A Contribution to the Etiology of Racemose Cysticercosis

S. Lachberg, R. C. A. Thompson, and A. J. Lymbery, Division of Veterinary Biology, School of Veterinary Studies, Murdoch University, Western Australia, 6150

ABSTRACT: An aberrant case of proliferating coenurosis caused by Taenia serialis in immunosuppressed mice is described. The coenuri closely resembled examples of racemose cysticerci described in the literature and the present results are discussed with reference to human cases of asexually proliferating larval cestode infections.

The precise etiology of the many cases of infection by accephal asexually budding cestode larvae (metacestodes) recovered from humans and other animals is uncertain (Beaver and Roland, 1981). Although determination of the species of cestode may remain equivocal, several types of metacestodes are thought to be involved, including aberrant spargana, cysticerci, cysticercoids, and tetrathyridia (Beaver and Roland, 1981). Of the forms described, the racemose cysticercus ("cysticercus racemosus") refers to aberrant proliferating cestode larvae that manifest as an unencapsulated bladder or several bladders that "bud" exogenously to form a multilocular cyst resembling a bunch of grapes (Slais, 1967; Manson-Bahr and Bell, 1987). Specific identification is difficult as this form is usually devoid of scoleces. As such, and because generally it is localized in the human central nervous system, it is commonly regarded as an aberrant cysticercus of Taenia solium. However, it has been suggested that racemose cysticercus may, in some instances, be a sterile coenurus (Hermos et al., 1970; Beaver and Roland, 1981; Benger et al., 1981; Jung et al., 1981). Assigning these atypical forms of coenuri to a particular species was based on hook size and shape where scoleces were present. Unfortunately, even with "normal" coenuri, hook size does not provide reliable identification. Table I shows the overlap in size of hooks among the 3 species associated with human coenurosis: Taenia multiceps, Taenia serialis, and Taenia brauni (Acha and Szhyres, 1987). It is clear that identification is difficult without carrying out animal feeding experiments using tapeworm-free dogs for rearing adult worms (Benger et al., 1981). In this way, strobilar characters, in particular the anatomy of the female reproductive system, can be observed, enabling a definitive identification (Soulsby, 1982).

Our laboratory recently received a subcutaneous cyst from a rabbit. The cyst was identified as T. serialis on the basis of its characteristic location in the intermediate host, gross morphology, and rostellar hook size. Several proto-
scoleces were fed to a dog. Praziquantel (Droncit, Bayer) was administered to the dog 21 days prior to infection and no subsequent evidence of helminth infection was detected in the feces during this period. Thirty-six days postinfection (PI) (when gravid segments were first noticed in the feces), the dog was killed with an injection of sodium pentobarbitone via the radial vein. The small intestine was removed quickly, dissected along its length, and the adult worms collected, placed in phosphate-buffered saline, and positively identified as *T. serialis*. Eggs were teased from the worms and 10 male mice (5 Quackenbush Swiss [QS], 5 CBA/CaH) were infected with approximately 1,500 eggs each via a stomach tube. Mice were immunosuppressed with betamethasone using the protocol described by Esch (1964).

Three of the mice (1QS, 2 CBA/CaH) autopsied 6 wk PI had developed fertile subcutaneous cysts that ranged from 0.5 to 1.0 cm in diameter. The life cycle was subsequently maintained by feeding approximately 10 protoscoleces from 1 cyst to a dog. From the second adult generation only 1 worm was recovered. Eggs were teased from this worm and used to infect another group of 10 immunosuppressed male mice (5QS, 5CBA/CaH). Three large subcutaneous cysts were found in 3 of the immunosuppressed mice (1QS, 2 CBA/CaH) when autopsied 8 wk PI. These mice also harbored cysts in their peritoneal cavity. One of these cysts was a large (approximately 5 cm diameter), asymmetrical, proliferating structure devoid of scoleces (Fig. 1). It closely resembled examples of racemose cysticercus described in the literature (Jung et al., 1981; Manson-Bahr and Bell, 1987). The other peritoneal cysts were smaller (approximately 2 cm diameter), proliferative in nature and, in 1 case, possessed a single scolex. This example is reported as it is the only case of such aberrant development by a taenid metacestode where the original species is known with certainty. Although we have shown that *T. serialis* can be a cause of so-called “cysticercus racemosus,” this does not preclude the involvement of other species, such as *T. solium*, in this disease.

**LITERATURE CITED**


ŚLAIAS, J. 1967. The morphology of *Cysticercus ra-

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**Figure 1.** Asymmetrical, sterile, proliferating coenurous of *Taenia serialis* removed from the peritoneal cavity of an immunosuppressed mouse 8 wk after infection (scale bar = 0.5 cm).

**Table 1.** A comparison of the range of larval hook size amongst 3 species of *Taenia* thought to be involved in human coenurosis.*

<table>
<thead>
<tr>
<th>Species</th>
<th>Larval hook</th>
<th>Size range (µm)</th>
<th>Mean (±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>T. multiceps</em></td>
<td>Large</td>
<td>157–177</td>
<td>166.71 (5.3)</td>
</tr>
<tr>
<td></td>
<td>Small</td>
<td>109–136</td>
<td>125.0 (5.8)</td>
</tr>
<tr>
<td><em>T. serialis</em></td>
<td>Large</td>
<td>145–170</td>
<td>155.8 (5.4)</td>
</tr>
<tr>
<td></td>
<td>Small</td>
<td>95–125</td>
<td>111.0 (6.2)</td>
</tr>
<tr>
<td><em>T. brauni</em></td>
<td>Large</td>
<td>139–150</td>
<td>144.8 (2.6)</td>
</tr>
<tr>
<td></td>
<td>Small</td>
<td>102–114</td>
<td>108.2 (2.7)</td>
</tr>
</tbody>
</table>

* Data from Verster (1969), who originally considered *T. brauni* to be a subspecies of *T. serialis*.
Leishmania braziliensis in the Squirrel Monkey: Development of Primary and Satellite Lesions and Lack of Cross-immunity with Leishmania donovani

Ricardo Luján*, Willie L. Chapman, Jr.,†, William L. Hanson, and Vida A. Dennist, Department of Parasitology, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602. *Present address: Centro de Investigaciones en Enfermedades Tropicales, Universidad del Valle de Guatemala, Apartado Postal 82, 01901, Guatemala, Guatemala. †Department of Pathology, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, and to whom reprint requests should be addressed. ‡Present address: Delta Regional Primate Research Center of Tulane University, Three Rivers Rd., Covington, Louisiana 70433

ABSTRACT: Three female and 2 male adult laboratory-reared squirrel monkeys (Saimiri sciureus) that previously had been inoculated with Leishmania (Leishmania) donovani and had recovered from experimental visceral leishmaniasis were each inoculated intradermally at the dorsal base of the tail with 2.2 × 10⁷ culture-derived promastigotes of Leishmania (Viannia) panamensis. The progression and regression of subsequent lesions were examined for 36 wk in all 5 monkeys after which 3 of the monkeys were killed (1 with a primary lesion and all with satellite lesions) and the 2 surviving monkeys (1 with primary lesion and both with satellite lesions) were treated with 104 mg/kg/day of meglumine antimoniate for 10 days. All of the monkeys developed a primary lesion at the site of injection of the parasite and later developed satellite lesions peripheral to the primary nodule. The primary lesions had disappeared from 3 of the 5 monkeys by 36 wk, whereas satellite lesions persisted on all at this time. Satellite lesions were present at 52 wk after treatment and persisted for 169 wk in the 2 surviving monkeys. The histopathologic appearance of the lesions was characterized as granulomatous inflammation. Our results indicated that squirrel monkeys that had recovered from visceral leishmaniasis remained susceptible to infection with L. (V.) panamensis.

American cutaneous–mucocutaneous leishmaniasis are caused by hemoflagellate protozoa parasites primarily of the Leishmania braziliensis and Leishmania mexicana complexes. These diseases are recognized as a serious and increasing problem in the western hemisphere (Lainson and Shaw, 1979, 1987).

Cutaneous leishmanial lesions in humans may be single or multiple and may have various cutaneous manifestations ranging from frank ulcerative to proliferative forms. The classical lesion is circular and ulcerated, with elevated discolored borders and a central crater (Deane and Grimaldi, 1985; Lumberas and Guerra, 1985; Scorza et al., 1985; Zeledon, 1985; Walton, 1987).

The lesions produced by Leishmania braziliensis panamensis differ little in their development or appearance from other forms of cutaneous leishmaniasis (Zeledon, 1985; Walton, 1987). Satellite lesions (lesions peripheral to the original site of infection) have been reported in humans infected with L. b. panamensis in Costa Rica (Chu et al., 1983) and in other Latin American countries (Lovelace, pers. comm.). Lesions may heal spontaneously, but there is a tendency for the lesion(s) to persist for 10 yr or longer (Zeledon, 1985).

Recovery in monkeys (Cebus apella apella) from infection with L. braziliensis in most cases results in firm resistance to infection against L. mexicana, but the reverse was not true (Lainson and Shaw, 1977). No cross-immunity was found between L. donovani and Leishmania tropica in humans (Manson-Bahr, 1961, 1971), and a similar lack of cross-immunity would be expected in monkeys although no report of this is available. These studies were conducted to study the susceptibility of squirrel monkeys to Leishmania (Viannia) panamensis following recovery from Leishmania (Leishmania) donovani and to char-

*cemosus and the determination of the Cysticercus species. Folia Parasitologica (Praha) 14: 27–34.


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