ATRIAL FIBRILLATION AND RENAL DYSFUNCTION: CURRENT STATE OF THE PROBLEM AND THE PROSPECTS OF FURTHER STUDY

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The review of literature is devoted to interrelations between atrial fibrillation (AF) and kidney function. The focus is on the most probable mechanisms of renal dysfunction in patients with atrial fibrillation such as fibrosis, inflammation, neurohormonal activation. The impact of central and renal hemodynamics disorders on chronic kidney disease development in atrial fibrillation patients is under consideration. The problem of atrial fibrillation in end-stage renal disease patients also gets the authors' attention. We identified unresolved issues and the prospects for their further research.

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AF — atrial fibrillation, ATII — angiotensin II, CAD — coronary artery disease, CHF — congestive heart failure, CKD — chronic kidney disease, CRP — C-reactive protein, CVD — cardiovascular disease, DM — diabetes mellitus, GFR — glomerular filtration rate, HTN — arterial hypertension, MMPs — matrix metalloproteinases, PIIINP — N-terminal propeptide of type III collagen, RAAS — renin angiotensin aldosterone system, SNS — sympathetic nervous system, TGF- β 1 — tissue growth factor β 1, TIMPs — tissue inhibitors of metalloproteinases, TNF- α — tumor necrosis factor- α .

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ФИБРИЛЛЯЦИЯ ПРЕДСЕРДИЙ И ФУНКЦИЯ ПОЧЕК: СОВРЕМЕННОЕ СОСТОЯНИЕ ПРОБЛЕМЫ И ПЕРСПЕКТИВЫ ДАЛЬНЕЙШЕГО ИЗУЧЕНИЯ

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Обзор литературы посвящен анализу взаимосвязей фибрилляции предсердий и функции почек. Рассмотрены наиболее вероятные механизмы формирования почечной дисфункции при фибрилляции предсердий: фиброз, воспаление, нейрогуморальная активация. Обсуждается роль нарушений центральной и почечной гемодинамики в развитии хронической болезни почек у больных мерцательной аритмией. Освещена проблема фибрилляции предсердий у пациентов с терминальной почечной недостаточностью. Определены нерешенные вопросы и перспективы дальнейших исследований по данной проблеме.

Atrial fibrillation (AF) is revealed in 1-2% of total population [1]. Severe complications and adverse outcome make AF lead among all arrhythmias, which is responsible for stroke, embolism, heart failure and a two-fold mortality increase [2].

Cardiovascular and renal diseases are known for their close and multifaceted relationship. Cardiovascular disease (CVD) is the most frequent cause of death for patients with chronic kidney disease (CKD). The latter at the same time is an independent risk factor for adverse outcome of coronary artery disease (CAD), arterial hypertension (HTN), congestive heart failure (CHF) [3].

Considerably less investigation is devoted to renal function in AF patients. The most evident reason for such relationship might be the commonness of pathologic processes underlying both AF and CKD, namely, HTN, atherosclerosis and diabetes mellitus (DM) [4]. Yet, the analysis of major prospective researches has shown that such association persists in patients without CVD and DM [5]. Molecular mechanisms have not conclusively been Российский кардиологический журнал 2016, 4 (132), Англ.: 198–201 http://dx.doi.org/10.15829/1560-4071-2016-4-eng-198-201

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revealed to give insight into the reason behind the relationship between AF and CKD including the concomitant CAD and HTN.

Renal function assessment is gaining a particular urgency on the background of wide introduction into clinical practice of new oral anticoagulants for stroke prevention in AF, as their safe administration is closely related to renal function due to a high renal clearance. The present literature review is devoted to the current state of the art.

Renal function at AF

In patients with AF, the occurrence of CKD is 18-21% [6, 7] while in total population it is 10-13% [5]. A number of researches have uncovered a direct relationship between AF frequency and severity of kidney dysfunction. Thus, as the glomerular filtration rate (GFR) decreased, the probability of AF development increased. To the contrary, AF presence suggested the most probable occurrence of CKD [5].

In patients with AF coupled with CHF a noticeable GFR decrease was associated with a high mortality risk [8]. AF presence was related to the increased level of serum

cystatin C [9], a more precise marker of renal function than creatinine [10]. In CAD patients the decrease of GFR calculated by cystatin C up to 30 ml/min/1.73 m² and less was followed by a threefold increase in the likelihood of AF development [4].

The direct dependence of albuminuria/ proteinuria degree on AF presence was revealed. Specifically, in patients with AF on the background of CAD and CHF, AF was linked to proteinuria development [4-6, 11]. In NIIGATA study (2009) proteinuria development risk in AF patients increased 2,2 times [5]. G. E. Gendlin at al. (2010) showed that in patients with permanent AF on the background of CHF albumin excretion with urine was almost two times higher than at sinus rhythm of 31,4 and 16,7 mg/l, respectively [8]. Injury of kidney parenchyma is likely to enlarge as AF is progressing: in paroxysmal AF proteinuria was found in 8.5% of cases, whereas in persistent AF — in 13,7% [11]. At the same time, some investigations do not give evidence in favour of AF and GFR association [7].

What are the plausible mechanisms of relationship between AF and renal function? Today there is no a single justified theory providing an explanation for the interdependence of these pathological processes. The analysis of the carried out investigations has made it possible to distinguish three closely interrelated pathophysiological processes that can be responsible for both AF and CKD pathway. These are tissue fibrosis, inflammation and neurohormonal activation. Besides, AF might be the cause of kidney injury due to changes in central and renal hemodynamics. Conversely, severe uremic poisoning instigates AF development.

Atrial and kidney fibrosis in AF and CKD

Atrial and kidney fibrosis is a morphological basis for both AF and CKD. Atrial fibrosis changes the cardiomyocyte electrophysiological properties and therefore becomes a substrate for AF emergence and maintenance [12]. Degradation of extracellular matrix proteins is generally caused by matrix metalloproteinases (MMPs). Thus, MMPs regulate the exchange of matrix, catalysing the breakdown of its components and changing the activity of growth factors and signalling molecules [13]. MMPs activity varies with recurrent rhythm disturbance including AF. In paroxysmal and persistent AF patients, MMP-9 level was twice as high while in permanent AF patients it was more than threefold in comparison to the healthy [14].

The tissue inhibitors of metalloproteinases (TIMPs) suppress MMP activity that allows treating them as fibrosis indicators. Reduced TIMP-4 and TIMP-1 levels are found to be associated with AF presence [15]. Obviously, a high MMP-9 activity and/or a drop in TIMP-1 regulation account for the excessive stimulation of collagen synthesis in the myocardium, which facilitates the atrial fibrosis formation and instigates AF development. Yet, the lack of tissue specificity of the markers in question does not permit to assert conclusively that their levels are related to atrial fibrosis rather than to fibrosis on the whole [16].

In kidneys over the whole nephron length, MMPs encoded by MMP-2, MMP-3, MMP-9 genes are revealed [17]. A number of researches have noted the link of MMP-2, MMP-9 activity variations and their TIMP-1, TIMP-2 tissue inhibitors with kidney fibrosis [18]. The increase in MMP-9 activity associated with microalbuminuria has been ascertained as well [17, 18].

Transforming tissue growth factor $\beta 1$ (TGF- $\beta 1$) is proinflammatory cytokine. Its excess expression stimulates the fibroblasts growth, extracellular matrix synthesis and fibrosis progressing. The level of TGF- $\beta 1$ in AF patients is known to be higher than in patients with sinus rhythm [19]. This, however, is inconsistent with the data of Cardiovascular Health Study (CHS, 2014) according to which the growth of TGF- $\beta 1$ was not associated with the new AF cases in the elderly [20]. On the other hand, it boosts glomerulosclerosis and interstitial fibrosis [21]. An only study on animals concerning the AF and kidney fibrosis interrelation showed that induced AF paroxysm raised TGF- $\beta 1$ level in renal tissue [22].

N-terminal propeptide of type III collagen (PIIINP) accounts for the collagen III content and is considered lately as a fibrosis marker [23]. The level of PIIINP in AF patients was higher than in patients with sinus rhythm. The increase of PIIINP in blood was suggested as a predictor of arrhythmia recurrence after cardioversion [24]. At the same time, a high concentration of PIIINP in urine was observed in the tubulointerstitional fibrosis cases. A double gain in PIIINP in urine regardless of GFR and albuminuria severity was followed by acceleration rate of CKD progressing in 1.2 times [25].

The data presented allow some fibrosis indicators (MMP, TIMP, TGF- β 1 μ PIIINP) to be considered as common kidney injure and AF markers though no special investigations were conducted earlier to substantiate this hypothesis.

Atrial inflammation and CKD

A convincing body of evidence has been amassed for inflammation in the atrial myocardium of the AF patients. The extent of structural changes in atria was found to depend on the level of inflammation markers [26]. AF presence as well as the risk of its recurrence after cardio-version were linked to the rise in high-sensitive C-reactive protein (CRP) [27]. In paroxysmal AF patients the level of tumor necrosis factor- α (TNF- α) in blood was 3.8 times higher than in the ones with sinus rhythm [28]. The interleukin-15 level directly correlated to the number of AF episodes [29]. Regaining the sinus rhythm resulted in the statistically significant decrease of elevated levels of inflammation markers [26].

However, cause-effect relationship between inflammation and AF has not been ultimately ascertained. The inflammatory cell infiltration and calcium overload are considered to contribute to atrial oxidative stress and fostering the atrial fibrosis development [12]. This may upset the mechanical and electrophysiological functions of atrium. Yet, the first paroxysm of arrhythmia is believed to occur independently while inflammation supports arrhythmia recurrence [26]. Finally, there is evidence for inflammation being a consequence of AF, whereas the rise in the inflammatory markers is due to the disease underlying AF development [30]. Inflammation does not seem to be the sole reason for atrial arrhythmogenic but is most likely to play a part in building up "readiness" for AF paroxysm development [26].

At the same time, the rise in the inflammatory markers is also observed in CKD patients [31]. CRP level has been found to correlate directly with the severity of albuminuria in hypertensive patients [32]. The increase in TNF- α and interleukin 1 and 6 content was associated with both the drop in GFR and the rise in albuminuria [33].

Therefore, there are grounds to believe that subclinical inflammation is one of the reasons for AF and CKD association. Whether it manifests itself as a single systemic process or these are separate unrelated inflammatory changes still remains unclear.

Neurohormonal activation at AF and CKD

Activation of the renin angiotensin aldosterone system (RAAS) is known to be typical for AF patients. Several mechanisms of structural and electrophysiological atria remodelling under the action of RAAS mediators aldosterone and angiotensin II (ATII) have been described. These are the increase of atrial tissue fibrosis, stretching and dilation of the atria, a direct impact on the ion channel function and cardiomyocyte intercellular interaction [34]. Besides, aldosterone induces local inflammation in coronary artery endothelium and myocardial perivascular spaces [2]. AF patients showed the increase of type 1 receptors to AT II density and a growing level of angiotensin converting enzyme in the left atrial tissue [35].

RAAS activation has been proved for CKD patients. In CKD patients, the plasma aldosterone level was found to grow with the disease worsening [36]. AT II is responsible for the glomerular arterioles spasm, predominantly of the efferent one, bringing about hyperfiltration, glomerular hypertrophy and, as a consequence, focal glomerulosclerosis [37].

It is reasonable to suggest, that the rise in RAAS activity, e.g. in high-renin form of HTN or CHF, stimulating AF development results in kidney fibrosis. Furthermore, ATII and aldosterone activate NAD(P)H oxidase to promote endothelial dysfunction as well as heart remodelling and kidney damage [38].

An elevated activity of sympathetic nervous system (SNS) can also provoke AF. Excessive catecholamines affecting myocardium and ion transport induce persistent tachycardia, heart rate variability violation and QT dispersion increase. This lowers the threshold of arrhythmias [39].

The role of SNS hyperactivity is also considered for CKD pathogenesis. It is common knowledge that 30% of norepinephrine excreted in urine and up to 70% of epinephrine come from blood to kidney. The remaining amount is released directly into the kidney from renal nerve endings. Sympathetic hyperactivity and RAAS acti-

vation underlie the glomerular efferent arteriola spasm and hyperfiltration development [40, 41]. Furthermore, catecholamines of high concentration are apt to exert a direct damage on renal interstitium. Parenchymal sclerosis and renal ischemia, in their turn, are primarily responsible for afferent signals to integrating brain nuclei in response to which the synthesis of catecholamines takes place by feedback mechanism [42].

Disorders of intrarenal and systemic hemodynamics in AF as the reason for renal dysfunction

Kidney embolism is another mechanism of AF negative effect on kidneys with a distinct cause-effect relationship. In population, renal artery embolism is encountered seldom enough. The incidence of peripheral embolism is 0.4% [43] and that of visceral embolism is 15% [43]. In AF patients, kidney embolism is registered in 3% of cases [44]. The most common reason for renal artery embolism is atrial flutter [43]. As a rule, acute renal failure is progressing in the case of large-sized emboli. Microembolization results in gradual decline in renal function due to ischemia, inflammation around emboli and hypertension [45]. However, this phenomenon is quite difficult to verify for lack of marked clinical manifestations, which does not allow us to judge reliably its prevalence.

In AF cardiac output decreases by 5 to 15% with further decline in the minute volume of the heart and blood pressure [46] which leads to renal hypoperfusion [47] with the development of ischemia and kidney damage.

AF in patients with end-stage renal disease

Of special consideration is the AF, which develops on the background of end-stage renal disease. 11-27% of patients on chronic haemodialysis are revealed to have a developing AF, which is 10-20 times more frequent than in general population [48]. Other evidence is reported for AF to be diagnosed in each sixth dialysis patient. The frequency of arrhythmias in dialysis patients amounts up to 40-76%[49]. In its turn, mortality of the patients in this category is twice as high as that of the patients without AF [50].

For the most part the researchers are agreed here that namely severe uremia and associated with it oxidative stress are the reasons for AF development [51]. Besides, during haemodialysis AF can be provoked by intradialytic hypotension, haemorrhage, electrolyte shifts as well as iatrogenic factors: receiving of psychotropic, anticonvulsive drugs and certain antibiotics. In the intradialytic interval, arrhythmias can occur because of hyperkalemia. AF is also developing at ineffective dialysis syndrome [52].

Conclusion

The literature analysis suggests pathogenic relationships to be found between AF and kidney disease. The core of a single pathophysiological mechanism can be attributed to systemic inflammation and neurohormonal activation, primarily RAAS and SNS, being responsible for excessive collagen deposition in the myocardium and electric atrial remodelling on the one hand, and kidney fibrosis, intraglomerular hypertension and endothelial dysfunction of glomerular capillaries, on the other. Most often, this mechanism is at work in atherosclerosis, HTN and CHF. AF may result immediately in renal damage due to renal artery embolism. Conversely, severe renal failure

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may initiate AF. Cause-effect relationship between AF and CKD still remain obscure. The idiopathic AF impact on renal function is not conclusively established, which calls for further specially designed epidemiological and experimental studies.

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