Villous Hypoplasia of Small Intestine in Neonatal Foals

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The small intestine of ungulates, particularly in the immediate post partum period, plays an important role in acquisition of maternal immunoglobulin from the colostrum of the dam through the immature mucosal epithelial cells [3]. Recently, a clearer understanding of the structural changes occurring in the small intestine has been required in neonatal foals affected with malabsorption of colostral immunoglobulin [5, 8], since such changes have received little attention previously [4, 11].

This note describes hypoplastic villi of the small intestine in neonatal foals.

The involved foals were three thoroughbreds and born at full-term after normal delivery (Table 1). They were weak, and of low birth weight (30-40 kg) in comparison with the mean weight for full-term thoroughbred foals (49.6 ± 0.45 kg) [10]. At autopsy, the length of the small intestine in each of the three foals was found to be slightly shorter than that of 12 aborted fetuses and 19 necropsied foals. The intestinal wall was thin throughout, but no structural abnormalities were noted on the surface of the mucosa and serosa.

Histologically, the villi were markedly shortened throughout the entire length of the small intestine in comparison with that of four age-matched foals without intestinal structural abnormalities (Figs. 1-3). The intervillous spaces were

wide, and the distribution of the villi was sparse (Figs. 3 and 4). Most of the villi were short and finger-shaped with conical tips or occasionally tongue-shaped. The villous epithelium consisted of cuboidal and columnar absorptive cells with

Fig. 1. Measurements of small intestinal mucosa in neonatal foals.

■ : Foals with villous hypoplasia.

□ : Foals without structural abnormalities of the intestine.

Bar : Standard deviation

D : Duodenum

UJ : Upper jejunum

MJ : Middle jejunum

LJ : Lower jejunum

I : Ileum

Table 1. Foals examined

Case	Breed	Sex	Age	Cause of death	Serum immunoglobulin ^{a)}	Body weight	Mare age
1	T ^{b)}	M ^{c)}	16 h	Euthanasia (scoliosis)	85	40	4
2	T	F ^{d)}	17 h	Euthanasia (twin)	280	30	14
3	T	M	60 h	Euthanasia (hydrocephalus)	170	37	

a) Measured by single radial immunodiffusion test (Miles Laboratories, Inc., Illinois, U.S.A.).

b) Thoroughbred.

c) Male.

d) Female.

Fig. 2. Villi of middle jejunum from a neonatal foals aged 10 hr without intestinal structural abnormalities. $\times 75$.

Fig. 3. Villi of middle jejunum of Case 3. The villi are short with conical tips. $\times 75$.

Fig. 4. Middle jejunum from Case 2 showing villous hypoplasia. HE stain. $\times 75$.

Fig. 5. A shortened and distorted villus with shallow crypt from Case 3. The lamina propria around the crypt is poor in loose connective tissue. HE stain. $\times 300$.

some goblet cells (Fig. 5). The nuclei were located in the upper part of the cytoplasm (Cases 1 and 2), or in the lower part (Case 3). A very sparse glycocalyx was present over the tips of the microvilli (Fig. 6-A), which showed a slightly sparse distribution (Fig. 6-A). In Case 3, the microvilli were arranged irregularly, and variable in size (Fig. 6-B). In Cases 1 and 2, invagination of the plasma membrane between the microvilli, apical complexes of anastomosing tubules and/or vesicles primarily consisting of non-dilated tubules, and small irregular-shaped droplets with high electron density were seen in the supranuclear regions (Fig. 6-A). In Case 3, large vacuoles and vesicle clusters containing amorphous substances of low electron density were observed in the supranuclear region. Most mitochondria were round and large (Fig. 6-A), and located chiefly in the infranuclear region. The rough endoplasmic reticulum-Golgi apparatus was poorly developed. The crypts of Lieberkühn were very sparsely distributed in the lamina propria mucosae, and narrow and shallow (Figs. 4 and 5). The basophilic columnar epithelial cells of the crypts were narrow and crowded (Fig. 5). Their mitotic activity was low. Immediately below the epithelia of the villi and crypts, a thick argyrophilic basal membrane was found (Fig. 7). The central lacteals and capillaries were poorly developed in the villous core. The villous lamina propria and the lamina propria mucosae around the intestinal crypts were also poorly developed (Fig. 5). The loose connective tissue in the lamina propria was composed of mesenchymal tissue of an embryonal type. Masson's trichrome stain revealed fewer collagen fibers in the mesenchyme, while reticulin meshes were evident by Wilder's reticulum stain (Fig. 7). The lamina muscularis mucosae, and the inner circular and outer longitudinal layers of the tunica muscularis were extremely thin (Fig. 4). Development of the connective tissues and blood vessels in the tunica submucosa was extremely poor (Fig. 4).

In general, short intestinal villi occur due to

various causes [1]. Familial enteropathy or congenital microvillous atrophy, which is characterized by short villi, has been reported in human infants in recent years [2, 9]. The present cases were characterized by poor development of all the intestinal wall including the villi. Namely, both epithelial and mesenchymal tissues were involved, suggesting to be hypoplastic change occurring during the fetal period. Moreover, the thick argyrophil basement membranes and mesenchymal cells of embryonic type in the lamina propria resemble those in the fetal intestinal villi at the mid to late stage of intrauterine life (unpublished data). The connective tissue has a large proportion of argyrophilic fibers in the fetuses, while it has large quantities of collagen fibers in adults [6]. The features of the basement membrane and connective tissue in the lamina propria in the present cases suggest the developmental retardation of the villous interstitium.

It is accepted that development and differentiation of the epithelium during the fetal period require the presence of the mesenchyme, and are influenced by its quality and quantity [1, 6, 7, 12]. The immature connective tissue observed in the lamina propria of our cases would probably have been responsible for the morphological changes of the villi.

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Fig. 6-A. Apical portion of an absorptive epithelial cell in middle jejunum from Case 2. Microvilli are sparsely distributed. Invaginations of the plasma membrane (arrows) and apical complexes, consisting mainly of non-dilated tubules (arrowheads). $\times 12,000$.

Fig. 6-B. Microvilli showing irregular length. Case 3. $\times 10,000$.

Fig. 7. A villus and crypts from Case 3. Lamina propria consisting chiefly of argyrophilic fibers, resembling the immature connective tissue in the fetal period. Wilder's reticulum stain. $\times 340$.

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要 約

新生仔馬の小腸にみられた腸絨毛低形成(短報): 及川正明・吉原豊彦・兼子樹広・吉川 堯¹⁾(日本中央競馬会競走馬総合研究所, ¹⁾北里大学獣医畜産学部獣医病理学教室)——新生仔馬の剖検例中小腸の腸絨毛の矮小化の著しい3例が偶発的に見出された。これら腸絨毛の著しい矮小化は従来報告されてきた各種疾患における腸絨毛萎縮像とは相違し, 胎生期における上皮及び間葉系組織の発育障害に基づく腸絨毛低形成像と解された。