قسم الباثولوجيا كلية الطب البيطري _ جامعة القاهرة (فرع بني سويف) رئيس القسم : أ د / صلاح ديب

قابلية الأغنام للاصابة بفيروس التهاب الأنف والقصبة الهوائية النفطي المعدي

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أجريت الدراسة الباثولوجية على عدد اثنين من الماعز النافقة ضمن قطيع يعاني من أعراض تنفسية مما رجح الاصابة بمرض التهاب الأنف والقصبة الهوائية النفطي المعدي وكانت أصابته أساسا بالجزء العلوي من الجهاز التنفسي مع التهاب رئوي بيني مع وجود وظهور التنكرز في كل من الكبد والكلى والقلب واحتقان في الغدد الليمفاوية وهذا وقد شوهدت الاجسام المحتواه الحمراء المميزة للمرض داخل أنوية الخلايا الطلائية للشعيبات الهوائية وكذلك في خلايا الكبد واستنتجت هذه الدراسة أن الأغنام قابلة للاصابية الطبيعية بفيروس التهاب الأنف والقصبة الهوائية النفطي المعدي وأن العوامل المنهكة تلعب دورا كبيرا في اظهار الصورة الحقيقية للمرض وقد كانت التغيرات الباثولوجية في هذه الحالات مشابه الى حد كبير لتلك التغيرات التي تشاهد في الأبقار و

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SUSCEPTIBILITY OF GOATS TO INFECTIOUS BOVINE RHINOTRACHEITIS VIRUS (With 6 Figs.)

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SUMMARY

The pathological study of two goats, which died from severe respiratory illness, revealed lesions suggestive of IBR. The lesions consisted of congestion of the upper respiratory tract, interstitial pneumonia, focal degeneration and necrosis in the liver and kidneys, myocardiosis and congestion of the lymph nodes. Acidophilic intranuclear inclusion bodies were seen in the alveolar and bronchiolar epithelium and in the hepatic cells. It was concluded that goats are susceptible to natural infection with IBR-virus, however, predisposing factors are thought to play a great role for the animals to react typically. The lesions in the affected goats were more or less similar to those cattle.

INTRODUCTION

Controversial results have been reported with respect to the susceptibility of goats to infectious bovine rhinotracheitis virus infection (IBR-virus). MACKERCHER, et al. (1958) found that goats are susceptible to IBR-virus. An elevation in temperature and mild clinical illness occurred in animals experimentally exposed to the virus. In contrast, VAN HOUWELLING (1966) reported that goats were not susceptible to infection with IBR virus either by intranasal or intracerebral inoculation and that infection was not associated with clinical signs or serum antibodies; however, the virus was recovered from nasal secretions of infected goats.

MOHANTY, et al. (1972) could isolate a viral agent identified as IBR virus from the nasal swabs of 2 goats with signs of severe respiratory illness and from ocular swabs of one of these goats that had severe keratitis with keratoconus.

PIRAK, et al. (1983) found that, in goats experimentally infected with a Belgian strain of IBR-virus (BHV-1 strain); virus excretion occurred in each animal for 5-13 days. The authors reported, moreover, that a rise of neutralizing antibodies was observed but no clinical signs were noted.

No information on the lesions of the natural or experimental IBR-virus infection was available in the literature, thus, the present study deals with these changes.

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MATERIAL and METHODS

In February 1987, two from 9 dead goats were submitted to Animal Health Institute, Dokki, for postmortem examination. The animals came from a flock of 20 goats belonging to the animal station of the Faculty of Agriculture, Al-Azhar University, Cairo. The animal station includes beside goats, sheep and cattle. Unfortunatally, exact information about the clinical status of these sheep and cattle were not available.

Specimens from different organs were collected for histopathological examination, paraffin sections were prepared and stained with haematoxylin and eosin.

RESULTS

Grossly, the gastrointestinal tract was inflammed. The larynx and trachea were severely congested. The lungs were pneumonic and consolidated. The kidneys were swollen and pale. The heart muscle showed features of degeneration. The spleen was slightly enlarged and the various lymph nodes were congested.

Microscopically, changes in the lung consisted of interstitial pneumonia. The alveolar wall was thickened due to aggregation of mononuclear cells which also obliterated the alveolar lumina. Desquamated epithelium and cellular debris were found inside the alveoli. The epithelial linning of many small bronchioles was destroyed and the bronchiolar lumina were filled with inflammatory cells, mainly mononuclears and neutrophils (Fig. 1). Multinucleated giant cells were frequently seen in the inflammed areas of the lung parenchyma and also lie free inside the bronchiolar lumina. These giant cells may show features of active phagocytosis (Fig. 2). Both in the alveoli and bronchioles, the cellular changes of the lining epithelium consisted of margination of nuclear chromatin, swelling of the nucleoli, and occurrence of intranuclear inclusion bodies (Fig. 3 a.b.c). These inclusion bodies were acidophilic, irregular, and structureless. They were seen also in desquamated alveolar and bronchiolar epithelium.

Degeneration and coagulative necrosis (peripherolobular) occurred in the liver (Fig. 4). The portal triads were slightly infiltrated with mononuclear cells. The sinusoids and central veins were dilated and engarged with blood. Acidophilic intranuclear inclusion bodies were seen in the hepatic cells adjacent to the necrotic areas (Fig. 5). Rarely, intranuclear inclusion bodies were found in Kupffer cells.

The main renal lesions were coagulative necrosis in the cells of the renal tubules especialy the convoluted ones (Fig. 6). No inclusion bodies could be demonstrated inside the cells of the renal tubules.

Focal haemorrhages occurred in the heart. Occasionally, the cardiac muscle bundles and Purkinjie fibers showed degeneration and necrosis. There was deposition of excessive amount of haemosiderin in the spleen. In the lymph nodes, depletion of lymphocytic alements was observed.

DISCUSSION

Pathological investigation of two goats, in the present study, revealed the occurrence of changes quite similar to IBR infection in cattle. In the latter, animals exposed to infection with the virus usually show inflammatory and degenerative changes in the upper respiratory

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tract, conjunctivitis. interstitial pneumonia, catarrhal bronchopneumonia, inflammatory changes in the digestive tract and erosions, as well as focal necrosis in the liver, kidneys, adrenals, spleen and lymph nodes (CRANDELL, et al. 1959; WEBSTER and MANKETLOW, 1959; BAKER, et al. 1960; HUGHES, et al. 1964; KRUNINGEN and BARTHOLOMEW, 1964). These lesions were associated with the occurrence of eosinophilic intranuclear inclusion bodies in the upper respiratory tract, lungs, liver, kidneys and brain (BAKER, et al. 1960; KRUNINGEN and BARTHOLOMEW, 1964; SAITO, et al. 1974; BERROIS, et al. 1975; BAYOUMI, 1983; MOLNAR, et al. 1984; OMRAN, 1985).

Variable results have been found in goats experimentally infected with IBR-virus compared to naturally affected cases. The clinical picture described in experimentally infected animals consisted of fever, mild clinical illness (MACKERCHER, et al. 1958), profuse bilateral nasal and ocular discharges accompanied by fibrinonecrotic ulceration of the nasal septum and hyperaemia of the nasal mucosa (SAITO, et al. 1974; BERROIS, et al. 1975), or that no clinical signs were noted (VAN HOUWELLING, 1966; PIRAK, et al. 1983). In naturally infected goats, severe keratitis with keratoconus and signs of severe respiratory illness were reported (MOHANTY, et al. 1972).

Pathologically, no data was available about the experimentally infected goats. As was found in the present study, the lesions in the naturally infected cases included congestion of the upper respiratory tract, interstitial pneumonia, degeneration and necrosis in the liver and kidneys, myocardiosis and congestion of lymph nodes. Intranuclear inclusion bodies, characteristic of the disease in cattle, were seen in the lungs and liver of infected goats.

The virus of IBR was isolated from both experimentally and naturally infected goats. It was recovered from the nasal secretion of experimentally infected animals (VAN HOUWELLING, 1966) 5-13 days after infection (PIRAK, et al. 1983). In naturally infected cases, a viral agent identified as IBR virus could be isolated from the nasal swabs of animals with signs of severe respiratory illness and from ocular swabs of a goat that had keratitis (MOHANTY, et al. 1972). No attempt for viral isolation was carried out in the present study.

Differences were also reported with respect to antibody response of goats experimentally infected with IBR virus and those naturally exposed to it. VAN HOUWELLING (1966) reported that intranasal or intracerebral inoculation of the viurs was not associated with serum antibodies. In contrast, PIRAK, et al. (1983) found a rise of neutralizing antibodies in experimentally infected goats. Serological field investigation was carried out on Nigerian livestock (1974–1980) showed that 11.2% of 501 goats were positive for IBR (NAWATHE and LAMONDE, 1982). In Nigeria also, survey of IBR virus infection in goats by the International Livestock Center for Africa in 1984, resulted in the detection of antibodies in 26% of 196 samples. However, a serological investigation was conducted by LAMONTAGNE, et al. (1985) to detect antibodies against bovine respiratory viruses including IBR-virus in goats. The authors found that there was no antibodies to IBR/IPV in 112 goats tested.

According to the above-mentioned data, the result of exposure of goats either experimentally or naturally to IBR-virus can be summerized as follows:

	Experimental Infection	Natural Infection
Clinical signs	mild or absent	severe
Lesions	?	+
Antibody response	+	+
Virus shedding	+	+

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It can thus be concluded that, although goats are susceptible to IBR-virus, predisposing factor (s) may play a major role for the manifestation of different types of reaction.

It is known that the clinical picture, lesions and antibody response depend not only on the host or its environment, but also on the characteristics of the virus strain. The identity of IBR viurs to the virus of infectious pustular vulvovaginitis (IPV) or coital exanthema as previously termed has been suggested by GILLESPIE, et al. (1959) and was later confirmed by many investigators. Experimental infection of goats with IPV showed variable results; however, typical lesions may be seen (BINDRICH, 1960; MANNINGER, et al. 1962). In cattle, the application of IPV-virus usually leads to rhinotracheitis (GILLESPIE, et al. 1959; MACKERCHER, et al. 1959; MANKETLOW and HANSEN, 1961; MACKERCHER, 1963; STUDDERT, et al. 1964 a.b).

In an experimental study by PIRAK, et al. (1983), virus excretion occurred in each of 7 goats for 5-13 days after intranasal inoculation of a Belgian strain of IBR virus (BHV-1 strain). These authors, however, found no excretion of the virus after two dexamethazone treatments applied one month and three months after the experimental infection. They concluded that IBR virus does not seem to remain latent in goats.

REFERENCES

- Baker, J.A.; K. MacEntee and J.H. Gillespie (1960): Effects of infectious bovine rhinotracheitisinfectious pustular vulvovaginitis (IBR-IPV) viurs on newborn calves. Cornell Vet., 50: 156-170.
- Bayoumi, A.H. (1983): Personal communication.
- Berrois, P.E.; D.G. McKercher and H.D. Knight (1975): Pathogenicity of a caprine herpesvirus.

 Am. J. Vet. Res., 36: 1763-1769.
- Bindrich, H. (1960): Untersuchungen über des virus des Bläschshlages des Rindes (Exanthema coitale vesiculosum bovis). Arch. Exp. Vet. Med., 14: 656-675.
- Crandell, R.A.: W.J. Cheatham and F.D. Maurer (1959): Infectious bovine rhinotracheitis. The occurrence of intranuclear inclusion bodies in experimentally infected animals. Am. J. Vet. Res., 20: 505-509.
- Gillespie, J.H.; K.M. Entee; J.W. Kendrick and W.C. Wagner (1959): Comparison of pustular vulvovaginitis virus with infectious bovine rhinotracheitis virus. Cornell Vet., 49: 288-297.
- International Livestock Centre for Africa (1984): Serological survey of some viral infections in goats in Southern Nigeria. Sheep and goats in hind West Africa. Proc. Workshop on Small Ruminant Production System, pp. 23-26.
- Hughes, 9.P.; H.J. Olander and M. Wada (1964): Keratoconjunctivitis associated with infectious bovine rhinotracheitis. J. Am. Vet. Med. Ass., 145: 32-39.
- Kruningen, H.J. and R.B. Bartholomew (1964): Infectious bovine rhinotracheitis diagnosed by lesions in a calf. J. Am. Vet. Med. Ass., 144: 1008-1012.
- Lamontagne, L.; J.-P. Descoteaux and R. Roy (1985): Epizootiological survey of parainfleunza-3, reovirus-3, respiratory syncytial and infectious bovine rhinotracheitis viral antibodies in sheep and goat flocks in Quebec. Canad. J. Comp. Med., 49: 424-428.
- MacKercher, D.G. (1959): Infectious bovine rhinotracheitis. Advances Vet. Sci., 5: 299-328. MacKercher, D.G. (1963): Studies of the etiologic agents of infectious bovine rhinotracheitis and Bläsenschlag (coital vesicular exanthema). Am. J. Vet. Res., 24: 501-509.
- MacKercher, D.G.; J.K. Saito; E.M. Wada and O.C. Straub (1958): Current status of the newer virus disease of cattle. Proc. 62nd Ann. Meeting U.S. Livestock san A., pp. 136-156.

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- Manketlow, B.W. and N.F. Hansen (1961): The isolation of a cytopathogenic agent resembling the virus of infectious bovine rhinotracheitis from an outbreak of pustular vulvovaginitis in cattle. New Zealand Vet. J., 9: 136-140.
- Manninger, K.; A. Barthe; A. Juhasz and Szent-Ivanyi, T. (1962): Studies of the aetiology and pathology of the so-called coital vesicular exanthema in cattle. Magyar Allatorvosok Lapja, 17: 193-199.
- Mohanty, S.B.; M.G. Lillie, N.P. Corselius and J.B. Beck (1972): Natural infection with infectious bovine rhinotracheitis virus in goats. J. Am. Vet. Med. Ass., 160: 879-880.
- Molnar, T.; R. Glavits, and V. Paffi (1984): Unusualform of IBR/IPV virus infection in young calf. Magyar Atlatorvosok Lapja, 39: 661-665.
- Nawathe, D.R. and A.G. Lammonde (1982): The impact of viral diseases on the Nigerian livestock economy and some suggestions on the control of rabies in Nigeria. Bull. Anim. Health Prod. Africa, 30: 309-314.
- Omran. Rawhia M.A. (1985): Pathological studies on infectious bovine rhinotracheitis (IBR) among newly born calves in Egypt. M.V.Sc. Thesis, Cairo Univ.
- Pirak, M.; E. Thiry, B. Brochier and P.-P. (1983): Infection experimentale de la chevre par le virus der la rhinotracheite infectieuse bovine. (bovine herpes virus 1) et tentative de reactivatin virale. Recueil de medecine Vet., 45: 1103-1105.
- Saito, J.K.; D.H. Gribble; P.E. Berrois; H.D. Knight and D.G. McKercher (1974): A new herpes virus isolate from goats: Preliminary report. Am. J. Vet. Res., 35: 361-362.
- Snowdon, W.A. (1964): Infectious bovine rhinotracheitis and infectious pustular vulvovaginitis in Australian cattle. Austr. Vet. J., 40: 277-288.
- Studdert, M.J.; C.A.V. Baker and M. Savan (1964 a): Infectious pustular vulvovaginitis virus infection of bulls. Am. J. Vet. Res., 25: 303-314.
- Studdert, M.J.; E.M. Wada, W.M. Kortum and F.A. Groverman (1964 b): Bovine infectious pustular vulvovaginitis in the Western United States. J. Am. Vet. Med. Ass., 144: 615-619.
- Van Houwelling, C.D. (1966): Susceptibility of goats to infectious bovine rhinotracheitis. Cornell Vet., 56: 38-41.
- Webster, R.G. and B.W. Manketlow (1959): Some observations on infectious bovine rhinotracheitis in New Zealand. New Zealand Vet. J., 7: 143-148.

Description of Figures

- Fig. (1): Lung showing desquamated epithelium and inflammatory cell infiltration (H & E, X 400).
- Fig. (2): A giant cell in the bronchilar lumen. Notice the presence of inclusion bodies in the nuclei of the cell (H & E, X 400).
- Fig. (3): Lung. Occurrence of acidophilic intranuclear inclusion bodies in the epithelial linning of a small bronchiole (H & E, X 400).
- Fig. (4): peripherolobular coagulative coagulative necrosis of the hepatic cells (H&E, X 100).
- Fig. (5): Liver. Acidophilic intranuclear inclusion bodies in the hepatic cells (H & E, X 1000).
- Fig. (6): Kidney showing necrotic changes of the convoluted renal tubules (H & E, X 100).















