kim et al. 2002). Later, Pampiglione and Gustinelli (2008) resolved the case as hepatic capillariosis.

Spurious infections

Spurious infections are associated with the consumption of infected game or soil, where noninfective, unembryonated eggs are ingested and passed through the digestive tract and could be found in stool diagnosis. More than 70 cases of spurious infections are documented in literature (Table 6). Most of those cases were reported in the first half of the twentieth century (e.g. Lubinsky 1956 summarized 34 cases) or from the Amazon region in Brazil in the last decade. More than half of all authors described spurious infections in tribal communities or immigrants: descendants from Jamaica in Panama, Bunun aboriginals (Taiwan), Surui Indians (Brazil) and the Golds (East Siberia, Russia). Spurious infections were documented in patients of various ages and both sexes (Palhano-Silva et al. 2009).In 1939, Foster suggested that the eating of infected livers of animals commonly used as food may cause spurious infections in humans (Foster and Johnson 1939). The consumption of (raw) liver is considered to be a delicacy by many indigenous groups in Brazil (reviewed by Sianto et al. 2009). The intake of wild animals, e.g. gravy out of squirrel liver, was also documented by several other authors (Engler and Sanchez 1950; Camargo et al. 2010; McQuown 1954).

Eggs of *Capillaria* sp. were found in several palaeoparasitological settings in Europe (Switzerland, 5,900–4,900 BP; Belgium, sixteenth century AD) and in South America (Argentina, 8,920 BP) (reviewed in Sianto et al. 2009). Fugassa et al. (2008) analysed a coprolite supposed

Table 6 Human cases of spurious infections with *C. hepatica*

Author (year)	Reported cases (n=78)	Country
Skrjabin et al. (1929)[a]	3	Russia (Amur Region, Eastern Siberia)
Vogel (1932)[a]	1	Guinea (former French Guinea)
Sandground (1933)[a]	1	Zimbabwe (Mt. Silinda, former Southern Rhodesia)
Wright (1938)[a]	16	Panama (Chagres R. Basin)
Faust (1931)[a], Faust and Martinez (1935)[a]	9	Panama (Chagres R. Basin)
Brosius et al. (1948)[a]	1	Panama
Engler and Sanchez (1950)[a]	1	Panama (Almirante)
McQuown (1954)	1	USA (Louisiana)
Coimbra and Mello (1981)	2	Brazil (State of Rondonia)
Fan et al. (2000)	1	Taiwan (Sanmin District)
Unknown[b]	7	Taiwan
Machado et al. (2008)	1	Brazil (Uberlândia)
Palhano-Silva et al. (2009)	28	Brazil (Brazilian Amazonas)[c]
Camargo et al. (2010)	6	Brazil (State of Rondonia)

[a] Reviewed by Lubinsky (1956)

[b] Mentioned by Fan et al. (2000)

[c] *Capillaria* sp.

to be from a canide from an archaeological site in Patagonia, Argentina (6,450±110 years BP). They analysed 149 eggs of *Capillaria* sp. and subdivided them in four morphotypes. 13.4% (n=20) of those eggs were compatible with current *C. hepatica* eggs. It is assumed that those eggs were ingested with the consumption of infected prey.

Spurious infections may lead to mild diarrhoea (McQuown 1954) but seem to be asymptomatic in most cases. Care should be taken to avoid mix-ups with eggs of other nematode species with comparable morphology, e.g. *Trichuris trichiura*, *Huffmanela* sp. (histozoic parasites in various tissues of marine and freshwater fishes) and other species of the genus *Capillaria* (e.g. *C. philippinensis*) (Moravec and Garibaldi 2000; Schouten et al. 1968).

Serologically confirmed cases

Several cases of infestations with *C. hepatica* were confirmed by serological techniques (Table 7). The findings of antibodies against *C. hepatica* in two employees of the zoological garden in Vienna, Austria, were reported by Juncker-Voss et al. (2000). Furthermore, *C. hepatica* was diagnosed in house mice caught in the area of the zoological garden. One employee presented an antibody titre of 1:40, which stayed stable until a follow-up 2 months later. A second employee presenting a borderline positive titre of 1 in 40 gave a negative result at follow-up. None of them had any signs of symptoms of a severe hepatic capillariosis. No increase in the activities of the liver-associated enzymes was observed, and all blood parameters were in normal range.

Camargo et al. (2010) reported the finding of antibodies in inhabitants along the Petro River in Brazil, who lived under poor sanitary conditions. A weak, median or strong positive serology at a dilution of 1 in 150 was observed in 84 (34.1%) of 246 participants of this study, but only two remained positive at titres of 1 in 500 and 1 in 1,000. However, all laboratory parameters (e.g. haemogram, AST, ALT) of those two participants were in normal range, and no abnormalities were observed at ultrasonography of the liver.

Nine out of 500 children (1.8%) from a slum in Salvador, Bahio, Brazil, presented high antibody titres against *C. hepatica*. Of those, four shed eggs with their faeces which lead to the suggestion of spurious infections, but the other five participants were assumed to have infestations with *C. hepatica* (Galvão 1981).

Table 7 Serologically confirmed cases of infestations with *C. hepatica* in human

Author (year)	Reported cases (n=13)	Country
Galvão (1981)[a]	7 male + 2 female children (including 4 shedding eggs)	Salvador, Bahia, Brazil
Juncker-Voss et al. (2000) Juncker-Voss et al. (2004)	2 males	Vienna, Austria
Camargo et al. (2010)	4 years, male; 34 years, female	Pietro River, Rondonia State, Brazil

[a] Reviewed by Schmidt (2001)

High seroprevalences are associated with low socio-economic and sanitary conditions, leading to an increased risk to ingest embryonated eggs (e.g. from carcasses of infected animals exposing eggs to the environment).

Therapy

Antihelminthic drugs (e.g. albendazole, thiabendazole, ivermectin) which attain high levels of plasma concentrations and inhibit the laying of eggs by adult female worms are used for therapy (Schmidt 2001). Choe et al. (1993) desribed albendazole (10–20 mg/kg/day) for 20 days and thiabendazole (25 mg/kg/day) for 27 days, in combination with prednisolone (10 mg/d) for therapy. Prednisolone, Prednisone and other corticoids decrease the inflammation reaction and help to control fever. However, treatment with thiabendazole is proven to eliminate adult *C. hepatica*, but fibrous tissue surrounding the eggs and the egg shells itself inhibits and protects the eggs from the drug effect (Choe et al. 1993; Keven et al. 2001). Mebendazole (200 mg) given twice daily for 2 months was documented to be effective against ova of *C. hepatica* (Keven et al. 2001).

In animal models, partial hepatectomy is described as an option to treat hepatic capillariosis. A lobectomy of the right lobe of the liver led to an uneventful recovery in an adult female patient (Koea and Smith 2008).

Conclusions

Although *C. hepatica* is a worldwide-distributed parasite, the incidence and prevalence of human capillariosis are rather low. However, *C. hepatica* can cause severe hepatic capillariosis which may end in the patient's death if misdiagnosed. None of the patients with serologically confirmed infestations with *C. hepatica* presented severe symptoms. These circumstances lead to the suggestion that the distribution of hepatic capillariosis is much more frequent than previously thought. If patients (especially children) present a symptom triad of persistent fever, hepatomegaly and leucocytosis with eosinophilia and increased AST, ALT and LDH levels, an infestation with *C. hepatica* should always be taken into consideration. Further investigations are necessary to simplify diagnosis and improve therapy.

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