Enterocyte ultrastructural alterations following intestinal obstruction in rats[1]

Alterações ultra-estruturais do enterócito após obstrução intestinal em ratos

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ABSTRACT
Purpose: To analyze the small intestinal mucosa ultrastructure, and to characterize the enterocyte lesion severity caused by mechanical intestinal obstruction combined or not with an ischemia of the mesenteric marginal vessels arch. **Methods: I**t was used 47 Wistar rats divided into 4 groups as follows: Group 1- Control rats (C), Group 2- sham rats (S), Group 3- rats submitted to intestinal obstruction excluding marginal vessel (OEV), Group 4- Intestinal obstruction including marginal vessels (OIV). Rats of groups 3 and 4 were allotted into two subgroups for the removal of small intestinal tissue samples, one at the proximal (P), and the other at the distal (D) segments from the obstruction site. Samples of groups 2, 3, 4 were obtained 24, 48, and 72 hours after post operation care. Small intestinal tissue samples of group 1 were excised following laparotomy. Imaging in Light and Transmission Electronic Microscopy were used for morphological and morphometric studies. The results were analyzed by using the ANOVA and Newman-Keuls tests. **Results:** No irreversible lesion was observed. In the 24 hours microvilli volume of group 3 turned down at the proximal site henceforth enlarging very slowly within the next 72 hours. At the distal site significant microvilli shrinkage was observed up to 48 hours. Then they recovered their volume after 72 hours. In the 24 hours microvilli volume of group 4 grew twice in comparison with the microvilli of group 1 rats but after 72 hours there was drastic volume shrinkage, shape alterations, and severe flatness, especially in the distal segments of the obstruction site. **Conclusions:** Terminal ileum mechanical obstruction with mesenteric marginal arch ischemia led to reversible ultrastructural alterations after 72 hours, and the injury is proportional to the persistence of the obstructive process. Furthermore the mesenteric vessels of the marginal arcade play an important role in the maintenance of mucosal integrity, when such obstructive disorder is present.
Key words: Microscopy Electron, Transmission. Intestinal Obstruction**.** Intestinal Mucosa. Intestine, Small. Animal Experimentation. Rats.

RESUMO
Objetivo: Analisar as alterações ultra-estruturais da mucosa do intestino delgado e caracterizar a severidade das lesões causadas por uma obstrução intestinal mecânica, associada ou não a isquemia da arcada marginal mesentérica. **Métodos:** Foram utilizados 47 ratos, da linhagem Wistar, distribuídos em quatro grupos, da seguinte forma: Grupo 1 - Controle (C), Grupo 2- Simulação (S), Grupo 3- Ratos com obstrução intestinal sem inclusão de vaso marginal (OEV), Grupo 4 - Obstrução intestinal com inclusão de vaso marginal (OIV). Os animais dos grupos 3 e 4 foram redistribuídos em dois subgrupos com coleta de amostras do intestino delgado, à montante (P) e à jusante (D) do ponto de obstrução. Nos grupos 2, 3 e 4, as amostras foram colhidas com 24, 48 e 72 horas de pós-operatório. No grupo 1, este material foi retirado após laparotomia. Realizaram-se estudos morfológicos e morfométricos dos microvilos através das Microscopias Óptica e Eletrônica de Transmissão. Os resultados foram analisados mediante os testes estatísticos de ANOVA e Newman-Keuls. **Resultados:** Não foram observadas lesões irreversíveis. No grupo 3 com 24 horas, o volume dos microvilos diminuiu, à montante, com discreto aumento em 72 horas. Á jusante, houve redução significante até 48 horas, com recuperação em 72 horas. No grupo 4, o volume dos microvilos quase dobrou em relação ao grupo 1, com 24 horas, mas reduziu-se, drasticamente, em especial à jusante, com 72 h de evolução, apresentando deformidade e achatamento severos, achados estes estatisticamente significantes. **Conclusões:** A obstrução intestinal mecânica do íleo terminal, associada ou não a isquemia da arcada mesentérica marginal, causa alterações ultra-estruturais reversíveis dos enterócitos, cuja gravidade é diretamente proporcional à duração do processo mórbido obstrutivo, até 72 horas de evolução. Aduz-se que, os vasos mesentéricos da arcada marginal exercem um papel relevante na preservação da mucosa intestinal, na presença destes quadros obstrutivos.
Descritores: Microscopia Eletrônica de Transmissão. Obstrução Intestinal. Mucosa Intestinal. Intestino Delgado. Experimentação Animal. Ratos.

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Introduction

Crucial value for the surgeon is the knowledge of the pathophysiology of the mechanical intestinal obstruction. This disorder is one of the most incident and prevalent in the context of the acute abdomen. Process understanding is imperative in the face of experimental data disparity including contradictory morphological results, and also considering reserved prognostics for ischemia or enteromesenteric vascular impairment.

Medical literature shows clearly that 12 to 24 hours of simple mechanical intestinal blockade ends in minor morphological intestinal wall alterations manifested by edema, and small blood stasis[1-8]. On the other hand intestinal mechanical obstruction plus mesenteric arterial occlusion causes several injuries with brush border displacement, nucleus pinocytosis, sometimes necrosis of the intestinal wall[9-11].

However, scientific works published in known reviews, describing enterocyte irreversible alterations and total necrosis of the intestinal layers in rats submitted to sole mechanical intestinal obstruction for minutes or few hours were catching us with surprise[6,12-17]. Based on their presumptions, then severe mucosal injury should be recognized in all cases of intestinal obstruction, even in the absence of vascular lesions; and an extensive radical surgery ought to be made from the initial stage of this morbid process. In fact the patients' outcome after an intestinal obstruction treatment does not endorse such findings. Such publications are neither in accordance with the day-to-day experience of surgeons nor with the published anatomo-pathological reports and therefore cannot be used to outline any clinical or surgical approaches.

Most of the experimental studies dealing with intestinal ultrastructural changes and vascular occlusion do not evaluate the role of the mesenteric marginal arch. Thus the lack of information about this particular condition and its relevance to the pathophysiology motivated us to develop this research.

Methods

Forty-seven adults Wistar male rats (*Rattus norvegicus albinus*), weighing about 250g, were used. They were supplied by the Center of Experimental Surgery of the Faculty of Medicine - Federal University of the Rio de Janeiro, placed in suitable cages, maintained in constant temperature with a 12 hours circadian cycle. They were fed with industrial standard diet formulation and water *ad libitum*.

This research had the approval of the Ethics Committee for the Use of Laboratory Animals of the Faculty of Medicine of Federal University of the Rio de Janeiro and followed the Council of International Organization of Medical Sciences ethical code for animal experimentation.

The 47 rats were randomly divided into 4 groups: Group 1- Control (C) - normal rats, n = 4; Group 2- sham rats (S), n = 15; Group 3- rats submitted to intestinal obstruction excluding marginal vessel (OEV), n = 14, Group 4- Intestinal obstruction including marginal vessels (OIV), n = 14.

Rats of groups 2,3, 4 were allotted into three subgroups with 4 to 6 animas for each one in accordance with the survival post operatory intervals: 24, 48, 72 hours.

After 12 hour fasted, intraperitoneal anesthesia with 0.5 mg/kg Ketamine hydrochloride and 0.15 mg/kg Xylazine was used. Ileum samples from group 1 were taken off after 72 hours of observation. Laparotomy with only terminal ileum manipulation was done in group 2 sham rats; and samples were withdrawn at 24, 48, 72 hours intervals in the subgroups of five animals. In group 3, the external mesenteric wall of the terminal ileum was completely tied up at a 5 cm range from the ileocecal junction with a 4-0 cotton thread; taking care to exclude the marginal mesenteric vessel (OEV). In this group four rats were-operated within 24 hours, four others within 48 hours and the remaining six within 72 hours. A careful abdominal inspection was performed. In group 4 the procedure was the same as in group 3 with the exception that the marginal mesenteric vessel was also tied (OIV). After the surgical procedure, animals were kept on 12 hours with water *ad libitum* followed

Before the excision of the intestinal fragments, the peritoneal cavity was moistened with 0.09% cold physiological solution. Earlier than samples fixation, 1 cm extension of the intestinal wall, proximal and distal of the ligature point was cut out, washed and cleared of debris and accumulated intestinal organic material.

It was performed both morphological and morphometrical analysis of the microvilli by using Light (LM) and Transmission Electronic Microscopy (TEM), respectively with 10 and 40 times and 7.000 and 30.000 times of magnification.

Samples of distal ileum were fixed in formaldehyde, dehydrated in ethanol, embedded in paraffin, sectioned parallel to the villus-crypt axis (5μm), stained with hematoxylin and eosin and finally examined by light microscopy at 40X and 100X magnifications. The presence of inflammatory cell infiltration, mucosal swelling, villi edema or disarrangement was graded from light to severe injury. At least five measurements were made on each fragment.

The method of the Ultrastructure Hertha Meyer Laboratory of the Biophysical Institute was taken as a basis in transmission electronic microscopy (TEM) as follow.

Intestinal transversal cuts less than 2 mm thin were processed according to the protocol described below:

a. Immersion in Karnowsky solution (4% paraformaldehyde + 1% glutaraldehyde in a 0.1 M sodium cacodylate buffer) for 2 hours at least

b. Triangular sample slices were cut as to get an uncovered intestinal mucosa when embedded.

c. Tissue were washed three times in 0.1 M sodium cocadylate buffer

d. Post fixation in 1% osmium tetroxide mixed with 0.8% potassium ferrocianyde and 5 mM calcium chloride in 0.1 M cacodylate buffer for 30 minutes at room temperature.

e. Three washes in 0.1 M sodium cacodylate buffer

f. Tissues were dehydrated in ascending grades of acetone (30%, 50%, 70%, 90% and 100%) three times lasting ten minutes each.

g. Tissues were infiltrated in a 1:2 mixture of resin and Epon/Acetone for 2 hours; samples were left overnight in a 1:1 proportion Epon/acetone and finally soaked in fresh Epon for 8 hours.

h. Tissue embedment in Epon for casting and polymerization in 60°C temperature for 48 hours.

i. 60 to 70 nm cuts of embedded tissue with the Reicher microtome were caught in 300 mesh copper grids.

j. Tissue was bathed in aqueous solution of 0.5% uranyl acetate for 1 hour added to lead citrate pH of 11.5 for the necessary contrast.

K. Grid drying for 24 hours.

Microphotographies were taken with a Zeiss 900 microscope. A total of 250 photographies with 7 thousand times and 30 thousand times magnified images were obtained. The obstructive process was standardized as follows: minor lesion

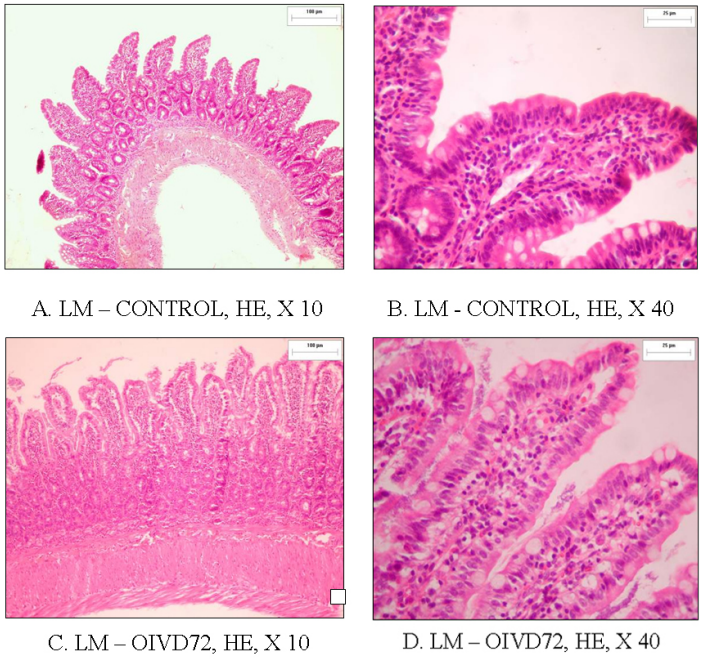
characterized by the loss of microvilli regularity, presence of slight cellular swelling, terminal web structure thickness, Golgi complex and endoplasmic reticulum swelling and finally finding of dense bodies. Moderate lesion met the following requirements: outstanding edema, shortening and breakdown of the integrity of the microvilli, chromatin condensation next to the nucleus membrane, quantitative mitochondrial degradation and increase of dense bodies. Heavy injury was identified by the presence of apical cellular membrane blebs, severe alteration of the endoplasmic reticulum, significant increase of the terminal web structure thickness, severe microvilli abnormality with shortening.

Microvilli mean volumes were evaluated by stereological studies and analyzed through the statistical ANOVA test, and the Newman-Keuls test for multiple comparisons was used. Fifty microvilli length and width for each subgroup were measured, magnified 30 thousand times and transformed into volume units ($10^3 \mu m^3$). The difference among normal and pathological samples were considered significant for statistical values of $p < 0.05$.

Results

Light microscopic (LM) findings

There were no morphologic differences between groups 1 and 2 samples. In group 4, slight to moderate swelling of the intestinal mucosa were seen at the segments proximal to the site of the obstruction but an intense edema of the lamina propria, flattening of the villi and vacuolization of the epithelial cells could be observed (Figure 1).



A. LM – CONTROL, HE, X 10 B. LM - CONTROL, HE, X 40

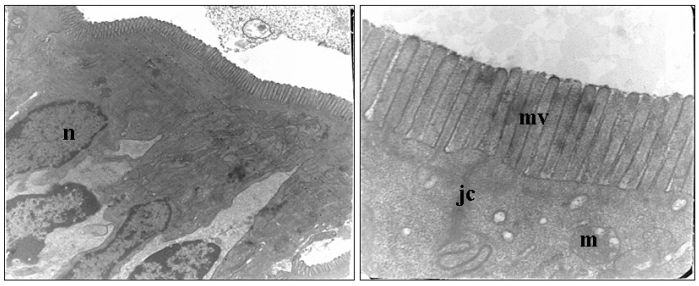
C. LM – OIVD72, HE, X 10 D. LM – OIVD72, HE, X 40

FIGURE 1 - Light Microscopy, HE, groups 1 and 4. (Control and OIVD72) – In Control group mucosal integrity is seen in contrast to group 4 with a severe inflammation of the intestinal mucosa with vacuolar degeneration, nuclear deformity and both architectural shortening and distortion of the villi

Electron microscopy findings

All animals survived the full 3 day period of total obstruction. At the third day it was observed distention of the segments proximal to the obstruction point with a diameter twice bigger than the distal ones.

In all the time intervals for group 2, the results were identical to group 1 for the enterocyte lumen surface, the microvilli, the nucleus, the chromatin, the junction complex, and the intercellular membranes (Figure 2).

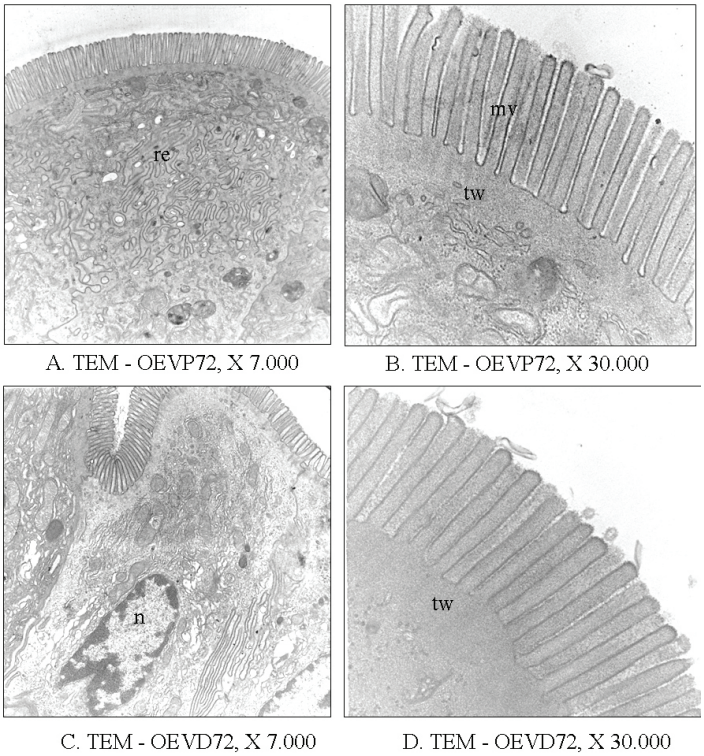


A. TEM - CONTROL, X 7.000 B. TEM - CONTROL, X 30.000

FIGURE 2 - TEM photomicrography, Control group – Integrity of the of the enterocyte ultra structure with the standard morphologic features of the microvilli (**mv**), nucleus (**n**), mitochondria (**m**) and junctional complex (**jc**)

No significant alteration was observed at group 3 (OEV24) up to 24 hours, except a slight edema.

Slight cellular swelling, and curtailed lumen sinuosity were noted at the proximal (OEVP48) and distal (OEVD48) segments from the intestinal knot after 48 hours in rats of group 3. Moderate cellular and terminal web space edema take place at the proximal (OEVP72) segments within 72 hours. At distal segments (OEVD72), positioned after the site of the obstruction, plentiful lipid containing vacuoles were exhibited, enterocyte nuclei were more roundish, and associated to condensed chromatin next to the nuclei membranes. However their regular shape microvilli base lines were not as straight as the corresponding ones at proximal segment (Figure 3).

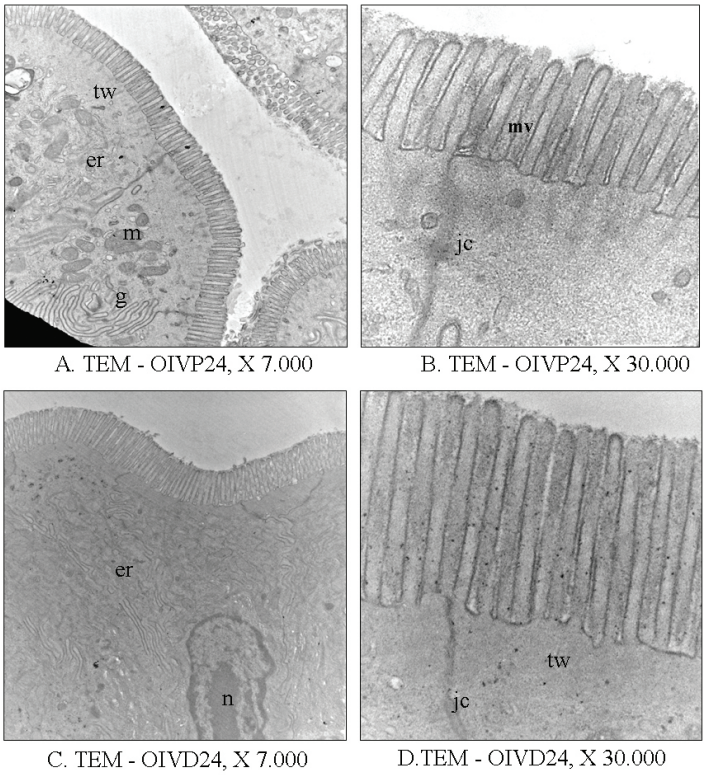


A. TEM - OEVP72, X 7.000 B. TEM - OEVP72, X 30.000

C. TEM - OEVD72, X 7.000 D. TEM - OEVD72, X 30.000

FIGURE 3 – TEM photomicrography, group 3. A and B, (OEVP72) - Cellular and endoplasmic reticulum edema (**re**), preserved microvilli (**mv**) –C and D (OEVD72) – Less dense cytoplasm, Nucleus chromatin next to nucleus membrane (**n**), discreet increase of the terminal web space (**tw**)

In group 4, at 24 h proximal (OIVP24) and distal (OIVD24) enterocyte apical plasmatic membranes slightly lost their sinuosity due to cellular swelling; microvilli base lines were not so regular, and their extremities were not so clearly seen. The terminal web was thicker; the Golgi complex and the endoplasmic reticulum were swollen; small vacuoles of lipidic nature, and dense bodies could be seen in the enterocyte cytoplasm of the distal segments (Figure 4).

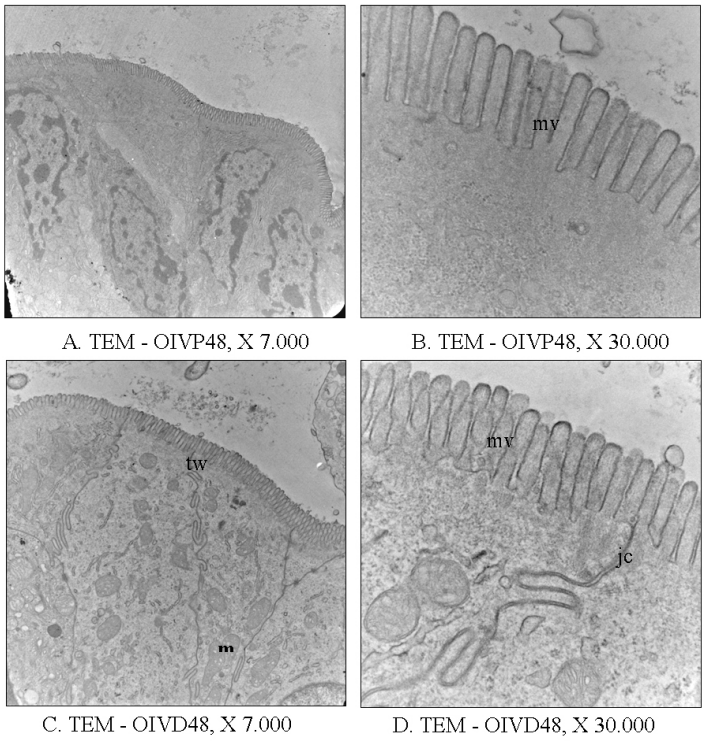


A. TEM - OIVP24, X 7.000 B. TEM - OIVP24, X 30.000

C. TEM - OIVD24, X 7.000 D.TEM - OIVD24, X 30.000

FIGURE 4 - TEM photomicrography, group 4, A and B (OIVP24) and C e D (OIVD24). Altered microvilli base line symmetry (**mv**), slight cellular edema and enlargement of the terminal web space (**tw**), golgi complex (**g**), endoplasmic reticulum (**er**), and junctional complex (**jc**) without abnormalities

At 48 h proximal (OIVP48) as much as distal (OIVD48) samples, it was seen edema and loss and deformity of cellular membrane, decrease of microvilli number notably at distal segment (after the point of obstruction). The terminal layer was thicker and could be more clearly seen than in the other groups of rats. The lengthened nuclei showed chromatin condensation next to the nucleus membrane. Swollen mitochondrions were seen, roundish, with loss of density, and with pale.cristae inside. Apparently the junctional complex was not altered; basolateral membrane invaginations were less tortuous. In the distal samples of intestinal segments moderate to severe damage were seen including various vacuoles of lipid nature and excessive dense body (Figure 5).



A. TEM - OIVP48, X 7.000 B. TEM - OIVP48, X 30.000

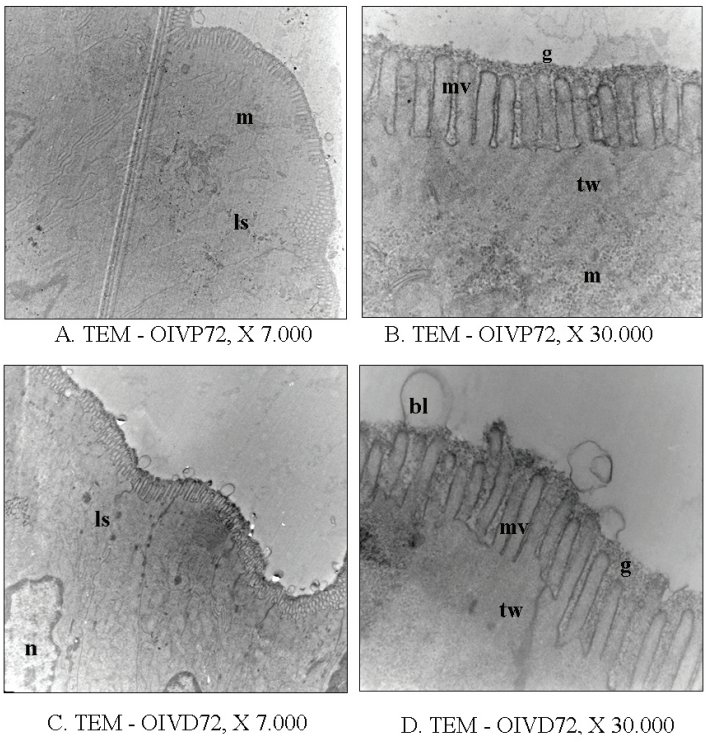
C. TEM - OIVD48, X 7.000 D. TEM - OIVD48, X 30.000

FIGURE 5- TEM photomicrography, group 4 -A and B (OIVP48): pronounced microvilli shrinkage and deformity (**mv**); C and D (OD48): cytoplasm edema confounding with the terminal web (**tw**), swollen mitocondrions (**m**), and outstanding deformity of microvilli (**mv**)

At 72 h proximal (OIVP72) and distal (OIVD72) alterations as pronounced cellular edema, heavy terminal web thickening and sparse, scattered and shortened microvilli were observed. Enhanced bacterial lay down in the glycocalyx was at a higher degree in the distal segment between the microvilli, reaching the base lines or in other words the apical cytoplasmic membrane. Important decrease of the cytoplasmic density, and condensed chromatin next to the membrane of a still visible nucleus were found. Microvilli base line was very atypical, chiefly at distal segments. The intercellular space lost its sinuous characteristic on account of the cellular swelling. Mitochondrions were enlarged, and had misshaped, vague cristae. For the first time structures that could be associated with the impairment of mitochondria function such as abnormal dilatation of apical cellular membranes resembling pseudopods or blebs, derived from the disordered endoplasmic reticulum were noticed. This description is equivalent to a severe structure injury (Figure 6).

Morphometry findings

The morphometry was done by stereological studies calculating the mean value of the microvilli volumes for each group, and in the different times. Comparison between all groups showed differences statistically significant ($p < 0.05$) except as follow: C vs. OEVP48; S72 vs. OIVP48; S72 vs. OEVD72; OEVP24 vs. OEVD24; OIVP24 vs. OEVD24; OEVD48 vs. OIVP72; OIVP48 vs. OIVD48; OIVP48 vs. OEVP72; OIVP48 vs. OEVD72; OIVD48 vs. OEVP72 (Table 1).



A. TEM - OIVP72, X 7.000 B. TEM - OIVP72, X 30.000

C. TEM - OIVD72, X 7.000 D. TEM - OIVD72, X 30.000

FIGURE 6 - TEM photomicrography, group 4, A and B (OIVP72): cellular edema, less dense cytoplasm, increased space of the terminal web (**tw**), fainted mitocondrions (**m**), secondary lysosome (**ls**), microvilli atrophy (**mv**), bacterial lay down on the glycocalyx.(**g**). C and D (OIVD72): more heavy alterations, great microvilli deformity and spacing (**mv**), bleb formation (**bl**) along of the luminal surface.

Discussion

Mechanical obstruction with a priori no vascular hazard may set traps and postpone a surgical treatment, with resulting sepsis and severe hydrolytic disturbance. Even in those cases of mechanical obstruction with intestinal ischemia as in strangulated hernia, volvo, and intussusception the symptomatological sequence in the first hours is insidious, and may be misinterpreted as a simple occlusion.

Objectively, as a rule, in case of an intestinal obstruction with vascular occlusion, intestinal resection is required with or without primary anastomosis, associated or not to estomae, adherence disruption, thromboendarterectomy or embolectomy with or without segmental excision of the intestinal loop. Morbid conditions resulting from the treatment may take the patient to severe postoperative hemodynamic and/or systemic complications and to the short bowel syndrome. The need for a detailed preoperative critical examination aiming an intestinal vascularized bed protection if at all possible is critical not only for a morbid-mortality index decrease but also for achieve a better quality of life.

In fact the relevance of the vascular unit in face of the surgical strategy relative to intestinal obstructions has been disregarded with exception for the correlation to the clinical findings when abdominal complications are present. Only the superior mesenteric vessel obstruction and its principal branches have been considered in the scope of researchers and clinical surgeons. Very few studies on small intestinal ultrastructure in mechanical obstructive syndrome have been published and references on the involvement of the third or fourth order blood vessels are still scantier. We emphasize that in our study the ligature of the marginal vessel interrupted the entire blood supply to a loop of intestine at only one place and because of the collateral supply this surgical maneuver avoided huge lesions such as necrosis seen after a long-term

TABLE 1 - Microvilli volume with means and standard deviation (SD) for the different groups

GROUPS	OBSTRUCTION SITE	FOLLOW UP (HOURS)	MEAN 10^3 MM³	SD (±)10^3 MM³
C			7.83	0.05
S		24	14.82	0.15
		48	9.73	0.05
		72	8.23	0.32
OEV	P	24	6.74	0.13
		48	7.53	0.12
		72	8.69	0.11
	D	24	6.74	0.09
		48	6.32	0.06
		72	8.29	0.09
OIV	P	24	11.72	0.23
		48	8.35	0.12
		72	6.28	0.12
	D	24	11.67	0.12
		48	8.72	0.13
		72	4.66	0.06

C - Control group; **S** – Sham group; **OEV** - Obstruction excluding marginal vessel; **OIV** - Obstruction including marginal vessels; **P** - Intestinal segment proximal to the site of the obstruction; **D** – Intestinal segment distal to site of the obstruction, 24, 48, and 72 hours – intervals of follow-up

ischemia. But despite of the intestinal obstruction, the supplying artery occlusion was determinant to worsen the intestinal damage. So, additional explanation should be pursued in order to explain why broad intestinal wall necrosis was found in rats submitted to a small intestinal obstruction for some hours without tying the superior mesenteric artery as showed by Yamamoto[16,17]. Deitch[1,18-19] and Santacroce[20] in some studies using light and electronic transmission microscopy. These results and conclusions are conflicting with surgical practices earned in the long run with human being surgeries. If they were reproducible, intestinal necrosis and severe sepsis should be expected within a few hours of evolution for patients with uncomplicated obstruction, and therefore conservative managements should be revised.

In any experimental model of mechanical intestinal obstruction with surgical approach, manipulation of the abdominal cavity sets free a paralytic ileus as a reflex from an underlying neuroendocrine and metabolic response. This was reduced by working against time and through the afore mentioned technique to get a less traumatic intestinal blockade.

No more than two rats were operated in a day with the purpose of making no mistake in the cuttings of the samples, and their fixations. With this rigid standardization there was no possibility of having morphological interferences or sample exchanging.

Physiological 0.9% saline solution at temperature near the freezing point was shed into the celoma space before samples was taken out. The stiffness of these fragments enabled the cutting of very tiny slices of tissue and it was also useful to protect the cells from an early autolytic degradation keeping their original characteristics. Light microscopy findings do not show irreversible injuries. For the Transmission Electronic Microscope (TEM) the specimens were preserved by fixing them in Karnowsky solution for two hours since the fixation speed is about 1 mm in one hour. By doing this, enterocyte ultrastructure was kept in good condition as near as possible to their in vivo natural state. Embedded fragments were sectioned in longitudinal slide-way, a necessary step for keeping undamaged the enterocyte columnar form. The mucosa border in the cut was discarded as in this inclined plane the enterocytes inevitably were injured despite their being fixed in the first minutes after submersion.

We started this experimental study with an intestinal blockade without vascular involvement, for a 24 hours time (OEV24), because it was reported a heavy epithelial injury in all pertinent publications we looked for. Unlikely, in our work no significant lesion was evidenced in all tested animals at the same period. In the presence of these contradictory results of the literature, the interval of intestinal obstruction was increased to 48 and 72 hours. Even though mucosal lesions were less severe than those described in some publications. The researchers do not mention the inclusion of the marginal blood vessel during the intestinal obstruction and for us this omission might have been related with the cause for the observed different enterocyte injuries. This hypothesis leads us to add the group 4 (OIV).

All the tested rats in all groups endured the three days evolution. Intestinal distention proximal to the obstruction site was seen but no intestinal necrosis, perforation or peritonitis was observed. There was no failure in our intestinal blockade technique such as recanalization of the lumen.

We based our work on the Gracey's researches[12] with sole ileum obstruction in rats, showing enterocyte villi vulnerability chiefly at their top[3,18,19]. Even with closed-loop obstruction this author found from moderate to heavy ultrastructural alterations in 10 to 35% of the enterocytes. These findings were useful to draw attention to the scientific disagreement in some publications of high impact[16] in which optical microscopy showed severe injuries, muscular tissue necrosis after 45 minutes of obstruction. However, in our study the microvilli of the enterocytes were preserved as observation with TEM after greater blockade time intervals. Blebs formation on the enterocyte apical membrane after an obstruction as short as 10 hours was one of the appraised morphological aspects. Only in the OIVD72 subgroup, the alterations were close to those described by Yamamoto[16] with a 24-hour blockade. Thus peripheral vascular factor might have been approached in such studies because of the paramount significance of marginal mesenteric vessels for the pathophysiology of the small intestine obstruction. In another work Yamamoto[17], modified the same experiment by occluding a secondary branch of the superior mesenteric artery in addition to the intestinal mechanical blockade. However statistical and morphological results on groups of rats with or without vascular tying showed amazing likeness among the different groups which was ascribed to vessels intramural compression. In fact it is astounding that the result of the simple intestinal obstruction was the same as that observed with mesenteric artery occlusion, which the effect is broader and much more severe.

Titova[13] in late nineties observed significant microvilli shrinkage in a study with TEM after a rat ileum obstruction which excluded blood vessels for 72 hours. However this author did not mention if the blocked vessels were mesenteric central or peripheral.

It should be considered that intestinal sample handling, and their processing for TEM analysis may play an essential role for divergent lesions grievousness. The size of the intestinal samples and the time taken from the sample removal to its suitable fixation are elements which require the greatest care. The differing processing not mentioned in most conflicting studies are indispensable to standardize all the steps of the sample preparation and necessary for good results. As long as these criterions are not complied with, the samples for electronic microscopy will not be suitable for further ultrastructural study as they will be sources of artifacts and will show inexistent injuries[2-4,7,8,10-12,19,20].

The assessment of the ultrastructural changes of the small intestinal mucosa after the ileal mechanical obstruction demonstrated that only reversible ultrastructural lesions of enterocytes were observed after obstruction up to 72 hours. However damage becomes great when mesenteric marginal vessel arch is also injured. The comprehension of those findings could not only help surgeons to understand the physiopathology of the intestinal obstruction but also be useful for the surgical management when major arterial branches are not involved.

Conclusion

Reversible ultrastructural alterations of rat enterocytes were observed after a terminal ileum mechanical obstruction lasting up to 72 hours. These findings were more severe in the presence of mesenteric marginal vessel arch injury.

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