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Functional impairment of the kidneys with their acute damage in patients with peritonitis

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Abstract

The aim of our study was to study renal function and processes in patients with peritonitis. The results of a study of 32 patients with peritonitis compared with 11 healthy individuals showed that patients showed signs of acute kidney injury (AKI), judging by the presence of urinary syndrome. Two periods of APF have been identified: preazotemic and azotemic with the development of acute renal failure, in which the glomerular filtration rate decreases. Proteinuria and sodium excretion have been shown to increase, but not definitively. Whereas when converted to a unit of glomerular filtration rate, they increase significantly. The proposition about the primary tubular-tubular compensation mechanism, which turns into the tubular-glomerular one, which reduces glomerular filtration and prevents sodium loss, is substantiated.

Key words: peritonitis; acute kidney injury; acute renal failure; sodium excretion; proteinuria.

It is known that various causes can cause functional disorders of the kidneys, defined as acute kidney injury syndrome (AKI) [1, 2]. Signs of pathology are also recorded in peritonitis with the possibility of AKI [2, 3, 4] up to the development of acute renal failure (ARF).

However, most patients do not develop ARF, and the signs of renal impairment that are detected in such patients are considered manifestations of the ARF syndrome. Previously, we studied the signs of renal impairment in peritonitis during the development of ARF and suggested that it be considered as the initial stage of kidney damage, which only in some patients with peritonitis progresses to ARF [5].

We have shown that in all patients with peritonitis, APN can be diagnosed by the presence of urinary syndrome [6]. However, at the same time, it is not known what disorders may occur in APN from the renal processes - glomerular filtration rate and especially reabsorption. Meanwhile, it is known that the most sensitive to nephron damage is sodium transport in the renal tubules [7, 8]. In connection with the above, we have conducted studies on the functional state of nephrons in patients with peritonitis.

Materials and methods of the study. We examined 11 healthy individuals and 32 patients with peritonitis. We took into account general clinical indicators and functional characteristics - daily diuresis, urinary creatinine and sodium concentration, protein, number of erythrocytes and leukocytes, urine density and well-known laboratory methods.

In the blood plasma, the concentration of creatinine, urea and the content of erythrocytes and leukocytes were determined. The glomerular filtration rate (GFR) was calculated, and the excretory function indicators were converted to 100 ml of GFR.

All patients were divided into two groups - without signs of ARF (24) and with signs - 8.

The data presented in Table 1 indicate that the main features that distinguish patients with ARF from those without ARF are significant differences in plasma creatinine and urea concentrations, which significantly exceed normal values. This clearly indicates that the level of GFR in ARF decreases with the development of ARF.

Table 1 - Some blood parameters in patients with ARI with peritonitis

Study groups	Creatinine content in blood plasma, μmol/l	Urea content in blood plasma, mlmol/l	Leukocyte count, thousand/ml	Red blood cell count, million/ml	Platelet count. thousand/ml
CONTROL	72,6 ± 1,5	5,7 ± 0,3	6,0 ± 1,7	5,1 ± 0,1	250 ± 5
Patients with peritonitis with AKI	81,1 ± 4,5	6,0 ± 0,6	7,8 ± 2,2 $P_1 < 0,05$	4,7 ± 0,3	270 ± 11
Patients with peritonitis with ARF	150,3 ± 5,8 $P_1 < 0,001$	17,1 ± 2,3 $P_1 < 0,01$	11,2 ± 2,5 $P < 0,05$	4,0 ± 0,5 $P < 0,01$	300 ± 25

P – significance of the difference relative to control,

P_1 – reliability of the difference between AKI and ARF

At the same time, in ARF, the number of leukocytes increases significantly, which is a direct consequence of the inflammatory process in the abdominal cavity and indicates the development of signs of systemic inflammation, due to which kidney disorders certainly occur, as evidenced by the data presented in Table 2. Thus, in AKI and ARF, proteinuria develops and increases, as well as sodium excretion, erythrocytes and leukocytes also appear in the urine, at the same time, the density of urine increases against the background of a decrease in daily diuresis. All this occurs against the background of a tendency to decrease GFR in AKI and a significant drop in ARF by more than three times. In general, these data confirm the presence of signs of urinary syndrome in patients with peritonitis, the degree of which increases in ARF. It should be noted that the appearance of erythrocytes and leukocytes in the urine indicates the presence of urinary syndrome. Moreover, the increase in the number of leukocytes exceeds erythrocytes. In our opinion, this confirms that although glomerular capillaries can be damaged in AKI, other disorders prevail, probably in the tubular part, in which, due to cell damage, there is a consequence of active leukocyte emigration as a combination of systemic inflammation and local alteration phenomena.

Table 2 - Indicators of renal excretory activity in patients with peritonitis, M±m

Study groups	Daily diuresis, ml	Protein excretion, g/24 h.	Sodium excretion, mmol/24 h.	Potassium excretion, mmol/24 h.
CONTROL	1291,1 ± 20,1	0	60,1 ± 6,2	40,1 ± 0.4
Patients with peritonitis with AKI	1195,5 ± 25,2	2,91 ± 0,35	75,4 ± 4,8	43,7 ± 0.5
Patients with peritonitis with ARF	690,1 ± 23,4 P < 0,001 P < 0,01	3,2 ± 0,4	80,1 ± 5,6 P < 0,01	50,1 ± 4,9

P – significance of the difference relative to control,
P_1 – reliability of the difference between AKI and ARF

It should be noted that the data presented in the table indicate both a violation of the renal glomeruli and, most likely, the tubular part of the nephron. Moreover, filtration disorders prevail over the degree of changes that characterize the tubular part of the nephron, as evidenced in particular by a rather moderate increase in sodium excretion and even an increase in urine density. Although the latter clearly indicates that the stage of oliguria begins in ARF. Meanwhile, it has long been known that the most sensitive process in the nephron is sodium reabsorption, which should lead to an increase in its excretion [8, 9, 10]. In addition, the data presented in Table 2 indicate that when GFR decreases threefold, sodium excretion increases

by only 26%. In this regard, we calculated the excretion of protein, sodium and potassium, as well as leukocytes and erythrocytes in relation to GFR values.

Table 3 - Kidney function indicators per 100 ml of glomerular filtrate

Study groups	Glomerular filtration rate, ml/min	Protein excretion, g/100 ml	Sodium excretion, mmol/100ml	Potassium excretion, mmol/100ml	Urine density
CONTROL	115,1 ± 1,3	0	0,51 ± 0,04	0,21 ± 0,03	1018,1± 0,12
Patients with peritonitis with AKI	98,1 ± 4,0	1,91 ±0,31	0,75 ± 0,11 P < 0,05	0,27 ± 0,04	1015,3± 2,2
Patients with peritonitis with ARF	70,1 ± 1,8 P < 0,001 P_1 < 0,05	4,51 ± 0,57	0,99 ± 0,1 P < 0,001 P_1 < 0,05	0,31 ± 0,1	1031,1± 2,1 P < 0,05 P_1<0,01

P – significance of the difference relative to control,
P_1 – reliability of the difference between AKI and ARF

The data presented in Table 3 indicate that conditionally each functioning nephron loses protein, sodium, potassium more in AKI and especially in ARF. At the same time, the excretion of leukocytes and erythrocytes increases. Previously, we defined the calculations of proteinuria on GFR as specific proteinuria [9, 11]. Of course, the increase in protein excretion is most likely associated with glomerular damage, which increases its entry into the ultrafiltrate in such an amount, and the mechanisms of protein reabsorption are insufficient to compensate for glomerular disorders, i.e. proteinuria is of mixed glomerular-tubular genesis [8]. By the way, erythrocyturia also indicates glomerular disorders, since it is a direct consequence of glomerular damage. An increase in leukocyturia indicates nephron damage, although its mechanism is complex, because leukocytes activated during inflammation can also emigrate to other segments of the nephron. However, the most interesting was the increase in sodium excretion by functioning nephrons. Although the total sodium excretion increased moderately, which, in our opinion, indicates that even with nephron damage, the general volume-regulating function of the kidneys is not significantly impaired, and this prevents significant renal sodium losses. But at the same time, when converted to 1 ml of glomerular ultrafiltrate, it is shown that each nephron loses more than three times as much sodium. This allows us to state that in patients with peritonitis, the processes of tubular sodium reabsorption are significantly impaired in AKI, which indicates disorders that occur at the tubular level [12, 13], probably due to a violation of energy-dependent sodium transport. It is important that at this stage of the disease, signs of

damage to the tubular part of the nephron are detected, while the value of GFR practically does not change. Moreover, it seems that the proximal part of the tubules is damaged mainly, since the increase in sodium excretion occurs simultaneously with the increase in proteinuria, which indicates that protein reabsorption in the proximal tubules does not provide protein return. These data confirm our opinion that in acute pathological processes in the kidneys, the proximal tubules are mainly damaged [8, 9]. Whereas in such cases compensation occurs due to increased sodium reabsorption in the distal tubules, i.e. the mechanism of tubule-tubule balance is triggered. This idea is confirmed by the increasing concentration of potassium in the urine, which is secreted in the distal tubules in exchange for sodium reabsorption. Additional evidence is the increase in the specific gravity of urine, the increase of which occurs precisely in the distal tubules and collecting ducts, as a result of which diuresis even decreases, which is a consequence of increased water reabsorption [11].

Thus, the results obtained indicate that most patients with peritonitis develop AKI syndrome, which in its development goes through two stages [12]. In the first, the tubular part of the nephron is mainly damaged, which is manifested by the appearance of urinary syndrome, this is the pre-azotemic stage. In the second azotemic stage of AKI development, tubular disorders and urinary syndrome are accompanied by a decrease in GFR, which leads to retention azotemia, i.e. ARF develops [13, 14].

The above necessitates the identification of early manifestations indicating the possibility of AKI transitioning to ARF, which will allow monitoring the dynamics of the pathological process in the kidneys and taking them into account in the treatment of AKI and prevention of ARF.

References

1. Vozianov O.F., Gozhenko A.I., Fedoruk O.S. Acute renal failure. – Odesa, 2004. – 488 p. (in Ukrainian)

2. Waikar S.S., Lu K.D., Chertow. Diagnosis, epidemiology and outcomes of acute kidney injury. Clin. J. Am. Soe Nephrol, 2008, 3(3), 844-861.

3. Peerapornratana S., Manrique-Caballero C.L., Gomez H. & Kellum J.A. (2019). Acute kidney injury from sepsis: current concepts, epidemiology, pathophysiology, prevention and treatment. Kidney International Supplements, 10(1), 1-18.

4. Bouman C., Kellum J.A., Lanuere N., Levin N. Definition of acute renal failure. Acute Dialysis Quality initiative. 2 nd International Consensus Conference, 2002.

5. Saensus M.A., Fedoruk O.S. Features of the functional state of the kidneys in peritonitis. //Current problems of transport medicine. – 2025. – No. 2 (80). – P. 69-71. (in Ukrainian)

6. Gozhenko A.I. Energy supply of the main renal functions and processes in norm and with kidney damage. Abstract of the doctoral dissertation. – Kyiv, 1987. (in Russian)

7. Pathophysiology of the kidneys / M.V. Kryshtal, A.I. Gozhenko, V.M. Sirman: Odesa, "Phoenix", 2020. – 144 p. (in Ukrainian)

8. Gozhenko A.I. Kidneys and homeostasis // Actual problems of transport medicine. 2023; 1-2 (71-72):13-29. (in Ukrainian)

9. Functional renal reserve: physiology, pathophysiology and diagnostics. Kyiv, 2019. – 151 p. (in Ukrainian)

10. Poston J.T. & Koyner J.L. (2019). Sepsis associated acute kidney injury. BMJ, 364, k4891.

11. Gozhenko A.I., Pishak V.P., Rogovy V.E. Tubulo-interstitial syndrome. – Chernivtsi: Medical Academy, 2002. – 221 p. (in Ukrainian)

12. Gozhenko A.I., Fedoruk O.S. Functional classification of renal failure //Physiological Journal. – 2000. – Vol. 46, No. 2. – P. 114. (in Ukrainian)

13. Gozhenko A.I., Fedoryuk A.S. Classification of renal failure // Urology. - 2011. - No. 5. - P. 35-38. (in Russian)

14. Adiyeke E.et. al. (2023). Clinical Courses of Acute Kidney Injury: A Multistate Model Analysis. arXiv: 2303.06071.

15. Zarbock A., Gomez H. & Kellurn J.A. (2014). Sepsis-induced acute kidney injury revisited: pathophysiology, prevention and future therapies. Current Opinion in Critical Care, 20(6), 588-595.