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СОВРЕМЕННАЯ КОНЦЕПЦИЯ — ПОЧЕЧНЫЙ КОНТИНУУМ (ОСТРОЕ ПОВРЕЖДЕНИЕ ПОЧЕК, ОСТРАЯ БОЛЕЗНЬ ПОЧЕК, ХРОНИЧЕСКАЯ БОЛЕЗНЬ ПОЧЕК)

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Modern concept — renal continuum (acute kidney injury, acute kidney disease, chronic kidney disease)

Резюме

В лекции представлены современные представления о почечном континууме, отражающие взаимосвязи между острым повреждением почек (ОПП), острой болезнью почек (ОБП) и хронической болезнью почек (ХБП). Остается нерешенным вопрос ранней диагностики ОПП, несмотря на многочисленные исследования, посвященные биомаркерам острого повреждения почек. Недостаточно изучена эпидемиология, клиническое и прогностическое значение ОБП. Необходима осведомленность как врачей, так и пациентов, о «почечном континууме» и возможностях своевременной диагностики и профилактики почечных осложнений.

Ключевые слова: почечный континуум, острое повреждение почек, острая болезнь почек, хроническая болезнь почек

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Abstract

The lectures present the current understanding of the renal continuum, reflecting the relationship between acute kidney injury (AKI), acute kidney disease (AKD) and chronic kidney disease (CKD). The issue of early diagnosis of AKI remains unresolved, despite numerous studies on biomarkers of acute kidney injury. The epidemiology, clinical and prognostic significance of AKD have not been sufficiently studied. Awareness of both doctors and patients about the «renal continuum» and the possibilities of timely diagnosis and prevention of renal complications is required.

Key words: renal continuum, acute kidney injury, acute kidney disease, chronic kidney disease

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ACS — acute coronary syndrome, AKD — acute kidney disease, AKI — acute kidney injury, CCS — chronic coronary syndrome, CKD — chronic kidney disease

Current concepts in medicine are based, among other things, on the sequence and interconnection of the following events: cardiovascular continuum [1], cardiorenal continuum [2], cardiorenal syndrome [3] with the suggestion to distinguish a type when it is impossible to define the cause and the consequence [4]. “Peripheral” branches of continua keep improving. In cardiology, these are acute coronary syndrome (ACS) and chronic coronary syndrome (CCS) [5]. In nephrology — acute kidney injury (AKI), acute kidney disease (AKD), and chronic kidney disease (CKD) [6–8].

Chronic kidney disease is defined as decreased renal function or structural changes/markers of kidney injury for more than three months [8]. Acute kidney injury is rapidly decreasing renal function, and its development is limited to seven days [7]. Currently, the definition of acute kidney disease raises many questions [9]. There is a definition by KDIGO [7] where acute kidney disease is considered as AKD “AKI, or GFR <60 ml/min/1.73 m², or markers of kidney damage for ≤3 months, or decrease in GFR by ≥35% or increased SCr by >50% for ≤3 months. At the consensus conference of North American and European Nephrologists (Improving Global Outcomes (KDIGO) Consensus Conference, 2020), the KDIGO definition was revised, and it was recommended to stop using AKI as a synonym for AKD, considering AKI *only* as a disorder that developed over one week and lasts ≤3 months (“Avoid the use of ‘acute kidney injury (AKI)’ ‘as a synonym for AKD’. AKD refers to kidney diseases and disorders with a duration of ≤3 months, whereas AKI refers to kidney diseases and disorders with onset within 1 week”) [10].

AKD is widespread, but its significance is underestimated despite the increased risk of death and the development or aggravation of previous CKD [11–12].

Though seemingly straightforward, there are certain difficulties in the diagnosis of AKI associated with various approaches to the interpretation of the initial (basal) creatinine: from the time of hospitalization to parameters in medical records for seven days or even up to a year or more, which leads to a large scatter of data on epidemiology and diagnosis of AKI. Also, diagnosis of AKI by creatinine changes requires time, and exact calculation of diuresis can be performed with a permanent catheter placed in the bladder. A large number of studies on biomarkers are yet not included in common recommendations for the diagnosis of AKI [7]. In addition to

the well-studied NGAL (neutrophil gelatinase-associated lipocalin) and KIM-1 (kidney injury molecule-1), tissue inhibitor of metalloproteinase-2 (TIMP-2) and insulin-like growth factor-binding protein 7 (IGFBP-7) are of great interest as early markers of AKI that precede increased creatinine and (or) decreased urine output [13, 14]. One of the factors of AKI pathogenesis is renal hypoxia that causes increased erythropoietin level in blood serum, which can be used to predict AKI development [15]. The number of papers on biomarkers of acute kidney injury performed with the hope of finding “renal troponin” is growing. However, at present, biomarkers of AKI have not been widely used in clinical practice. At the same time, automated systems for predicting the development and early diagnosis of AKI were created; they demonstrated good results in the hospital population by optimizing the follow-up and examination of patients with high risk of acute kidney injury [16].

In several cases, it is impossible to assess what is going on at the time of hospitalization — AKI or AKD since it is not known for how long serum creatinine level has increased. Moreover, this situation is usually regarded as AKI in everyday clinical practice.

Numerous publications over the past 20 years include data on the clinical significance of AKI and CKD; epidemiology, clinical and prognostic value of AKD were not studied enough, which, in particular, was confirmed by the KDIGO consensus conference, 2020 [10]. Today there is no doubt that CKD can develop as one of AKI outcomes [17, 18]. According to a large American Registry, 31% of patients who had AKI develop CKD within one year [19]; repeated episodes of AKI exacerbate the situation [20]. It should be borne in mind that even if kidney function has recovered after AKI, there is a long-term risk of CKD that was demonstrated by a ten-year follow-up of patients who suffered acute kidney injury [21].

Unfortunately, the awareness of patients with the history of AKI about possible CKD development is extremely low [22]. Meanwhile, it is in this category of patients that it is important to know and correct risk factors for CKD. On the other hand, there is no doubt that CKD is a risk factor for AKI [23]. The problem is becoming increasingly important due to the increasing number of AKI cases worldwide, especially in countries with a high standard of living [24–25]. Today we can talk about a «renal continuum» that shows the interconnections between AKI, AKD and CKD (Fig. 1).

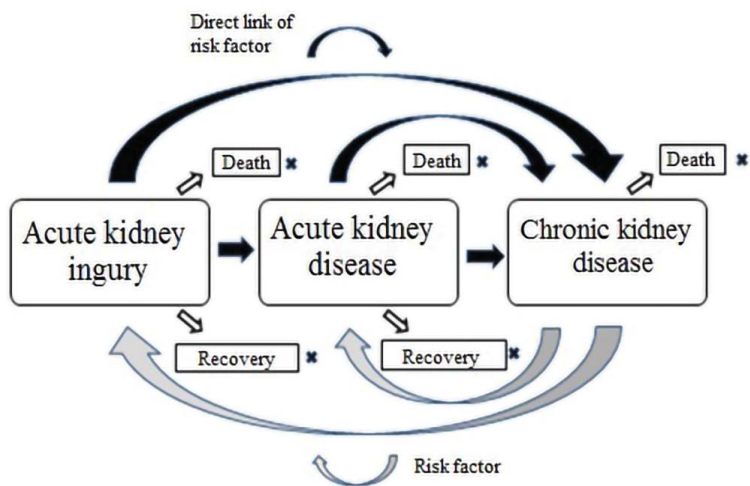


Figure 1. Renal continuum

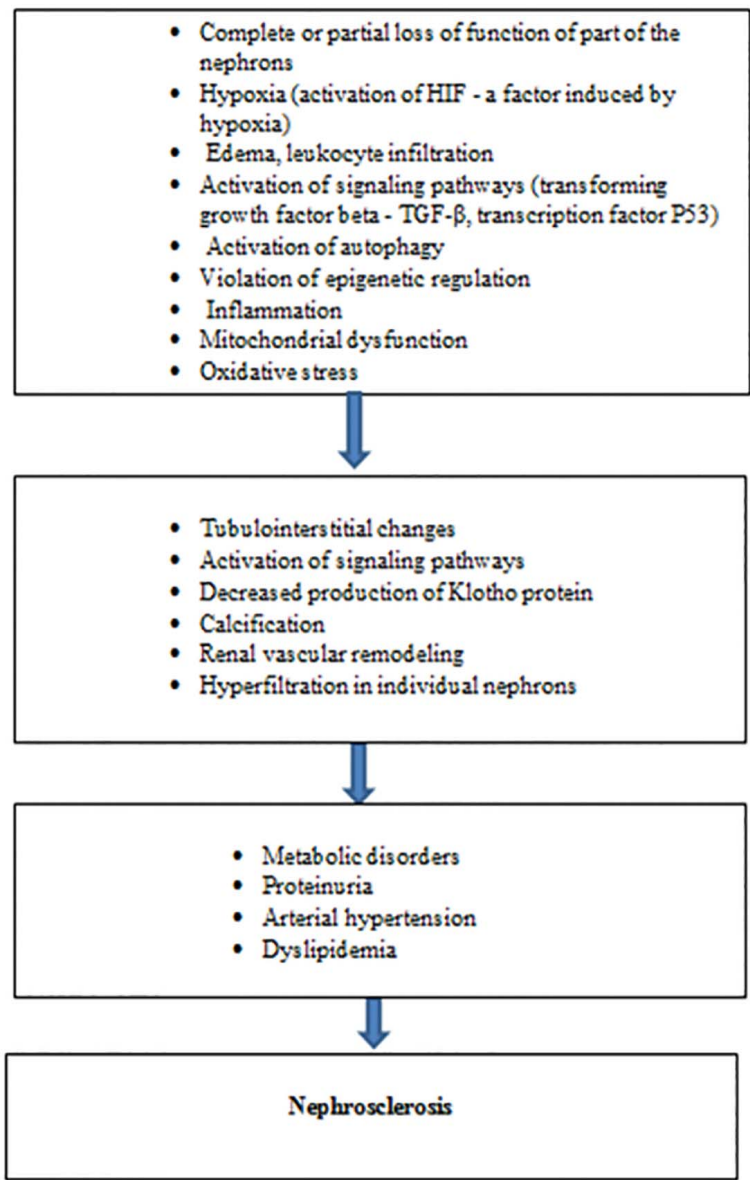


Figure 2. Mechanisms of formation of chronic kidney disease caused by acute kidney injury

Pathogenesis of CKD after AKI is multifactorial. Roles of hemodynamic factors, proteinuria, oxidative stress, metabolic disorders, inflammation, hypoxia, and other factors are discussed (Fig. 2).

A fairly complete picture of the relationship between acute kidney injury, acute kidney disease, and chronic kidney disease was developed. We definitely should try to establish the cause of AKI, AKD, or CKD. It is equally important to identify potentially modifying risk factors for CKD in patients after AKI or AKD, as well as factors predisposing to the development of acute kidney injury in patients with CKD. Both physicians and patients ought to be aware of the «renal continuum».

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