

CELLULAR AUTOLYSIS IN INTRAPARTUM DEATH. SOME STRUCTURAL CHARACTERISTICS

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Abstract

Asphyxia is one of the implicit causes of intrapartum death. The most affected are the organs of respiratory and digestive systems, filled with different substances or structures of the feto-maternal metabolism. Inside these organs, the cells of the mucosal epithelium suffer cellular autolysis and are completely removed. The loss of structure can reach the depths of the mucous membrane. In some organs on the normal skeleton we can observe regions of cellular autolysis (kidneys).

Keywords: intrapartum death; structural characteristics; cellular autolysis.

Introduction

Postmortem autolysis is the cellular death similar to necrosis, which takes place in this case, along with the death of the entire organism. Postmortem autolysis is the decay of the organism induced by the substances found inside (cellular enzymes) or in different compartments of the organism (intestines) (Manolescu N., 1999, Keeling J., 2007, Gunn A., 2009).

Tomita Y and col. (2004) enumerate in a study conducted on rats the main immediate transformations which take place in the postmortem cell: cellular edema. amorphous deposits in mitochondria, decrease of glycogen granules, dilatation of the endoplasmatic reticule tanks, perinuclear and cluster arrangement of the chromatin and/or its condensation.

In some organs, like the pancreas, transformations remind both autolysis and apoptosis.

The transformations associated with postmortem autolysis differ from one system to another.

Some organs (bones, hair, nails, and teeth) are more resistant to the action of cellular enzymes or extra-cellular factors. The most vascularized organs (liver, kidneys, pancreas, muscles) will be affected first, others will be affected more slowly (cornea) (Iftenie, V., 2006).

The embryo and the fetus develop inside the mother's body, in the amniotic cavity filled with amniotic fluid. The latter appears in the beginning through the action of the amniotic cells and afterwards through the action of the mother's body and of the fetus.

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At 37 weeks, the quantity of amniotic fluid is of 1000 ml. The amniotic fluid is ingested by the fetus through the digestive and respiratory tracts, reaching the kidneys, the stomach and the intestines, from where is exuded in the same environment from which it was produced. In normal cases, the amniotic liquid effectuates a continuous circulation between the fetus's body, the amniotic cavity and the mother's body.

Materials and methods

The tissues derive from two bodies deceased intrapartum (bodies were kept at 4°C); the sampling was made at 24 hours after death. They were preserved in 4% formic aldehyde, put in

Results and discussions

Knowing that postmortem autolysis is influenced by a series of factors (temperature, period of time passed after death, cause and manner of death, type of tissue, environment where death has occurred) we analyzed a few cases of intrapartum death.

On the sections of brain studied we

The chemical composition is of approximately 99% water, proteins, carbohydrates, fats, enzymes, hormones, fetal desquamated epithelial cells (Moore K.L., 1993, Sirbu V. 2004).

In cases of intrapartum death, the lungs are filled with amniotic fluid, secretions of the tracheal mucous glands and fetal desquamated epithelial cells.

paraffin and coloured with hemalaun-eosin. We analyzed samples from brain, lungs, heart, kidney and intestines. The sections were studied and photographed with an Olimpus microscope.

observed different patterns of necrosis in the grey substance (the neuronal bodies) and the white substance (the glial cells). Lesions appear in the parasagittal areas, in the depths of the cerebral grooves. We notice on extended areas of cerebral softening and thromboses (multiple focuses of ischemic infarct) (fig.1).



Fig. 1. Brain, intrapartum death, ischemic infarct, hemalaun eozin, MO 20X

The lungs have an unoxygenated fetal lung aspect. Both the pulmonary channels and the alveolar sacs are filled with amniotic fluid, squamiferous fetal cells, erythrocytes, macrophages and meconium. Meconium is perceived as a set of granulations in the alveolar sacs. At the level of main bronchia we observe the necrosis, and all the epithelium is desquamated. The parenchyma presents regions of chronic stasis (passive congestion).

Fetal hypoxia has induced the amniotic alveolitis (fig.2).



Fig. 2. Lungs, intrapartum death, a. pulmonary channels and the alveolar sacs filled with amniotic fluid (arrow), b. squamiferous fetal cells (arrow), c. meconium in the alveolar sacs (arrow) d. main bronchia with desquamated epithelium (arrow), hemalaun eozin, MO 20X, 40X, 90X, 20X.

The reins present regions of necrosis on a normal structural skeleton, but the regions affected by necrosis are limited. Necrosis is particularly distinguished in the renal tubules. It begins with the condensation of the nuclear chromatin, small intracitoplasmatic vacuoles, and cytoplasm disorganization. The rest of the parenchyma is hemorrhagic (fig.3).



Fig. 3 Reins, intrapartum death, regions of autolysis (arrows) on a normal structural skeleton hemalaun eosina, MO20X

The myocardial striated tissue of the heart is integrally affected by necrosis.

Inside the gastro-intestinal tract, transformations take place very fast, because of the presence of digestive fluids. Inside the intestine we can see the total lysis of the mucosa and a thick content (fig.4).

The cells inside the organs studied are affected either separately, the neurons and the glial cells, or as a group, the epithelia cover. The lack of vascularization and the feeding by osmosis of the epithelia influence the hypoxia by intrapartum asphyxia to increase the speed of cellular autolysis apparition. Another factor which influences this process of the epithelia is the epithelial arrangement towards the organ's lumen, consequently the direct contact with extracellular enzymes. Intrapartum asphyxia stops the alimentation with oxygen of the blood (hypoxia), increasing the quantity of CO_2 in blood (hypercapnia) and induces acidosis by accumulation of lactic acid in the tissues. As the fetus lies in an aquatic environment, the deprivation of oxygen by suppressing maternal infusion increases the speed and the violence of the phenomena stated previously.

Organs with a faster metabolism (liver, brain, spleen) are affected first.



Fig. 4. Intrapartum death: a. heart, myocardial autolysis b. intestine, lysis of the mucosa (arrow), hemalaun eosin, MO20X.

Conclusions

Asphyxia is one of the implicit causes of death intrapartum. The most affected are the organs of respiratory and digestive sistems, filled with different substances or structures of the feto-maternal metabolism.

Inside these organs, the cells of the mucosal epithelium suffer cellular autolysis and are completely removed.

The loss of structure can reach the depths of the mucosa.

In some organs on the normal skeleton we can observe regions of cellular autolysis (kidneys).

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