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Mutation of connexin 26 (M34T, V37I, R127H) at psoriatic erythroderma

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Different skin chronic diseases with the imposed phenotypes are caused by mutations in five various connexin genes. Some of these diseases is caused by mutations in GJB2 gene that implicated most of researchers to focus on the connexin 26 Calcium – the key regulator of differentiation of keratinocytes also participates in maintenance of a cellular homeostasis, especially at patients with an erythroderma where damage of skin covers more than 80-90% of an integument. Erythroderma – skin diseases with not clear etiopathogenesis. Search of mutations in the coding area of a gene of GJB2 among patients with various forms of an erythroderma and also assessment of level of ions of Ca^{2+} in plasma of peripheral blood of patients of the same groups. The prospective research was conducted at 56 patients with an erythroderma undergoing examination and treatment on bases of dermatological offices: GBUZ Lenoblcentre of specialized types of medical care, GORKVD of St. Petersburg the period from 2013 to 2018. Indicators Ca^{2+} at patients with various forms of erythroderma are studied. In all examined groups the intake of peripheral blood in vakuumteyner with EDTA in the morning, on an empty stomach for the first days of arrival of the patient in a hospital has been carried out. Genomic DNA was emitted from leukocytes of peripheral blood with method of phenolic and chloroformic extraction (Maniatis et al., 1984). Amplification of the coding area of a gene of GJB2 was carried out with use of the primers described earlier. In the real research all patients were divided on the basis of the anamnesis, a clinical picture, histological and immunohistochemical analyses into 4 groups. Also in the real research direct sequencing of the coding area of a gene of GJB2 among patients with various forms of an erythroderma has been carried out. Mutations of M34T, V37I, R127H in a heterozygotic state in three separate cases at patients with a psoriatic erythroderma have been revealed. Frequency of these mutations was 16,7% among patients with the psoriatic erythroderma and 8,8% among all researched patients with different forms of an erythroderma. Our results mean that the mutation of a gene of a connexin 26 – M34T, V37I, R127H (GJB2) probably is fundamental in development of a psoriatic erythroderma. The last promotes changes of a cellular homeostasis and possibly bring to emergence of signs of an erythroderma.

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