



EEHV LIKE HAEMORRHAGIC MYOCARDITIS IN A WILD ASIAN ELEPHANT (*Elephas maximus*)

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INTRODUCTION

Elephas maximus is classified as endangered according to the IUCN red list of threatened species. Western Ghats, a biodiversity hotspot enriched with a wide variety of flora and fauna on the peninsular India is one of the natural habitats of the species. Habitat shrinkage and defragmentation of elephant corridors are major threat for the survival of the species. In this context, newly emerging diseases like Endotheliotropic elephant herpes virus (EEHV) may cause even greater threat to the species survival. This case is dealing with an elephant died with lesions similar to EEHV lesions, which was tested negative in PCR analysis. This gains importance since there are similar cases reported from various parts of Kerala, India with similar lesions.

OBSERVATIONS

Death of an elephant was reported by forest officials near Athirapilly waterfalls, Kerala, India. On examination it was a female elephant of approximately 10 years old. The cadaver was observed close to the river, indicating that the animal was on its way to water. The animal was lying on the right lateral recumbence with the limbs extended. Rigor mortis has passed and post-mortem bloating has started. The animal had died 24 hours earlier. No external wounds were present except few skin aberrations on the trunk. There was oedema in the throat, temporal and mandibular regions and on the forelimbs (Fig.1). Abdomen on opening revealed a straw coloured fluid in excess. Examination of mouth revealed cyanosis of tongue. The stomach and small intestine was empty. Stomach was infested with



Fig. 1

100 to 200 maggots of *Cobboldia elephantis*, causing severe gastric myiasis. Congestion was noted in the mesenteric vessels. Intestinal mucosa revealed congestion in several regions. Hypertrophy of Payer's patches was noted on various regions of small intestinal wall. Large intestine contained small amount of partially digested feed material. Liver was oedematous with rounding of edges.



Fig. 2

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Lungs exhibited diffuse haemorrhage in the parenchyma. Heart on examination revealed ecchymotichaemorrhage on the myocardium. Endocardium revealed severe haemorrhage with severe involvement of cardiac valves (Fig.2). Generalised myocardial oedema with myocarditis was present. Samples were collected from heart, liver and lung on ice for DNA extraction and PCR analysis. The result of PCR analysis to detect Endotheliotropic Elephant Herpes Viral DNA performed at Wildlife Disease Research Laboratory, Wayanad was negative.

DISCUSSION

Haemorrhagic myocarditis with pericardial effusion is the typical symptom of EEHV. Other symptoms observed in EEHV infection are haemorrhage throughout the peritoneal cavity, hepatomegally, cyanosis of the tongue, intestinal haemorrhage and ulceration. Early clinical signs of EEHV infection are oedematous swelling of head, neck and thoracic limbs and cyanosis of tongue starting from the tip of tongue (Richman *et al.*, 2000). In the present case peritoneal haemorrhage was absent, but peritoneal cavity was filled with very large quantity of straw coloured fluid as reported by Garner *et al.* (2009).

Elephant stomach bot flies (*Cobboldia elephantis*) have been reported in Asian elephant population by many scientists (Easwaran *et al.*, 2002 and Sanyathiseree *et al.*, 2009). In this case there were too many larvae on the stomach wall causing gastric myiasis. Adult flies lay eggs near the mouth. The larvae hatch and develop in the mouth cavity and later move to stomach. All three stages of larvae may be observed on stomach wall. Matured third stage larvae exit from the mouth and drop to the ground to pupate (Fowler and Mikota, 2006).

African elephants act as reservoir for herpes virus and which causes disease in two elephant species. Death in EEHV is a result of cardiac failure resulting from herpes virus induced capillary injury with extensive myocardial haemorrhage and oedema. In most animal species the herpes virus are

epitheliotropic or a predilection for nervous tissue. In case of EEHV the virus multiplies in vascular endothelium and forms amphophilic to basophilic intranuclear inclusion bodies which may be considered as pathognomonic of the disease syndrome.

CONCLUSION

In the present case lesions described are similar to lesions of EEHV, but PCR analysis was negative for viral DNA. EEHV if spreads to the wild population of Asia, it will devastate the already fragile and critically endangered Asian elephant population. There need to be a better understanding of the epidemiology of EEHV in wild elephant population, so that a better control strategy may be devised, which is totally lacking today.

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