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Protective Action of Xymedon on the Morphological Structures of the Pancreas of the Rat in Ischemia

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Abstract

Pathologic processes associated with the effect of ischemia on the pancreas are not yet fully investigated and searching for ways of reduction of the damaging effect of ischemia for providing full organ regeneration are of certain interest. Protective properties of xymedone with regard to pathologic processes in pancreas were studied on a model of total ischemia created by ligation of the coeliac and cranial mesenteric artery for 0 to 90 min. Pathologic structural changes in the pancreas in case of ischemia lasting up to 30 min are minor and therefore fully reversible. On the contrary, ischemia duration of more than 60 min in the group of animals not injected with xymedone can be seen as critical. We conclude that prior intraperitoneal injection of xymedone in the dose of 3.3 mg/kg helps to delay the development of pathologic processes in pancreatic parenchyma by approximately 30 min.

Keywords Ischemia · Pancreas · Pyrimidine derivative · Xymedone

1 Introduction

The issue of ischemic damages to various organs and tissues remains one of the most pressing and understudied in the medicine. In spite of seemingly well-studied consequences of hypoxia for many organs and tissues, in general, the mechanisms of ischemic damages to various cells and the possibility of their future regeneration remain underinvestigated. The effects of ischemia on the heart [1], brain [2], and pancreas [3] are being studied by many scientists and, increasingly frequently, using new study methods.

In our opinion, issues associated with the effect of ischemia on the pancreas and the maximal reduction of the damaging effect of ischemia for providing full organ regeneration as well as delaying the irreversible changes as much as possible are of certain interest. The approach involving pharmacological correction of ischemic lesions can be promising in this respect.

Molecular and clinical signs of organ ischemia can contribute to multiple organ dysfunction syndrome as a result of oxidation processes and inflammatory response after reperfusion [4]. The resulting active forms of oxygen are very unstable. They can cause cell damage, primarily due to lipid peroxidation in cellular membranes and mitochondria, as well as DNA degradation [5]. It has been proven that overproduction of oxygen free radicals in organs causes suppression and decreased ability to absorb own endogenous antioxidants through the lipid peroxidation component of the mitochondrial and cellular membrane, which leads to cell death [6].

At the present time, to reduce the damaging effect of free radicals, a wide range of drugs is being used such as antioxidants, various enzyme blockers, and drugs improving microcirculation. Recently, protective properties of a drug from the group of pyridine bases have been studied on various models. Among them, xymedone (1,2-dihydro-4,6-dimethyl-*N*-(β-oxyethyl)-pyrimidone-2) synthesised in 1966 by A.E. Arbuzov Institute of Organic and Physical Chemistry of Kazan Scientific Centre of the Russian Academy of Sciences and by the Kazan State Medical University is of principal interest.

The preparation is a white or touched with pink crystalline powder, without odour, bitterish, with the melting temperature of 139–143 °C, freely soluble in water, spirit, and normal saline. According to the literature, xymedone has antioxidant [7], apoptosis-regulating [8], burn-treating [9, 10], and antimutagenic [11] effects, as well as immunomodulatory effect [12]. There are data confirming the positive effect of xymedone on the treatment of ischemic ulcers [13], systemic sclerosis [14], atopic asthma [15], peritonitis [16], and sinusites [17]. In our previous study, we showed that intraperitoneal introduction of xymedone in the

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