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ETIOPATHOGENETIC STRUCTURE AND LABORATORY DIAGNOSIS OF ACUTE KIDNEY INJURY

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In pathogenetic terms, AKI should be considered as a set of mechanisms associated with damage to various compartments of the kidney and leading to organ dysfunction, primarily as a result of impaired glomerular filtration and excretion, with subsequent disturbances in systemic homeostasis [3,4,5,6].

Keywords: acute kidney injury, homeostasis, glomerular filtration, renal dysfunction, systemic disorders, renal diseases;

A separate problem is the timing of the onset of renal dysfunction after adverse effects or the development of acute disease (including renal disease). It has been conventionally accepted that the onset of acute renal dysfunction after exposure to a damaging factor should occur between 0 and 48 hours. 48 hours is the critical time required to record hypercreatininaemia (serum creatinine increase is delayed in relation to the damage). However, in a particular clinical situation, this time frame may vary considerably [1,2,3].

This issue should be decided on a case-by-case basis. The causes of AKI are divided into three main groups, which are the basis for the pathogenetic classification of this condition:

1) prerenal (associated with renal hypoperfusion);

2) Renal (associated with direct damage to the main compartments of the organ - intrarenal vessels, tubules, tubules and interstitium);

3) postrenal (associated with postrenal obstruction of urine flow) [4,5,6].

The mechanisms of AKI and reduced CKF in different compartments of the kidney, such as the vasculature, tubules, tubules and interstitium, may overlap to a large extent. Therefore, it is often impossible to distinguish between the different pathogenetic variants of AKI. For example, prerenal AKI may lead to ischaemic tubular necrosis (ITN) and progress to renal AKI [7,8,9,24,25].

The epidemiological pattern of acute respiratory failure depends significantly on the aetiology. In particular, the prevalence of acute respiratory failure depends on whether it occurs in the prehospital phase ('out-of-hospital acute respiratory failure') or develops in the hospital ('in-hospital acute respiratory failure') [10,11,12].

More precise information on the etiological structure of hospital-acquired acute respiratory failure can be obtained from the work of X. Zeng et al. [3]. According to the data obtained by the authors of this work, the most frequent complications of OPP are sepsis (68.4%), pneumonia (52.5%), cardiovascular pathology (47.4%). CKD is also frequently complicated by OPP ('OPP on CKD'; 45.6%) [14,15].

It is recommended to assess the physico-chemical properties of urine and microscopic examination of urine sediment for the diagnosis of OPD [13,23].

The study of the physico-chemical properties of urine at the patient's bedside is carried out by visual examination of the urine and its examination with test strips. This approach at this preliminary stage is already diagnostically significant in 97% of cases [11]. The colour and transparency of urine are visually assessed, and test strips are used to determine the presence of haematuria (haemoglobin and myoglobinuria), proteinuria, bilirubinuria, leukocyturia. Nowadays, the data of general urinalysis retain their differential diagnostic value [16,17].

Serum creatinine concentration is a low-sensitivity index. A distinct increase in serum creatinine level occurs when the global functional capacity of the kidneys decreases approximately twice [12,13,15].

In addition, the peculiarities of creatinine kinetics in the organism are such that the growth of its concentration is significantly (more than a day) delayed after a sudden decrease in SCF. At the same time, the slowest increase in Scr (at least in relative terms) occurs in patients with initially reduced renal function (initially low CKF), which may create problems in the diagnosis of 'RRF on CKD' or in the differential diagnosis of RRF and CKD [14,15].

Among the principles for stratifying the severity of AKI noted above, attention should be paid to the 'basal (baseline) level of renal function'. In the vast majority of patients with suspected AKI, neither Scr nor CKF baseline levels are known. In this regard, a table can be used to provide a quick reference for appropriate baseline Scr values. A value of 75 mL/min has been taken as such an appropriate baseline. [15,16,17].

In addition to the limitations of serum creatinine estimation for the diagnosis and stratification of the severity of ARF, another point worthy of attention in terms of monitoring the individual patient with ARF is the effect of hydration level on Scr. It has been shown that in hyperhydrated patients, serum creatinine levels can be significantly reduced, which naturally underestimates the severity of ARF [18,19,20,21,22].

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