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PRADER-WILLI SYNDROME, DIAGNOSTICS AND CURRENCY FEATURES

Abstract. Five boys with Prader-Willi syndrome were examined at the endocrinologist by 2016. All children had minimal diagnostic signs of the syndrome, namely: muscular hypotension, hypogonadism, obesity, mental retardation of varying severity, small hands and feet. In two children there was a disruption of glucose tolerance. All patients had the manifestation of hypergonadotropic hypogonadism. If a minimal diagnostic criteria are found in the newborn, a genetic analysis is necessary.

Key words: Prader-Willi syndrome, obesity, children, hypogonadism.

Introduction. The Prader-Willi syndrome (PWS) was first described by the Swiss pediatricians A. Prader and H. Willi in 1956. Its frequency in the world is 1 per 25,000-10,000 newborns.

The Prader-Willi syndrome arises from the deletion of the parent copy of the imprinted SNRPN gene of the small nuclear ribonucleoprotein N polypeptide and the neclin gene, which is adjacent to the mRNA clusters SNORD64, SNORD107, SNORD108 and two copies of SNORD 109, 29 copies of SNORD116 (HBII-85) and 48 copies SNORD115 (HBII-52). They are located on the 15th chromosome in the region 15q11.2-q13. This is the so-called PWS / AS region, which can be lost as a result of the action of one of several genetic mechanisms in most cases as a result of mutations. There are other more rare mechanisms of development of this syndrome: maternal isodisomy, that is, when both chromosomes 15 are obtained from the mother, random mutations, chromosomal translocations and gene deletions [5, 7].

The risk of the birth of a sick child in a family where there is already one patient is completely dependent on the genetic mechanism that caused the disorder. The probability of a sick child's birth is less than 1% if he has a gene deletion or isodisomy, but if the child has a mutation of the region, which is characterized by the phenomenon of imprinting, then the risk rises to 50%, in the case of chromosomal translocations,

the occurrence of the disease can be predicted in the next child in 25%. For the diagnosis of all known mechanisms, prenatal testing should be used [1].

Children with Prader-Willi syndrome usually are born full-term with insignificant intrauterine hypotrophy, often in asphyxia, 10-40% previa gluteus. There are two phases of the syndrome [2]. The first is inherent in children of 12-18 months. Immediately after birth, severe muscle hypotension is noted. The symptom is very pronounced, children do not make spontaneous movements and can not suck. As a result, the formation of static and locomotor functions is sharply delayed: they do not hold their heads, they do not sit. The physiological reflexes of the newborns are decreasing or absent: Moro's reflex, step reflex, and Bauer's response. There is also a tendency to hypothermia. There are other anomalies: a high, narrow forehead; almond-shaped incision of the eye slits with thin, drooping eyelids; skin and hair are lighter than all other family members have, hypopigmentation of the iris (in 75% of cases); microdontia, hypoplasia of the cartilages of the auricles, scoliosis, ectropion (eyelid eversion), glaucoma. The second phase of the disease develops in a few weeks, months or until the end of the first - the beginning of the second year of life. Hypotension gradually decreases and bulimia develops: the child constantly experiences hunger, actively searches for food and, as a result, obesity develops.

Subcutaneous fatty tissue is distributed unevenly, most of it on the trunk and proximal parts of the limbs - hips, shoulders. The feet and hands are disproportionately small. This phase begins to attract attention to mental retardation [3]. Hypogonadism is also characteristic. Typically, patients also have a deficiency in FSH and LH secretion, which can lead to a delay in puberty and underdevelopment of the sex glands [4]. In boys, hypoplasia of the penis is observed, in girls hypoplasia of large and small labia and in 50% of cases - the uterus. Further development of diabetes is characteristic. In the blood biochemistry there are no abnormalities [6].

Differential diagnosis is performed with other syndromes accompanied by severe muscle hypotension, delayed psychomotor development (myopathy, spinal amyotrophy, Opitt-Frías syndrome) with obesity syndromes (Lawrence-Moon-Barde-Biddle, Alström, Cohen syndrome, adiposogenital dystrophy, etc.) [8].

Objective of the study was to analyze the course of the Prader-Willi syndrome in children of the Chernivtsi region.

Material and methods. Five children with Prader-Willi syndrome were examined, who were on the endocrinologist's monitoring in 2016.

Results of the study and their discussion. All children with this disease were male. Up to a year, the diagnosis is made for two children (40%), up to 4 years also for two, and one child for 14 years. All children had minimal diagnostic signs of the syndrome, namely: muscle hypotension, hypogonadism, obesity, mental retardation of varying severity, small hands and feet.

General clinical tests showed no abnormalities. Two children had a disruption of glucose tolerance, which was proved with a glucose tolerant test. Biochemical blood test did not show any other abnormalities.

Three children had a true bilateral cryptorchidism. So, as in all patients hypogonadism was clinically diagnosed, in all children the level of sex hormones was studied.

The level of hormones in children was: FSH - 14.04 mlOd / ml, testosterone 1.1 ng / ml, LH - 12.6Mod / ml, that is, hypergonadotropic hypogonadism was detected.

All children underwent ultrasound of the thyroid gland. Also, children with suspected autoimmune thyroiditis were assessed for TSH, T3

and T4 levels and the detection of antibodies to thyroid peroxidase. Two children confirmed the diagnosis of "autoimmune thyroiditis, hypertrophic form, euthyroidism."

Two children had short-sightedness and one child suffered from spinal amyotrophy of Wernig-Hoffmann.

All patients received the following treatment: massage, exercise therapy, monitoring changes in the musculoskeletal system, correction of nutrition and cognitive abnormalities, treatment of endocrinological pathology, surgical interventions.

Conclusions. If a child has a low weight and height in the case of full term pregnancy at birth; previa gluteus, some microanomalies of development; pronounced hypotension of the muscles, reduced pigmentation of the skin, iris of the eyes and hair, it is recommended to perform molecular-genetic testing by the FISH method to detect the microdeletion of the 15th chromosome.

Prospects for further research. To study the features of genetic disorders in children of the Chernivtsi region with the Prader-Willi syndrome.

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CONTENT:

Galagdina A.A., Dmytrenko R.R., Bambuliak A.V. Diagnostics of ischemic-reperfusion damage of the brain in rats afflicted with diabetes mellitus	3
Guranych S.P., Voronych-Semchenko N.M., Guranych T.V. Macro- and microelement status of rats with insulin resistance against the ground of iodine deficiency	6
Fedyshyn T.V., Maliar V.V., Maliar V.A. Peculiarities of utero-placental blood circulation formation in women with spontaneous and recurrent miscarriages associated with vagina dysbiosis	10
Rusnak V.F., Bedyk V.V. Growth of the pharynx at the end of the fetal stage of human ontogenesis	13
Teplytskyi S.S. Formation and development of the skin on the palmar surface of the hand throughout the period of prenatal ontogenesis and its importance in dermatoglyphics	16
Tkachuk N.P., Bilookyi V.V., Gyrla Ya.V., Sheremet M.I. Evaluating the efficiency of the scale for prediction of post-operative relapse in patients with nodular goiters	20
Yemelyanenko N.R. Anatomical transformations of the nasal septum in childhood	24
Kavun M.P. Morphogenesis of the hepatic-duodenum ligament in early ontogenesis of the human	26
Kotyuzhinskaya S.G., Umansky D.A. Functional state of lipitransport system in patients with atherosclerosis with fatty load	28
Lomakina Yu.V., Burdeina M.P. Stress-associated changes in the excretory function of the kidneys in old rats under the conditions of a usual light period	32
Malyar V.V. Structural and functional features of fetal membranes in pregnant women with moderate idiopathic oligo- and polyhydramnios	35
Nesterak R.V., Gasyuk M.B. Pilot investigation of the method of interactive training of patients at the stage of medical rehabilitation and treatment	38
Pecheryaga S.V., Marinchina I.M. Features of hemodynamic changes in spiral arteries with low placentation at the early gestational age	42
Pschytychenko V.V., Chernov V.S., Frenkel Yu.D. The status of extraorganic blood flow in pineal gland of rats under conditions of acute stress and twenty-four hour darkness	44
Reshetilova N.B., Glubochenko O.V., Kulish N.M., Dudko A.G. Formation of anterior cerebral vesicle cavities at the 5th week of the embryonic period	47
Riznichuk M.O., Galitskaya V.O., Dyhodyuk Yu.V., Kravchuk Yu.V., Vakaryuk O.V. Prader-willi syndrome, diagnostics and currency features	50
Shalamay U.P., Pavlikivska B.M., Voronich-Semchenko N.M. The state of autonomous heart regulation in adolescents with light iodine deficiency and latent iron deficiency	52
Shutova N.A., Nikolayeva O.V., Sulkhdost I.O. Electromagnetic radiation impact on the cellular protective mechanisms in experiment	58
Yasnikovska S.M., Hrytsak H. Evaluation of clinic-laboratory and ultrasonic research results in different forms of the chorion's pathology in the first three-month of gestation	61
Yashchyshyn Z.M., Zaiats L.M., Yurkiv I.Y., Maslyak K.T., Vodoslavskaya N.Y., Sikomas M.T. Changes in neuroglial interrelation of muscle-intestinal nerve plexus of esophagus after one-sided crossing of vagosympathetic trunk	64
Navarchuk N.M., Kosteniuk S.V. Morphogenesis of the dentognathic apparatus during the early times of the human ontogenesis	67
Rusnak V.F., Bedyk V.V. Features of pharyngeal morphogenesis in five-week embryos	70
Talanova O.S., Apt O.A. Specifics of distribution of glycosaminoglycans in the white pulp of the spleen and stroma of rats after experimental modeling injection inside the fetus of antigens of different nature	72
Pivtorak K.V., Mazur I.A., Voloshin M.A. Correction of metabolic disorders caused by non-alcoholic fatty liver disease	74
Rozhko V.I. Research of content correlation of immunoglobulins and lisozyme in oral fluid of children with rampant caries against the background of gastro-intestinal diseases	78
Karavan Ya.R., Havaleshko V.P. Up-to-date anesthetic possibilities in dentistry practice in diagnosis of the patient's allergic status	80



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