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HEPATOBILIARY CHANGES AND THEIR CORRECTION WHEN THE DRUG" CORAL ZINC " IS DILUTED IN PATIENTS WITH ACUTE ALOPECIA DEPOSITION

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Abstract:
Migraine alopecia disease is a disease of a polyethiological nature, the causes that cause the disease in its formation are neurogenic, infectious, the presence of migraine infections, changes in blood vessels, functional changes in the central and vegetative nervous system, pituitary, adrenergic and hormonal disorders at the cell level, which in turn cause hair follicles to enter the body of
The role of neurotrophic factors in the development of alopecia is large. Neurotrophic factors control the proliferation process of epidermis cells, control the state of apoptosis, the development cycle of hair follicles, as well as the process of melanogenesis. Nervous and emotional arousal are responsible for the development of neurovegetodistonia, which in turn leads to the appearance of deep disorders in the hair follicles in this disease. The disease is often observed to occur after stressful situations. In particular, such a condition is of particular importance in children. To the opinions of the authors of the series, the impact of a stress condition, one of the triggers of the environment in the occurrence of AA disease in children, is from 9.5% to 80% gacha.

Keywords: Malone dialdehyde, cyclic aminosinmonophosphate, cyclic guanosinmonophosphote, ribonuclease, guanosinmonophosphate, Alani aminotransferase.

Introduction

Acute alopecia (AA), a type of hair loss disease, is common among skin diseases, the causes and pathogenesis of which have not been adequately studied to date. According to data in the medical literature, AA disease accounts for about 4% of all skin diseases, and 1-3% of dermatological diseases accompanied by hair disease, including 4-8% of skin diseases found in children. Humans have a 1.7% lifetime risk of developing the disease. The reasons for the occurrence of the disease are based on the presence of neurogenic, endocrine, immune system activity, violation of the state of

microcirculation, metabolic changes, infections with Foci. The fact that the incidence of the disease increases at the next time is assessed not only because the etiopathogenesis, diagnosis, clinical trials of this disease are not well studied, but also because there is insufficient development of effective methods of treatment of the disease and preventive measures [2, 6, 9].

Currently, the study of the importance of genetic factors in the course of AA disease is of particular importance. Assessing the state of hereditary predisposition of the disease and how it occurs, as well as its repeated course, answering these questions makes it possible to study and analyze the nature of the disease. The high incidence of OEA disease in families and among the patient's relatives gives reason to come to the opinion that on the basis of this disease lies a state of hereditary predisposition to the disease[10, 14, 21, 54, 59]. It is known that the incidence of recurrent OEA disease in families has been confirmed to be high, as well as the incidence in probands to be 4-24%. The increased frequency of occurrence of AA disease is causing the study of conditions that are a factor in the occurrence of this disease. Of particular importance in this regard is the study of factors of the internal environment, including the state of hereditary predisposition to the disease, in addition to external muxit factors. But in the emergence of the disease, it will not always be possible to clearly show the place of hereditary factors in its development. In this regard, in the realization of genetic information in the patient phenotype, it is important to take into account the influence of external mukhit factors. Such a characteristic characteristic of the disease is a sign that AA disease is a disease in the form of a polygen. The role of hereditary factors in the occurrence of OEA disease in children can be said to have been studied to an almost lesser extent. The issue of solving such a problem is of particular importance in the area in which we live, where children have a high rate of termination [2, 13].

Research Objective

To study and practice hepatobiliary changes and their correction when the drug "coral zinc" is diluted in patients with acute alopecia deposition.

Inspection Tools and Materials

The diagnosis of AA in children was based primarily on clinical observations. Once the alopecia foci have been identified, patients are given urgu for location, number, constipation, atrophy, and teleangectasia. The structure of the furnaces, the possibility of joining, and the condition of the absence of hair in the furnaces were determined. The condition of observing the "area of frizzy hair" sign was determined by plucking the hair with the fingers of the hands around the foci of alopecia where the hair fell. Cases of the appearance of a loose hair root (dystrophic hair in the form of a telogensymon, exclamation mark) and a vellus condition, that is, the observation of thin, depigmented hair, were found. Observations revealed the condition of the nail plates, dermographism,

and pilomator reflexes. Kulagin V. in the diagnosis of AA disease.I. (1992) based on the proposed classification.

Patients were examined by various Soha-doctors, including pediatricians, ENT, ophthalmologists, dentists, pediatric jarrochi, traumatologists, endocrinologists, infectionists, according to necessity, and blood, urine, population, as well as biochemical examinations were carried out in each patient child, which were the basis of general clinical examinations.

Research Results

Until observations, the duration of the disease was from 3 months to 3 years. Patients were denied that 54(45.0%) of children were urban and 66 (55.0%) were rural.

The gestation period was found to be 74 (61.7%) patients with toxicosis, 62 (51.7%) patients with anemia were found to be accompanied by eclampsia in 16 (13.3%) patients, and 19 (15.8%) cases with normal delivery. 45 (37.5%) of patients were found to be born in the family from I-pregnancy, 39 (32.5%) from II-pregnancy, 17(14.2%) from III-pregnancy, 17 (14.2%) from IV - pregnancy, and 2 (1.7%) from V-pregnancy. The course of the birth process was moderate in 99 (82.5%) cases and asphyxia in 11 (9.2%) cases. Babies are 49.7 cm tall at birth. ni, while the weight of the body is on average 3238.5 gr. organized. After completion, 117 (97.5%) of infants under the age of 1 were in natural feeding and 3 (2.5) were in artificial feeding. Artificial vaccinations in 104 (86.7%) sick children were performed during the break, while 16 (13.3%) sick children were noted to have no vaccinations. 109 (90.8%) of children with AA disease are URVI, 37 (30.8%) are influenza, 34 (28.3%) are anemia, 47 (39.2%) are caries, 85 (70.8%) are angina, 25 (20.8%) are bronchitis, 16 (13.3%)are gastritis, 46 (38.3%) are hepatitis, 22 (18.3%) are pneumonia diseases and in 7 (5.8%) cases, various operas were postponed. The median age for Sick Children's fathers was 30 years, and the median age for mothers was 27 years.

It was noted that 36 (30.0%) patients had the disease in Spring, 33 (27.5%) had the disease in summer, 26 (21.7%) had the disease in autumn and 25 (20.8%) had the disease in winter when children were observed to have the disease late in the year (Table 2.). In 78 (65.0%) cases, the incidence of AA worsened as patients aged, while in 40 (33.3%) cases remained unchanged, while in 2 (1.7%) cases the incidence was reversed, with improvement.

Before arriving at the hospital, it was found that patients with OEA were treated in various treatment facilities, mainly in an outpatient state. During this time, vitamins (92; 76.7%), antibiotics (15; 12.5%), enzyme drugs (20; 16.7%), sedatives (44; 36.7%), immunomodulators (45; 37.5%), topical physiotherapy (45; 37.5%), and tour li ointments were administered and treated. As a result of the treatments obtained, 60 (50%) cases showed that the process remained unchanged, while 47 (39.2%) cases showed temporary good, and 13 (10.8%) cases showed worsening of the process. In 22 (22%) cases, it was noted that patients were not treated at all.

On arrival at the hospital, patients were found to have started AA with itching in the foci in 7 (5.8%) cases, ogriq in 1 (0.8%) cases, and 102 (85%) cases without any signs. The average number of days the patient spent children in the hospital was 14.7 days. 39 (32.5%) patients had enlarged perepheric lymph nodes in children, while 2 (1.7%) patients had their lameness.

The course of the disease was adenoid in 4 (3.3%) patients, rhinitis in 3 (2.5%) patients, caries in 85 (70.8%) patients, gastritis in 16 (13.3%) patients, enterocolitis in 36 (30.0%) patients, hepatitis in 83 (69.2%) patients, hepatitis, hepatocholesistitis, constipation in 55 (45.8%) patients, and dysbacteriosis in 34 (28.3%) patients, 39 (32.5%) patients were found to be accompanied by comorbidities such as vomiting, ascaridosis, lyambliosis, deafness chain.

UA was delayed by irritability in 23 (19.2%) patients, and by contrast melancholy in 2 (1.7%) patients. 22 (18.3%) patients experienced increased brain pressure, while 24 (20.0%) patients experienced head augmentation. 21 (17.5%) patients were found to experience process Hyperteriosis in children, and 3 (2.5%) patients were found to experience hypotheriosis.

When children with AA were observed, 94 (78.3%) patients were reported to have brown skin color, while 23 (19.2%) patients had white and 3 (2.5%) patients had yellow.

Conclusions

1. AA disease occurs mainly in children over the age of 1 and is observed in 94.2% (iz) cases between the ages of 3 and 16. The disease is reported in the form of single foci in 24.2% (29) cases, multiple foci in the form of co-location in 55.8% (67) cases, and the occurrence of the local form of the disease in 58.3% (70) cases. The condition of the onset of foci of the disease from smooth skin areas of ensa 62.5% (75) later manifested in the form of Total and universal clinical forms.whereas IRNA damage was observed in 4 (3.2%) patients.

2. The atopic 14.2% (16) and hypetensive 12.5% (15) types of AA disease in children were poorly visible in terms of prognosis of early onset, rapid development, prolonged course of the disease, and in these types the local clinical form of the disease was distinguished by a tendency to uta to switch to clinical forms of the head.

3. Hereditary predisposition to OEA in children was attributed to patient parents in 11.7% (25), relatives in 4.5% (39), and inbred environment conditions in 11.6% (14) cases, and the disease was reported to occur mainly in families in 68.4% (82) of children born from pregnancy 1 and pregnancy 2. This state of predisposition to AA's disease was manifested unconnected to Mendelian law, that is, in the form of a polygen.

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