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PATHOLOGICAL ROLE OF AUTOPHAGY IN OSTEOARTHRITIS

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The incidence of osteoarthritis is influenced by many factors. Among them are mechanical, genetic, senescence-associated, ecological and other. Autophagy is considered a key factor in the pathogenesis of OA. Arising in healthy cartilage cells as a protective mechanism, it becomes defective and leads to development of the osteoarthritis.

Keywords: autophagy, osteoarthritis, senescence, inflammation, aging, chondrocytes, cartilage.

Osteoarthritis is a type of chronic joint disease that is characterized by the degeneration and loss of articular cartilage and hyperplasia of the synovium and subchondral bone. Articular cartilage is very susceptible to the senescence-related changes, because of the low turnover of chondrocytes and extracellular matrix. There are very small part of proliferating cells in mature cartilage. According to this, cells are prone to accumulate changes related to trauma, mechanical or oxidative stress over time. These changes include reduction of oxidative defense system, aberrant gene expression, which is responsible for abnormal protein synthesis, and altered responses to growth factors and cytokines. These mechanisms are critical to maintenance of chondrocyte survival and normal function.

Autophagy has role in pathogenesis of several diseases. It also regulates the aging process. It is a highly regulated cellular mechanism with both beneficial and pathogenic effects. Cellular homeostasis require a well-regulated balance between protein synthesis and degradation. There are two basic mechanisms for degradation in eukaryotic cells by the proteasome and autophagy. So autophagy involved in the degradation of long-lived proteins, whereas the ubiquitin proteasome system degrades specific short-lived proteins. Autophagy protects against neurodegeneration, heart diseases, infections and even cancer. The autophagy mechanisms are loosen with aging and related to the failure of the lysosomal hydrolases. That leads to accumulating of protein catalysis products and slow clearance of autophagosomes in the aging tissues. In addition, there are some hormonal changes.

In articular cartilage, the role of autophagy in the maintenance of cellular homeostasis and function is particularly important, due to the low rate of chondrocyte proliferation. Autophagy is considered a key factor in the pathogenesis of OA. Homeostasis in chondrocytes is maintained by intercellular interaction, organelle functioning and normal biosynthesis functioning. A common feature of degenerative diseases (including OA) is the accumulation of destructive macromolecules, which leads to the loss of the extracellular matrix, cell dysfunction, and death. Chondroptosis is the term, which describes the death of chondrocytes in articular cartilage. This process includes apoptosis and autophagy. In OA patients, autophagy activation function is missing, thus leading to chondrocyte death and tissue destruction.

The functional deficiency of autophagy can lead to mitochondrion dysfunction and abnormal accumulation, further increasing the risk of OA. For instance, lack of effective mitochondrial coupling in OA causes deficiency of the reparation ability of articular cartilage. Additionally, pathological chondrocytes contain large amount of reactive oxidants. Moreover, the increase in oxidative stress, reduction of chondrocyte proliferation, inflammation, and death of chondrocytes are all related to mitochondrial dysfunction. The mitochondrial dysfunction plays an important role in OA pathogenesis. Autophagy can be activated to combat the dysfunction of the mitochondria in human chondrocytes. It is one of the indispensable regulatory mechanisms for intracellular homeostasis.

Autophagy does not only regulate nutrient provision but can also play an important role in the removal of dysfunctional organelles and macromolecules, an activity that can be confirmed in OA. Animal studies have indicated that the activation of autophagy can prevent cartilage from mechanical damage in OA. With aging chondrocytes lose some mechanisms of autophagy, which helps to defend them and maintain homeostasis. Therefore, autophagy became defective and leads to OA development.

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GESTOSIS AS PATHOLOGICAL CONDITIONS OF THE SECOND HALF OF PREGNANCY

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Despite many years of research, the problem of gestosis remains not fully understood. From point etiology, gestosis is a multifactorial disease (complication) of pregnancy. According to the WHO, gestosis accounts for approximately 14% of cases of maternal mortality (MS) and takes the 2nd place in the structure of its causes.

Keywords: Gestosis, pregnant, edema, proteinuria, hypertension.

Gestosis – pathological conditions of the second half of pregnancy, characterized by a triad of the main symptoms: edema (hidden and visible), proteinuria (the presence of protein in the urine), hypertension (persistent increase in blood pressure).

The diagnosis of gestosis is made on the basis of a characteristic clinical picture, taking into account predisposing factors [1].

In Belarus, the incidence of late gestosis is from 7,3 to 10,5%, while in Russia it is 20–25%, in the USA 23–28 %, and in developing countries it reaches 30–35% [1].

Gestosis is considered a classic complication of pregnancy; it aggravates gestation in 6–8% of pregnant women in developing countries and 0,4% in developed countries. Annually late gestosis affects 1,5 – 8 million women in developing countries and 50 – 370 thousand pregnant women in developed countries [4].

Uncomplicated arterial hypertension in pregnant women does not worsen the outcome of gestation, but with the development of gestosis, the frequency of complications and mortality of mothers and newborns increases [2].

In a Parkland hospital (USA) over a 25-year observation period, the gestosis rate was 1 case per 1750 births.

In the US National Statistical Report, the frequency of gestosis is indicated as 1 case per 3250 births in 1998, i.e. the frequency of gestosis gradually decreases. To date, late gestosis in the United States accounts for 15 % of premature births and 17,6 % of maternal deaths.

An epidemiological study carried out under the auspices of the WHO in China determined a 10,4 % incidence of hypertensive disorders in pregnant women, with a histosis rate of 0,2 %.

In the Russian Federation in recent years there has been an increase in the number of cases of gestosis and its severe forms, respectively, the proportion in the structure of maternal mortality has increased from 9,4 to 15,6 % with fluctuations in the regions from 6–8 to 29,6 % [3].

An analysis of the prevalence of late gestosis in the Republic of Belarus over 10 years from 1996 to 2005 was carried out. Based on data from annual statistical reports (form No. 32) of healthcare institutions in all its regions. According to the results, it was determined that from 1996 to 2003. in Belarus, there was a clear tendency to increase the incidence of gestosis from 7,7 to 10,3 % of women who completed a pregnancy, that is, an increase of 3,1 % [3].

Since 2004, the republic has seen a tendency to reduce the frequency of this pregnancy complication to 9,9 %, in 2005 – to 9,1 %. Analysis of studies indicates that in the Republic of Belarus for the period from 1996 to 2005. 22 women died, the pregnancy of which was complicated by gestosis, which amounted to 18,3 % of all cases of maternal mortality that occurred during this time period [2].

Late gestosis continues to be one of the most frequent and serious complications in the process of pregnancy development, childbirth and the postpartum period, not only in our country, but also abroad. The frequency of these gestosis is constantly growing [4].

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