antibodies indicate acute infection because they are not usual in acquired immunity and very rare in chronic infections. They further reported that the presence of IgA together with IgG antibodies in circulation indicate recent acquired infection because IgA antibodies persist over 3 or 4 months following infection in acquired toxoplasmosis and low titers of IgG antibody are usual in patients with active toxoplasmic chorioretinitis, and IgM antibody cannot usually be detected.

Direct statistical comparison of the performance of UAgE with DT on one hand and with LAT on the other may be erroneous since the respective tests are detecting different components in circulation. Moreover, antibodies once produced against antigens are known to remain in circulation for a prolonged period even after drug therapy whilst antigens gradually reduce in quantity and go out of circulation. However, detection of *Toxoplasma* antigens in urine from patients whose sera were negative for anti-*Toxoplasma* antibodies is suggestive of early stage of fresh (or primary) infection, which was evident in this study. In other words, DT sero-negative serum samples that were positive by UAbE and/or UAgE could possibly be due to a putative active infection (IgM/IgA+ but IgG−). Additionally, in the UAgE system, antigens from samples that indicated weak positive results bound to murine-IgM and IgA antibodies, which appear in circulation earlier than IgG. Among the patients whose sera were positive for both DT and LAT but negative for urinary antigens by UAgE were two with high IgG titers (1:1024–1:2048). This is expected because both DT and LAT are reported to measure primarily IgG antibodies low titers of which may persist for life whilst some patients have persistently high titers (e.g. 1:1000–1:4000) for years. Consequently, the titer does not correlate with the severity of illness (Anderson and Remington, 1975). The comparable efficacy of DT and LAT in determining seropositivity was evident in this study (Table 1).

Considering the high prevalence of parasitic diseases like falciparum malaria, schistosomiases and soil-transmitted helminthiasis in Ghana, and more so with the existence of a urinary antigen detecting membrane-based dipstick assay for urinary schistosomiasis (Bosompem et al., 1997), it was necessary to clarify possible cross-reactions due to any of these parasites' antigens. None of the parasites detected do seem to have influenced the results of the membrane-based tests since urine samples from some individuals with mixed parasitic infections were negative for those tests.

Cryptosporidiosis is in Ghana but no comprehensive studies have been conducted. Addy and Aikins-Bekoe (1986) recorded a prevalence of 12.9% (61/474) in children from 2 to 60 months old and prior to our study a hospital-based research conducted by Otchere in 2001, using the same method on watery and normal formed faecal specimens from Ghanaian patients, yielded no oocysts (personal communication). That no oocysts were detected in any of the stool samples by the formalin–ether sedimentation technique was not surprising but does not rule out also inclusion of patients with antigenemia for, and/or antibodies against other relevant coccidian species like *C. parvum*, *Isospora belli* and *Cyclospora cayetanensis*. Current research methods like the use of fluorescein isothiocyanate-labelled anti-parasite monoclonal antibodies, SDS-PAGE and Western blotting techniques as well as ELISA could be employed to detect parasite antigens (Smith, 1998; Ungar, 1990) in a more elaborate study.

The 22 normal samples were confirmed by the study as sero-negative and could therefore be considered as true controls.

In this study, the UAbE and UAgE systems appear to have good prospects as their outcome are consistent with the intentions of development. However, their sensitivity and specificity need to be enhanced, and detailed cross-reactivity studies conducted with respect to infections from other coccidian species, using well-defined and larger study populations, especially, for obstetrics/gynaecology patients.

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CASE REPORT

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Strongyloidiasis in a patient with acquired immunodeficiency syndrome

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Abstract Rhabditiform larvae, transforming larvae from rhabditiform to filariform, and eggs of *Strongyloides stercoralis* were identified in the sputum of a Thai woman with acquired immunodeficiency syndrome (AIDS), and stool microscopy also showed a heavy load of rhabditiform larvae of *S. stercoralis*. She was treated with 12 mg ivermectin once a day for 2 days for the strongyloidiasis, with good therapeutic results being obtained. Strongyloidiasis may be a curable disease through the use of an appropriate therapy, even in a patient with AIDS.

Key words *Strongyloides stercoralis* · HIV · Ivermectin

Introduction

Strongyloidiasis stercoralis is an infectious disease caused by *Strongyloides stercoralis*, a small intestinal nematode, and is usually symptomatic or asymptomatic in immunocompetent persons. This parasitic infection is distributed widely in tropical and subtropical areas. Okinawa Prefecture and the southwestern part of Kagoshima Prefecture, which are subtropical areas, have been known as endemic regions in Japan, but strongyloidiasis is uncommon and Japanese physicians are unfamiliar with this infectious disease outside the areas mentioned above. Some patients coinfected with human immunodeficiency virus (HIV) and *S. stercoralis* have been reported.[1,2] The effectiveness of ivermectin against strongyloidiasis in immunocompetent patients is well known; however, its usefulness has not been well studied in regard to strongylodiasis in patients coinfected with HIV. Here we present a case of strongyloidiasis treated with ivermectin in an acquired immuno deficiency syndrome (AIDS) patient.

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Case report

A 46-year-old Thai woman, who came to Japan in 1998 and married a Japanese man, was admitted to a hospital in Tokyo complaining of fever and dyspnea on June 16, 2002. She was diagnosed as having pneumonia and treated with meropenem, but her condition did not improve. She was referred to our hospital on June 21, 2002, because her serum anti-HIV antibody was positive and *Pneumocystis carinii* pneumonia was suspected. She also complained of soft, but not bloody, stool (1–3 times/day) on admission; however, the onset date of her soft stool was unknown. She did not complain of nausea or vomiting.

On admission to our hospital, she was alert; her height was 153 cm, body weight 45 kg, body temperature 37°C, blood pressure 110/60 mmHg, pulse rate 102/min and regular, respiratory rate 40/min, and SpO_2 was 82% in room air. There was no jaundice, and cervical lymph nodes were not palpable, but oral candidiasis was found. Heart sounds were normal, and mild coarse crackles were audible on her right-side chest. Abdominal examination revealed no tenderness, normoactive bowel sounds, and no ascites. Pretibial edema were absent.

White blood cell count on admission was 10800/mm^3 (neutrophils 96.0%, lymphocytes 2.6%, monocytes 1.0%, eosinophils 0.4%), red blood cells were 387 × 10^4/mm^3, hemoglobin was 9.4 g/dl, and hematocrit was 29.7%. Plasma HIV-RNA levels and CD4$^+$ lymphocytes were 1.5 × 10^5 copies/ml and 3/mm^3, respectively. The numbers of white blood cells (with eosinophil percentages) were as follows: 5900/mm^3 (2%) on June 24, 7600/mm^3 (2%) on June 26, 11200/mm^3 (0%) on June 28, 6500/mm^3 (0%) on July 4, and 4400/mm^3 (3%) on July 8. Serum levels of total protein,

Fig. 1. Transforming larva from rhabditiform to filariform in sputum. Papanicolaou stain, ×182

albumin, aspartate transaminase, lactate dehydrogenase, potassium, iron, C-reactive protein, immunoglobulin G, immunoglobulin A, and β-D-glucan on admission were 7.7 g/dl, 2.5 g/dl, 71 IU/l, 782 IU/l, 128 mEq/l, 15 mg/dl, 10.3 mg/dl, 2050 mg/dl, 732 mg/dl, and 153.7 pg/ml, respectively. Cysts of *P. carinii* were identified in her sputum, and moreover, rhabditiform larvae, transforming larvae from rhabditiform to filariform (Fig. 1), and embryonated ova of nematodes were all identified in her sputum under Papanicolou staining. Stool microscopy showed a heavy load of rhabditiform larvae, and occult blood examination in her stool was positive. The mean size of the rhabditiform larvae found in her sputum with Papanicolou staining was 200 × 20 μm. An esophagus with a club-shaped anterior portion and a posterior bulbus was found in the rhabditiform in her sputum, with a buccal cavity that is short and of a small diameter. A longer esophagus was found in the transforming larvae, but a posterior bulbus was not identified. The rhabditiform in her stool had a genital primordium halfway down the midgut, an esophagus with a club-shaped anterior portion and a posterior bulbus, as well as a short buccal cavity. They were thought to be larvae and ova of *S. stercoralis*.

She was then diagnosed as having acquired immunodefieciency syndrome (AIDS) with *P. carinii* pneumonia, oral candidiasis, and strongyloidiasis. Sulfamethoxazole trimethoprim and predonisolone were administered from June 21, 2001, for the treatment of *P. carinii* pneumonia, and clotrimazole was administered orally for the oral candidiasis. She was treated with a single oral dose of 12 mg ivermectin on June 24 and again on July 1, 2002. Her defecation rate changed to between 0 and 2 times daily beginning June 27, 2002. Ivermectin caused no adverse side effects. Her condition significantly improved, and she was discharged on July 16, 2002; a highly active antiretroviral therapy with sanilvudine, lamivudine, and efavirenz was started at an outpatient clinic beginning August 15, 2002. *S. stercoralis* was not found in her stool by a direct smear method on November 11, 2002, or by a direct smear method, filter paper culture method, or ordinary agar plate culture method on February 6, 2003.

Discussion

There are two possible explanations for the findings of larvae and eggs of *S. stercoralis* in the sputum from our patient. First, eggs were expelled by matured worms in the lungs where larvae were developing. Second, her sputum was contaminated with duodenal juice. In our patient, not only intestinal strongyloidiasis but also pulmonary strongylodiasis may have been present, as she did not complain of nausea or vomiting.

It has been reported that eosinophils play an important role in protecting the host against fulminant strongyloidiasis,[3] and eosinopenia may be associated with a poor prognosis of strongylodiasis.[3–5] If strongyloidiasis had been overlooked in our patient, her prognosis might have been poor, because her eosinophil count was low or normal. Further studies of the relationship between eosinopenia and strongyloidiasis are anticipated, however.

Ivermectin is the most useful drug for the treatment of strongylodiasis in immunocompetent conditions,[6,7] and appears to be promising for the treatment of strongyloidiasis in patients coinfected with HIV.[8,9] Because the number of cases is few, however, the effectiveness of ivermectin against strongyloidiasis in patients infected with HIV has not been fully studied. The disappearance of *S. stercoralis* and its symptoms after the administration of ivermectin in our patient may indicate that strongylodiasis is a curable disease, even in patients with AIDS, and at the same dose as for patients infected with *S. stercoralis* without HIV. Our patient is thought to have become infected with *S. stercoralis* outside Japan, because she had not been to any *S. stercoralis* endemic areas in Japan. She probably acquired the causative organism in Thailand. Our present report indicates that it is important to investigate stool and sputum for *S. stercoralis* in HIV-positive patients who have been in a *S. stercoralis* endemic area and are complaining of diarrhea, because strongyloidiasis may be curable through the use of an appropriate therapy, even in a patient with HIV.

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LARVA MIGRANS BY *BAYLISASCARIS TRANSFUGA*: FATAL NEUROLOGICAL DISEASES IN MONGOLIAN JIRDS, BUT NOT IN MICE

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ABSTRACT: Raccoon roundworms (*Baylisascaris procyonis*) and other *Baylisascaris* species cause patent or latent larva migrans (LM) in a variety of mammals and birds, including humans. It is not clear whether LM by *Baylisascaris transfuga*, roundworms of bears, is associated with clinical neurological disorders. To clarify this issue, ICR and BALB/c mice as well as Mongolian jirds (*Meriones unguiculatus*) were orally inoculated with 2,000–5,000 embryonated eggs of *B. transfuga*. In mice, the ascarid caused symptomatic LM of limited extent and duration, whereas the infection was fatal in jirds; i.e., they exhibited general signs such as severe depression and emaciation on days 8–11 postinfection (PI) and died, or they developed progressive and fatal neurological disorders after day 14 PI. Histological examination showed *B. transfuga* larvae in the brain of all mice and jirds examined, and the larvae collected from them developed to a size comparable with that of *B. procyonis*. There existed, however, critical differences in host reactions against larvae localized in the brain of mice and jirds; *B. transfuga* larvae found in mice were surrounded by granulomatous reactions and immobilized, whereas larvae found in jirds were free from any host reaction and mobile, causing extensive malacia.

Because of its zoonotic impact on animals and humans, larva migrans (LM) by the raccoon roundworm (*Baylisascaris procyonis*) has been investigated extensively using laboratory rodents as well as accidentally infected domestic or wild animals. Because raccoons (*Procyon lotor*) are endemic in North America, a dozen human cases of *B. procyonis* LM with severe or fatal neurological manifestations, as well as dozens of patients with visual disorders related to diffuse unilateral subacute neuroretinitis (DUSN) caused by this ascarid, have been documented almost exclusively from that continent (Huff et al., 1984; Fox et al., 1985; Goldberg et al., 1993; Cunningham et al., 1994; Boschetti and Kasznica, 1995; Park et al., 2000; Rowley et al., 2000; Gavin et al., 2002; Kazacos et al., 2002; Mets et al., 2003; reviewed by Kazacos, 1997, 2001). Recent and progressive naturalization of imported raccoons in other continents is ominous of an expansion of the disease worldwide in the near future. Raccoons exported to West Germany and Russia in the mid-1930s became feral, expanding their distribution into Germany, France, The Netherlands, Turkestan, Azerbaijan, Uzbekistan, Kirgiz, Belorussia, Ukraine, and Caucasia. In these countries, raccoons cause ecological, economic, and public health problems. For example, a human case of DUSN and serologically suspected human cases of asymptomatic LM caused by *B. procyonis* have been reported in Germany (Küchle et al., 1993; Conraths et al., 1996). More than 20,000 raccoons had been exported to Japan as pets during the past 3 decades until 2000, and several thousand raccoons have become feral in this country (Miyashita, 1993; Kawanaka et al., 2001). After the report of *B. procyonis* causing fatal infection in rabbits kept at a wildlife park where visitors, including infants and children, had common access to an egg-contaminated rabbitry and had close contact with infected rabbits (Sato et al., 2002; Sato, Kamiya et al. 2003; Furuoka et al., 2003), this ascarid LM became an important public health problem in Japan as well.

After the first reports of *B. procyonis* LM in Japan, histological slides of ascarid LM in zoo animals were sent to our laboratory for identification of the causative species (Sato, Matsuo et al., 2003). One example was Japanese macaques (*Macaca fuscata*) kept in a safari park, which shared living space with American black bears (*Ursus americanus americanus*) harboring *B. transfuga*. In contrast to *B. procyonis*, only a few studies have been conducted on *B. transfuga* LM, although larval migration to the viscera, eyes, and brains has been demonstrated in laboratory mice (Sprent, 1955; Papini et al., 1994; Papini, Lo Piccolo, and Casarosa, 1996; Papini, Renzoni, Lo Piccolo, and Casarosa, 1996) and rabbits (Papini, Demi, and Croce, 1996), suggesting the zoonotic potential of this ascarid species.

In this study, we conducted several experiments to determine the clinical and histological changes of LM caused by *B. transfuga, B. procyonis*, and *Toxocara canis* in mice and Mongolian jirds (*Meriones unguiculatus*), which has been reported to be the best laboratory model for DUSN caused by *B. procyonis* larvae and other ascarid ocular LM (Takayanagi et al., 1999; Akao et al., 2000, 2003). The results suggest that *B. procyonis* LM is fatal to both rodent species, whereas *B. transfuga* LM is fatal only in jirds but not in mice. This difference in clinical manifestations is ascribed to different host reactions against *B. transfuga* larvae in the central nervous system (CNS) of jirds and mice; specially jirds could not encapsulate and immobilize invading larvae, whereas mice could.

MATERIALS AND METHODS

Animals

Mice of outbred (ICR) and inbred (BALB/c) strains and Mongolian jirds were bred in the Institute for Animal Experiments, Hirosaki University School of Medicine. They were housed in plastic boxes and provided with commercial pellets (MF; Oriental Yeast Co., Ltd, Tokyo, Japan) and water ad libitum. After oral inoculation of embryonated eggs of ascarids, rodents were kept in a closed room to avoid environmental contamination with eggs or biohazards to other workers. Equipment used to keep infected rodents was subsequently cleaned in boiling water.

Parasites

Adult *B. transfuga* were expelled after anthelmintic treatment from a polar bear (*Ursus maritimus*) kept in a zoological garden. *Baylisascaris procyonis* was obtained from raccoons in a wildlife park, where LM due to this ascarid was found in kept rabbits in 2000 (Sato et al., 2002). *Toxocara canis* was collected from naturally infected dogs after the administration of anthelmintics. Fertile eggs were collected from the uteri of adult females and incubated for 1 mo at 27 C to obtain embryonated eggs, then kept at 4 C until used.

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TABLE I. Fate of larva migrans caused *Baylisascaris transfuga*, *Baylisascaris procyonis*, and *Toxocara canis* in mice and jirds.

Parasite	Animal	Eggs inoculated*	Rate of clinical disease	Date of the first signs of disease	Date of death or killing due to severe clinical disease	Date of the end of the experiment
B. transfuga						
	ICR mice (21-wk-old, male)	2,000	1/7	21	—	60
	BALB/c mice (12-wk-old, female)	2,000	3/8	21	28†	56
	Jirds (7-wk-old, female)	5,000	12/12	1	9.8 ± 1.1 (8–11)	11
	Jirds (9-wk-old, female)	4,000	13/13	20.3 ± 4.2 (14–28)	25.8 ± 3.7 (21–31)	31
B. procyonis						
	ICR mice (16-wk-old, male)	200	5/7	14.0 ± 2.2 (10–21)	25.6 ± 3.6 (21–31)	60
	BALB/cmice (8 to 12-wk-old, female)	100‡	24/24	13.0 ± 2.7 (9–18)	16.0 ± 3.4 (12–21)	56
	BALB/c mice (12-wk-old, female)	200	5/6	22.6 ± 3.1 (21–28)	23.2 ± 2.7 (22–28)	56
	BALB/c mice (12-wk-old, female)	100	2/6	31.5 ± 5.0 (28–35)	34.0 ± 8.5 (28–40)	56
	Jirds (11 to 13-wk-old, female)	100‡	16/16	13.6 ± 2.3 (9–15)	16.6 ± 3.4 (12–21)	21
	Jirds (8-wk-old, female)	200	10/12	19.2 ± 7.5 (15–39)	27.1 ± 5.9 (21–39)	60
	Jirds (7-wk-old, male)	200	13/13	21.7 ± 5.0 (14–31)	30.3 ± 11.9 (21–60)	60
T. canis						
	ICR mice (14-wk-old, male)	2,000	0/7	—	—	77
	BALB/c mice (12-wk-old, female)	2,000	0/6	—	—	56
	Jirds (9-wk-old, female)	2,000	0/15	—	—	93

* Embryonated eggs with viable larvae were used to infect rodents in all experiments, but *B. procyonis* eggs except for those marked by (‡) were preserved in vitro for more than 1 yr before use.
† One mouse only.
‡ Explanation appears in the footnote marked by *.

Infection, monitoring of clinical manifestations, and necropsy

Rodents were inoculated orally with embryonated eggs using a metal gastric sonde under light anesthesia (Table I). Approximate numbers of eggs used for infection were estimated after counting eggs with mobile larvae inside per unit volume. Rodents were observed daily to check the onset and progression of clinical signs. When the clinical condition deteriorated, i.e., spinning around the longitudinal axis, continuous circling or lateral recumbency, rodents were killed on that day. Animals not exhibiting clinical signs after inoculation with eggs of *Baylisascaris* spp. were killed on days 56 or 60 PI. A part of mice and jirds infected with *T. canis* were treated with immunosuppressive agents such as prednisolone or anti-T cell monoclonal antibody (mAb) mentioned below or after day 71 PI, and killed on days 77 and 93 PI, respectively. Animals were skinned and dissected. Pieces of the intestinal wall, liver, lungs, heart, kidney, femoral muscle, and whole parts of the brain were fixed in 10% neutral-buffered formalin. Other parts of the body were minced and digested in physiological saline containing 0.8% pepsin and 0.8% HCl at 37 C for 3–4 hr. Digested material was passed through a 125-μm metal mesh sieve and washed several times by simple sedimentation using physiological saline.

Immunosuppressive treatments

To evaluate the effects of immunosuppressive treatment on clinical manifestations associated with chronic *T. canis* LM, 4 of each of the mice and jirds were injected subcutaneously with 4 mg of prednisolone tertiary-butylacetate (Suspension of Codelcortone®-T.B.A., Merck & Co., Inc., Rahway, New Jersey) on day 71 PI. Similarly, 4 jirds were treated with anti-jird T cell murine mAbs, HUSM-M.g.13 of IgG1 isotype and HUSM-M.g.14 of IgG2a isotype, from day 71 PI. The epitope recognized by these mAbs is identical to HUSM-M.g.15 of IgG2b isotype, which has been used for successful depletion of functional T cells from jirds (Sato et al., 2000; Sato et al., 2004). Preparation of semipurified mAbs from mouse ascitic fluid and injection schedule were based on the methods described previously (Sato et al., 2000), except for the injection dose of 0.5 mg protein/injection. Complement-fixing mAb HUSM-M.g.14 exhibits the same effects as HUSM-M.g.15, but complement-unfixing mAb HUSM-M.g.13 exhibits no immunosuppressive effect and was used as a negative control mAb to assess the effect of T-cell depletion by mAb HUSM-M.g.14.

Histological examination

Formalin-fixed tissue blocks were dehydrated in a series of alcohol, cleared in xylene, and embedded in paraffin. Sections, 5 μm thick, were stained with hematoxylin and eosin.

Measurements of recovered larvae

Morphological examination and measurements of ascarid larvae were conducted using parasites collected by artificial digestion of body muscles and viscera. Measurements were calculated with the aid of camera lucida.

Statistical analysis

Data are expressed as mean ± SD with range values provided in parentheses. Differences between 2 groups were examined for significance using the Student's *t*-test. A P value less than 0.05 denoted the presence of statistical significance.

RESULTS

Clinical features

As shown in Table I, 14 experiments of ascarid LM were conducted to compare the pathogenecity of *B. transfuga*, *B. procyonis*, and *T. canis* larvae in mice and jirds. Clinical signs ascribed to *B. transfuga* LM were rarely noticed in mice; there was 1 case of continuous circling among 7 infected ICR mice and 3 cases of locomotor incoordination among 8 infected BALB/c mice. Except for 1 fatal case, all other mice showed full recovery from these conditions within 1 wk. In contrast, *B. transfuga* LM caused fatal infection in jirds. One group of 7-wk-old jirds inoculated orally with 5,000 eggs exhibited roughened hair coats within a few days after infection, became highly emaciated, and died by day 11 PI. Another group of 9-wk-old jirds inoculated orally with 4,000 eggs survived the acute phase of the disease but had clinical neurological signs such as torticollis, spinning around the longitudinal axis, and continuous

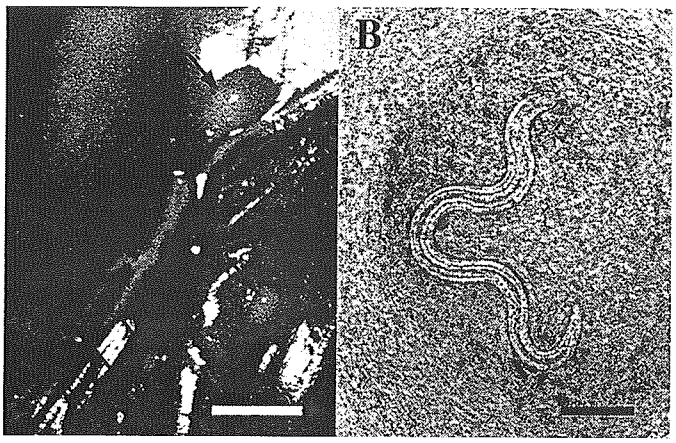

FIGURE 1. Granulomatous lesions elicited by *Baylisascaris transfuga* larva migrans. **A.** Granulomas of variable sizes (arrows) in the fascia lata femoris. Bar = 2.0 mm. **B.** Image of a press specimen of 1 granuloma containing the larva. Bar = 200 μm.

circling or lateral recumbency on days 14–28 PI, and died by day 31 PI. *Baylisascaris procyonis* LM induced severe neurological signs in both mice and jirds, and the infection was usually fatal for both host species. No neurological sign associated with *T. canis* LM was detected in either mice or jirds.

Gross and histological findings

Examination of jirds that died from acute *B. transfuga* LM by day 11 PI showed fibrinous exudate trapping numerous larvae in the peritoneal cavity with marked pulmonary hemorrhage and edema. No granulomatous lesion could be detected in these jirds. The other group of jirds that died from neurological disorders caused by *B. transfuga* LM had disseminated granulomatous lesions on the serosal surface of abdominal and thoracic organs, particularly the posterior part of the ileum, cecum, and colon, as well as in the subcutaneous tissue or on the surface of muscles of the trunk (Fig. 1). Mice also had numerous disseminated granulomas in various organs, similar to jirds. Similarly, mice and jirds with *B. procyonis* LM had several granulomatous lesions. No gross granulomatous lesion was noticed in mice and jirds with *T. canis* LM. No gross lesion was found in the brain of any mice or jirds with *B. transfuga*, *B. procyonis*, and *T. canis* LM, even though some animals manifested severe neurological signs.

Histological lesions associated with ascarid LM in organs other than the CNS were characterized by formation of granuloma around migrating larvae, albeit the cellular composition varied in animals infected with different ascarid species or examined on different days of infection. The most conspicuous histological differences were noted in the CNS, i.e., localization of the lesions and host reactions surrounding ascarid larvae. Figure 2 depicts the distribution of malacia, gliosis, and larvae themselves in a series of sections from the frontal cerebrum to the cerebellum and pons of jirds with *B. transfuga*, *B. procyonis*, and *T. canis* LM. *Baylisascaris procyonis* larvae caused malacic lesions, usually in the tissues beneath the aqueductus cerebri and the ventriculus quartus cerebri, whereas malacia and subsequent accumulation of foamy macrophages in association with *B. transfuga* larval migration were prominent in the tissues above the ventriculus quartus cerebri or in the cerebellar medulla. *Baylisascaris procyonis* larvae in jirds and mice or *B. transfuga* larvae in jirds were found free from host reactions or adjacent to malacic foci (Fig. 3A, B), whereas *B. transfuga* larvae in mice were surrounded or embedded in granulomatous reactions (Fig. 3C, D). Histological examination strongly suggested that the larvae were immobilized in these locations, although they were alive and well developed. *Toxocara canis* in mouse CNS induced little tissue damage or host reaction (Fig. 3E), whereas the ascarid in jird CNS caused disseminated foci of foamy macrophage accumulation (Fig. 3F), indicating that they caused substantial damage to the nervous tissue. There was no difference in clinical manifestations or histological changes in the CNS between immunosuppressed and competent mice or jirds with chronic *T. canis* LM.

The size of migrating larvae

Table II compares measurements of 3 ascarid species mainly from infected jirds. The average worm length and maximum width of *B. procyonis* larvae collected from infected rodents were always larger than those of *B. transfuga* larvae. Although differences in these values were sometimes statistically significant when comparisons were made between samples collected at the same time, the values significantly overlapped with each other.

DISCUSSION

Baylisascaris includes species of roundworms in Carnivora, Rodentia, and Marsupialia as follows: *B. columnaris* (Leidy, 1856) of skunks, *B. devosi* (Sprent, 1952) of martens and fishers, *B. laevis* (Leidy, 1856) of marmots and ground squirrels, *B. melis* of badgers, *B. procyonis* (Stefanski et Zarnowski, 1951) of raccoons, *B. schroederi* (McIntosh, 1939) of giant pandas, *B. tasmaniensis* (Sprent, 1970) of carnivorous marsupials, and *B. transfuga* (Rudolphi, 1819) of bears. Among them, in addition to *B. procyonis*, which is a well-known cause of zoonotic LM in birds and mammals, including humans, with neurological manifestations (Kazacos et al., 1981, 1984, 1985; Kazacos, 1997, 2001), *B. columnaris* and *B. melis*, followed by *B. devosi*, *B. tasmaniensis*, and *B. transfuga*, are considered potential causes of LM when a substantial number of eggs are ingested (Sprent, 1953b, 1955; Matoff and Komandarev, 1965; Sprent et al., 1973; Kazacos, 2001). The lower pathogenecity of the latter 5 species is manifested by delayed onset of neurological disorders and no progression of disease to death, with occasional recovery of affected mice. The distinct pathogenecity of *B. procyonis* from other *Baylisascaris* spp. is explained partly by the faster growth rate and larger size of larvae on tissue migration as well as their predilection for the CNS, in contrast to the predilection of *B. devosi* for the anterior carcass musculature and that of *B. tasmaniensis* and *B. transfuga* for the intestinal wall or mesentery (Sprent, 1952, 1953a; Tiner, 1953a, 1953b; Matoff and Komandarev, 1965; Berry, 1985; Kazacos, 2001). According to Sprent (1952), Matoff and Komandarev (1965), and a series of studies by Papini et al. (Papini and Casarosa, 1994; Papini, Lo Piccolo and Casarosa, 1996; Papini, Renzoni et al., 1996), the number of embryonated *B. transfuga* eggs (2,000, 4,000, or 5,000 eggs/animal) used for

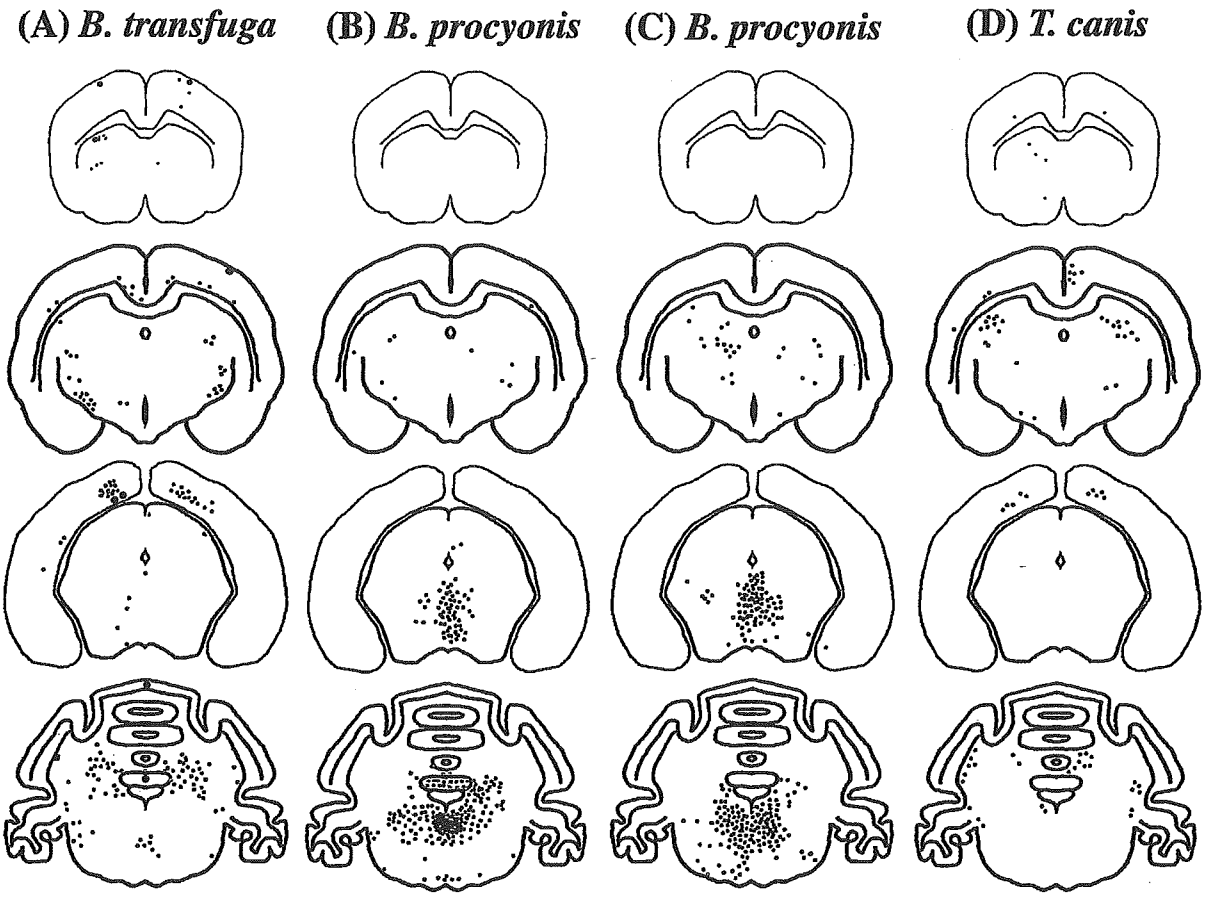

FIGURE 2. Diagram showing distribution of larvae (red dots) and histopathological changes such as malacia and gliosis (green dots) or granuloma formation (blue dots) associated with larva migrans caused by *Baylisascaris transfuga* (**A**), *Baylisascaris procyonis* (**B**, **C**) and *Toxocara canis* (**D**) in jirds. Serial transverse sections at 4 levels of the brains as shown here were prepared from 21, 23, and 15 jirds inoculated orally with embryonated eggs of *B. transfuga* (4,000 eggs), *B. procyonis* (100 eggs for B; 200 eggs for C) and *T. canis* (2,000 eggs), respectively. The foci detected on these sections were plotted accumulatively for each ascarid species. Note the distinct distribution of histological lesions in the brains of jirds infected with different ascarid species.

infection in this study was determined to be 10, or more, times the number of *B. procyonis* eggs (100 or 200 eggs/animal).

In this study, a group of jirds died on days 8-11 PI and were found at necropsy to have numerous fibrin-trapped larvae in the peritoneal cavity and marked pulmonary hemorrhages. Sprent (1952) noted that a considerable number of larvae migrated to the lungs within the first day, at which time the lungs showed hemorrhagic spots and patches that increased to a uniform congestion by the third day. After oral infection with 2,000 or 5,000 eggs, Matoff and Komandarev (1965) also noted numerous larvae in the peritoneal cavity until day 7 PI, then the number of localized larvae diminished markedly. They also noticed that encapsulation of live larvae was evident also in the subcutaneous tissues of the trunk, which was also found in our study. In the same study, Matoff and Komandarev (1965) found multiple larvae in the CNS throughout the observation period of up to day 22 PI. Sprent (1955) reported that *B. transfuga* larvae, 1.0 on average, were found in CNS of mice orally infected with 2,000-30,000 eggs. These authors did not notice neurological disorders in their mice despite the presence of multiple *B. transfuga* larvae in CNS tissues.

Papini and Casarosa (1994) investigated whether *B. transfuga* larvae in the CNS can cause neurological manifestations in mice. They argued that inoculation of high doses of infective eggs could induce neurological disorders in mice; they found neurological signs in 1 of 40 mice (2.5%), 2 of 30 mice (6.7%), and 2 of 10 mice (20.0%) inoculated with 3,000, 4,000, and 50,000 eggs, respectively, and 16–26 larvae were detected in the CNS of these 5 mice. On the basis of these results, Papini and Casarosa (1994) concluded that *B. transfuga* larvae were less pathogenic, compared with *B. procyonis*, *B. columnaris*, and *B. melis*, and ascribed lower pathogenicity of *B. transfuga* larvae partly to their smaller sizes attained on migration in mice. As shown in this study, the size of *B. transfuga* larvae found in laboratory rodents was comparable or overlapped significantly with that of highly pathogenic *B. procyonis* larvae, although larval size is commonly referred to as a critical factor in determining the outcome of ascarid neural LM. In addition to the predilection of the larvae to the CNS, the critical factor responsible for induction of neurological diseases in laboratory rodents seems to be host reactions against *Baylisascaris* spp. larvae in the CNS; when migrating larvae are encapsulated and immobilized in the brain, the affected rodent does not show neurological signs, as observed in mice with *B. transfuga* LM. Sheppard and Kazacos (1997) described 1 asymptomatic white-footed mouse (*Peromyscus leucopus*) with neural LM by *B.*

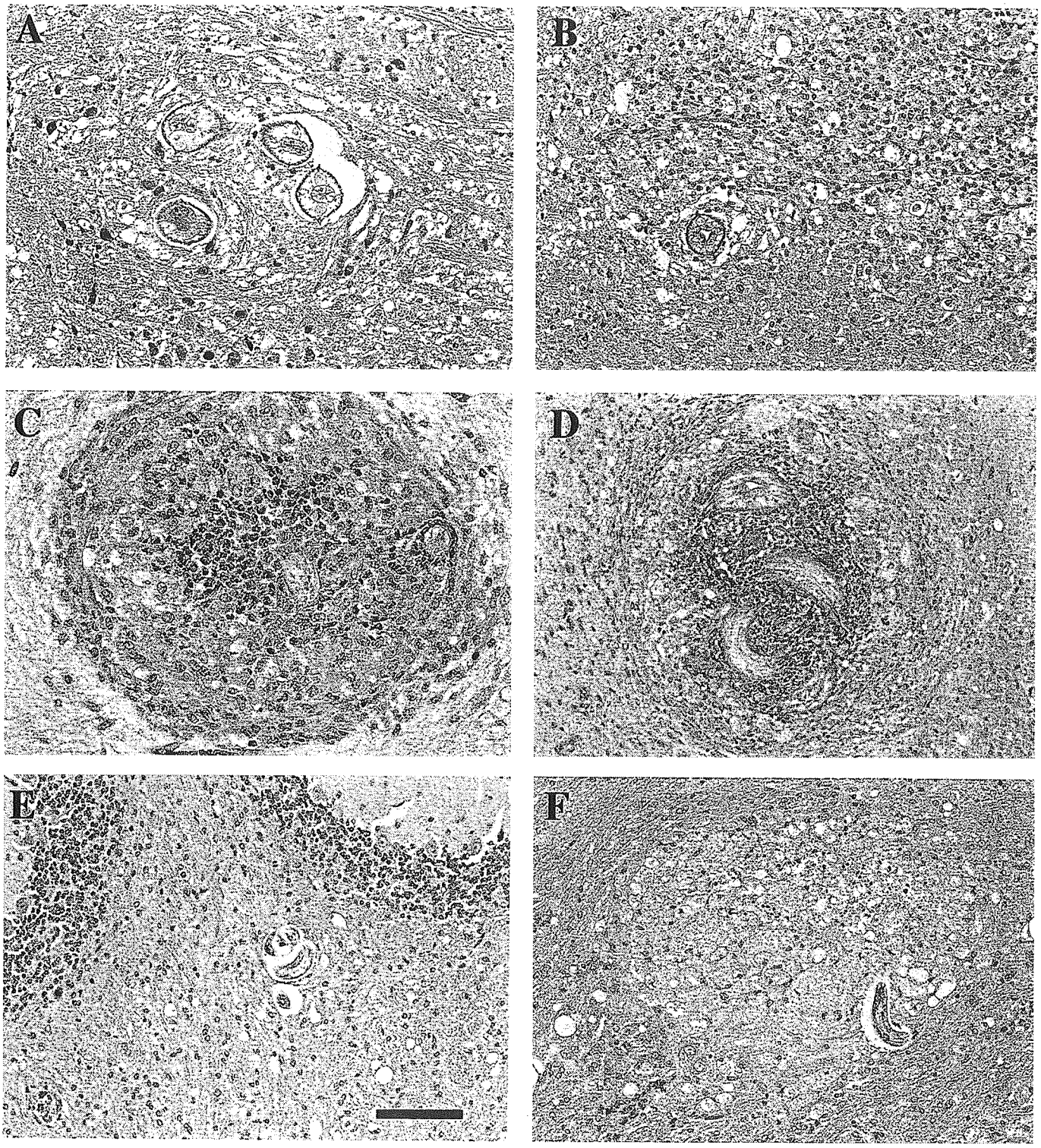

FIGURE 3. Histological changes associated with neural larva migrans by *Baylisascaris transfuga*, *Baylisascaris procyonis*, and *Toxocara canis* in jirds and mice. **A.** *Baylisascaris procyonis* larva in the jird cerebellum on day 31 PI. **B.** *Baylisascaris transfuga* larva and malacia with replacing accumulation of foamy macrophages in the jird cerebellum on day 9 PI. **C.** Granuloma immobilizing a *B. transfuga* larva in the hippocampus of an ICR mouse on day 28 PI. **D.** Granuloma immobilizing a *B. transfuga* larva in the cerebrum of an ICR mouse on day 60 PI. **E.** *Toxocara canis* larva in the cerebellum of an ICR mouse on day 77 PI. **F.** *Toxocara canis* larva and malacia with replacing accumulation of foamy macrophages in the jird cerebrum on day 93 PI. All figures except for (C) are at the same magnification; bar shown on (E) = 50 μm for (C); and 100 μm for other photographs.

TABLE II. Measurements of *Baylisascaris transfuga*, *Baylisascaris procyonis*, and *Toxocara canis* recovered from mice or jirds (in mm).

Parasite	Host	Day of collection	No. of worms examined	Worm length	Maximum worm width	Length of esophagus	Length of tail
B. transfuga							
	ICR mice	28	10	1.33 ± 0.14 (1.15–1.54)	0.070 ± 0.018 (0.071–0.093)	0.207 ± 0.033 (0.187–0.247)	0.124 ± 0.0017 (0.099–0.148)
	ICR mice	60	9	1.23 ± 0.28 (0.76–1.58)	0.061 ± 0.018 (0.039–0.088)	0.199 ± 0.039 (0.154–0.258)	0.120 ± 0.0021 (0.088–0.154)
	Jirds	26	10	1.28 ± 0.13 (1.08–1.52)	0.065 ± 0.013 (0.044–0.077)	0.217 ± 0.021 (0.187–0.253)	0.098 ± 0.0011 (0.082–0.115)
	Jirds	31	10	1.38 ± 0.13 (1.11–1.58)	0.079 ± 0.012 (0.060–0.099)	0.199 ± 0.046 (0.115–0.247)	0.113 ± 0.0013 (0.099–0.137)
B. procyonis							
	Jirds	26	7	1.41 ± 0.18 (1.17–1.57)	0.071 ± 0.010 (0.060–0.088)	0.199 ± 0.012 (0.187–0.214)	0.117 ± 0.0012 (0.099–0.132)
	Jirds	31	9	1.57 ± 0.07 (1.48–1.65)	0.084 ± 0.008 (0.071–0.093)	0.228 ± 0.011 (0.214–0.253)	0.120 ± 0.0016 (0.104–0.154)
	Jirds	60	8	1.59 ± 0.12 (1.39–1.79)	0.078 ± 0.010 (0.066–0.093)	0.231 ± 0.008 (0.220–0.242)	0.126 ± 0.0013 (0.104–0.143)
T. canis							
	Jirds	93	7	0.37 ± 0.02 (0.33–0.40)	0.021 ± 0.004 (0.016–0.027)	0.068 ± 0.009 (0.055–0.077)	0.062 ± 0.0005 (0.055–0.066)

procyonis due to encapsulation and immobilization of a single larva invading the CNS. Although *B. procyonis* larvae are rarely encapsulated in the CNS of rodent hosts and a single larva can induce clinical signs, the formation of granuloma around the larvae is a common finding in human and non-human primate cases of LM with encephalitis (Kazacos et al., 1981; Garlick et al., 1996; Kazacos, 1997; Rowley et al., 2000). In humans, it is speculated that CNS tissue damage by cytotoxic eosinophil granule proteins may contribute to the neurologic symptoms of *B. procyonis* infection (Hamann et al., 1989; Moertel et al., 2001). In rodent hosts, infiltration or accumulation of eosinophils around ascarid larvae, including *B. procyonis*, is not prominent; therefore, we might exclude possible modification or aggravation of the lesion by infiltrated eosinophils.

In mice with *T. canis* LM, a substantial number of larvae (1.1–6.1% relative to the number of inoculated eggs) were detected in the CNS, particularly the telencephalon and the cerebellum (Holland and Cox, 2001; Good et al., 2001). Although neural LM by *T. canis* is suggested to affect social behavior of affected mice, it is often difficult to detect neurological or locomotory changes associated with the infection (Sprent, 1953a, 1955). Indeed, we could not find any clinical signs except for roughened hair coats and depression in the acute phase of infection. In the mice in this study, histological examination showed no evidence of tissue damage, despite the localization of larvae in that area. In contrast, *T. canis* larvae localized in the CNS of jirds was associated with the appearance of several foci of foamy macrophages, indicating tissue damage. At this time, we could not find any clinical signs related to these histological lesions in these jirds. However, the size of larvae does not seem to be associated directly with distinct pathogenecity of different ascarid species as found in different clinical outcomes of *B. procyonis* and *B. transfuga* neural LM in mice. We are interested in defining the neural pathogenecity of ascarid larvae of different species by characterizing the biochemical properties of the parasite surface in association with host immune reactions. In addition, the histological sequels to migration of *T. canis* larvae in CNS tissues of mice and jirds are intriguing because the same ascarid larvae cause no tissue damage in mice CNS, but induce clear, but latent, tissue damage manifested solely by replacing accumulation of foamy macrophages.

Although distinct pathogenecity of *B. transfuga* larvae compared with *B. procyonis* larvae was demonstrated in this study, the morphological features and dimensions of the larvae were quite similar. It is practically very difficult to identify the causative agent for natural LM cases caused by *Baylisascaris* spp. An example is the primate cases mentioned in the Introduction. In addition, the distribution of feral raccoons is expanding and their population is increasing in mountainous Japan, where wild bears *Ursus arctos* and *Ursus thibetanus*, harboring *B. transfuga*, are common (Uni et al., 1995). To diagnose or determine the prevalence of *B. procyonis* LM in zoo and wild animals, a new approach involving molecular biology for differentiation of *B. procyonis* from *B. transfuga* might be critical, which should provide complementary data to routine parasitological methods involving morphological examinations. When referring to expanding distributions of raccoons worldwide, including Japan, efforts of continuous and critical monitoring of the

disease in the regions where raccoons are endemic or feral become an important public health issue.

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特集

イヌからうつる感染症

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SUMMARY

・イヌからヒトにうつる寄生虫には，①糞便内に存在する病原体が感染する，②体表に寄生する外部寄生虫が感染する，③イヌ固有の寄生虫が媒介動物によって感染するものがある．
・これらの中には悪性腫瘍と誤診された症例や治療法のない寄生虫，将来感染者の増加が懸念されている寄生虫もある．
・臨床医は患者の動物飼育歴を正しく把握し，動物由来感染症の可能性をつねに考慮に入れておく必要がある．

はじめに

現代社会においては，イヌは単なる愛玩動物としての存在から，伴侶動物として広く受け入れられるようになってきた．それにつれて，ヒトとイヌが密接に接触する機会が増えてきている．1999年に施行された，「感染症の予防および感染症の患者に対する医療に関する法律（感染症新法）」は動物由来感染症の予防対策を推進する上で重要な転換点となった．また，近年の医療の進歩は動物由来感染症の診断治療技術をめざましく発展させたが，イヌとヒトとの密接な接触に起因する感染症が過去の病気となってしまったわけではない．

ここでは，イヌからうつる寄生虫感染症のうち日常診療で遭遇する可能性の高いものについてその症状と検査法を中心に解説する（**表1**）．

Ⅰ　イヌの糞便内に病原体が存在する

① イヌ回虫幼虫移行症（トキソカラ症）

イヌの糞便内に排泄された回虫卵は適度の湿度と温度下で，2週間足らずのうちに幼虫包蔵卵にまで発育する．これがヒトに誤飲されると，消化管内で孵化して幼虫が体内に侵入する．公園などでの砂遊びによって感染する小児の感染症と考えられてきたが，最近ではニワトリなどの待機宿主

表1　イヌからうつる寄生虫症

寄生虫名	ヒトへの感染源	ヒト体内での寄生型	検査方法	抗体検査の重要度
糞便内に病原体が存在する				
イヌ回虫	幼虫包蔵卵（経口感染）	幼虫（300×20μm）	血清（眼内液）中の抗体検査	必須・重要
アライグマ回虫	幼虫包蔵卵（経口感染）	幼虫（2000×40μm）	血清（髄液）中の抗体検査	重要
エキノコックス	六鉤幼虫卵（経口感染）	包虫（体内で増殖）	血清中の抗体検査，病理組織検査	必須・重要
ランブル鞭毛虫	嚢子（経口感染）	栄養型と嚢子	糞便内の栄養型，嚢子の検出	利用可能
体表に寄生する外部寄生虫がヒトに感染する				
イヌノミ（ネコノミ）	成虫・虫卵（接触感染）	成虫	病変部からの虫体検出	不要
イヌヒゼンダニ	成虫・虫卵（接触感染）	成虫	病変部からの虫体検出	不要
媒介動物によってイヌを固有宿主とする寄生虫がヒトに感染する				
イヌ糸状虫	媒介蚊内の感染幼虫	幼若成虫	病理組織，血清中の抗体検査	利用可能
瓜実条虫	イヌノミ（ネコノミ）内の擬嚢尾虫	成虫	排泄された片節の形態	不要
マンソン裂頭条虫	第2中間宿主，待機宿主内のプレロセルコイド	プレロセルコイド（まれに小腸で成虫にまで発育）	病理組織，血清中の抗体検査	利用可能

の肝臓や筋肉の生食（幼虫が潜伏している）によって感染する成人例が増加してきている[1]．侵入した幼虫は消化管から肝臓，肺臓を通り全身の骨格筋や中枢神経系に移行する．

一度に多数の幼虫が侵入すると1～2週間で全身症状が現れる．その病型は以下の4つに分けられる．①**内臓移行型**：発熱，肺炎様症状，肝障害，皮疹．末梢血中の好酸球増多が必発する．②**眼移行型**：ぶどう膜炎（眼内炎型，後極部肉芽腫型，周辺部腫瘤型を区別する）を起こし，失明することもある．好酸球増多は著明ではない．眼型の発病機序や潜伏期間については不明な点が多い．③**中枢神経移行型**：てんかん様発作やアトピー性髄膜炎の原因となるといわれている[2]．④**不顕性感染型**：臨床症状がなく血清中の抗体のみが陽性で，軽度の好酸球増多やアレルギー症状を伴う．

診断は病変部位の生検によって幼虫断端を組織学的に証明することにより確定されるが，幼虫は300μm×20μmと非常に小さく，検出例は少ない[3,4]．内臓移行型ではCTやX線検査で肝臓や肺臓に多発性の小結節像を認める．補助的診断として，幼虫の排泄物抗原を用いた血清学的検査法が実施されている．眼型では硝子体液中の抗体が陽性となる．

治療は駆虫薬であるアルベンダゾールやメベンダゾールの経口投与が試みられている．眼型では駆虫剤の投与とともに病変部位のレーザー照射や凍結凝固により視力の低下を防ぐ措置が講じられている．砂遊び後の手指洗浄の励行や公園などでのイヌの排便を防ぎ，待機宿主となりうる動物の肝臓などの生食を控えることで予防できる．

② アライグマ回虫症

アライグマ回虫はアライグマの小腸に寄生する回虫であるが，イヌにも感染する．ヒトへはトキソカラ症と同じく，外界で発育したアライグマ回虫卵の誤飲によって起こる．トキソカラ症と異なり，アライグマ回虫は体内で2mm程度まで発育し，中枢神経系に寄生した幼虫によって起きる炎症反応により，斜頸，旋回運動，運動失調が出現し，全身麻痺から昏睡に至り死亡する．神経症状が現れる前には，嘔吐，易疲労感，言語障害がみられる．幼虫が眼球内に侵入すると瀰慢性片眼性亜急性視神経網膜炎（DUSN）を起こし失明する．

末梢血や髄液中の好酸球増多症がみられ，アライグマとの接触の機会があれば本症を疑う．診断はアライグマ回虫幼虫に対する特異抗体を血清中に証明するか，病理組織学的に幼虫を確認する．神経症状を呈した症例の治療は対処療法のみで，予後は不良．有効な駆虫薬は知られていない．

③ エキノコックス

イヌの小腸内に寄生する体長3mm程度の多包条虫が産出した虫卵をヒトが誤飲して感染する．北海道ではキタキツネの50％以上に多包条虫の寄生が確認されている．産卵時，虫卵内には六鉤幼虫が形成されており，外界に出た虫卵はすでにヒトへの感染力を持っている．ヒトへの感染後，幼虫は血流に乗って肝臓に至り，ここで嚢胞（多包虫）を形成して発育を開始する．ヒトは固有宿主ではなく中間宿主であり，包虫のまま増殖する．しかしその発育は緩徐で，感染後十数年間は無症状のまま経過する．

肝臓内で包虫が大きくなるにつれて，腹部膨満感，腹痛などの症状が現れ，さらに進行すると発熱，貧血，腹水貯留などの肝機能不全状態となる．肺臓や骨組織などの他臓器への転移もしばしば起こる．患者の大多数は北海道居住歴を有しているが，居住歴のない症例も本州各地から報告されている．最近，北海道内において飼育犬の感染が確認されたことから，イヌの移動に伴う感染拡大が懸念されている[5]．

病巣の外科的摘除がもっとも確実な治療法となるが，正常組織との境界が明瞭でなく，完全な摘出が困難な場合が多い．摘出組織のPAS染色で特徴的な構造を認める（**図1**）．薬物療法としてはアルベンダゾールが用いられているが，長期間の投与を必要とし，発育速度を抑えるのには有効であるが，著効例は少ない．

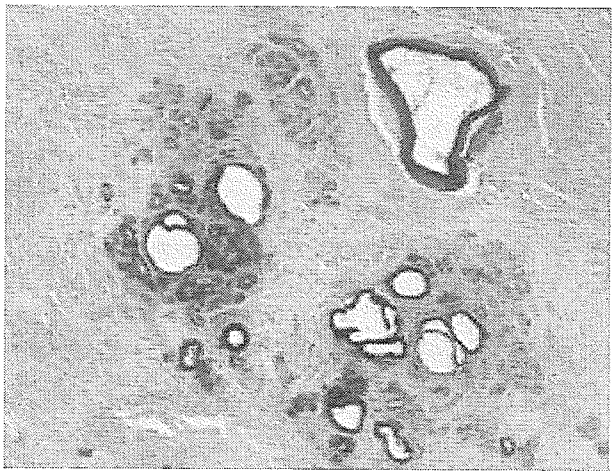

図1
北海道居住歴のない福井県在住62歳女性の肝臓にみられた多包虫症．大小不同の多数の嚢胞が認められる．PAS染色により包虫壁は濃染する．

④ ランブル鞭毛虫症（ジアルジア症）

Itoh, et al（2001）の調査では1035頭のイヌのうち151頭（14.6％）からランブル鞭毛虫の嚢子（シスト）を検出し，その感染率は幼犬において有意に高かったと報告されている[6]．ヒトへの感染は糞便中に排泄されたシストの経口感染による．潜伏期間は2～3週間で，ウイルス性や細菌性下痢症，食中毒，腸アメーバ症，病原性大腸菌感染症などとの鑑別が必要である．小児では吸収不良症候群がしばしばみられる．

診断は生鮮標本で糞便内に活発に運動する栄養型を検出するか，ホルマリン酢酸エチル沈澱法後のヨード染色標本によってシストを確認する．

Ⅱ　イヌの体表に寄生する外部寄生虫がヒトに感染する

イヌの体表にノミやダニなどの外部寄生虫が見つかることがある．通常はイヌからイヌへの接触感染によって感染が広がるが，イヌからヒトへの感染例もしばしば報告されている．イヌにみられ

るノミはそのほとんどがネコノミである．また，イヌと同衾してイヌヒゼンダニによる皮膚炎を生じた例がある．

Ⅲ　イヌを固有宿主とする寄生虫が媒介動物によってヒトに感染する

① イヌ糸状虫症

イヌの肺動脈内に寄生する15cm（雄）～25cm（雌）の糸状の線虫で，コガタアカイエカ，ヒトスジシマカ，トウゴウヤブカなどの蚊によって媒介される．イヌの血液内に産出されたミクロフィラリアが，蚊の吸血時に蚊体内に取り込まれると，約12日で感染幼虫となって唾液腺に集まり，次の吸血時にその刺し口から侵入する．ヒトへの感染は感染幼虫を持った媒介蚊の刺咬によって起こる．大部分の感染幼虫は侵入局所で死滅するが，まれに幼若成虫が肺臓内の末梢小動脈に栓塞して梗塞病変（**銭形陰影**）を作り，結核や肺癌と誤診されることがある（**図2**）．皮下や内臓諸臓器への異所寄生例も報告されている．

② 瓜実条虫症

イヌに広く感染がみられる条虫で，成人の寄生はまれであるが，乳幼児の感染がしばしば報告されている．イヌノミやネコノミが中間宿主となる．虫卵を摂取したノミの体内で擬嚢尾虫となり，ヒトはこれらを誤飲して感染する．気密性の高い住居で室内犬を飼育し，イヌノミやネコノミが繁殖する環境であれば感染は容易に起こりうる．ノミの誤飲から3～4週間で成虫にまで発育する．

乳児では，オムツの交換時に母親によって片節の排泄に気づかれることが多い．片節は白色のウリの種状でよく動く．少数寄生では無症状であるが，時に腹痛や下痢を伴うこともある．また，肛門周囲の瘙痒感や蕁麻疹を訴えることもある．

③ マンソン（幼）裂頭条虫症（マンソン孤虫症）

成虫はイヌの小腸内に寄生する．排泄された虫卵は第1（ケンミジンコ）および第2中間宿主（両生類，爬虫類，鳥類，ほ乳類などの多くの動

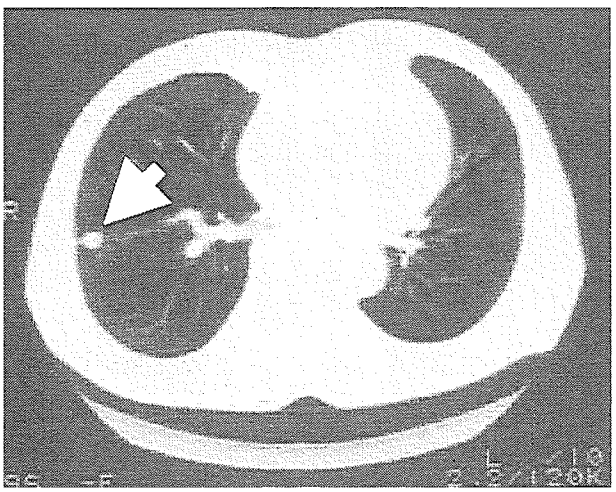

図2
肺イヌ糸状虫症の胸部CT所見．胸壁に接して銭形陰影を認める（矢印）．

図3
腹壁皮下に腫瘤を形成したマンソン幼裂頭条虫症のコッサ硝酸銀染色所見．幼虫（プレロセルコイド）内に多数の石灰小体を認める．

物）を経て成長し，第2中間宿主内でプレロセルコイドと呼ばれる白色紐状の幼虫になる．ヒトへの感染はプレロセルコイドを持った第2中間宿主の生食（いわゆるゲテもの喰い）によって起こる．ケンミジンコに汚染された飲料水からの感染もある．

通常ヒトの体内では成熟せず，幼虫のまま皮下に移動性の索状腫瘤をつくる．しかし，まれにではあるが小腸で成虫にまで発育することもある．成虫寄生例では腹痛，下痢などの消化器症状が強く現れ，末梢血好酸球増多もみられる．

幼虫は骨組織以外のさまざまな部位に移行する．好発部位は腹壁，胸壁，大腿部，乳房などの皮下組織であるが，乳房寄生例では乳癌と誤診され手術時に虫体が確認されることもある．深部眼球組織の寄生や脳内寄生では失明や神経症状が現れる．虫体断端の組織学的検査ではコッサ硝酸銀染色で黒褐色に染まる石灰小体が多数みられる（**図3**）．幼虫に有効な駆虫薬はなく，外科的摘除を行う．成虫寄生に対してはプラジカンテルを投与する．

おわりに

以上解説してきたように，ヒトを固有宿主とする寄生虫と異なり，動物由来の寄生虫の中にはヒトに対して不可逆的で重い障害を与えるものもある．動物由来感染症に対する正確で最新の知識が第一線の臨床医に求められている．

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どのようなときに疑うか？

特集

日常診療で役に立つ寄生虫情報システム

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SUMMARY

・寄生虫症に関する一般的，教科書的な情報を得るには，日本寄生虫学会ウェブページにアクセスする（http://jsp.tm.nagasaki-u.ac.jp/welcome-2.html）．またはウェブブラウザ上でグーグル検索を利用する．
・具体的な症例を相談する場合は，日本寄生虫学会ウェブページ表紙中央の「Consultation」をクリックする．parasite@jsp.tm.nagasaki-u.ac.jp 宛にメールを送るフォームが開くので，質問・相談内容を書き込み，送付する．

はじめに

医学雑誌の特集で，このような話題（特定疾患情報へのアクセス）がひとつの独立した項目として成立するのは珍しい．次の項で簡単に触れるが，寄生虫情報に限らずどのような情報でも今ではインターネットを駆使すれば，日常診療のために十分な情報を入手することが可能であり，取り立てて「情報システム」という項目を独立させる理由はない．にもかかわらず本特集にこの項目が存在する理由は，日本寄生虫学会がとくに寄生虫（疾患）情報へのアクセス整備に力を入れてきたからである．

寄生虫学会は，寄生虫疾患に日常の診療の中で具体的に対応するためには，寄生虫（疾患）に関する適切な情報が，いつでも，どこでも，誰でも，簡単に手に入れることができる状態が必要と考えてきた．その実現のため，早くから，「情報システム」の存在こそ最も必要なものと認識して，インターネットの普及と軌を一にして情報提供シス

テムの整備を行ってきた．その一旦を披露するのが本項目の主要な目的である．

ここでは，まず寄生虫（疾患）情報へのアクセスについて一般的な解説を行う．続いて**日本寄生虫学会が運営している「寄生虫疾患診断システム（コンサルテーション）」**を紹介する．

Ⅰ　寄生虫病に関する情報「システム」の必要性

国内で寄生虫疾患に遭遇する機会は，昭和40年代を境に急激に減少した．卒後一度も寄生虫を見たことがない医師も多く，患者糞便中の白くて長いものが寄生虫かそれ以外の虫か，それとも未消化のモヤシかさえ見分けることが難しい．寄生虫どころか，ミミズやヒル，果てはボウフラさえじっくり手に取って見る機会が少ない現代日本の状況では無理からぬことではある．実際に寄生虫症を経験した医師は多くはない．

一方で，寄生虫症の減少はまた，国内における寄生虫病学の重要性も相対的に低下させた．この十数年，医学部における寄生虫学関連の講義，実習は減少傾向で，寄生虫学関連教室も減る一方である．医学教育の中でも寄生虫学は相対的に軽視され，寄生虫症をよく知る医師や寄生虫専門家を見つけることすら困難になっている．

これに加えて，遭遇する寄生虫疾患も以前とは異なってきた．従来の，検便で診断できる古典的寄生虫病，腸管寄生虫病に代わって，輸入寄生虫病の増加，検便では捉えることのできない幼虫移行症，食生活の変化による寄生虫種の変化など，寄生虫疾患の多様化が進んでいる．かつての古典的寄生虫に関する知識や技術では追いつかない寄生虫病が相対的に増加している．

これらの結果，寄生虫疾患に対する知識，診断技術，経験ともに不十分な，若い世代の医師が急激に増加している．従来は各医師が周囲に相談者を見つけて容易に処理していたと考えられる寄生虫，例えば回虫などのごく基本的なものでさえ，大学の寄生虫学教室や医動物学教室などが直接しかも遠方から相談を受けるケースが増えている．寄生虫情報システムの必要性は，寄生虫症，寄生虫専門家，両者の減少に起因している．

Ⅱ　インターネットによる情報収集

情報システムといえばインターネットである．インターネットの普及は過去十年ほどで革命的に世の中を変え，寄生虫情報に限らず医療情報一般にも大きな影響を与えた．とくにIE（Internet Explorer），Netscapeなどのウェブブラウザで閲覧するWWW（world wide web, webpage）や電子メールはいまや情報メディアのスタンダードである．多くの医師が日常的に利用するようになった今では改めて解説する必要もないが，まず簡単に触れる．

❶ WWWとグーグル革命

情報通信革命すなわちインターネットが革命と呼ばれた所以は，第一に情報の入手手段の大変革である．インターネットが普及する前，医療情報は学術書，学術誌，公的機関からのニュースレターなどの印刷物（ハードコピー）に頼るしかなかった．そのためにはそれらを購入するか，大学の図書館を利用するか，どちらにしても時間もかかった．それが今では，インターネットという手