AUTOPHAGY GENES EXPRESSION IN THE INFANT AND ADOLESCENT HUMAN OVARY

Albamonte, Maria Itat*i[1]; Calabro, Lara[2]; Albamonte, Mirta Susana[2]; Zuccardi, Luis[3]; Stella, Ines[4]; Vitullo, Alfredo Daniel[1]

[1] Maimonides University. Conicet, [2] Maimonides University, [3] Hospital De Niños Dr Ricardo Gutierrez, [4] Hospital Bernardino Rivadavia

Abstract Body

From fetal life to menopause, the human ovary suffers a constant elimination of germ cells that markedly diminish the germinal reserve leading to a total intra-ovarian loss of more than 99.9% of the original pool by the end of the reproductive lifespan. Several studies have revealed apoptosis as the main responsible mechanism for the loss of germinal mass. Nevertheless, the involvement of other concurrent cell death mechanisms cannot be neglected. For instance, germinal exfoliation has been shown to contribute to germ cell elimination in the peri-natal ovary. Other possible cell death mechanisms such as autophagy or non-lysosomal degradation that may concurrently participate in germ cell elimination have not so far been investigated in the human ovary. We analyzed by immunohistochemistry the spatio-temporal expression pattern of autophagy proteins BECLIN 1, LC3b and LAMP1 and the apoptotic protein BCL2 in the infant/adolescent (7-19 years old) ovary, in patients (n=28) with extra-gonadal oncologic pathology. BECLIN 1 was detected in oocyte and granulosa cells in all types of healthy follicles and atretic follicles; advanced antral follicles showed positive signal especially in theca cells. LC3b showed a similar expression pattern as BECLIN1. LAMP1 was detected in the oocyte from all follicular stages with a punctuated cytoplasmic localization. BCL2 was detectable in oocyte and granulosa cells in all follicle types; in antral follicles, BCL2 signal was mainly detected in theca cells. As far as we could track in the literature, this is the first description of autophagy markers immunolocalization in the infant/adolescent ovary. The expression of autophagy markers evaluated in conjunction with the spatio-temporal distribution of the BCL2 and related BCL2-family members suggests that both mechanisms might coordinately act in determining death or survival of the germinal reserve.