TREATMENT OF STEROID-RESISTANT NEPHROTIC SYNDROME IN CHILDREN

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Steroid-resistant nephrotic syndrome (SRNS) is one of the leading types of nephropathy with progressive course, often leads to the development of chronic renal failure.

OBJECTIVE

to evaluate the effectiveness of steroid-resistant nephrotic syndrome therapy in children using immunosuppressants and without using once.

MATERIAL AND METHODS

SRNS patients were divided into 3 groups. The first group included 34 patients on immunosuppressive therapy (IST) (chlorambucil, cyclophosphamide, cyclosporin A and mycophenolate mofetil), the second group - 22 patients on immunosuppressive therapy in combination with inhibitors of angiotensin -converting enzyme (ACE), third group included 21 patients treated with ACE inhibitors and symptomatic therapy.

RESULTS AND DISCUSSION

We traced catamnesis on maintenance of renal function in patients (by determination of GFR) during 4 years (Fig. 1). Remaining of renal function in patients receiving immunosuppressive therapy was higher than in patients being treated with ACE inhibitors and symptomatic therapy.

Thus, the use of immunosuppressive therapy in patients with steroid-resistant nephrotic syndrome led to a significant improvement in clinical and laboratory parameters, which is important for the further prognosis of disease. The combination of ACE inhibitors and immunosuppressants is more favorable option for preservation of renal function and prognosis in general.

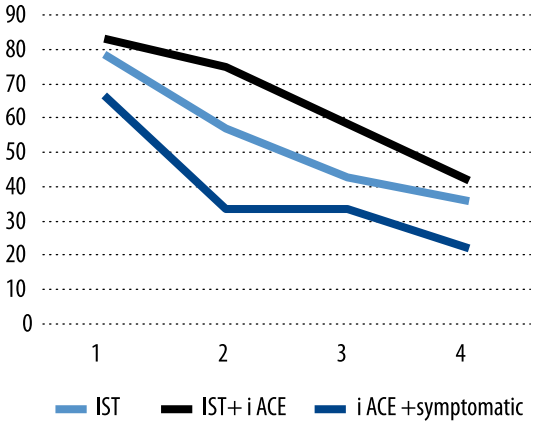

Fig. 1. *Survival of renal function in patients with SRNS on IST background and without it*

CLASSIFICATION OF HYPERTENSION IN THE PULMONARY CIRCULATION

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Developing classification of pulmonary hypertension, taken into account, above all, the fact that the latter is not a separate ontological unit, and the syndrome occurs when a number of illnesses of lungs, heart and blood vessels. Therefore, as it seems to us expedient was to highlight the main etiological factors, resulting in increased pressure in the bloodstream of a small circle, pathogenic forms of pulmonary hypertension, clinical variants and their types. Furthermore, as classification should reflect the stage of the disease in several of clinical manifestation in each stage.

Set that this syndrome is manifested in two main causal factors leading to the increase in pressure in the small circle.

A – Factor – Organic (anatomic)

B – Factor – Functional

Each of these factors includes pathogenic form, and recent clinical types. To organic factor include the following pathogenic form.

1. Pre-capillary blood, which is manifested in two clinical variants:

Intravascular and outside the vascular intravascular variant occurs either due to disease, covering the lumen

of the arteries, violation the arterial patency (primary intravascular block), such as thrombosis and embolism pulmonary artery and its branches, thromboarteritis, obliterating arthritis, idiopathic the obliteration of the pulmonary artery disease extravascular clinical variant is associated with diseases, localized outside vessels and compress them as such, the outcome of which is not polysegmental share, or a total pulmonary cirrhosis. Under this option, in addition to the above internal mentioned a chance and extrapulmonary types, as, for example, in connection with compression of the lungs due to the high standing of the diapulmonary (syndrome Pickwick), chest injuries with fractures of lungs.

2. To organic factor includes also the alveolar capillary (diffusion) pathogenetics form. Diffusion of gases, oxygen carbon dioxide are:

Through the membrane, which is the only partition (shell) between the alveoli and capillary types of sensors. In violation of the permeability of the membrane (diffusion blocks) diffusion slows down or stops, and them, due to hypoxia and hypercapnia, reflex limited to the inflow of arterial blood by spasm of the arteries) to the alveolar capillaries. This happens with inflammatory processes in the alveoli, capillary arterioles, venules (alveolitis, exciting significant part of the respiratory surface), as, for example, when dangers and chronic pneumonia syndrome Hamman-rich alveolar.

ALVEOLAR PROTEINOSE: THESE PATHOGENIC FORMS TO FUNCTIONAL FACTORS

3. Pre-capillary pathogenetic form, arising again on the ground of primary organic or functional partial unit (gateway) in the air sacs ways of determining ventilation (external tons of breath deficiency alveolar hypoxia and reflex spasm of the arteries of the small circle (reflex citaev) (3). Thus, a second pre-capillary causes ventilation (alveolar – hypoxi) pathogenic form hypertension pulmonary (4). This form impacts at different stages of the diseases such as diffuse pneumosclerosis and other diseases like emphysema and atelectasis, bronchial asthma (4) and other diseases, "immured" pre-capillary arterioles.
4. Post capillaries pathogenetic form. This form is linked with the localization of the block in venous channel small circle: secondary arterial bloc. Of course, at a certain stage of the development process, joint the block and arterial the tideway of the small circle: secondary arterial block this pathogenetic form of clinically manifested in two versions: mild venous – pulmonary and heart. In the basis of the first clinical variant is increases of venous pressure due to "exhaustion" reserve capacity venous bed of a small circle associated with the "wall" pulmonary veins chronic inflammatory – sclerotic process. As a consequence – increase of pressure in the blood stream. The mild venous – pulmonary option include two clinical types: inside the vascular and out – vascular. The first is related to the primary pulmonary unit as a result of thrombosis. The second – most frequently in chronic poly – segmental pneumonia in the conditions of mild emphysema.

In the basis of cardiac pathogenetic variant lies outside pulmonary process leading to primary pulmonary venous hypertension. Most often, this type develops with mitral stenosis, because the stenosis of the left venous bed, venous hypertension, sclerosis (cardiac pneumosclerosis). This stasis leads to thrombosis, pulmonary, heart attack, stagnant pneumonia.

5. Hyperkinetic (Hyper perfusion) pathogenetic form. This form occurs in connection with pathologically increased perfusion, sometimes combined increases volume of blood circulation. This form is manifested in two clinical variants: a cordial and outside of the heart. The first option occurs when, in connection with certain congenital heart defects arterial and between the ventricular walls, cleft batalova duct, complete, or incomplete arterial – ventricle channel anastomosis between the lung and the subclavian artery, as well as operations: stenosis of the mouth of the pulmonary artery (operation bunting), shunt between artery system and arterial blood circulation. The second variant is associated with diseases involving acceleration of blood flow: hyperthyroidism, anemia etc. On both versions, the pressure in the small circle increases slightly.
6. It should be noted, however, that in the conditions of clinic, isolated pathogenic variant, is observed, if the latter is due to organic etiological factor. Pathogenic form, such clinical variants and styles, due to functional factor, usually, to a certain extent, combined, are intertwined. Therefore, it begs the appropriateness of the allocation of mixed pathogenetic form. The following tables present a classification scheme for hypertension in the pulmonary circulation.