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THE PATHOLOGY OF NEONATAL ENTERITIS IN CALVES WITH OBSERVATIONS ON E. COLI, ROTAVIRUS AND CRYPTOSPORIDIUM

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Abstract

The mucosa of the small intestine of neonatal calves responds in a similar way to a variety of infectious agents. However, samples should be removed under general anaesthesia to avoid post-mortem artefacts. These include the separation of epithelium from the villous lamina propria and denudation of villous tips within a few minutes of death, and occur more rapidly in challenged animals. Pathological changes consist of blunting and fusion of the villi with a reduction of the columnar epithelium to cuboidal and occasionally squamous epithelium. A surface inflammatory exudate may be present, especially within 24 hours of challenge with enteropathogenic E. coli. Examination of several sites from the small intestine indicates a variable distribution for enteropathogens. Rotavirus is seen by immunofluorescence microscopy in epithelial cells principally in the proximal half of the small intestine, although they may occasionally be found in the distal half. By contrast, E. coli organisms adhere to enterocytes in the distal half of the small intestine. Cryptosporidia inhabit the brush border of the enterocytes enclosed within host cell microvillous membranes, principally in the distal half of the small intestine. Examination of the small intestine of neonatal calves for pathological changes and the presence and distribution of infectious agents complements bacteriological, virological and immunological techniques in the diagnosis of neonatal enteritis.

In recent years there has been a greater understanding of the pathology of neonatal enteritis in calves. This has largely been due to the experimental reproduction of diarrhoea with infections involving single agents (Mebus et al., 1971, 1973; Pearson et al., 1978a; Bellamy and Acres, 1979). Furthermore histological, immunofluorescence and transmission electron microscopic techniques have provided good correlation between the association of microorganisms with the intestinal mucosa and the presence of pathological lesions.

It is important however to distinguish between post-mortem artefacts and genuine pathological changes. Denudation of epithelium from villus tips has been described as part of the pathological process in certain virus diseases of calves (Mebus et al., 1971; Morin et al., 1974; Doughri and Storz, 1977) and samples removed from control calves did not show this change. However, post-mortem artefacts have been recognised in other species for some years (Badawy et al., 1957; Fell, 1961). When the intestine of a calf infected with both rotavirus and E. coli was sampled under anaesthesia and again from the same site after death (Pearson et al., 1978b), epithelium entirely covered the villi in the sample removed under anaesthesia, but denuded tips, similar to those described as a pathological change, were found in the sample taken after death (fig. 1). It was further shown (Pearson and Logan, 1978) that artefacts occurred as early as 3 min after death and denuded villi were present by 7 min. By contrast the mucosa of
an unchallenged calf remained intact for up to 10 min after death. These findings have since been confirmed by other workers (Hadad and Gyles, 1982). Therefore desquamation of epithelium from the villi in the absence of a polymorphonuclear cell reaction on the surface, should be considered to be artefact since such an appearance can be readily reproduced by collecting samples after death.

The small intestinal mucosa of the neonatal calf consists of long finger-like villi and when challenged by a variety of infectious agents the pathological changes are similar. The main pathological lesion consists of stunting and thickening of the villi (fig. 2). Frequently the villi are fused leading to a flat mucosa in the most severe cases (fig. 3). The epithelium covering such villi is generally cuboidal, and in rotavirus infections squamous epithelium covering the tip of some villi may be seen. Elongation of the crypts of Lieberkühn is more noticeable in calves challenged with rotavirus and cryptosporidium than in calves with E. coli infection but may reflect the survival time of the calf following challenge, since calves with E. coli infection were killed by 4 days of age (Pearson et al., 1978a). When calves are challenged experimentally with E. coli there is an early and transient polymorphonuclear leukocyte exudation into the ileal lumen. This was seen to be most prominent at 12 h, post inoculation (p.i.) in a sequential study (Pearson and Logan, 1979) and regarded as a diagnostic feature in E. coli infections in calves examined at 24 h p.i. (Bellamy and Acres, 1979).

There is an increase in mononuclear and polymorphonuclear cells in the lamina propria causing thickening of villi. Fusion of the epithelial surface of adjacent villi is frequently seen. Occasionally there is continuity of the lamina propria between adjacent villi (Pearson, 1980). By transmission electron microscopy bridges of epithelial cells between villi are clearly seen and increased desmosomal attachments between the cells appear to be the mechanism of adhesion (Pearson and Logan, 1982a).
Erosion of small areas of epithelium has been observed in both *E. coli* and rotavirus infections (Pearson *et al.*, 1978a and b) and was invariably accompanied by a polymorphonuclear leukocyte response in the lamina propria and on the epithelial surface. It could thus be readily distinguished from artefactual changes. In control calves epithelial cell loss from the extrusion zone at the villus tips was rarely seen. By contrast epithelial extrusion was readily seen in challenged calves (Pearson and Logan, 1982a). Cells were lost from the villi by extrusion of single effete cells or by forming ribbons, or rounded groups of cells prior to exfoliation into the lumen.

The association of organisms with pathological lesions has been correlated using immunofluorescent, light, scanning and transmission electron microscopy. Enteropathogenic *E. coli* possessing the K99 antigen can be seen lying on the villous surface (fig. 4) as a layer of organisms. However they are separated from the mucosa by a gap of approximately 200 nm comprised of the fimbriae of the organism and the glycocalyx of the microvilli (Bellamy and Acres, 1979; Pearson and Logan, 1982a). Rotavirus antigen is observed within epithelial cells by immunofluorescence microscopy (fig. 5) and virus particles by transmission electron microscopy (Stair *et al.*, 1973). Cryptosporidia are found in the brush border of epithelial cells (fig. 6). They occupy an apical, intracellular location, being surrounded by an envelope which is continuous with enterocyte microvilli (Pearson and Logan, 1983).

Studies on the pathogenesis of experimental *E. coli* infection (Pearson and Logan, 1979) indicates that organisms adhere to the mucosa in small numbers as early as 3 h p.i. Thereafter they increase in number and distribution, involving the lower half of the small intestine. Lesions occurred abruptly between 8 and 9 h p.i. and consisted of thickened stunted villi. By 12 to 21 h p.i. lesions were well established in association with bacterial adhesion to the mucosa.

Differences in the distribution of agents along the small intestine may influence the quantity of diarrhoea produced. In normal calves there is a net fluid resorption in the distal small intestine (Bywater and Logan, 1974), therefore pathological changes in this region probably play an important role in the quantity of diarrhoea produced. *E. coli*
are found adhering to the mucosa consistently in the distal small intestine (Moon et al., 1978) and this region is also favoured by cryptosporidia (Morin et al., 1976; Pearson et al., 1982b). In experimental studies with rotavirus, pathological changes associated with immunofluorescence against rotavirus antigen were located predominantly in the proximal half of the small intestine (Logan et al., 1979). These calves had only mild diarrhoea and this may have been a reflection of the distribution of the virus within the small intestine. Other workers however attribute no particular distribution for rotavirus (Mebus et al., 1971; Morin et al., 1976).

In conclusion, experimental studies on calves with diarrhoea have demonstrated the pathogenesis and distribution of lesions in the small intestine. Histological examination of moribund calves therefore complements microbiological and immunological techniques in the diagnosis of field cases of neonatal enteritis.

References


**Question**

*From Dr. Lecce to Dr. G.R. Pearson*

The neonatal pig’s upper third of the small intestine is far more resistant to rotaviral infection than the lower 2/3. This is just the opposite of what is seen in the calf.

**Answer**

We found similar results with experimental rotavirus in pigs.

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**Question**

*From Dr. Viso to Dr. Pearson*

Had you found any combined infection with Rotavirus and Cryptosporidia?

**Answer**

Yes. In an experiment where calves were challenged with rotavirus, only one calf had severe diarrhoea. This calf was subsequently found in a post-mortem examination to have cryptosporidia in the distal half of the small intestine.

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**Question**

*From Dr. Contrepois to Dr. Pearson*

Have you observed lesions of the abomasum in rotavirus infections as we noted in experimental infections a few years ago?

**Answer**

No.

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**Question**

*From Dr. P. Yvore (F) to Dr. G.R. Pearson (G.B.)*

In France we have observed two causes of human cryptosporidiosis. In one of these cases the strains of Cryptosporidium inoculated to young goats are pathogenic (diarrhoea).

1. Have you observed contagion from ruminant to man?
2. Have you any opinion on the possible relationships between microflora and Cryptosporidium?

**Answer**

1. No. There was a recent report from N. America on transmission of cryptosporidiosis from calves to veterinary personnel.
2. In field cases, diarrhoea mixed infections including cryptosporidia are very common.

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**Question**

*From Dr. Nabuurs to Dr. Pearson*

Are villi apart from being thickened, also shortened in *E. coli* infections?

**Answer**

Yes. *E. coli* infections cause villus atrophy in the small intestine, as do Rotavirus infections.

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**Question**

*From Dr. H.J. Greene (Ireland) to Dr. Pearson*

Can you rely on the location of lesions in the small intestine for diagnosis of field cases of bovine diarrhoea (Aetiology).

**Answer**

If there are lesions in the distal small intestine, *E. coli*, Coronavirus or Cryptosporidia may be responsible. If the lesion is in the proximal small intestine, the infection is not an *E. coli* but probably a Rotavirus is the cause.