



# Clinical manifestations in cats with feline panleukopenia

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Feline panleukopenia is a rapidly progressive viral disease of domestic cats caused by a single stranded parvovirus of the genus *Protoparvovirus*. The virus, owing to its ubiquitous nature, remains persistent in the environment for extended periods leading to frequent exposures in naïve cat population. Clinically, the disease is manifested as haemorrhagic enteritis characterised by vomiting, diarrhoea, dysentery, dehydration, shock and death. Previous studies conducted by Parthiban *et al.* (2014) and Koulath *et al.* (2017) detected feline panleukopenia in domestic cats of India using polymerase chain reaction (PCR) targeting *VP2* gene of the virus. However, data related to the clinical manifestations of this disease in cats in India is limited. The current paper deals with the clinical findings of cats affected with feline panleukopenia in Kerala.

A total of 40 cats showing signs of dehydration, diarrhoea and vomiting presented to Teaching Veterinary Clinical Complex, Pookode and other Veterinary Hospitals of Wayanad, Kozhikode, Thrissur and Palakkad districts of Kerala during November 2017 to July 2019 formed the subjects of the study. Animals were subjected to detailed clinical examination. The clinical signs were photo documented and clinical parameters recorded. Faecal samples were collected aseptically from sick cats and diagnosis was confirmed by PCR targeting *VP2* gene of the feline panleukopenia virus (FPV) (Koulath *et al.*, 2017).

Out of the 40 faecal samples, 34 samples were obtained from Wayanad, three from Kozhikode, two from Thrissur and one from Palakkad. Upon PCR, 698bp segment of *VP2* gene of FPV was detected in 34 samples, out of which 29 (85.29 per cent) samples were collected from Wayanad, three (100 per cent) samples from Kozhikode and one (100 per cent) sample from Palakkad. The FPV DNA was detected in one (50 per cent) out of two samples from Thrissur.

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Out of the 34 cats diagnosed with feline panleukopenia, clinical examination of the affected cats revealed pyrexia (greater than 39°C) in 26.40 per cent of the cases while hypothermia (lesser than 37°C) was noted in 23.50 per cent cases. Similar findings have been reported by Ichijo *et al.* (1976) and Porporato *et al.* (2018). In contrast, Kruse *et al.* (2010); Lister and Benjanirut (2014) and Mayur *et al.* (2016) reported fever in response to FPV infection in cats. This contradiction could be because pyrexia occurs only in the initial stages of the disease and subsides with advancement of the disease. Shallow respiration was noted in 26.40 per cent of cats while weak pulse rate (less than 140 per minute) was noted in 79.41 per cent cases. Pallor of mucous membranes was observed in 79.41 per cent of the cases (Fig 1). These findings are in accordance with Mayur *et al.* (2016) and Klainbart *et al.* (2017). Septicaemia associated with feline panleukopenia could be manifested as peripheral vasodilatation leading to hyperaemic mucosa followed by vasoconstriction resulting in pallor of mucous membrane with delayed capillary refill time and degraded pulse quality.

Vomiting was reported in 85 per cent of the cases (Fig 2). Similar findings have been reported by Lister and Benjanirut (2014), Awad *et al.* (2018), Porporato *et al.* (2018) and Barrs (2019). Gastritis induced stimulation of the emetic centre manifested as vomiting lead to bile tinged vomitus in affected cats. Watery, mucoid faeces were observed in all the 34 FPV positive cases among which dysentery was noted in 70 per cent of the infected cats (Fig 3). These findings are in accordance with Bayati (2016), Awad *et al.* (2018) and Porporato *et al.* (2018). Viral replication in the deeply situated crypt cells in the intestinal mucosa leads to stunting and malformation of the intestinal villi. Damaged intestinal villi result in diarrhoea due to malabsorption and increased cell permeability (Truyen *et al.*, 2009; Greene, 2012 and Stuetzer and Hartmann, 2014). Dehydration was noted in all the cats upon presentation that was assessed by skin turgor test which revealed 64.71 per cent, 20.59 per cent and 14.70 per cent cats to have ten to twelve per cent, six to eight per cent and four to eight per cent dehydration respectively as determined

by skin turgor examination. Similar findings were reported by Kruse *et al.* (2010), Lister and Benjanirut (2014) and Awad *et al.* (2018). Dehydration may be attributed to the damaged



Fig 1: Pallor of mucous membrane in a cat affected with feline panleukopenia



Fig 2: Bile tinged vomitus in a cat affected with feline panleukopenia



Fig 3: Diarrhoea in a cat affected with feline panleukopenia

intestinal villi cells leading to increased permeability and associated fluid loss (Greene, 2012 and Stuetzer and Hartmann, 2014).

Clinical signs indicative of respiratory distress were noted in 23.50 per cent cases with oculo-nasal discharge, shallow respiration and lateral recumbency. Pleural effusion was noted in 5.89 per cent out of 34 cats with respiratory signs. Similar findings have been reported by Klainbart *et al.* (2017) and Porporato *et al.* (2018). They suggested that tachypnoea, pleural effusion, pneumonia and other respiratory signs may occur in cats affected with feline panleukopenia which could be assumed because of secondary bacterial infections leading to pulmonary lesions.

Out of 34 cats infected with FPV, 5.89 per cent of the cats manifested nervous signs characterised by seizures, ataxia, hypersalivation and lateral recumbency. Similar findings were reported by Schatzberg *et al.* (2003) and Poncelet *et al.* (2013). Injury to the neuroblast cells due to viral replication may lead to development of cerebellar hypoplasia manifested as ataxia.

### Summary

Out of the 34 cats diagnosed with feline panleukopenia, mortality occurred in 19 (55.88 per cent) cats while 15 (44.12) cats responded to therapy. Mortality rate was higher in cats that showed clinical signs of hypothermia, vomiting, dysentery and dehydration. Vomiting which became apparent after resolution of pyrexia was the preliminary sign in cases which terminated with the development of severe dehydration and hypothermia leading to shock, lateral recumbency and death.

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