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Clinico-histo-ultra pathobiology of parvoviral enteritis of felines in and around Indore, Madhya Pradesh

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Abstract
In the present study, 50 cats were examined irrespective of age, sex and breed showing symptoms of haemorrhagic gastroenteritis. Out of these 50 cats, mortality of 5 cats was recorded and subjected to detailed post-mortem examination and samples of intestine, liver, spleen and heart were collected from 5 cats and were screened for gross, histopathological and ultrastructural studies. The confirmation of incidence was done by using sandwich lateral flow immune chromatography assay as per the protocol provided along with PARVO kit. The overall incidence of CPV infection in cats was reported as 10%. Persian female cats were more affected from first month onwards, showing decreased haemato-biochemical values and higher SGPT values indicative of liver damage, along with leukopenia. The values of total protein and potassium levels were less than normal in both Persian and non-descript cat breeds. On histopathology, intestines revealed haemorrhages in the mucosal villi and intervillous crypts, congestion in sub mucosa and serous surfaces, liver had fatty changes with mononuclear periportal infiltration, hyperplasia of splenic trabeculae with infiltration and degeneration, and necrosis of myocytes with haemorrhages, and infiltration was noticed in heart. Transmission electron microscopy (TEM) revealed presence of VLPs outside in crypts and inside intestinal epithelial cells, hepatocytes, splenic pulp. Other changes noticed were apoptosis of enterocyte, severe margination of nuclear chromatin, fat bodies disrupted RER and infiltration by lymphocytes. TEM also revealed changes in mitochondria and cytoplasmic organelles with myeline figures and vesicles formation.

Keywords: Cats, TEM, feline

Introduction
Feline panleukopenia, also known as feline distemper is a highly contagious viral disease of cats characterized by acute depression, anorexia, gastroenteric diarrhoea and vomiting, and leukopenia with high rate of mortality among nonimmune kittens (Clemens and Carlson, 1989) [12]. The highest rate of morbidity and mortality was recorded up to 12 months of age. In per-acute infections, mortality may reach up to 100 percent whereas it is 25 to 90 percent in acute cases. In cats, the disease is caused by CPV, generally appear to be much milder as compared to dogs infected with the virus, or those caused by FPV. Canine parvovirus (CPV) belongs to the genus Parvovirus and has a close antigenic and genomic relationship with Feline panleukopenia virus (FPV), Mink enteritis virus (MEV) and Raccoon parvovirus (RPV). Canine parvovirus (CPV) is a small (diameter of 25 nm), non-enveloped virus. The virion consists of a spherical capsid, which is composed of three proteins namely VP1, VP2 and VP3 and contains a linear, single-stranded DNA molecule. The main source of infection is the faecal material of infected cats as a large number of virus particles are excreted in faeces.

Materials and Methods
Sample size
For the present study, a total 50 cats of either sex were screened from January 2021 to December 2022, brought with the suspected clinical symptoms (vomiting and bloody diarrhoea) of parvovirus infection to VCC Veterinary College, Mhow and from different private clinics of Mhow and Indore.

Sample collection
Collection of blood sample
About 2 ml blood samples of cats were collected aseptically from the saphenous or cephalic

vein and was equally distributed into two vials, one with anticoagulant and the other without anticoagulant for analysing haematological and biochemical parameters, respectively.

Collection of faecal swabs
The faecal samples were collected by inserting the sterile swab deep in the rectum of suspected cats.

Sandwich lateral flow immune-chromatography assay/CPV antigen rapid testing
After sample extraction, the swab was immersed in the diluents buffer provided in the kit and mixed well. The sample was left for a short time (20 seconds) and finally 4 drops of supernatant was added to the sample hole of the cassette provided in the kit. The results were read after 5 to 10 minutes. PV antigens in felines faeces bind to the PV specific antibody-gold particle conjugate and move on the kit membrane by capillary forces and show a red test line. Result was positive when control and test line were red and negative when only control line were red. If there was no control line re-testing was performed.

Collection of samples for histopathology
Felines positive by rapid antigen test kit for parvo viral infection and later succumbing to the disease were brought to the department for postmortem. After recording gross lesions, the tissue samples from intestine, liver, spleen and heart were collected in 10% buffered formalin for histopathological and in 2.5% gluteraldehyde for EM studies.

Detection of parvoviral antigen in the faecal samples
Parvo viral antigen in the faeces was detected by sandwich lateral flow immune chromatography assay as per the protocol provided along with the PARVO kit. As per manufacturer (Bynode, Korea), the kit shows specificity of 98.8% and sensitivity of 100%.

Haematological parameters
The haematological parameters carried out as per the procedure described by Jain (1986) were- Total Erythrocyte Count (TEC) (million/µl), Total Leucocyte Count (TLC) (thousand/cumm), Haemoglobin (Hb) Concentration (g/dl), Packed Cell Volume (PCV) (%), Differential Leukocyte Count (DLC) (%), Platelets count (thousand/cumm). The blood smear prepared were air dried and stained with Wright's stain and DLC was performed by counting one hundred leucocytes under oil immersion.

Biochemical parameters
The biochemical parameters carried out with the Semiautomatic biochemical analyzer Erba Mannheim make Transasia were - Total protein (g/dl), Alkaline phosphatase (IU/L), AST(IU/L), ALT(IU/L), Electrolytes- Na, K(mmol/L). The kits used for all the parameters were ERBA diagnostic manufactured by TRANSASIA BIO-MEDICALS LTD. Malpur Baddi, Dist. Solan (H.P.).

Histopathology
After recording the gross lesions, tissue sections from different parts of the intestine, liver, spleen and heart were collected from the carcass and subsequently preserved in 10% neutral buffered formalin for at least 24-48 hours. Further, these tissue samples were processed by the routine method of dehydration in graded alcohol, clearing in benzene and embedding in paraffin, as per the method described by Lillie (1976) [20]. Sections of 4-5 micrometre thickness was processed by conventional procedures using routine Haematoxylin and Eosin for histopathological studies.

Transmission electron microscopic (TEM) study
Samples were transferred to vials and fixed in 2.5% glutaraldehyde solution in 0.05M phosphate buffer at PH 7.2. Tissue pieces 1-2 mm in size were immersed in this solution for 24 hours at 4-10 °C, for post-fixation, 1% aqueous osmium tetroxide for 2 hours at 4-10 °C was used. After post-fixation, the tissue was dehydrated by immersing it sequentially, in a series of graded ethyl alcohol or acetone such as 30, 50, 70, 90 percent and absolute. The dehydration fluid was replaced by clearing fluid such as propylene oxide 22 (two changes of 15 min each) and tissues were embedded in hard Spurr's resin block.
Both semi-thin and ultra-thin sections were cut with a glass knife on the ultramicrotome. Semi-thin sections of 200-300 nm thickness were stained with toluidine blue and ultra-thin sections of 50-70 nm thickness were mounted on grids. Then the sections were stained with saturated aqueous Uranyl acetate and counter-stained with 4% lead citrate and introduced into the specimen chamber of the Transmission Electron Microscope column for observation (Bozzola and Russell, 1998) [8]. Samples were studied at AIIMS New Delhi by Hitachi 7500.

Statistical analysis of data
The data of all observations were subjected to statistical analysis of variance (Single way ANOVA) programmed CRD (Completely Randomized Design) as stated by Snedecor and Cochran (1994) [26].

Results and Discussion
The present investigation aimed to study the incidence of parvoviral enteritis and pathomorphological alteration resulting due to virus in the gastrointestinal tract of cats, with possible correlation with haematological and biochemical parameters. In the present study, 50 cats were examined for a period of one year (January to December 2021) irrespective of age, sex and breed with tentative apparent symptoms of hemorrhagic gastro-enteritis. The confirmation of incidence was done by using sandwich lateral flow immune chromatography assay as per the protocol provided along with the PARVO kit. The rapid antigen test kit showed a high degree of accuracy, sensitivity and specificity to detect the parvo virus subtypes in canine and feline faeces. Similar type of testing were conducted by Bayati *et al.* (2018) [6].
The overall incidence of parvovirus infection was recorded at 10% (5/50) in cats. The present observation of high incidence was in correlation with the work reported by other workers like Archana *et al*. (2010) [2], Yang *et al*. (2010) [28], Deka *et al*. (2013) [13], Raheena *et al.* (2015) [24], and Tanwar *et al.* (2020) [27].

Age-wise incidence of feline parvovirus infection
Cats between 0-3 months of age showed the highest incidence (20%) followed by cats above 12 months of age (16.67%) and 6-12 months of age (5%), whereas, the lowest incidence of FPV was reported at 3-6 months of age. The present observation of high incidence was in correlation with the work of workers like Chisty *et al*. (2020) [11] and in contrast

with observations of Awad *et al*. (2018) how reported lower incidence in kittens of 0 to 3 months of age.

Sex-wise incidence of feline parvovirus infection

Out of 50 cats having gastroenteritis, the sex-wise incidence revealed a higher prevalence in females (14.28%) as compared to males (8.33%). The present observation of high incidence in female cats was in correlation with the work reported by other workers like Awad *et al*. (2018) [3].

Breed-wise incidence of feline parvovirus infection

During the present study, cats of various breeds were examined for FIP infection including Persian and non-descript cats. The higher incidence was noticed in non- descript cats 12.50% cats, i.e., as compared to Persian cats 9.52%. The present observation of high incidence was in correlation with the work reported by other workers like Chisty *et al*. (2020) [11].

Haematological parameters of felines

A total of fifty faecal samples were collected from the suspected cats or kittens of different breed, age and sex out of which five samples were positive for FPV. The mean Hb concentration (g/dl), mean TEC (miilon/µl) and PCV (%) decrease in FPV affected cats and these observation correlated with the findings of Bayati *et al.* (2016) [6], Zenad and Radhey (2020) [30] and Kadam (2022) [16]. Anaemia in the affected cats might be due to loss of blood during intestinal and gastric haemorrhages and virus infect and replicates in early progenitor cells of bone marrow, and causes destruction of all myeloid precursor.

In the present study, TLC was significantly decreased in FPV affected cats , mild significant changes in mean platelet count was observed in FPV positive cats. The range in different breeds, sex and age group of FPV affected cats and also increased towards higher range values was noticed in lymphocyte, eosinophils, monocytes and platelets indicative of mild eosinophilia and thrombocytosis despite leucopaenia though haemorrhagic enteritis is there. The target cell of FPV are lymphocytes thus infect and causes lymphocytosis due to destruction of myeloid cells leading to lymphopenia, neutropenia and thrombocytopenia observed in FPV infected cats. The study was correlated with Zenad and Radhey (2020) [30] and Kadam *et al.* (2022) [16].

Biochemical parameters of felines

The present finding revealed that the Total protein levels were significantly lower in age, sex and breed wise in FPV infected cats. Such findings were similarly recorded by Baruah *et al.* (2007) [4], Kaur *et al.* (2005) [18] and Khare *et al.* (2020) [19]. This decrease in level of TP in CPV affected cats might be due to the leakage of serum protein through damaged capillaries of the villi of intestine and also due to less absorption through the damaged villi.

In present finding the level of ALP, AST in some cat breeds of diffrent age and sex showed higher to moderate elevation and these observations correlated with Kaur *et al.* (2005) [18], Baruah *et al.* (2007) [4] Bastan *et al.* (2013) [5] and Khare *et al.* (2020) [19]. Elevation may occur due to hepatic hypoxia secondary to severe hypovolemia or the absorption of toxic substances due to loss of the gut barrier. ALT level was lower in cats than the normal values mainly in breed wise the exotic cats with lower values might be due to sever hepatic toxicity.

A significant decrease in Sodium and Potassium values was observed in FPV infection. This finding is in accordance to the findings of Joshi *et al.* (2012) [15] and Katariya *et al.* (2020) [17]. Decreased levels of Sodium and Potassium might be due to loss of ions in vomiting and diarrhoea.

Gross, histopathology and ultrastructural lesions

In the present study, 50 cats examined irrespective of age, sex and breed and showing tentative symptoms of hemorrhagic gastroenteritis were screened grossly and later 3 dead cats were examined for any alterations in the tissues, these gross changes were recorded for further histopathological studies.

Intestine

Grossly, parvo-affected Persian cat showed severe catarrhal enteritis of small intestine and ballooning of large intestine, congestion of mesenteric blood vessels and focal areas of haemorrhages. The common histopathological lesion obtained from different species of cats revealed mostly increased intervillus gap with focal clumping of several villi at other places, severe infiltration in the mucosa of inflammatory cells. In addition, multiple haemorrhages in the submucosa of intestine of parvo affected Persian cat whereas intestine of parvo-infected nondescript cat showed hollow villi with complete loss of epithelium, presence of cellular debris and fibrin in the intestinal lumen to large lymphoid follicles in the submucosa. Ultrastructural examination of Parvo-affected Non-descript cat showed marked nuclear and cytoplasmic disorganization, disruption and degeneration, few slide showed clear nuclear and cytoplasmic degeneration , necrosis of adjacent cells with complete loss of different cytoplasmic organelles or cytoplasmolysis.

Liver

Grossly, parvo-affected non-descript cat showed mottled liver parenchyma with petechial haemorrhages pale areas and focal areas of ecchymotic haemorrhages, engorged gall bladder was also visible. In addition, Persian cat showed hepatomegaly with severe congestion and focal areas of haemorrhage. Microscopically, parvo-affected Persian cat showed extensive fatty degeneration around the central vein with an increase in the number of Kupffer's cells. In few cases, congestion of the central vein, centrilobular necrosis and infiltration, also periportal infiltration of inflammatory cells were observed. In non-descript cat severe congestion of central vein, haemorrhages and deposition of hemosiderin pigment and coagulative necrosis of hepatocytes. Electron dense particles, myelin figures and oval vesicles were commonly observed in ultra sections.

Spleen

On gross examination, Parvo-affected Persian cat showed congestion and mild splenomegaly and non-descript cat showed splenomegaly with loss of wrinkles and congestion. On Histopathological examination, Persian cat showed depletion of white pulp, congestion of splenic blood vessels, focal areas of haemorrhages and thickening of trabecular septum. Subcapsular edema, haemorrhages, depletion of lymphocytes, infiltration of inflammatory cells in the deeper parenchyma and fibrin deposition was also seen. In addition, non-descript cat showed venular microthrombi. The ultrastructural changes observed in Parvo affected Persian cat were mainly pyknosis and karyorrhexis.

Heart

Grossly, parvo-affected Persian cat showed hydropericardium & congestion of the myocardium and non-descript cat showed congested myocardium with focal haemorrhages. Histopathologically, parvo affected non-descript cat showed congestion haemorrhages, infiltration of lymphocytic cells with degeneration & necrosis of surrounding muscles fibres. Ultrastructurally, parvo affected Persian cat showed several mitochondrial degeneration and loss of cross striations in the cardiac myocytes, nuclear changes included chromatin condensation, loss of its membrane integrity and blebbing, and heart of parvo infected non-descript cat showed cardiac myocyte mitochondria with varying degrees of degeneration, including low to high amplitude swelling, loss of cristae or cristaelysis and even complete disruption of mitochondrial morphology. There was also an accumulation of electron dense bodies, which was likely calcium sequestration in mitochondria.

The present observation was in agreement with the research reported by workers like Meunier *et al.* (1985) [21], Parrish *et al.* (1995) [24], Bayati *et al.* (2017) [7], Oliveira *et al.* (2018) [23] and Ain-Fatin *et al.* (2020) [1] in canines and by other workers like Parrish *et al.* (1995) [24] and Bayati *et al.* (2018) [8] in felines. The organs understudy in the present research such as intestine, heart, liver and spleen showed different pathological lesion during viremia due to multiplication of virus. The high level of alkaline phosphatase were indicative of extensive of epithelial crypts and resulting haemorrhages and hardening of intestinal loop Greene (2012) [14]. Cardiomyopathy in kittens and pups was due to presence of rapidly dividing myocytes. Hyperplastic changes of spleen may occur due to severe viral infection with lymphoid depletion and leukopenia in addition to heavily concurrent secondary bacterial infection in later stage Boosinger *et al.* (1982) [9]. Presence of micro thrombi in feline spleen may be a result of FPV pathogenesis. The generalized systemic infection caused hepatic fatty changes, necrosis, haemorrhages and inflammatory response.

TEM revealed presence of VLPs outside in the crypts and inside intestinal epithelial cells, hepatocytes, splenic pulp and large group of virions in degenerated cardiac myocytes in a single pup (Norman, 2009) [22].

These results indicate that the pathogenesis and pathology produce by canine and feline parvo viruses is almost the same at cellular and tissue levels. However, there were minor differences depending upon the age, sex and breed of the animal. The standard viraemia pattern induces loss of cryptal epithelium and consequent regeneration with hypertrophy and hyperplasia, mesenchymal epithelial cell destruction in various organs like intestine, heart, liver, lungs, spleen, kidneys and bone marrow. Hypocellularity of T lymphocytes (inner cortex), B lymphocytes (follicles) and Peyer's patches is seen. Gut associated lymphoid tissue (GALT) and mast cells of the lamina propria are both crucial in pathogenesis. Functional pathology of diarrhoea, dehydration, hypovolemia, hypoglycaemia and acidosis due to energy generation by anaerobic glycolysis occur due to irreversible hypoxic injury and damage to cell membranes by consequent free radical injury (Zachary 2012) [29].

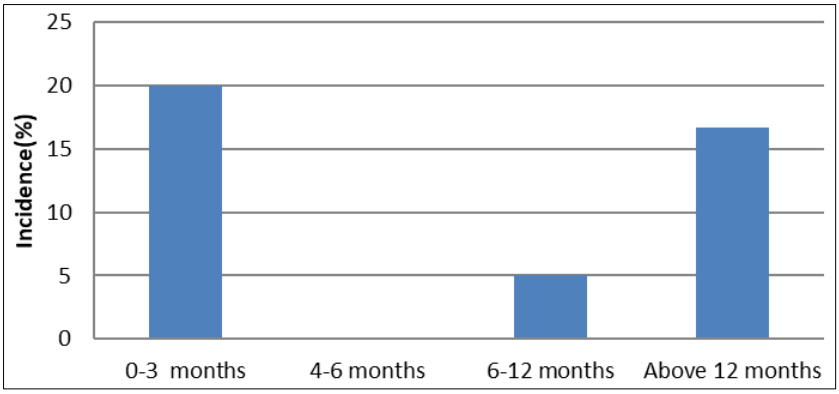

Fig 1: Age wise incidence of Feline parvovirus infection

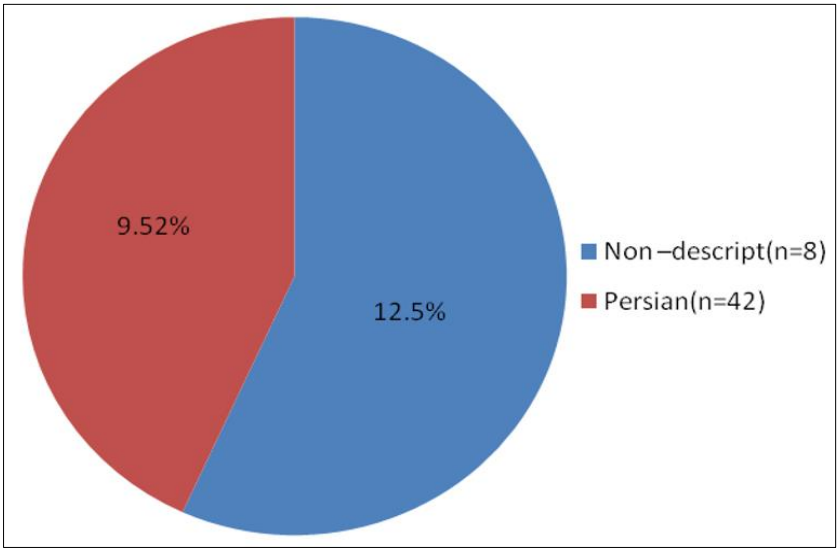

Fig 2: Breed wise incidence of feline parvovirus infection

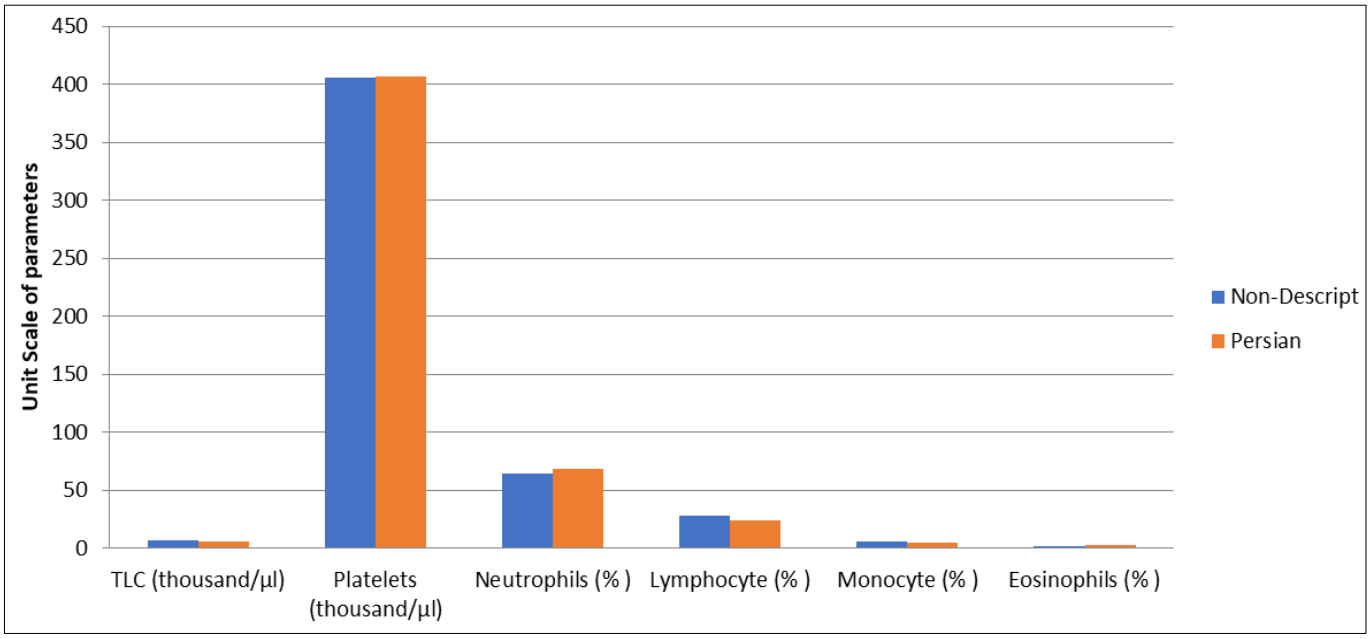

Fig 3: Breed wise mean haematological parameters of feline parvovirus infection

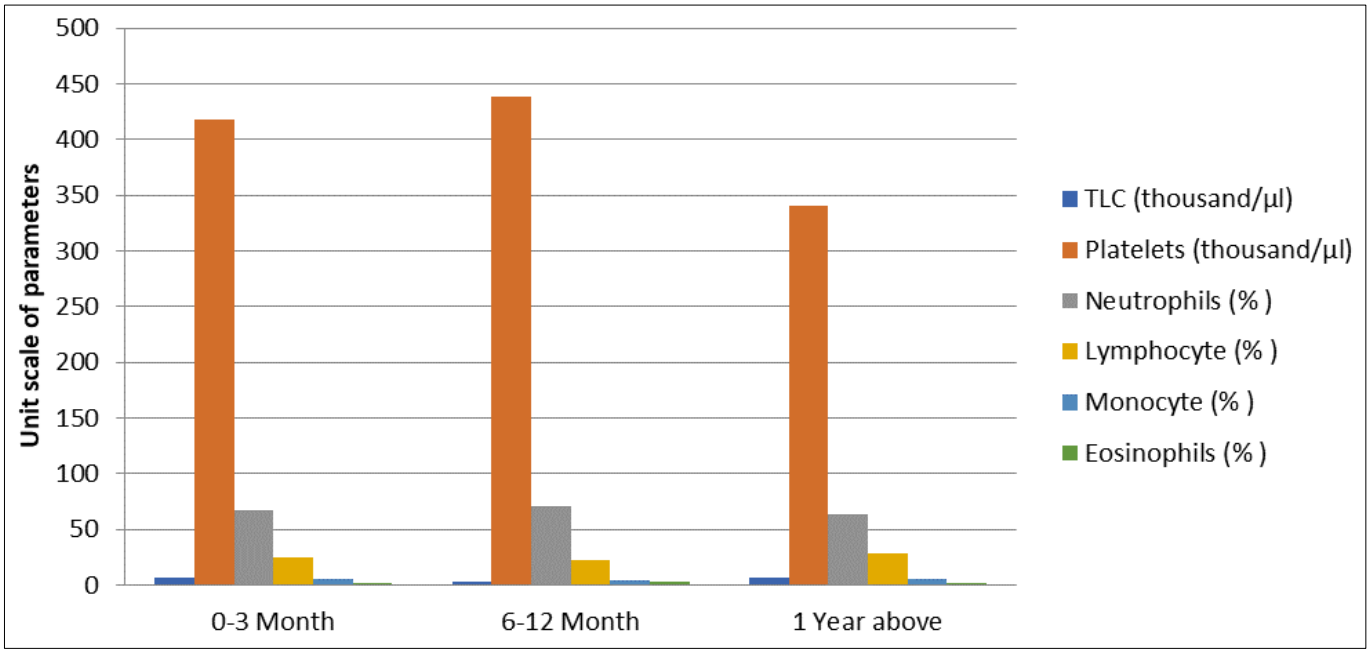

Fig 4: Age wise mean haematological parameters of feline parvovirus infection

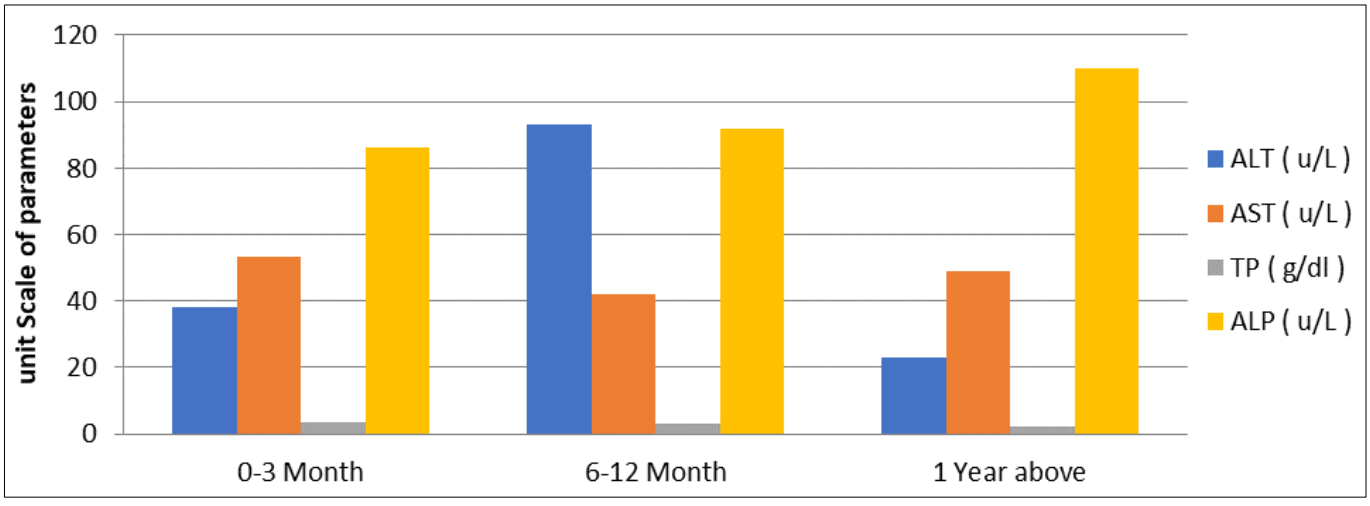

Fig 5: Age wise mean biochemical parameters of feline parvovirus infection

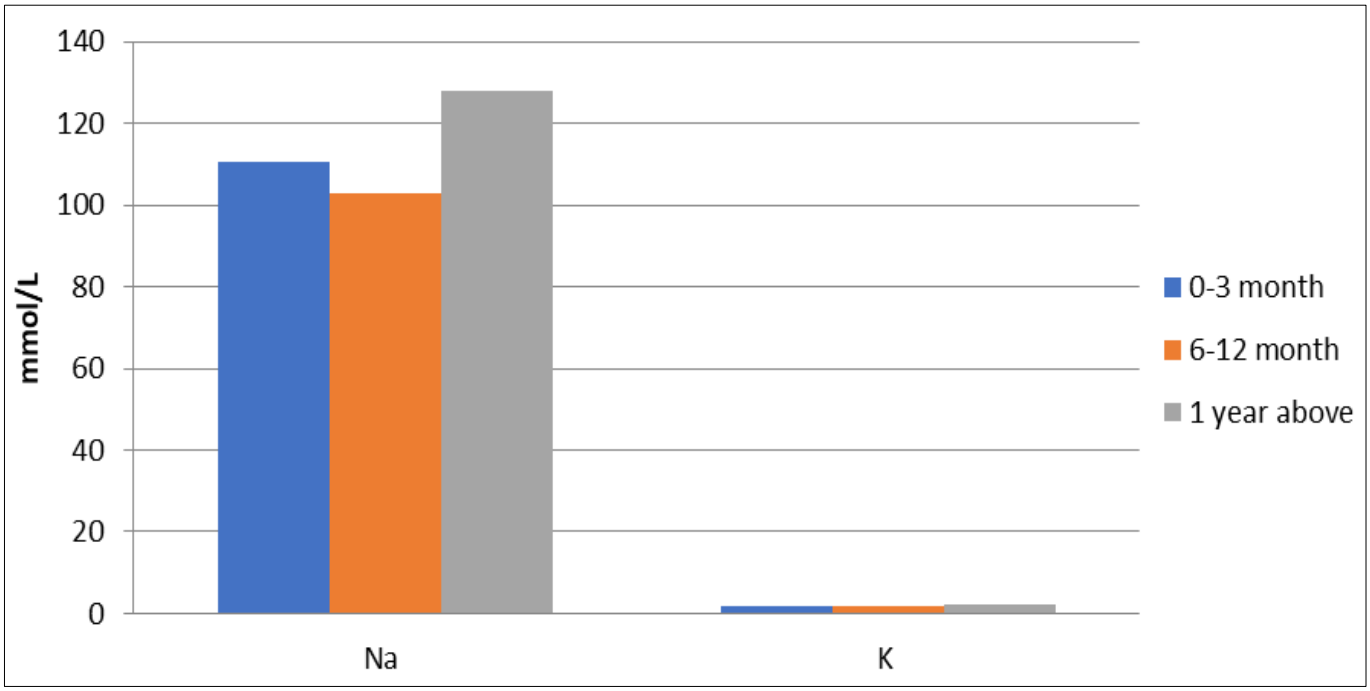

Fig 6: Age wise mean biochemical parameters of feline parvovirus infection

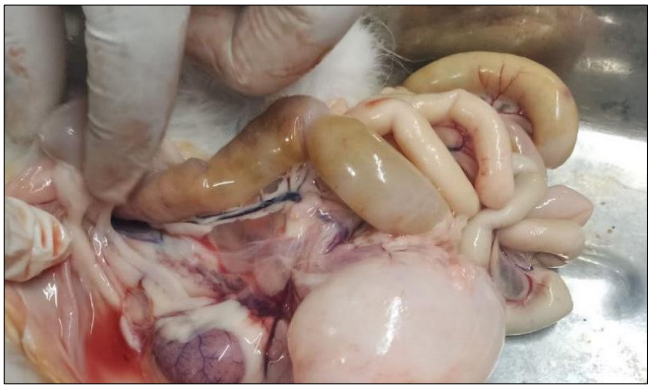

Fig 7: Gross photograph of intestine of parvo affected Persian cat showing sever catarrhal enteritis of small intestine and ballooning of large Intestine, congestion of mesenteric blood vessels and focal areas of haemorrhage

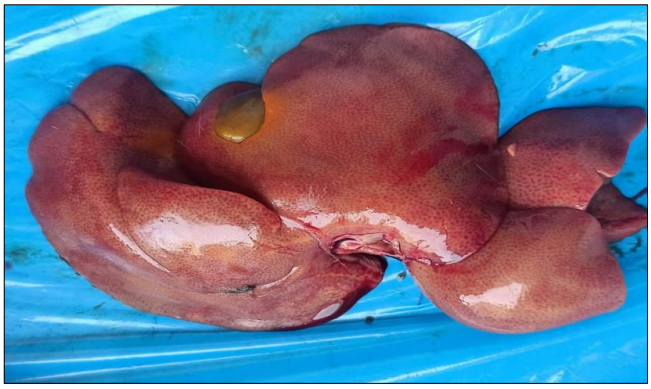

Fig 8: Gross photograph of liver of parvo affected non descript cat showing mottled liver parenchyma with petechial haemorrhages, pale fatty areas and focal areas of ecchymotic haemorrhages

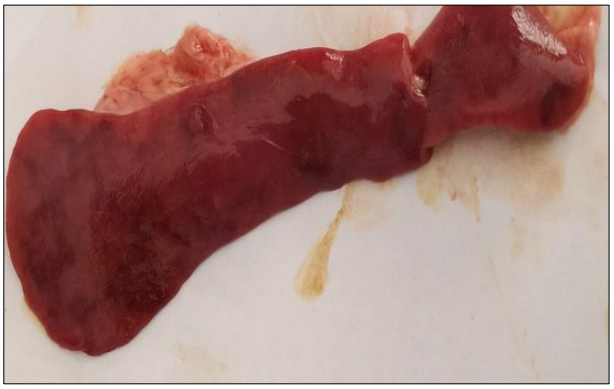

Fig 9: Gross photograph of spleen of parvo affected Persian cat showing congestion and mild splenomegaly

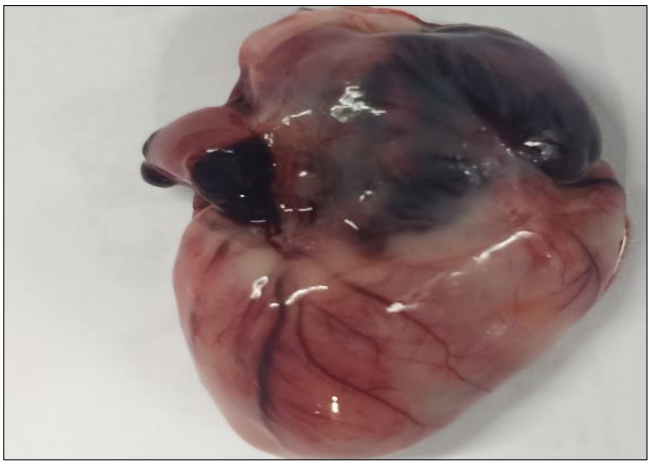

Fig 10: Gross photograph of heart of parvo affected Persian cat showing hydropericardium and congestion of myocardium

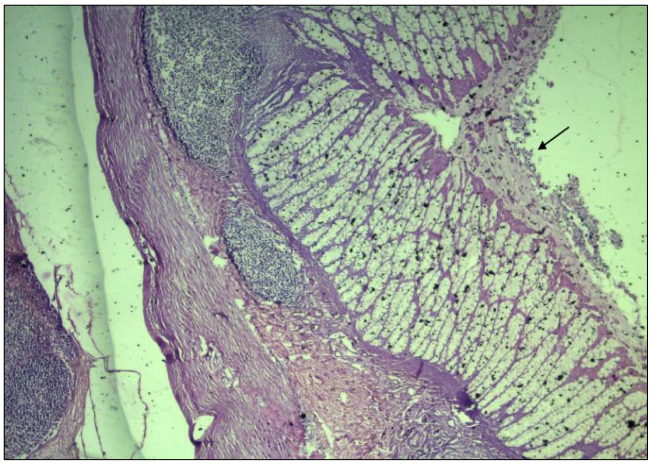

Fig 11: Photomicrograph of intestine of parvo infected cat (non-descript) showing villi with complete loss of epithelium , presence of cellular debris and fibrin in the intestinal lumen (arrow) and few large lymphoid follicles in submucosa (H & E, 4X)

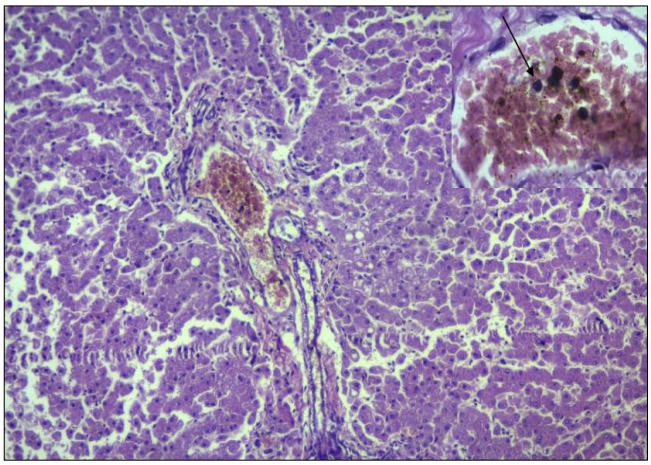

Fig 12: Photomicrograph of liver of parvo infected cat (non-descript) severe congestion of central vein haemorrhage and deposition of hemosiderin (arrow)pigment , also coagulative necrosis of hepatocytes (H & E, 20X)

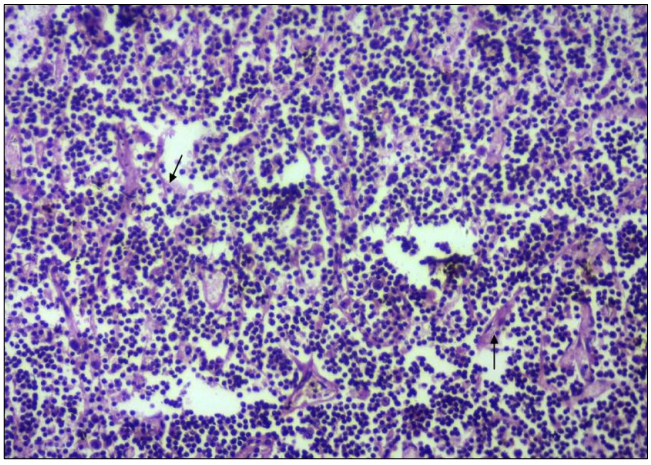

Fig 13: Photomicrograph of spleen of parvo infected cat (Persian) showing fibrin deposition ,thinly populated white pulp with deposition of fibrin in splenic parenchyma (arrows)(H & E, 20X)

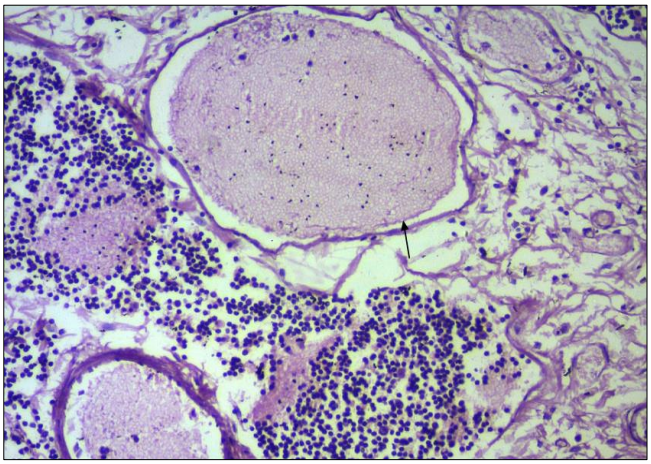

Fig 14: Photomicrograph of spleen of parvo infected cat (non descript) showing venular microthrombi (arrow) (H & E, 40X)

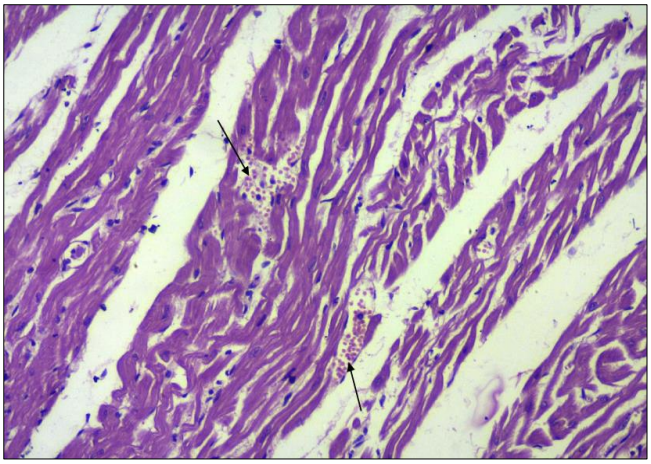

Fig 15: Photomicrograph of heart of parvo infected cat (non descript) showing in many muscle fiber congestion hemorrhages , infiltration of lymphocytic cells with degeneration & necrosis of surrounding muscles fiber (H & E, 10X)

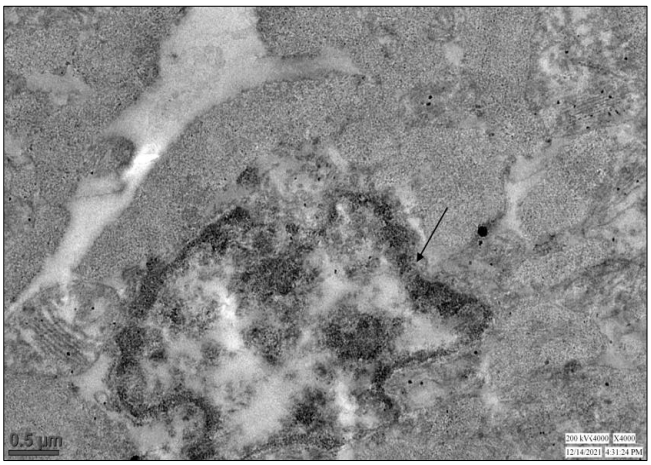

Fig 16: Ultrastructural photograph of heart of parvo infected cat (non descript) showing severe mitochondrial degeneration and loss of cross striations in the cardiac myocytes, nuclear changes include chromatin condensation, loss of its membrane integrity and blebbing (arrow)

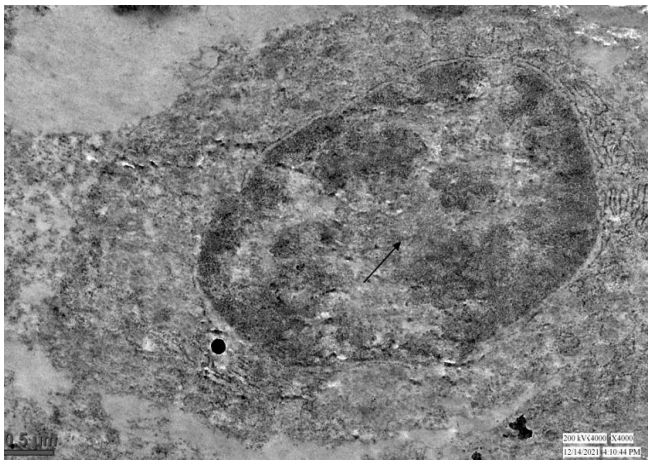

Fig 17: Ultrastructural photograph of intestine of parvo infected cat (non descript) showing marked nuclear and cytoplasmic disorganization (arrow), disruption and degeneration of enterocyte (X4000)

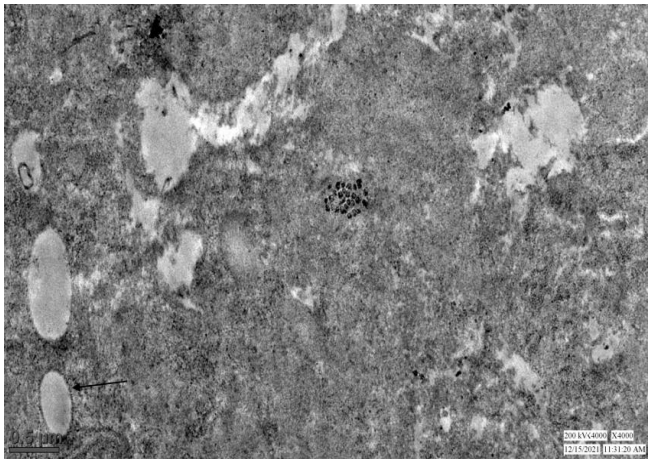

Fig 18: Ultrastructural photograph of liver of parvo infected Cat (Non descript) showing oval vesicals (arrow) (X4000)

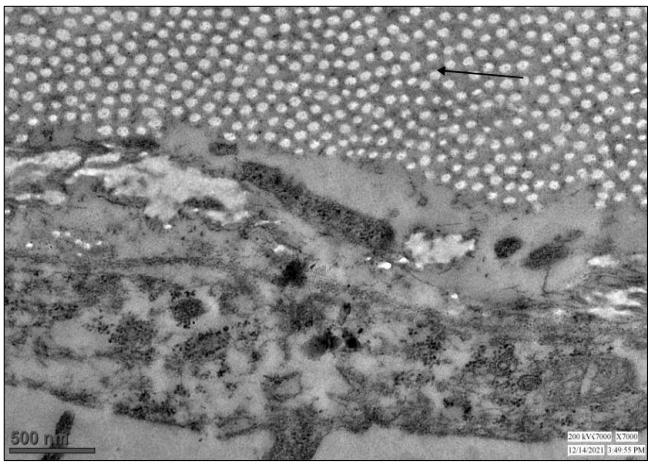

Fig 19: Ultrastructural photograph of heart of parvo infection showing large group of virions resembling parvo virus (arrow) (X7000)

Conclusion

Prevalence of parvo infection in cats of Indore district was determined using Parvo rapid test kit (Idex). Incidence was10% in cats depending 50 cases studied. Haemato-biochemical parameters revealed anaemia and leucopenia irrespective of breeds. However kittens between 0-3 months age affected more. Females were affected more than males. Potassium and sodium ionic concentration decrease in blood due to severe necrosis and fluid loss by infected injured epithelia. Gross lesions in intestine, liver, heart and spleen. Microscopically, intestine revealed haemorrhagic enteritis, fatty changes in liver, depletion of lymphocytes in splenic follicles and myocarditis with necrosis of myocardial fibres. Fibrinous changes were more with damage to blood vessels. By histopathology and EM studies, overall parvo viral pathogenesis in felines was more studied. However, due to fibrino protective effect, fatality was less, though mitochondrial and cell membrane damage was more indicative of free radical injury in early stages of disease.

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