childhood mortality. Early neonatal mortality takes away society's potential physical, social, and human capital.

Aim of the study: To evaluate the structure of the index of early neonatal mortality since 2009 to 2014.

Methods: A statistical analysis of the autopsy reports of the children's department of “Grodno Regional Postmortem Bureau” and the information that was obtained in Regional Statistic Department for the period from 01.01.2009 till 31.12.2014.

Results: In 2014 in Grodno region 13,253 children were born. This is 297 less than in the same period of 2013 (01.01.2013-31.12.2013 – 13,550 births). After analyzing of the birthrate in this region since 2009, we got the following numbers: 2009 -12,463, 2010 – 12,215, 2011- 12,562, 2012 – 13,208, 2013- 13,550, 2014 – 13,253. After analyzing the data of Children Pathology Department and Regional Statistic Department on early neonatal mortality it was found that in 2009 there were recorded 14 deaths of newborns during the first 7 days (rate per 1,000 live births was 1.1), in 2010 - 14 (1.1), 2011 - 18 (1.4), 2012- 14 (1.1), 2013 - 15 (1.1), 2014 - 13 (1.0) . It was found that the main causes of death in 2009 were internal malformation(IM), multiple internal malformation(MIM), which averaged 39% of the total figures for years, with some reduction in 2014 year to 23%. The second leading cause of early neonatal deaths remained pneumopathy. So from 2009 to 2014, they averaged about 12% of all cases of early neonatal death, with minimal growth in 2014 to 15%(2 cases). The most rare causes were hemorrhagic disease, pulmonary hemorrhage, diabetic embryopathy severe anemia at birth.

Conclusions: After analyzing the data about causes of early neonatal death the following conclusions were drawn: the most common cause of early neonatal mortality from 2009 to 2014 were the IM and MIM (averaged 39%).

After analysis of information for reasons of early neonatal deaths, to reduce infant mortality should to:

1. Improve ultrasound techniques to detect abnormalities of the fetus;
2. Observe for pregnant women to reduce the complications of gestation.

THE ROLE OF THE INSERTION-DELETION ACE GENE POLYMORPHISM IN TARGET ORGAN DAMAGE IN PATIENTS WITH ASTHMA AND OBESITY

Pasiyeshvili T.M.[1], Zheleznyakova N.M.[2]
Kharkiv National Medical University, Ukraine
Department of General Practice – Family Medicine and Internal Disease[1]
Department of Internal Medicine №12
Scientific adviser – MD, prof. Pasiyeshvili L.M.

To date, more than 300 million people in the world suffer from asthma and this number continues to grow every day, which allows us to consider asthma as a disease of civilization. The prevalence of asthma among obese persons is 11.9% in contrast to the general population, where the figure is 6.1%. At the same time pathogenetic mechanisms along with activation of cytokine link of immunity, hormonal spectrum of blood, lipid, and carbohydrate metabolism species can be the result of different gene polymorphisms [2]. Genetic changes in asthma are the result of

complex schemes haplotypical combinations of polymorphic genes. Variations in different groups of genes may influence on the development of atopic sensibilization, while other genetic changes - for the development of asthma [1]. At the same time, combining for asthma with any diseases of internal organs may make changes in gene polymorphism, thereby leading to the formation of complications. Unfavorable factor such comorbidity acts obesity, the presence of which in patients with asthma can cause damage of the cardiovascular system [1, 2]. Aim: to study the role of insertion-deletion polymorphism (I/D) gene angiotensin-converting enzyme (ACE) gene polymorphism in the formation of target organ damage in patients with asthma and obesity.

Materials and methods. The study involved 42 patients with isolated asthma (comparison group) and 61 patients with asthma and obesity (study group) at the age of 40.3 ± 6.2 and 41.7 ± 6.9 years old, respectively. The control group consisted of 50 healthy subjects of similar age and sex that allowed us to obtain control results. Endothelial function were assessed by the studying insertion-deletion polymorphism (I/D) gene ACE, which is regarded as the most significant marker of endothelial condition and the main factor that determining the concentration of this enzyme in the plasma. In the 16th intron of the gene, which is located on chromosome 17q23r, presents (I-insertio) or absents (D-deletio) a DNA fragment. It is consisting of the 263-287 pairs Alu sequences. The presence of these changes in a DNA fragment considered as an indicator of a mutation of the gene.

Results and discussion. Thus, among the 50 control subjects homozygous I/I ACE gene was detected in 16% of patients, heterozygotes I/D - 54%, and the mutant homozygote D/D - 30%. In the group with isolated asthma genotype distribution corresponded to 14.3%, 33.3% and 52.4% patients. In case of combination of asthma and obesity amount of patients with abnormal genotype increased in 2 times compared to control group and was 8.2%, 31.1% and 60.7% respectively. Pathological genotype of the ACE gene influenced on the time of occurrence of asthma. Thus, when comparing the clinical symptoms with genotype of the ACE gene were found that asthma exacerbations in 28 patients of the study group and 11 - the comparison group led to the appearance of cardiac symptoms: cardialgias, heartbeat, heart rhythm disturbances, increased blood pressure (abnormal genotype D/D - 35.1% and 13.2 %, respectively). Conclusion. Considering the fact that the ACE gene determines the same enzyme concentration in the plasma, we can assume that the increase in mutant homozygotes can lead to early development of endothelial dysfunction and cardiovascular events.

References:

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