PATHOLOGY OF NATURAL AND EXPERIMENTAL ADENOVIRUS ENTERITIS IN PIGS

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Introduction

Adenoviruses have been associated with diarrhea in several animal species such as cattle, dogs, sheep, horses, rabbits, chickens, turkeys, pigeons and pigs. In this last species adenoviruses could be isolated from faeces, brains and kidneys. One of the isolated strains was called 6618. With this and other strains several experimental transmissions were performed. A mild diarrhea sometimes was seen clinically, beside lesions such as hepatitis, interstitial nephritis, myocarditis, meningitis and encephalitis. Also natural cases of adenovirus enteritis were suspected.
The natural case of adenovirus enteritis and intestinal lesions of experimentally piglets are reported in this paper.

Methods

From the natural infected piglet the lower jejunum and ileum were examined by histology and electron microscopy. Twenty one hysterectomy-derived, colostrum-deprived piglets were used. They were infected with the porcine adenovirus strain 6618. Twelve piglets received $4.10^{5.3}$ mean TCID 50 of virus, five piglets received 4.10^{5} TCID 50 and one more piglet received 4.10^{7} TCID 50 virus on the second or third day of live. Three piglets were controls. The animals were killed at 12 and 18 h. one day (the piglet that received 4.10^{7}), 2,3,4,5,6,7,15,16,45 and 120 days after challenge. For histology tissue specimens of the tonsil, stomach, duodenum upper, middle and lower jejunum, the ileum, caecum, colon and rectum were taken. For transmission electron microscopy the lower jejunum of 16 piglets was examined. For scanning electron microscopy the terminal jejunum was examined. Intestinal contents were examined by negative staining.

Results

Natural case

In the ileum many intranuclear inclusion bodies were present in enterocytes of short villi. Affected nuclei were enlarged and round. A clear halo separated nuclear membrane from the inclusion body. Electron microscopy of affected nuclei revealed many adenovirus particles inside the nucleus. When many particles were present, crystalline arrays were found. When only a few were present, they were scattered throughout the nucleus. Sometimes caryolysis of enterocytes was seen. Also infection of goblet cells was seen.

Experimental piglets

The incubation period of the disease varied from 3 to 4 days, except in the piglet that received 4.10^{7} TCID 50 of virus. The duration of the diarrhea was 3 to 6 days.
At 24 h after infection, the above described piglet started to show diarrhea and histologically many intranuclear inclusion bodies were present in enterocytes of short villi in the terminal jejunum and ileum. Affected nuclei were located toward the microvillus border. From 48 h after infection on, affected nuclei had a narrow peripheral clear halo and four days after infection typical Cowdry type A inclusion bodies could be observed. Short villi and inclusion bodies were present till 15 days after infection. No lesions could be observed in the crypt epithelium. In the duodenum, upper jejunum and colon no lesions were seen.
By electron microscopy, virus containing epithelial cells were found in the lower jejunum and ileum from 24 h till 16 days after infection. Infected nuclei were round and swollen and contained many virus particles. The microvilli and apical cell margins were irregular. In the terminal stage of infection the cytoplasm was protruded into the intestinal lumen and the microvilli disappeared completely. The nucleus either ruptured or was released into the lumen. Scanning electron microscopy revealed shortening of villi in the terminal jejunum of infected pigs compared to the normals.
In the intestinal contents of three piglets adenovirus particles were found. In one of these piglets also 23 nm viruslike particles were detected. These were not found in the inoculum.

Discussion

The present study shows that, under experimental conditions, the porcine adenovirus strain 6618 is a pathogen for the pig intestine. In the natural case as well as in the experimentally infected pigs the intestinal lesions were the same. Presence of intranuclear inclusion bodies in experimentally infected animals has been described, although no clinical signs of diarrhea were observed. The animals however were challanged by the aerosol inoculation in contrast to the present oronasal route of infection. Other experimental infections with the same virus strain revealed an acute enteritis. The mechanism of diarrhea can be explained by the malabsorption syndrome. This is the result of digestive failure which is caused by destruction of villous absorptive cells.
Compared to TGE and CV777 coronavirus enteritis the number of enterocytes destroyed in the present adenovirus enteritis is much lower which reflects a not so decreased villus/crypt ratio as in TGE. The number of goblet cells also is decreased which will result in a decreased production of mucus. This can implicate a decreased protection of the intestinal wall. A third factor in the arise of diarrhea can be the malabsorption of bile salts in the ileum. Bile salts stimulate the secretion in the colon epithelium.

Selected references

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