Histopathological alteration of lung, small intestine and lymph nodes in calves suffering from typical clinical case of Foot and Mouth Disease.

H. kh. Ulaiwi
Coll. of Vet. Med. /Unive of Al-Qadisiyah

H. M. J. Al-Tamemy
Coll. of Vet. Med. /Unive of Basrah

N. H. Mansoor
Coll. of Med. / Unive of Misan

Abstract

This study was designed to investigate the histopathological changes that occur in some organs during infection of calves with foot and mouth disease. Autopsies from twenty six cases of calves aged 6 months to 1 year suffering from typical case of foot and mouth disease(FMD) were studied. The results of histopathological alteration of the lung revealed emphysema of in the lung, also there clusters of pigment –laden macrophages, hemorrhage, dilatation of alveoli and accumulation of amorphous exudate . The lesion of the intestine include hemorrhage, edema, thickening and hypertrophy of villi, also there are degeneration and necrosis of some intestinal gland. Alteration in the lymph nodes showed atrophy of lymphoid nodules and accumulation of collagen fibers with hemorrhage.

Introduction

FMD is an acute infection of cattle, sheep, pigs, goats, buffalo and many species of cloven-hoofed wildlife, caused by a single-stranded RNA virus belonging to the genus Aphthovirus, in the family Picornaviridae. There are seven distinct serotypes of FMD virus, and within each serotype there are numerous strains(1). The virus causes an acute disease of cloven-hoofed animals characterized by fever, lameness, and vesicular lesions of the feet, tongue, snout, and teats. These debilitating effects, rather than high mortality rates, are responsible for severe productivity losses associated with foot-and-mouth disease (FMD). The highly contagious nature of the virus and severity of economic impacts associated with the disease, determine FMD's status as the most important disease limiting trade of animals and animal products throughout the world(2).

Materials and Methods

This study conducted in the field by studying 26 cases of calves aged 6 months to 1 year suffering from FMD. After investigation of clinical signs and gross lesion, an autopsy from immediately dead calves were taken including specimens from lungs, intestine and lymph nodes to reveal the histopathological changes. The specimens fixed in 10% formalin and dehydrated by ascending concentrations of ethanolic alcohol, embedded in paraffin, cut at 5 μm, stained with hematoxylin and eosin and examined by light microscopy(3, 4).

Results

- Clinical signs

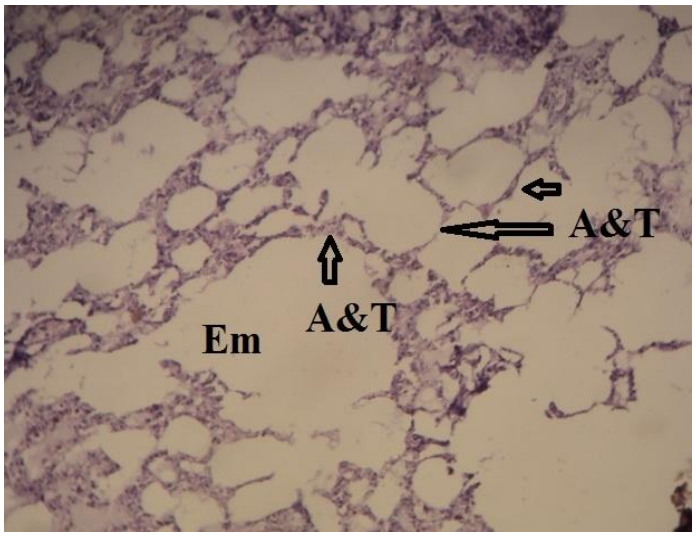
The clinical signs of the affected calves characterized by profuse salivation, erosion in the buccal cavity of the mouth hemorrhage with saliva, vesicle in the mouth and interdigital space and erosion and ulceration of the coronary band and respiratory difficulties.

- Gross pathological changes

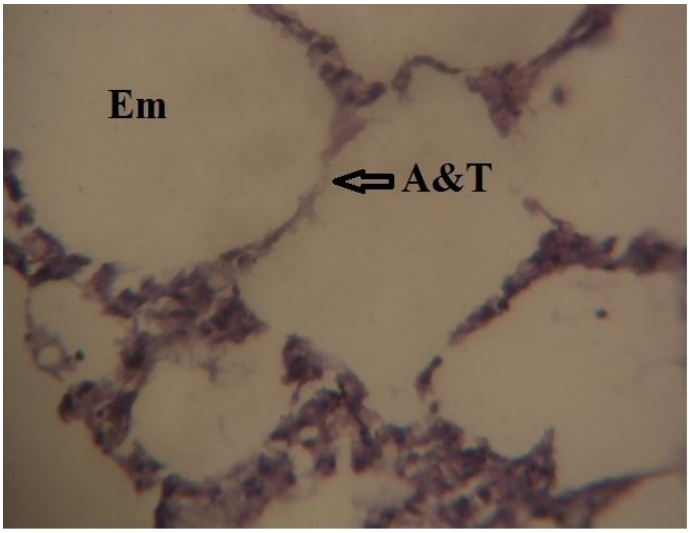
The gross lesion in the viscera include severe hemorrhage in the lungs with frothy hemorrhagic exudated in the trachea and presence of serous fluid in the thoracic cavity and hemorrhagic enteritis.

- Histopathological changes:

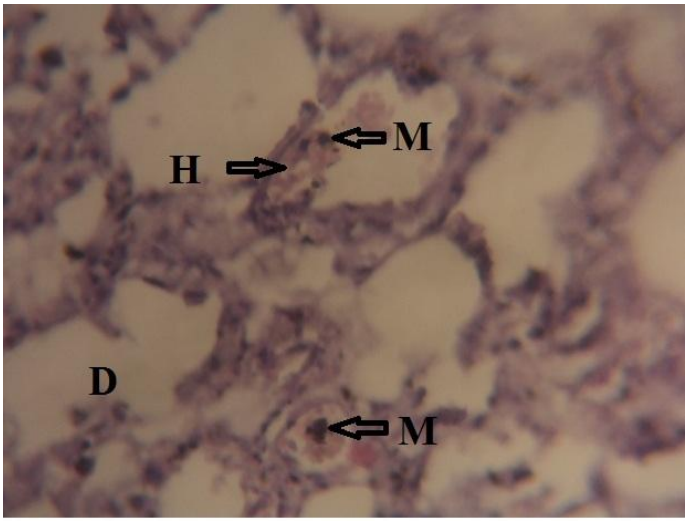
- Lungs: the results of histopathological alteration of the lung revealed emphysema of in the lung were the alveolar wall atrophied and thin(figures:1 and 2), also there clusters of pigment – laden macrophages, hemorrhage, dilatation of alveoli and accumulation of serous exudate (figures:3,4, 5 and 6).
- Intestine: the histopathological lesion of the intestine included hemorrhage, edema, thickening and hyperplasia of villi(figure:7), also there are degeneration and necrosis of some intestinal gland(figure:8).
- Lymph nodes: alteration in the lymph nodes showed atrophy of lymphoid nodules and accumulation of collagen fibers with hemorrhage(figures:9 and 10).



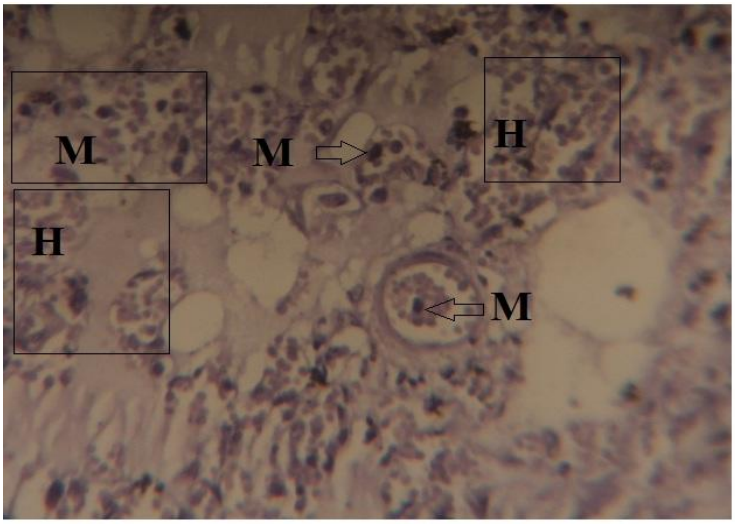
Figure(1): histological section of lung shows, the alveoli are thin(A&T) with emphysema(Em). H&E. 100X.



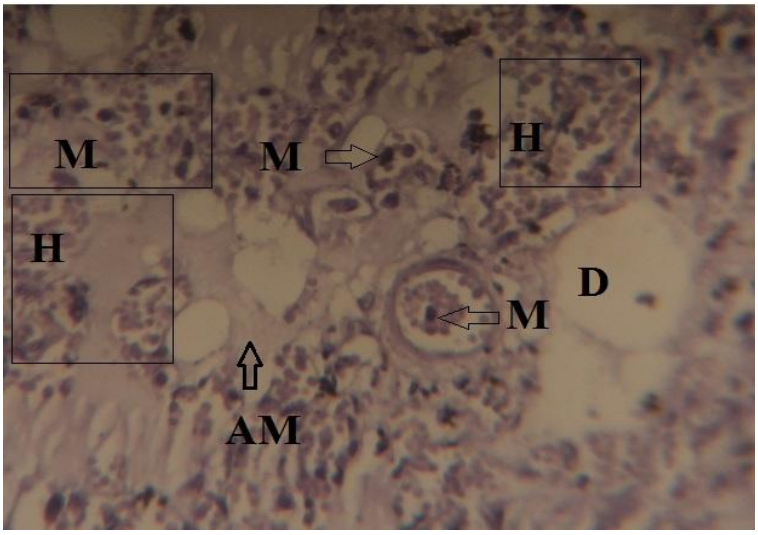
Figure(2): histological section of lung shows, the alveoli are thin(A&T) with emphysema(Em). H&E, 400X.



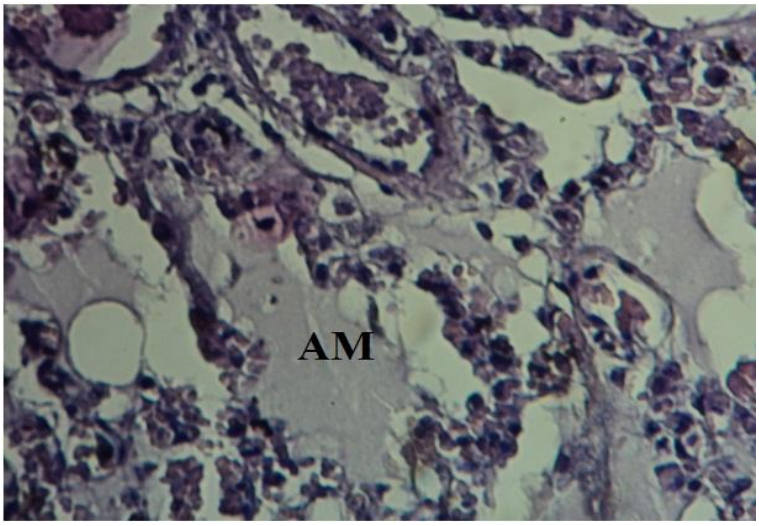
Figure(3): histological section of lung shows that there are clusters of pigment –laden macrophages(M), hemorrhage(H) and dilatation of alveoli(D). H&E, 400X.



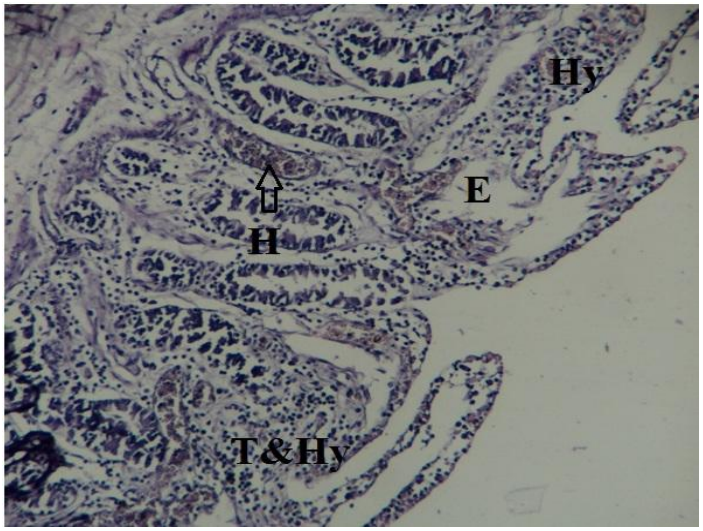
Figure(4): histological section of lung shows that there are clusters of pigment –laden macrophages(M) and severe hemorrhage(H). H&E, 400X.



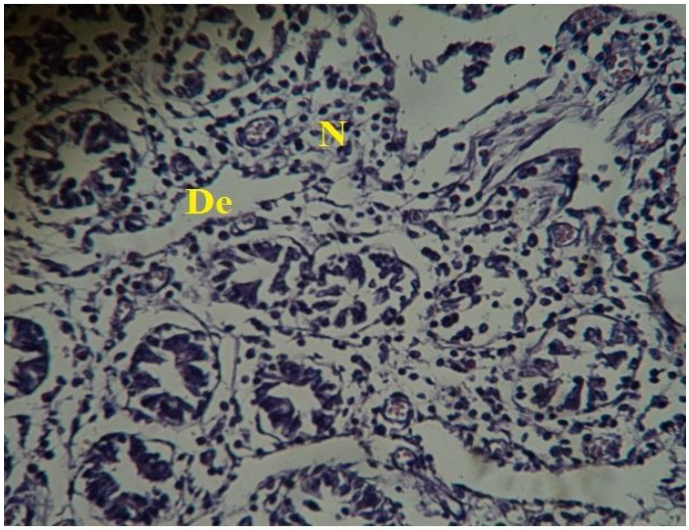
Figure(5): histological section of lung shows that there are clusters of pigment –laden macrophages(M), severe hemorrhage(H), dilatation of alveoli and accumulation of serous exudate in the alveoli (AM). H&E, 400X.



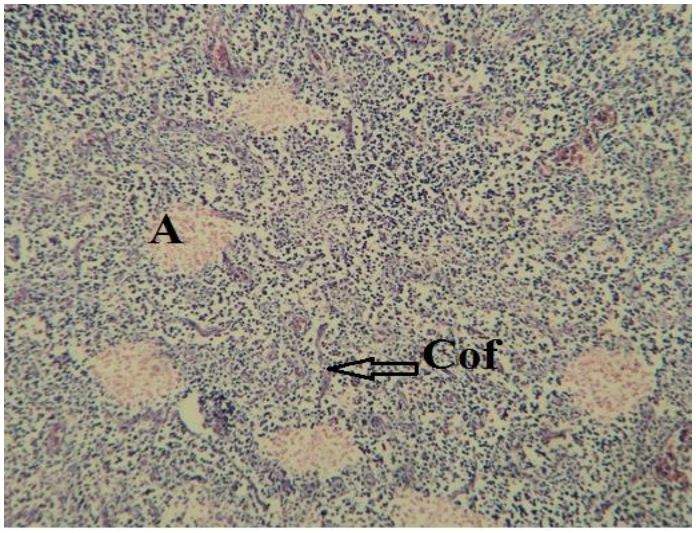
Figure(6): histological section of lung shows, presence of exudate(amorphous materials)(AM) in the alveoli. H&E, 400X.



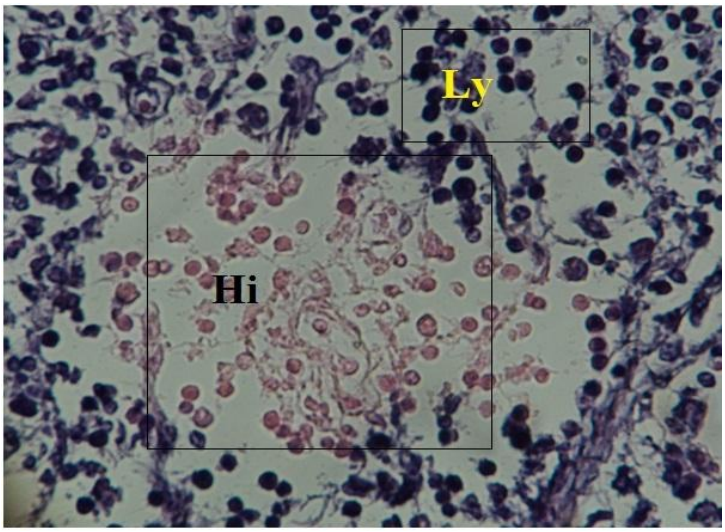
Figure(7): histological section of intestine shows that there are hemorrhage(H), edema(E), thickening and hyperplasia of villi(T&Hy). H&E, 100X.



Figure(8): histological section of intestine shows that there are degeneration(De) and necrosis(N) of some duodenal gland. H&E, 100X.



Figure(9): histological section of lymph node shows that there are atrophy of white pulp(A) and accumulation of collagen fibers(Cof). H&E, 100X.



Figure(10): histological section of lymph node shows that there are hemorrhage(Hi) extravasated between lymphocytes(Ly). H&E, 400X.

Discussion

Virus can gain entry through abrasions in the epithelium of the oral cavity, feet or teats, it is now generally accepted that the common portal of entry of the virus is by the respiratory tract (5). Most virus will be trapped in the upper respiratory tract, with subsequent multiplication in the mucosa of the oro-pharynx. However, after experimental pulmonary infection of cattle, FMD virus will multiply in lung tissue (6) and virus that reaches the alveoli can also pass readily into the blood stream (7). FMD virus is then distributed throughout the body, to reach multiplication sites such as the

epithelium of the oro-pharynx, oral cavity, feet and the udder.It has recently been suggested that FMDV replication in the lungs of cattle may substantially contribute to maintenance of high-titer viremia. FMDV was most consistently localized to nasopharyngeal tissues, thereby indicating this region as the most important site of primary viral replication. The earliest site of microscopic localization of FMDV antigens was the lymphoid follicle-associated epithelium of the pharyngeal mucosa-associated lymphoid tissue of the nasopharynx at 6 hours postaerosolization(8). Detection of high quantities of viral RNA, antigen, and infectious virus in pulmonary tissues starting at the onset of viremia combined with the large mass of the lungs. Onset of viremia coincided with marked increase of viral loads in pulmonary tissues and with substantial decrease of viral detection in nasopharyngeal tissues. These data indicate that subsequent to aerogenous exposure to FMDV, the temporally defined critical pathogenesis events involve (9) primary replication in epithelial cells of the pharyngeal mucosa-associated lymphoid tissue crypts and (10) subsequent widespread replication in pneumocytes in the lungs, which coincides with (11) the establishment of sustained viremia.A transient lymphopenia has been reported during the early stages of infection in swine which may be a consequence of early infection of T cells(12,13).The virus enters and multiplies in the pharynx and lungs, followed by viraemic dissemination to surface epithelium with subsequent lesion development at sites of mechanical or physiological stress such as oral and pedal epithelium and the teats of lactating animals(14). The (15) indicate that the bronchioalveolar pneumonia was characterized by diffuse infiltration of neutrophils and macrophages. There was also diffuse alveolar oedema with moderate active congestion in the affected areas of the lungs.

References

1.Regenmortel, M.H.V.; Fauquet, C.M.; Bishop, D.H.L.; Carstens, E.B.; Estes, M.K.; Lemon, S.M.; Manioff, J.; Mayo, M.A.; McGeoch, D.J.; Pringle, C.R. and Wickner, R.B. (2000). Virus Taxonomy: Classification and Nomenclature of Viruses. Seventh Report of the International Committee on Taxonomy of Viruses. Academic Press, SanDiego.

2.Jonathan, Arzt.(2010). Dissertation the early pathogenesis of Foot and Mouth Disease in Cattle after Aerosol inoculation. Dissertation of doctorate / Colorado State university.

3. Luna, L.G. (1968). Manual of Histological Staining methods of the Armed Forces Institute of Pathology. 3rd ed. New York, Mc Graw-Hill.

4.Bancroft, J.D.; Stevens, A. & Turner, D.R. (1990).Theory and Practice of Histological techniques.3rd ed. Churchill Livingstone.Pp:21-226.

5.Sutmoller, P.; McVicar, J.W. and Cottral, G.E.(1968). The epizootiological importance of foot-and-mouth disease carriers. I. Experimentally produced foot-and-mouth disease carriers in susceptible andimmune cattle. *Arch. Ges. Virusforsch*; 23: 227-235.

6.Eskildsen, M.K.(1969). Experimental pulmonary infection of cattle with foot-and-mouth disease virus. *Nord. Med. Vet.*; 21: 86-91.

7.Sutmoller, P. and McVicar, J.W.(1981). Pathogenesis of foot-and-mouth disease: The lung as an additional and simulated natural foot- and-mouth disease infection in cattle. *J. Comp. Path.*; 91:599-609.

8.Arzt, J.; Pacheco, J.M. and Rodriguez, L.L.(2010). The early pathogenesis of foot-and-mouth disease in cattle after aerosol inoculation: identification of the nasopharynx as the primary site of infection. *Vet Path*.;47(6):1048-1063.

9.Alexandersen, S. and Mowat, N. (2005). Foot-and-mouth disease: host range and pathogenesis. *Curr Top Microbiol Immunol.;* 288:9–42.

10. Alexandersen, S.; Zhang, Z.; Donaldson, A.I. And Garland, A.J.(2003). The pathogenesis and diagnosis of foot-and-mouth disease. *J Comp Pathol*.; 129:1–36.

11. Arzt, J.; Gregg, D.A.; Clavijo, A. and Rodriguez L.L.(2009). Optimization of immunohistochemical and fluorescent antibody techniques for localization of foot-and-mouth disease virus in animal tissues. *J Vet Diagn Invest*.; 21:779–792.

12. Bautista, E.M.; Ferman, G.S. and Golde, W.T.(2003). Induction of lymphopenia and inhibition of T cell function during acute infection of swine with foot and mouth disease virus (FMDV). *Vet Immunol Immunopathol.;* 92:61-73.

13. Diaz-San Segundo, F.; Salguero, F.J.; de Avila, A.; de Marco, M.M.; Sanchez-Martin, M.A. and Sevilla, N.(2006).Selective lymphocyte depletion during the early stage of the immune response to foot-and-mouth disease virus infection in swine. *J Virol.;*80:2369-2379.

14. Brown, C. C.; Baker, D. C. and Barker, I. K. (2007). Alimentary system. In: Maxie, M. G.; *Kennedy, J. and Palmer's pathology of domestic animals* (5th ed.). Saunders, Edinburgh: 135-137.

15. Berkowitz, A; Waner , T; King, R; Yadin, H and Perl , S.(2010). Description of the pathology of a gazelle that died during a major outbreak of foot-and-mouth disease in Israel. *J. S. Afr. Vet. Assoc*.; 81 (1).

التغيرات النسجية المرضية في الرئتين والأمعاء والعقد اللمفاوية في العجول المصابة بمرض الحمى القلاعية

حسن خلف عليوي حسان محمد جاسم نمارق هادي منصور
كلية الطب البيطري/جامعة القادسية. كلية الطب البيطري/جامعة البصرة كلية الطب/جامعة ميسان

الخلاصة

صممت هذه الدراسة للتعرف التغيرات النسجية المرضية التي تحدث في بعض الأعضاء الداخلية خلال إصابة العجول بمرض الحمى القلاعية. ستة وعشرون عينة نسجية أخذت من عجول تتراوح أعمارها ما بين ستة أشهر الى سنة تعاني من إصابات تميزت بأنها نموذجية لحالة الحمى القلاعية لدراستها. أظهرت نتائج الفحص النسيجي بوجود حالات نفاخ الرئة مع وجود تجمعات لخلايا البلاعم الحاوية على صبغات، كذلك وجود نزف وتوسع للاسناخ الرئوية مع تجمع للسوائل الالتهابية. الآفات الموجودة في الأمعاء الصغيرة تميزت بوجود نزف مع وذمه مع تثخن للزغابات المعوية وكذلك تنكسات مع تموت في بعض الغدد المعوية. أما التغيرات النسجية المرضية في العقد اللمفية فاوضحت وجود ضمور في العقيدات اللمفية مع تجمع للألياف الغر وانية مع نزف.