HISTOPATHOLOGIC CHANGES OF THE SMALL INTESTINE MUCOSAE IN PIGLETS WITH ANOXIA AND PARTURITION HYPOXIA

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Abstract: *The experiment took place a commercial closed circuit pig farm. From 10 still born piglets, squashed by sows and sacrificed in the first 24 hours of life, duodenum, jejunum and ileonum samples were harvested for the histopathological exam. The tissue samples were prepared with the classic histological techniques and were stained with haematoxylin-eosin (H.E.). 10 villi and crypts were measured with the analysis and image capturing programme of the Olympus C x 41 microscope. The vascular changes were described, as well as the changes of the villosity epithelium and of the enterocyte and goblet cell aspect, and the villi mucous integrity. Glycaemia, measured before the sacrifice with a Accucek device, appreciated the parturition hypoxia level. Piglets anoxic at birth (still born) presented at the small intestine mucosae level vascular lesions, expressed by blood and lymphatic stasis, edema in lamina propria and subepithelial in the duodenum, jejunum and ileonum. The enterocytes from the jejunum and ileonum present inversed polarity, and the other mucus secreting cells are hyperplasic. In the three segments of the small intestine, the villi atrophy took place, their height representing 49.5 % in the jejunum and 35.57 % in the ileonum in comparison to the live and healthy newly-borns.During the life interval at birth – 24 hours in piglets suffering from parturition hypoxia, the mucosae lesions were also of vascular type, manifested by local ischemia, followed by reperfusion. In relation to the ischemia seriousness in the small intestine mucosae, tissue edema, villi atrophy up to amputation, villi epithelium denudation and ulcers in the criptae area. Also, enterocyte hypertrophy with special nuclei migration, hypertrophy and hyperplasia of mucus secreting cells; in the lamina propria, subepithelial and in the villi axe there was a massive leukocyte infiltration. The villi height was reduced in the three segments of the small intestine, the percentage of the villi height being under 400 μm.*

Key words**:** *piglets, anoxia, parturition hypoxia, villi, criptae*

INTRODUCTION

The losses caused by mortality in suckling piglets range from 5 to 25 %, of which about 80 % are still born, hypothermic, starved and crushed.

Parturition hypoxia causes adrenaline and noradrenaline discharge followed by hyperglycaemia and vascular change in the small intestine mucous membrane, resulting in an attack on the mucous integrity (3).

The present experiment intends to evaluate, from a histopathological point of view, the morphological changes of the small intestine mucous in anoxic piglets and piglets suffering from parturition hypoxia, during their first day of life.

MATERIAL AND METHODS

The experiment took place at a commercial closed circuit pig farm. From 10 still born piglets, squashed by sows and sacrificed in the first 24 hours of life, duodenum, jejunum and

ileonum samples were harvested for the histopathological exam. The tissue samples were prepared with the classic histological techniques and were stained with haematoxylin-eosin (H.E.). 10 villi and crypts were measured with the analysis and image capturing programme of the Olympus C x 41 microscope.

The vascular changes were described, as well as the changes of the villosity epithelium and of the enterocyte and goblet cell aspect, and the villi mucous integrity. Glycaemia, measured before the sacrifice with a Accucek device, appreciated the parturition hypoxia level.

RESULTS AND DISCUSSIONS

No major differences were noticed regarding piglet ages and small intestine segments, the dominant aspect being represented by vascular changes. Thus, in the case of still born piglets, we appreciate that the piglets died of asphyxia in the uterus, as a result of the premature tearing of the umbilical cord.

Surprising is the gravity of the vascular lesions generated by sanguine and lymphatic stasis, edema in the lamina propria and under-epithelial, present in the duodenum as well as in the jejunum and ileonum. These vascular lesions appear uniformly in the three small intestine segments.

What is more, in the jejunum and the ileonum, we observed a reversed enterocyte polarity and goblet cells hyperplasia. Also, a non-uniform atrophy of villosities was observed in all small intestine segments.

The medium villosity height was observed in the duodenum, jejunum and ileonum, 274.3±53.5 μm, 317.8±83.9 μm and respectively 206.3±82.57 μm, and the criptae depth of 74.68±5.85 μm, 54.84±5.94 μm and respectively 55.6±4.02 μm. Comparatively, at birth, villosity height in live piglets was of 642 μm and 580 μm for the jejunum and ileonum, and the criptae depth of 96.4 μm and respectively 86.9 μm in the jejunum and ileonum (1). The result is a villosity height reduction to 49.5 % in the jejunum and of 35.57 % in the ileonum (figure 1). The criptae depth was reduced to 56.89 % in the jejunum and to 63,98% in the ileonum (figure 2). Proportion villi/criptae is presented in the table 1.

Table 1

Proportion of villi/criptae in small intestine segments between 0 and 24 hours

Age (hours)	Proportion of villi/criptae (μm)		
	Duodenum	Jejunum	Ileonum
Still born piglets (0)	3,67	5,79	3,71
1-24	3,03	5,60	3,94
Reference values			
0		6,65*	6,67*
0		8,02**	
20		7,23**	

* - Burrin, 2000 (2); ** - Biernat şi col., 2000 (1).

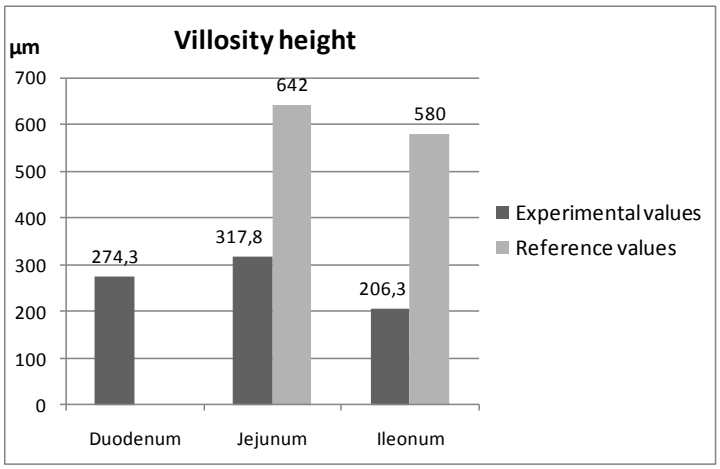

Figure 1: The average villosity height and reference values comparison in small intestine segment

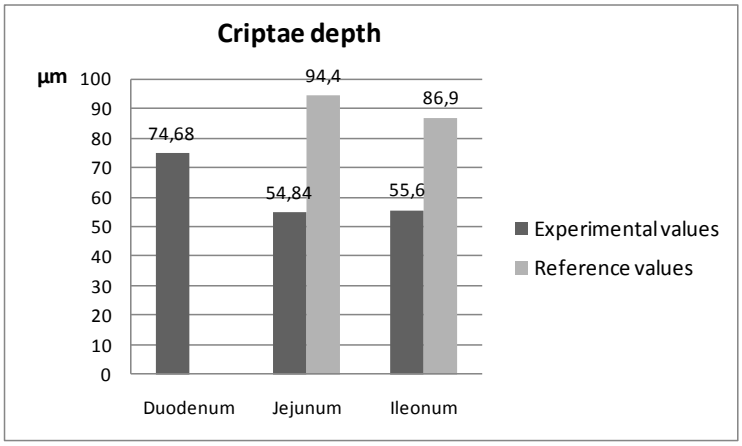

Figure 2: The average criptae depth and reference values comparison in small intestine segment

Anyway, villosities with a height < 400 µm represented 80 %, 71.19 % and 91.66 % from the villosity total measured in the duodenum, jejunum and ileonum. It was observed that relatively short lived ischemia triggers major morphological changes in the small intestine mucus, the ileonum being the most affected. A parturition hypoxia effect was appreciated in piglets up to 24 hours of age.

In one hour old, crushed piglets, with gastric content, proving the achievement of the first sucking, but with reduced viability, edema was observed in villosities, lymphatic stasis and discreet villosity atrophy in all three segments of the small intestine. Also, there was a change in enterocyte functional polarity, through apical nuclei migration (figure 3, 4 and 5).

At a 4 hour age, parturition hypoxia was maintained by the glycaemia value: 188 mg % compared to normal glycaemia at the same age, about 60 mg %; glycaemia increase was

produced as a result of adrenaline rush due to parturition hypoxia. Although the piglet was born with 1.52 kg, viability was reduced due to parturition hypoxia; in many cases these piglets do not manage the first sucking. The histopathological lesions noticed ranged from epithelium erosion to mucus ulcer; some villosities detached from the mucus, others presented stasis and edema. A small number of pro-inflammatory leukocytes appear in lamina propria; the described lesions are present in all small intestine segments (figure 6, 7 and 8).

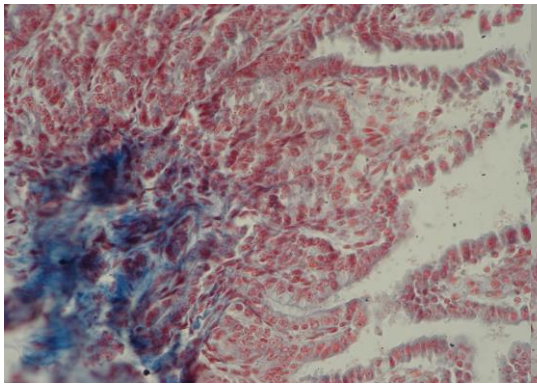

Figure 3 : Duodenum mucosae in still born piglet. Unballanced atrophy of the villosities, edema and lymphatic stasis (conjunctive ax with central chilifer dilatations)

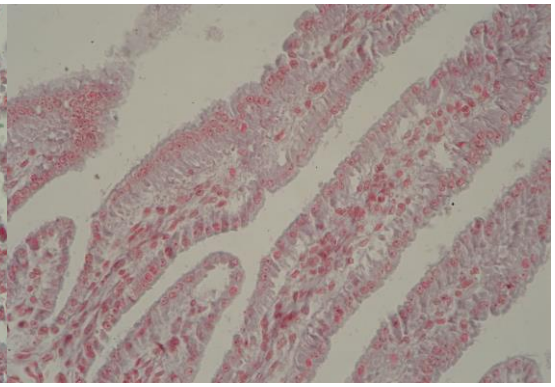

Figure 4: Jejunum mucosae in still born piglets. Unequal atrophy and marked dilatation of the central chilifer with most villosities; reversed functional polarity of the enterocytes

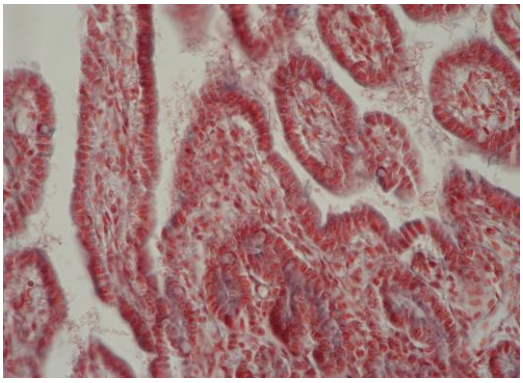

Figure 5: Ileonum mucosae in still born piglets. Variable height villosities, with a change in the functional polarity of the enterocytes

At the age of 11 hours, villosity atrophy is discreet in all small intestine segments, but the lymphoplasmocitary infiltrate is well represented in the lamina propria and in the villosity axe; the villosity edema is discreet. Calciform cells are hyperplasic and tend to replace enterocytes (absorbent cells) especially in the ileonum. The examined piglet came from a sow with mamma edema; its reduced viability caused inanition (empty stomach). The glycogen and body fat deposits were consumed, resulting in a1 kg body weight at killing.

At the age of 21 hours in piglets with a 1.02 kg body weight at birth, and a full stomach prior killing, the lesions were vascular: tissue edema in the villosity axe,

lymphoplasmacitic infiltrate abundant in the lamina propria; hyperplasic calciform (goblet) cells.

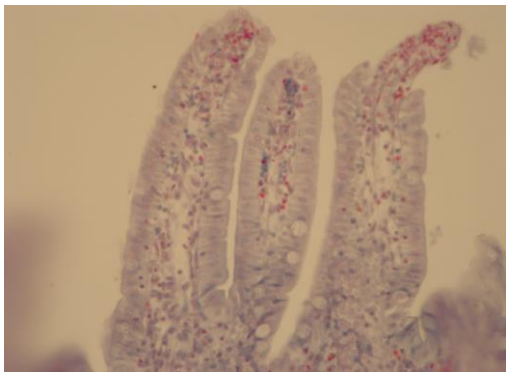

Figure 6: Duodenum mucosae in 4 hour old piglets with parturition hypoxia. Atrophy and duodenum mucosae ulceration with some villosity amputation; epithelium denudation on extended areas of the mucosae surface

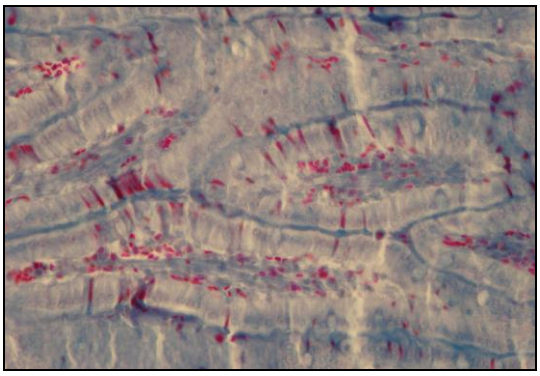

Figure 7: Jejunum mucosae in 4 hour old piglets with parturition hypoxia. Stasis at mucosae level with the extension of the villosity axes through edema and stasis; hyperplasia of the lymfoidal tissue associated with the mucosae and the formation of defused and nodular lymfoidal aggragates in the submucosae; lymfoidal infiltrations fragmentize the muscle mucosae

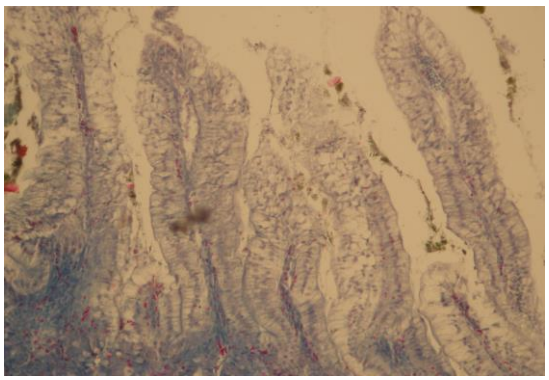

Figure 8: Ileonum mucosae in 4 hour old piglets with parturition hypoxia. Stasis at mucosae level with the extension of the villosity axes through edema and stasis; hyperplasia of the lymfoidal tissue associated with the mucosae and the formation of defused and nodular lymfoidal aggragates in the submucosae; lymfoidal infiltrations fragmentize the muscle mucosae; some lymfoidal follicles contain germinative centres; microerosions of the epithelium covering the villosities and the presence of the biliary pigments in the intestinal lumen.

During the first 24 life hours general lesions generated by parturition hypoxia were noticed, predominantly vascular lesions at the small intestine mucus level, which generate local ischemia, probably followed by reperfusion. This affects the morphological integrity of the small intestine mucus, varying, according to the hypoxia gravity, from tissue edema and slight villosity atrophy to villosity epithelium denudation, mucus ulceration in the criptae area and villosity amputation.

A viability score reduction in piglets also reduces the colostrum ingestion, heightening lesion gravity.

Alongside with vascular lesions, up to 24 hours of life, enterocyte hypertrophy and apical nucleus migration occur, as well as calciform cell hypertrophy and hyperplasia and massive leukocyte infiltration in the lamina propria, in villosities and under-epithelial. All these heighten vascular and close epithelium junction permeability, triggering diarrhoea.

All in all, villosity is reduced, average values amounting to 233.05±56.72 μm, 337.66±73.89 μm and 203.68±83.82 μm in the duodenum, jejunum and respectively ileonum. Compared to 20 hours old healthy piglets, observing a villosity height of 875 μm in the jejunum, in hypoxic piglets, villosity height represents only 38.59 %. Further more, the proportion of jejunum villosities with a length < 400 μm was of 64.68 %, between 400-600 μm, 34.6 % and > 600 μm, 0.72 %. In the duodenum, the proportion of villosities < 400 μm was of 92.58 % and between 400-600 μm, 7.42 %. In the ileonum 80 % of the villosities observed a length < 400 μm, 15.23 % between 400-600 μm and 4,77 % over 600 μm.

CONCLUSIONS

From a histopathological point of view, piglets anoxic at birth (still born) presented at the small intestine mucosae level vascular lesions, expressed by blood and lymphatic stasis, edema in lamina propria and subepithelial in the duodenum, jejunum and ileonum. The enterocytes from the jejunum and ileonum present inversed polarity, and the other mucus secreting cells are hyperplasic. In the three segments of the small intestine, the villi atrophy took place, their height representing 49.5 % in the jejunum and 35.57 % in the ileonum in comparison to the live and healthy newly borns.

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