VALVULAR ENDOCARDITIS IN A CAPTIVE MONKEY IN IBADAN, NIGERIA: A CASE REPORT

EMIKPE*, B. O., ADENIRAN¹, G. A., ALAKA¹, O. O., OHOBE¹, O. G., ANTIA¹, R. E., AJAYI¹, O. L. and OMÖBOWALE², O.T.

¹Department of Veterinary Pathology and ²Veterinary Teaching Hospital University of Ibadan, Ibadan - Nigeria.

Correspondence: E-mail: Tel: +234 805 030 3798

INTRODUCTION

Primates are present in large number in the game reserves and national parks throughout Nigeria and other parts of the world (Ayodele, 1988). Out of these primates, monkeys and baboons seem to be most abundant. The interrelationship between these primates and humans, as relates to disease similarity is of great importance (Trube and Bassett, 1978). Congestive heart failure which can be due to mural or valvular endocarditis has been found to be a product of a septicemic condition (Robinson and Maxie, 1985). There have been various reports of the pathology of heart related conditions in primates in different parts of the world (Isoun et al., 1972; McConnell et al., 1974; Levin and Carey, 1986 and Canfell et al., 1986). However, there is dearth of information on heart related condition in Nigeria (Chineme et al., 1978); hence this report presents a case of valvular endocarditis associated with hepatic necrosis and hemorrhagic pneumonia in a captive monkey (Cercopithecus torquatus torgilatus) at the University of Ibadan Zoological garden of the University of Ibadan.

KEY WORDS: Monkey, Endocarditis, Ibadan, Nigeria

CASE HISTORY

An adult male monkey of 25 years old was one of the 10 in a colony of monkeys housed at the University of Ibadan Zoo for 23 years. The animal was presented to the Veterinary Teaching Hospital, university of Ibadan with the signs of laboured breathing and epistaxis. The blood oozing from the nostrils was fresh and clotted. The animal later collapsed and died prior to medical intervention.

Pathology and Microbiology

When the animal was necropsied, the carcass was well fleshed, but showed swelling of the right eye. There was bloody discharge from the nostrils; some of the blood was clotted. A thin smear of the bloody discharge stained with giemsa and methylene blue was negative for anthrax bacilli. The trachea contained blood stained frothy exudates, while the bronchi were filled with frank blood. The lungs were oedematous and congested with focal areas of haemorrhage. The thoracic cavity contained clotted blood, while about 10 ml of serosanguinous fluid was present in the pericardial sac. The left ventricular wall was flabby and pale with focal yellowish areas. There was an irregularly shaped vegetative growth on the left atrioventricular (mitral) valve of the heart (Plate 1). Haemorrhages were observed in the stomach, duodenum, jejunum and ileum as dark starry contents, while the liver was shrunken with pale, rough and granular surface. The spleen was slightly enlarged.

Specimens of the heart, lungs spleen liver, and intestine were fixed in 10% buffered formalin. These samples were processed for histopathology and stained with haematoxylin and eosin. Histopathologic findings included pulmonary congestion, oedema and
haemorrhages. The macrophages were laden with haemosiderin in the alveoli and its wall.
The liver showed areas of diffuse necrosis (Plate 2) with disorganization of hepatic cords.
The heart muscles also had areas of necrosis, degeneration, and disorganization. There were
neutrophilic infiltrations of the myocardium and bacterial colonies (Plate 3). Samples from
the liver and the arterioventricular valve of the heart with vegetative growth and the liver were
submitted aseptically for bacterial cultures. Isolates were identified by the methods of
described by Carter et al (1995) as *Staphylococcus aureus* and *Listeria monocytogenes*.

Drug sensitivity tests of bacterial isolates using multidisc revealed the organisms to be sensitive
to chloramphenicol at 30 mcg, while *Staphylococcus aureus* was also sensitive to
gentamycin at 10 mcg.

**PLATE 1:** Irregularly shaped vegetative growth on the
left atrioventricular (mitral) valve of the heart (X2)

**PLATE 2:** The liver showed areas of diffuse
necrosis (Hematoxylin-eosin stain, 250 X)

**PLATE 3:** The valve shows inflammatory cells and
bacterial colonies (Hematoxylin-eosin stain, 150 X)

**DISCUSSION**

Valvular endocarditis has been reported as an
uncommon condition in monkeys (Roberts and
Innes 1966 and Wood et al., 1978). There has not
been documented report of a natural occurrence
of this condition in monkeys in Nigeria,
previously. The bacteria isolated in this report are
similar to those isolated in other parts of the world
(Robinson and Maxie, 1985 and Levin and Carey,
1986). The origin of the vegetative endocarditis
and its effect in the lungs and liver of animals may
not be easily explained, but it has been known to
occur as a result of persistent or recurrent
bacteraemia (Robinson and Maxie, 1985 and
Cantrell et al., 1986).

Pneumonia with left sided congestive heart
failure as seen in this report had been reported in a
baboon in the United States of America (Levin
and Carey, 1986). It has also been reported that
congestive heart failure is an important
predisposing factor to bacterial pneumonia
(Hook, 1979), and it can also develop as a
complication of pneumonia (Roth and Gleckman, 1985). In this case, the two conditions
were seen in the animal hence the difficulty in
determining the initial problem in the dead
animal. The marked pulmonary involvement may
be the cause of the epistaxis seen and death, as
there was presence of blood stained froth in the
airway at postmortem.

The antibiotic sensitivities of the bacteria were
consistent with the observations of Levin and
Carey (1986). Although the source of the infection in this case is not known, pathogenic bacteria have been isolated from the soil and faeces in white tail deer (McCrum et al., 1967 and Botzler et al., 1986) which corroborated the fact that the soil or faeces could be the source of infection. A disease from which the condition should be differentiated is anthrax which has been reported in captive carnivores and elephants in Nigeria (IkeDe et al., 1976 and Okewole et al., 1993). This was excluded by the stained thin smear and bacterial culture. It should then be noted that adequate veterinary care should be provided to primates as they grow in zoos in order to promptly detect infections and adequately institute proper therapy.

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REFERENCES


