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OUTBREAK OF CLINICAL AMOEBOSIS AMONG CAPTIVE CHIMPANZEES (*PAN TROGLODYTES*) AT THE SANDA KYARIMI PARK, MAIDUGURI, NIGERIA

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SUMMARY

An outbreak of clinical amoebosis due to *Entamoeba histolytica* was investigated among four captive chimpanzees (*Pan troglodytes*) at the Sanda Kyarimi Park in Maiduguri, Nigeria. One female out of four adult chimpanzees (aged 15 25 years old) in the colony manifested clinical amoebosis characterised by dysentery, slight dehydration, bouts of abdominal cramps, severe tenesmus and prolapse of the rectum. Clinical and laboratory investigations revealed that the affected chimpanzee had slightly elevated rectal temperature, capillary refill time, pulse, respiratory and heart rates, total white blood cell and neutrophil counts but reduced packed cell volume, haemoglobin concentration and red blood cell counts than the other colony mates. All the chimpanzees in the colony were shedding the ova of *Trichuris trichiura* and the cysts and trophozoites of *Entamoeba hystolytica* in their faeces at the time of the outbreak with the clinically sick female having higher counts of these parasites than the other colony mates. Although *Trichuris trichiura* infection had been recurrent in the colony, it is believed that the animals acquired their *Entamoeba* infection from one of the human attendants that was shedding both *Trichuris trichiura* ova and *Entamoeba histolytica* cysts in his faeces at the time of the outbreak.

***KEYWORDS*:** Clinical amoebosis, chimpanzees, Maiduguri, Nigeria

INTRODUCTION

Amoebosis or amoebic dysentery, caused by *Entamoeba histolytica*, is a common disease of man usually transmitted between hosts through the faecal-oral route in cyst contaminated food or water (WHO, 1985; Levine, 1973). The disease is zoonotic with man usually acting as the reservoir of infection for other primates, domestic and ild animals (Elsdon-Dew 1968, Levine 1973). Amoebosis constitutes a serious health problem in chimpanzee colonies in zoological gardens (Miller and Brays 1966, Levine 1973, Soulsby 1982). Presently, information is lacking on the status of the disease in domestic, wild and captive animals in Nigeria. This paper describes, the occurrence of clinical amoebosis due to *Entamoeba histolytica* in captive chimpanzees at the Sanda Kyarimi Park in Maiduguri, Nigeria.

CASE REPORT

The outbreak occurred at the Sanda Kyarimi Park (Zoological Garden) in Maiduguri, Nigeria. The park has four chimpanzees (2 males and 2 females) in its collection. The animals were acquired in 1980 and have shared the same primate colony complex since then. They were in apparently good health until 'Mary' one of the females (25 years old) showed bouts of abdominal cramps with a tucked up abdominal posture characterized by holding the lower abdomen with one hand and covering the eyes with the other. She had bloody diarrhoea; tenesmus and severe straining that resulted in the prolapse of the rectum by the second day. The other colony mates did not manifest any of these clinical signs.

Each of the chimpanzees was restrained by dart injection of 50 mg ketamine hydrochloride (Ketalar®) for routine clinical examination. Blood, rectal faecal and soil samples from various locations within the primate complex, were collected and examined routinely to estimate haematological parameters and detect haemoparasites, helminth ova and protozoal trophozoites and cysts (MAFF 1977; Fleck and Moody, 1988; Schalm *et al.*, 1995). The number of *Entamoeba* trophozoites was estimated per microscope field of a wet preparation (Levine, 1973; Fleck and Moody, 1988).

The four attendants in charge of the primate colony complex were referred to the State Specialist Hospital, Maiduguri for routine medical examination and treatment while each of the animals was treated with Metranidazole (flagyl®, May and Baker Nigeria Plc., 400 mg orally, 3 times daily for 7 days), Albendazole (Zentel®, SmithKline Beecham, France, 25 mg/kg orally, daily for 2 days) and Tetracycline hydrochloride (250 mg orally, 4 times daily for 5 days). The clinically sick female, in addition, was given the anti spasmodic, analgesic Diclofenac calcium (Cataflan®, Novartis Farma, Switzerland, 50 mg orally, 3 times daily for 4 days).

RESULTS AND DISCUSSION

The clinically affected chimpanzee was weak, slightly dehydrated, had diarrhoea and the lower abdomen was tender to touch. The other colony mates were in apparently good health. The sick chimpanzee also had relatively higher clinical parameters, total WBC and neutrophil counts but lower PCV, Hb concentration and RBC values than her other colony mates (Table I).

External and blood parasites were not encountered during clinical and laboratory examinations but all the chimpanzees were shedding the ova of *Trichuris trichiura* and the cysts and trophozoites of *Entamoeba histolytica* in the their faeces at the time of the outbreak (Table II). The sick chimpanzee had higher counts of all the parasite stages than the other colony mates. Large numbers of *Trichuris trichiura* ova were also recovered from soil samples taken from various locations within the primate complex housing the chimpanzees. One of the four attendants caring for the animals was shedding large numbers of *Trichuris trichiura* ova and the cysts of *Entamoeba histolytica* in the faeces at the time of the outbreak.

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The treatment regimen administered to the animals stopped the clinical manifestations and the faeces were negative for nematode ova and protozoan cysts and trophozoites within three

days of therapy. The infected attendant was also successfully treated at the State Specialists hospital.

The results of this study suggest that the clinically sick chimpanzee suffered from amoebosis rather than trichurosis. The manifested symptoms were indicative of ulcerative colitis, common in clinical intestinal amoebosis due to *E. hystolytica.* (Miller and Brays, 1966; Soulsby, 1982; Martinez-Palomo, 1987). Consequently, the fall in PCV, Hb concentration and RBC values of the affected chimpanzee may be due to bleeding ulcers and erosion of the intestinal mucosae usually associated with intestinal amoebosis while the elevation in rectal temperature and increased neutrophil and WBC counts may have resulted from secondary bacterial invasion of intestinal lesions caused by the amoeba (Smith *et al.,* 1972).

It is probable that the chimpanzees acquired their infection from the infected human attendant (Levine 1973, Martinez-Palomo, 1987). Consequently, the concurrent *Trichuris trichiura* infection may have produced the intestinal environment responsible for the manifestation of the clinical amoebosis in this outbreak. Several factors including captivity situations and the presence of concurrent trichuriasis have been shown to increase the susceptibility of chimpanzees to *Entamoeba histolytica* (Levine, 1973; Miller and Brays, 1966; Sepulveda and Martinez-Palomo, 1982). Previous reports suggest that *Trichuris trichiura* is endemic in the primate colony environment with constant resurgence of infection of the animals with the parasite (Nwosu, 1995).

In conclusion therefore, the results of the present investigation show that amoebosis may constitute a serious health problem to primates, zoo attendants and visitors. Consequently, the disease should be considered in the differential diagnosis of diarrhoea and dysentery for chimpanzees in captivity. They further emphasize the need for regular routine screening followed by treatment of zoo attendants and animals in order to prevent the transfer of infective agents from man to the zoo inmates and vice versa.

TABLE I. Routine clinic al and haematological parameters of the chimpanzees examined at Maiduguri, Nigeria.

Parameters	Male 1	Male 2	Female 1*	Female 2	All animals
Clinical parameters					
C)? Temperature (	37.6	37.8	40.6	37.4	38.4 ± 1.5
Pulse rate/minute	70	80	90	70	77.5 ± 9.6
Heart rate/minute	80	90	95	80	86.3 ± 7.5
Respiratory rate/minute	60	70	90	60	70 ± 14.1
Capillary refill time/sec.	2	2	4	3	2.8 ± 1.0
Haematological parameters					
PCV (%)	40	41.2	30	40.4	37.9 ± 5.3
Hb	12.7	10	9	12.9	11.2 ± 2.0
RBC	5.2	5.4	4.5	5.5	5.2 ± 0.5
WBC	5.7	5.5	8.4	4.3	6.0 ± 1.7
Neutrophil (%)	65.9	65.8	68.5	64.1	66.1 ± 1.8
Lymphocyte (%)	32.4	32.4	29.7	34.2	32.2 ± 1.9
Eosinophil (%)	0.8	0.8	0.9	0.8	0.8 ± 0.1
Monocyte (%)	0.3	0.4	0.4	0.3	0.4 ± 0.1
Basophil (%)	0.6	0.6	0.5	0.6	0.6 ± 0.1

*Female chimpanzee that manifested the clinical disease

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