

Study the histopathological changes accompanied with canine parvovirus infection

Hussein Ali Mohammad Al-Bayati Shony Meakhael Odisho Huda Abed Al-hammed
Coll. of Vet. Med. / Univ. of Baghdad
email: husseindoctor25@yahoo.com

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Abstract

This study considered to be the first in Iraq intended to identify the gross and microscopic pathological alterations in the susceptible organs of the naturally infected pups were comer to Private Vet. Clinics in Baghdad. These infected cases were checked previously by rapid test to detect canine parvovirus antigen in canine feces. After death of animal, the susceptible organs (intestine and heart) were taken and examined grossly, then prepared for histopathological examinations. Results were revealed presence of severe congestion and bleeding of small intestine, further to other organs. Also the microscopic examinations declare presence of extensive necrosis and loss of epithelial cells of the villi, with infiltration of mononuclear cells in lamina propria, in addition to changes in other organs that took to be pathognomic for infection.

Key words: Canine parvovirus, histopathology, puppies

دراسة التغييرات النسيجية المرضية المصاحبة للإصابة بفايروس بارفو الكلاب

حسين علي محمد البياتي شوني ميخائيل اوديشو هدى عبد الحميد الشعلان
كلية الطب البيطري / جامعة بغداد

الخلاصة

تعتبر هذه الدراسة الأولى من نوعها والتي تهدف تمييز الآفات العيانية والمجهريّة في الأعضاء الحساسة للإصابة الطبيعية بفايروس البارفو Parvovirus في الجراء الوافدة إلى العيادات البيطرية الخاصة في مدينة بغداد . فحصت هذه الحالات مسبقا بواسطة الاختبار السريع بوجود مستضدات الفيروس في براز الجراء المصابة ، حيث أخذت الأعضاء الحساسة بعد نفوق الحيوانات وفحصت عيانيا ومن ثم قطعت نسيجا لتفحص أخيرا التأثيرات المرضية. أظهرت النتائج وجود نزف شديد واحتقان في الأمعاء الدقيقة وكذلك بالنسبة إلى بقية الأعضاء ، واطهر الفحص المجهري وجود موات شديد وفقدان للظهارة المبطنّة للأمعاء مع ارتشاح الخلايا وحيدة النواة في طبقة تحت الظهارة وكذلك تأثيرات أخرى لبقية الأعضاء والتي تعتبر تشخيصية للمرض.

الكلمات المفتاحية : فيروس بارفو الكلاب ، الأمراض النسيجية ، الجراء.

Introduction

Canine parvovirus2 (CPV2) was the most important viral cause of enteritis in puppies at the age of two months and thereafter (1). The virus infects rapidly dividing cells in the intestinal epithelium leading to crypt necrosis and crypt dilation then villous atrophy that is diagnostic for CPV2 infection (2). CPV2 was emerged in 1978 worldwide and termed as CPV type 2 to distinguish it from CPV type 1 (minute virus of canine MVC) (3). The disease is a highly contagious viral disease of

canine characterized by vomiting, diarrhea typically appeared yellow to gray contain blood or mucous and fever (40-41) °C (4 and 5), and may lead to rapid dehydration (6). The disease is more dangerous in young puppies from weaning to six months of age (7). More than 80% of adult dogs show no symptoms (8). Mortality rate of those infected 16-48%, but reach in untreated cases 91% (9). Myocarditis may develop due to inutero exposure or under eight weeks of age

(8 and 10). Asymptomatic urinary tract infection developed in 25% of puppies following parvovirus enteritis (9).

Materials and methods

The autopsy samples were removed from animals passed away due to the virus infection and study of histopathological changes caused by the disease, including bowel, heart, spleen, tongue, kidney and

lung, ten (10) piece of each. These cases were checked previously had positive infection by rapid test and ELISA (11). Histopathological study was done by taken (1 cm³) from specimens include intestine, heart, spleen, lung which fixed at 10% buffered formalin, then embedded in paraffin, cut at 5µm, then stained with Hematoxylin and Eosin and examined by light microscope according to (12).

Results

The gross lesions of naturally infected pups characterized by severe congestion and hemorrhage in most parts of the gastrointestinal tract. Also exhibited a different size of pale areas in the intestinal serosa. At the heart, there are pale areas of myocardium with thin walls and congestion blood vessels (Fig. 1 A, B, C).

The histopathological sections of the tongue of naturally infected pups, characterized by vacuolar degeneration of epithelial cell of the mucosa with moderate mononuclear cell infiltration in sub epithelial layer (fig. 2 A). The sections of susceptible infected organs from dead pups show that the Kidney with atrophy of the glomerular tuft with dilatation of Bowman's capsule as well as acute cellular degeneration of the epithelial cell lining of renal tubule (fig. 2 B). The histopathological section of the lung, which showed edema in the lumen of alveoli with mononuclear cells infiltration (fig. 2 C).The

prominent pathologic changes of heart were degeneration of the muscular fibers (fig. 2 D), also there is interstitial edema, scattered mononuclear inflammatory cells infiltration between myofibers and there is congestion and thickening of the wall of a blood vessels with endothelial proliferation, many blood vessels contains few inflammatory cells in their lumens with desquamation of endothelial cells which lining the blood vessels within the lumen (fig. 2 F). The section of intestine showed extensive necrosis and loss of epithelial cells of the villi and crypts of Liberkuhn resulting in collapse and fusion of villi (fig. 2 E) and the scattered crypts are dilated and lined by remnant epithelial lining found deep in lamina propria and containing necrotic debris with mucous. Others are lined by hypertrophic regenerated epithelial cells with moderate infiltration of mononuclear cells within the lamina propria of mucosa and few in sub mucosa.

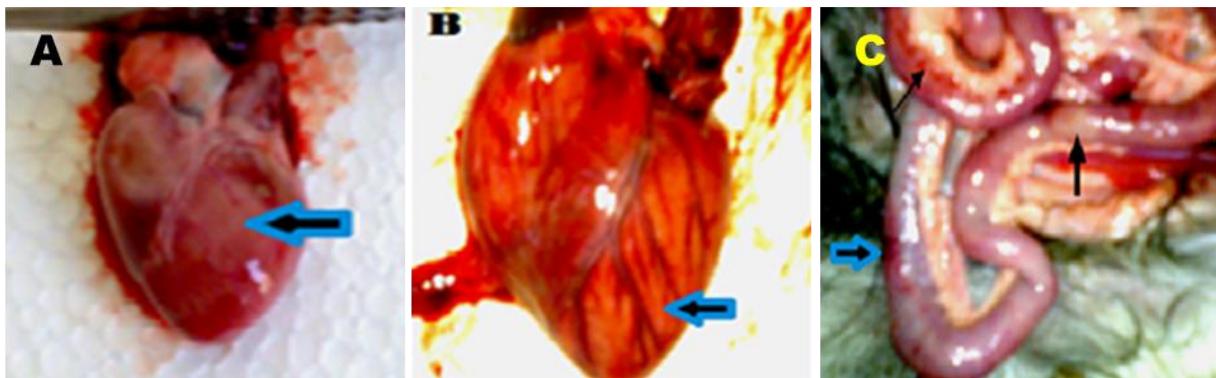


Fig. (1): A and B: Irregular pale areas of the myocardium of pups (1 and 2) months age respectively, died with both CPV2 signs (arrow). C: Small intestine there is congestion and petechial and echymotic hemorrhage (black arrow) on the serosal surface with pale areas (blue arrow) of the pup died with CPV2 enteritis.

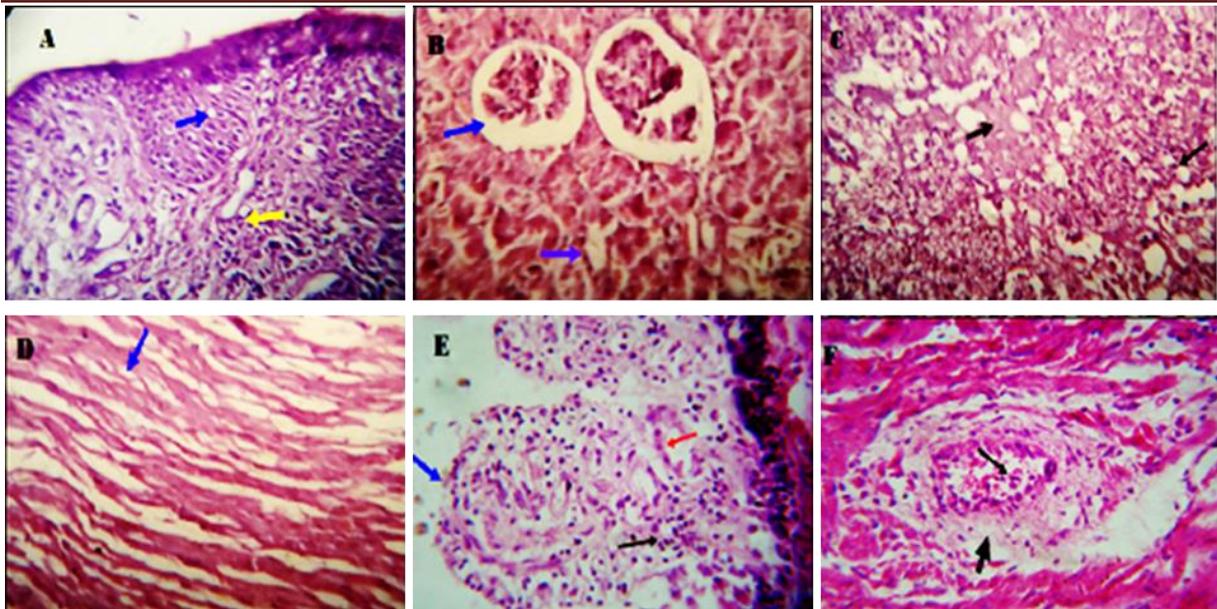


Fig. (2): A. Microscopic lesion of the tongue characterized by vacuolar degeneration of epithelial cell of mucosa with moderate mononuclear cells infiltration in sub-epithelial layer (arrows) (H&E, X40), B. Histopathological section in kidney showed atrophy of glomerular tuft with dilatation of Bowman's capsule as well as acute cellular degeneration of epithelial cell lining of renal tubule (arrows) (H&E, X40), C. Histopathological section in the lung showed edema in lumen of alveoli with moderate mononuclear cells infiltration (arrows) (H&E, X40), D. Histopathological section in the heart revealed fragmentation of muscular fibers (H&E, X40), E. Section of villi showed of desquamation of epithelial cell lining villi with collapse and fusion of villi with infiltration of mononuclear cells in lamina propria and deep mucosa the dilated scattered crypts are lined by hypertrophic, regenerated epithelial cells (arrows) (H&E, X40), F. Thickening in the wall of blood vessel with proliferate of endothelial lining cells causing the narrowing in the lumen. Few inflammatory cells are seen in the lumen (arrows) (H&E, X40).

Discussion

Canine parvovirus 2 spreads rapidly from dog to dog via oronasal exposure to contaminated feces, infected soil and fomites. Following ingestion of the virus replication begins in lymphoid tissues of oropharynx, mesenteric lymph nodes, and thymus specially attack rapidly dividing cells. The virus then disseminated to intestinal crypt by means of viremia. Marked plasma viremia is observed 1-5 days after infection (19). Subsequent to the viremia CPV2 localized predominantly in the gastro-intestinal epithelium that is lining the tongue, oral, esophageal mucosa and the small intestine. It was also localized in lymphoid tissues such as thymus, lymph nodes, and bone marrow. There was depletion of lymphocytes in

lymph nodes and necrosis in intestinal crypts. It may be also isolated from lungs, spleen, liver, kidneys and myocardium. All investigations mentioned were supported by (13) who shows that the virus may be isolated from kidneys and another organ, also in line with investigation of (14) who shows that the generalized infection of CPV leads to necrosis of developing renal tubular cells (13,14). In case of intestinal lining infection was cooperated with (15) who recorded that the virus localized predominantly in gastro-intestinal epithelium which lining the tongue and other organs of gut subsequent to viremia (15), also in line with (16) who showed that the virus cause lung hemorrhage also with (17) that recorded of pulmonary edema or

affiliates may be observed in dogs dying of complicating septicemia. There was support with (18) that showed the intestinal lesions characterized by necrosis of crypt epithelium

in the small intestine and the villi shortened or obliterated owing to lack of replacement by maturing crypt cells resulting in collapse lamina propria.

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