Shigella flexneri Type 2 Infection in Captive Nonhuman Primates

DEAR SIR:

Gastrointestinal diseases are among the most important problems of nonhuman primates (1) and there is ample evidence that shigellosis and salmonellosis contribute significantly to the overal problem (1-9). In this report, an outbreak of shigellosis due to *Shigella flexneri* type 2 among primates in the Granby Zoo (Quebec, Canada) is described.

On October 9, 1984, three adult gibbons were anorectic and depressed and a few hours later manifested a profuse diarrhea. They were given kaopectate, but the following day the diarrhea had increased and the animals were prostrate. They were then treated orally with amoxycillin and intramuscularly with trimethoprim-sulfamethoxazole (TMP-SMZ). Two days later, one of them, a two year old animal, died. The necropsy revealed lesions limited to the large intestine: acute colitis, appendicitis and hemorrhagic proctitis without visible erosions or ulcers. The bacteriological examination demonstrated the presence of a *Shigella* spp in the duodenum, jejunum, ileum and colon, but not in the other viscera. Antimicrobic susceptibility testing revealed that the agent was sensitive to cephradin, neomycin, gentamicin, TMP-SMZ and resistant to ampicillin, chloramphenicol, streptomycin, tetracyclines and sulfonamides.

In order to prevent the dissemination of this microorganism among other primates, particularly the gorillas, the two other affected gibbons were euthanized. They had the same lesions as the first animal and *Shigella* spp was isolated from the intestines.

The three isolates of *Shigella* spp were sent to the Laboratoire d'Entérobactériologie, Ministère des Affaires sociales, Sainte-Anne de Bellevue, Quebec, and all were identified as *Shigella flexneri* type 2.

On October 16, the two gorillas presented with diarrhea and one had hemorrhagic feces. Based on the previous antibiogram, it was decided to treat the gorillas with TMP-SMZ given orally mixed with bread and yogourt. They refused to eat these foods. Finally, they accepted the treatment when given in orange juice. At this time, chimpanzees were also treated with TMP-SMZ as a preventive measure.

On October 18, the gorillas recovered almost completely. Bacteriological results revealed that they had been infected with the same agent, *Shigella flexneri* type 2. Bacteriological examination of water accessible to the different species of monkeys did not reveal the presence of the microorganism. Furthermore, bacteriological examination on feces from the gorillas, carried out twice at monthly intervals, was negative for the presence of *Shigella*.

Outbreaks of *Shigella* dysentery have occurred in several zoo colonies, from which many types have been isolated (10). The most common has been *S. flexneri* (2,6). Control of an outbreak involves breaking the cycle of infection and transmission, by prophylactic treatment and the use of hygiene and physical barriers (7). Elimination of the carrier state is desirable since carriers represent a constant source of infection and many shigellae are not susceptible to commonly used antibiotics (2). Trimethoprim-sulfamethoxazole is a drug combination which has proven its efficiency in human (11) and simian (4) cases of shigellosis. In the present outbreak, this antimicrobial substance was able to eliminate the *Shigella* carrier state in the gorillas.

Our results of antimicrobial susceptibility tests on *Shigella* agree with those of previous authors (3,7). Indeed, multiple antibiotic resistance was reported to include tetracyclines, sulfonamide, streptomycin and chloramphenicol.

It was not possible to find the source of the infection as no monkey had been introduced into the zoo during the previous nine months and as no human case had been diagnosed among employees. Nonetheless it is highly probable that primates do acquire shigellae after contact with humans since a number of workers emphasize the failure to isolate the organism from free-living primates (1). Minimizing human contact is probably the most important factor in prevention of shigellosis (1).

Yours sincerely,

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Alopecia Areata in a Dog

DEAR SIR:

I wish to report a case of alopecia areata in an 18 month old Dachshund. Alopecia areata is a rare skin disorder in the canine, characterized by focal and multifocal patches of noninflammatory hair loss. The etiology is unknown. Therapeutic management of this disease in the canine is unclear, although systemic or intralesional glucocorticoids may be helpful in man with nonresolving or progressive alopecia.

The patient, a spayed female, was presented in September 1983. The animal had multifocal areas of alopecia on the head and shoulders. There was no pruritus, the skin was smooth and clean. Skin scrapings and fungal cultures were negative. The T4 was within normal limits.

A tentative diagnosis of alopecia areata was made in January 1984 and treatment with 10 mg IM of methylprednisolone acetate (Depo-Medrol, Tuco Products Company, Orangeville, Ontario) was given with no improvement. The lesions remained static for the next twelve months and in February 1985, a course of levothyroxine sodium (Synthroid, Flint Division of Travenol Laboratories, Malton, Ontario) was initiated for three months with no improvement in the lesions. Two skin biopsies were taken in June 1985, preserved in 10% formalin and sent for histopathology (Histovet, Guelph, Ontario). The first biopsy revealed a mononuclear cell perifolliculitis associated with degenerating hair follicles, in the telogen and catagen phase. The inflammatory cells were clustered around the hairbulb and were composed of lymphocytes, plasma cells and a few macrophages. The second biopsy had a more generalized perifolliculitis and periadenitis, with the inflammatory cells being chiefly mononuclear, as in the first biopsy. These findings are consistent with the diagnosis of alopecia areata in both dog and man.

Of interest is that the areas of hair loss appeared suddenly and had stabilized after four months. No appreciable change has occurred for almost two years.

Yours faithfully,

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ABSTRACTS

SHULL RM, HELMAN RG, SOELLACY E, CONSTANTOPOULOS G, MUNGER RJ, NEUFELD EF. Morphologic and biochemical studies of canine mucopolysaccharidosis I. *American Journal of Pathology* 1984; 114: 487-495 (Dep. Path., Coll. Vet. Med., PO Box 1071, Knoxville, Tennessee 37901-1071, USA).

A 6-month-old bitch with mucopolysaccharidosis I (MPS I; L- iduronidase deficiency) was observed for 13 months, then killed. This was the most severely affected animal in a litter which included 2 affected males, 3 normal puppies and 7 others of unknown enzyme activity. Gross pathology, light and electron microscopic findings, and tissue enzyme, glycosaminoglycan and sphingolipid levels were compared with those reported for human and feline MPS I. Results support the similarities between the canine disease and MPS I in man.

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BURSTEIN H, GILEAD M, BENDHEIM U, KOTLER M. Viral aetiology of haemangiosarcoma outbreaks among layer hens. *Avian Pathology* 1984; 13: 715-726 (Dep. Molecular Genetics, Hebrew Univ., Hadassah Med. Sch., POB 1172, 91.010 Jerusalem, Israel).

Outbreaks of neoplastic disease defined as haemangiosarcoma occurred among layer flocks of chickens on 60 farms in Israel. The disease caused bleeding tumours in the skin and internal organs of young layers, followed by anaemia, cessation of egg production and high mortality up to 20%. Avian leukosis virus was isolated from tumour cells containing several viral DNA copies integrated in the cell genome. The isolated virus induced haemangiosarcomas in more than 30% of birds inoculated on the day of hatching. Congenital transmission of viruses from tumour-bearing hens to their offspring was observed.

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LAIRMORE MD, ALEXANDER AF, POWERS BE, MILISEN WB, McCHESNEY AE, SPRAKER TS. Oxytetracycline-associated nephrotoxicosis in feedlot calves. *Journal of the American Veterinary Medical Association* 1984; 185: 793-795 (Coll. Vet. Med. State Univ., Fort Collins, Collins, Colorado 80523, USA).

Renal nephrosis and increased mortality were investigated in feedlot calves of 180-190 kg that had received excessive doses of oxytetracycline for the treatment of bronchopneumonia. Histological findings included moderate to severe cortical tubular nephrosis, and suppurative bronchopneumonia. Results of serum and peritoneal fluid analysis were consistent with severe renal disease. Renal toxicosis from the excessive oxytetracycline should be considered a possible side effect in stressed calves with concurrent respiratory disease.

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