Histopathological examination of Bovine parainfluenza type 3 in infected organs of experiment tally infected mice

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Abstract

Twenty mice (fifteen days old) were divided into 2 groups, first group include 10 mice were inoculated intracerebrally with 100 µl of isolated bovine parainfluenza type 3 virus and the second group, include 10 mice were inoculated intracerebrally with 100 µl embryonic bovine kidney (EBK) cell suspension media. After (3-7) days post infection, mice were killed three in each group. Histopathological examination for collected organs of BPIV-3 infected mice (Liver and heart) indicated histopathological changes were detected in comparison to non-infected liver and heart.

الفحص النسيجي المرضي لفايروس نظير الأنفلونزا ألبقري نوع 3 في الأعضاء المصابة للفئران المصابة تجريبيا

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الخلاصة

تم تقسيم عشرون من الفئران بعمر خمسة عشر يوم إلى مجموعتين، المجموعة الأول تتألف من 10 فئرات تم أصابتها تجريبيا بفايروس نظير الأنفلونزا البقري نوع 3 بجرعة **100** مايكرليتر عن طريق الدماغ إما المجموعة الثانية تتألف من 10 فئرات تم حقنها بجرعة **100** مايكرليتر عن طريق الدماغ بسائل خلايا الزرع النسيجي (خلايا جنين البقر غير المحقونة بالفايروس) وتركت كمجموعة سيطرة. بعد مرور (3–7) يوم تم قتل الفئران ثلاثة في كل مجموعة. اظهر الفحص النسيجي المرضي للاعضاء المصابة بالفايروس المعزول التي تم جمعها (الكبد والقلب) تأثيرات نسيجية مرضية مقارنة بأعضاء مجموعة السيطرة التي لم يتم حقنها بالفايروس.

Introduction

Bovine parainfluenza virus type 3 (BPIV-3) is an enveloped, non-segmented, negative-sense RNA virus within the genus Respirovirus and family paramyxoviridae (1), Viruses in this group include human parainfluenza virus types 1 and 3 (HPIV-1 and HPIV-3) and Sendai virus (2). The clinical signs of infections with BPIV-3 can vary considerably, ranging from asymptomatic infections to severe respiratory illness. In most cases where BPIV-3 is implicated in disease, mild clinical signs characterized by coughing, fever and nasal discharge are observed (3). In some cases where animals are also subjected to high stress, infection with BPIV-3 can participate to immunosuppression and tissue damage and result in severe bronchopneumonia due to secondary bacterial infections (4), the disease is known as bovine respiratory disease complex (BRDC) and is considered the most important disease associated with cattle in the USA (5). Most strains of BPIV-3 induced pathogenic effects when inoculated in mice (6). Mice were experimentally infected with isolated BPIV-3 in order to detect pathogencity of isolated virus and histopathological changes were examined in infected organs (liver and heart).

Materials and Methods

- Laboratory Animals: Twenty (20) mice (fifteen days old), Albino Swiss Mice were supplied by Iraqi National Center for Drugs Safety and Evaluation.
- Cell culture: Embryonic bovine kidney (EBK) cell culture was prepared according to (7).
- Virus strain: Isolated Iraqi bovine parainfluenza type 3 virus was used for experimental infection.

The experiment was conducted according to (8) method as follows: Fifteen days old mice were divided into 2 groups, first group include 10 mice were inoculated intracerebrally with 100 µl of isolated BPIV-3 virus and the second group, include 10

mice were inoculated intracerebrally with 100 µl of EBK cell suspension media. After three days and seven days post experimental infection, mice were killed three in each group. Collected samples (liver and heart) were used for histopathological examination; buffered 10% formalin was used to fix samples for 24 hrs. Then tissues were embedded in low- melting point paraffin and sectioned at 5 Mm thickness .Samples were stained with hematoxylin and eosin (9).

Results

Histolopathological sections in the liver and heart tissue revealed the followings:

- After three days post infection: histopathological section in the liver of infected mice showed mononuclear cells infiltration in the dilated congested central vein associated with sever degenerative changes in the hepatocytes (moderate to sever vacular changes in liver paranchyma) with single cell necrosis of some hepatocytes, as seen in (Fig. 1), but no histopathological changes were detected in liver tissue of non-infected mice as seen in (Fig. 2). The heart of infected mice showed Fragmentation and separation of cardiac muscles bundles and moderate intramuscular cellular infiltration with evidence of hyalinization of some muscles bundles that appear more esinophilic or acidophilic as seen in (Fig.3) but no histopathological lesions were detected on normal cardiac tissue as seen in (Fig.4):

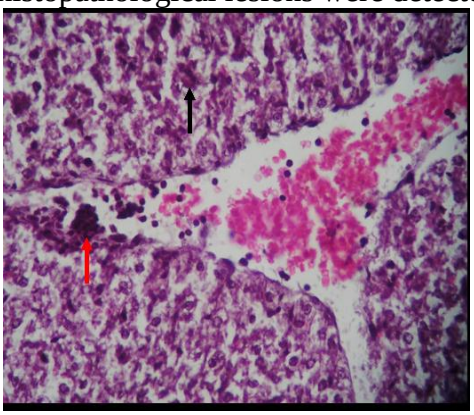

Fig. (1) histopathological section in the liver of infected mice showed mononuclaear cells infiltration (➡) in the dilated congested central vein associated with sever degenerative changes in the hepatocyte (moderate to sever vacular changes in liver paranchyma) with single cell necrosis of some hepatocytes (➡). Stained with H&E (100X).

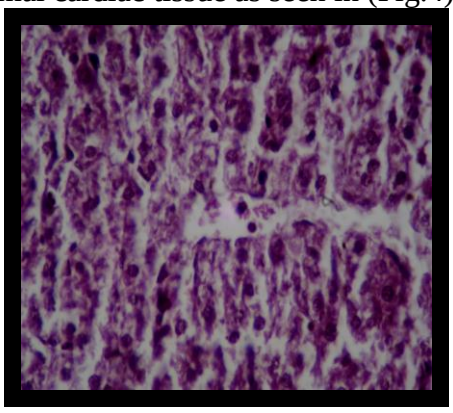

Fig. (2) histopathological section in the liver of non infected mice showed no clear pathogenic alterations observed in the liver tissue stained with H&E (100X).

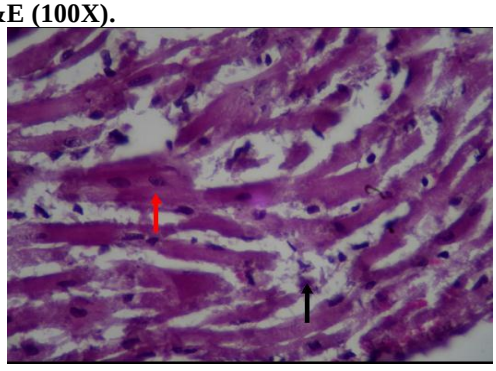

Fig. (3) histopathological section in the heart of infected mice showed Fragmentation and separation of cardiac muscles bundles and moderate intramuscular cellular infiltration with evidence of hyalinization of some muscles bundles (➡) that appear more esinophilic (➡) or acidophilic. Stained with H&E (400X).

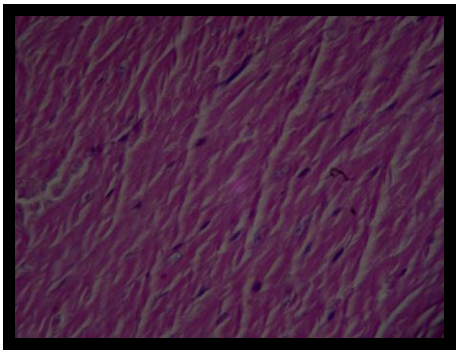

Fig. (4) histopathological section in the heart of non infected mice showed no histopathological lesions were detected on normal cardiac tissue stained with H&E (400X).

- After seven days post infection: histopathological section in the liver of infected mice showed Focal cellular infiltration of mononuclear cells seen around portal area and bile duct as seen in (Fig. 5), and also the liver of infected mice showed variable degrees of degenerative changes and necrosis in liver parenchyma with appearance of some apoptotic cells with blood vessels and sinusoid congestion as seen in (Fig.6) and (Fig.7) but no histopathological changes were detected in liver tissue of non-infected mice (Fig. 8). Histopathological section in the heart of infected mice showed sever destruction of cardiac muscle bundles with necrosis of some muscular bands and sever congestion of blood vessels as seen in (Fig.9) and also the heart tissue of infected mice showed thickening and hypertrophy of muscle bundles due to sever cellular swellings with evidence of intramuscular congestion (Fig.10) but no clear histopathological effects were detected in heart tissue of non infected mice as seen in (Fig.11).

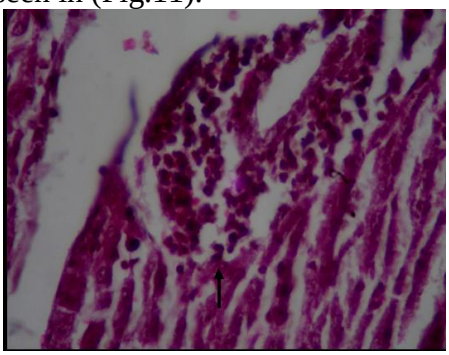

Fig. (5) histopathological section in the liver of infected mice showed Focal cellular infiltration of mononuclear cells seen around portal area and bile duct. Stained with H&E (400X).

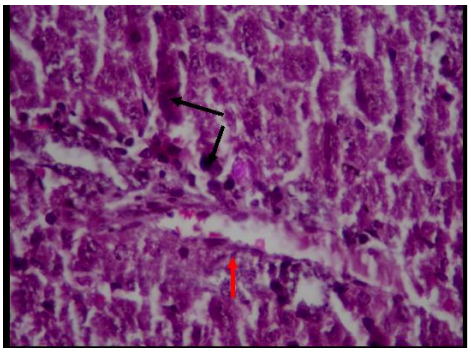

Fig. (6) Histopathological section in the liver of infected mice showed variable degrees of degenerative changes and necrosis in liver parenchyma with appearance of some apoptotic cells ( ) with blood vessels and sinusoid congestion ( ) stained with H&E (400X).

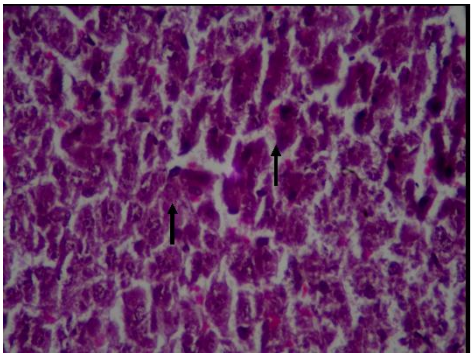

Fig (7) Histopathological section in the liver of infected mice showed variable degrees of degenerative changes and necrosis in liver parenchyma with appearance of some apoptotic cells. Stained with H&E (400X).

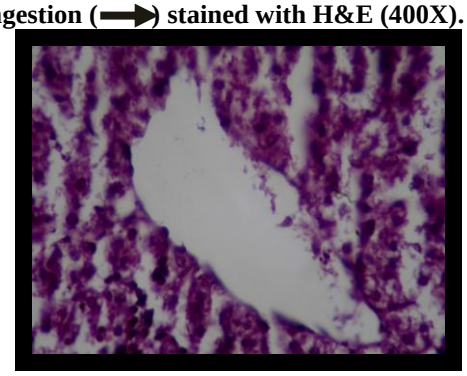

Fig. (8) Histopathological section in the liver of non infected mice showed no clear pathogenic alterations observed in the liver tissue stained with H&E (400X).

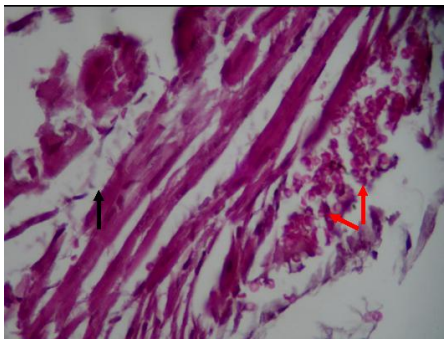

Fig. (9) Histopathological section in the heart of infected mice showed sever destruction of cardiac muscle bundles with necrosis of some muscular bands ( ) and sever congestion of blood vessels ( ) stained with H&E (400X).

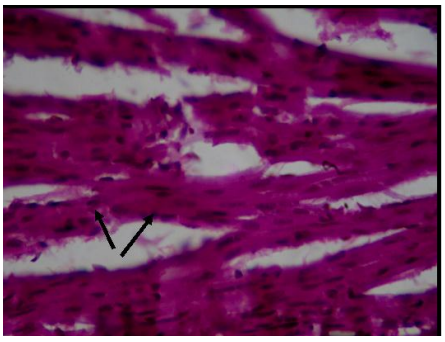

Fig. (10) Histopathological section in the heart of infected mice showed thickening and hypertrophy of muscle bundles due to sever cellular swellings with evidence of intramuscular congestion. Stained with H&E (400X).

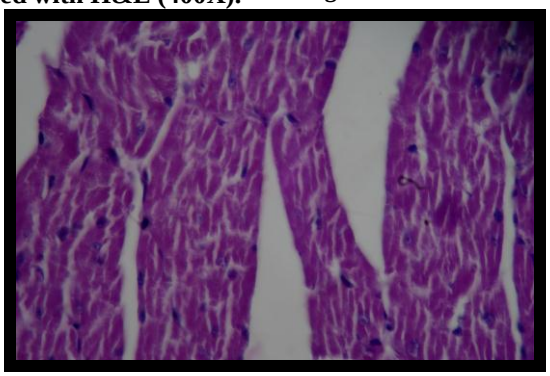

Fig. (11) Histopathological section in the heart of non infected mice showed no clear pathogenic effects were detected in heart tissue stained with H&E (400X).

Discussion

This study is the first study to detect histopathological effects of isolated bovine parainfluenza type 3 in infected liver and heart of experimentally infected mice, because most studies detected histopathological effects of BPIV- 3in infected lung and brain of infected mice because the target organ for BPIV-3 infection is lung (6, 10). In order to detect the spreading of infection with isolated BPIV-3 in other organs of infected mice, this study was conducted. As seen in Fig.(1, 3) the histopathological effects were appeared after three days post infection with infiltration of inflammatory cells and in Fig. (5, 6, 7, 9, 10) after seven days post infection in agreement with other studies which have been shown that, the histopathological changes of BPIV-3 beginning as early as 24 hrs. after infection accompanied by infiltration of inflammatory cells and these changes are most commonly between 2 and 7 days after infection (11). As seen in the in figure (6, 7, 9) there is variable degrees of degenerative changes and necrosis with appearance of some apoptotic cells in liver parenchyma and heart which agreed with (12) which has been explained that epithelial changes in the organ cultures was similar to those reported in the respiratory tract of calves with uncomplicated experimental BPIV-3 infection, and disagreed with other studies (13) which have been shown that no formal proof that BPIV-3 associated cytopathology is caused by necrosis, apoptosis or programmed cell death as in case of mouse parainfluenza type 1 infection in mice.

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