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Renal dysfunction in young adults with severe community-acquired pneumonia

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ABSTRACT

BACKGROUND: Pneumonia continues to be a serious public health concern in the 21st century because of its high prevalence and significant mortality, especially in adults, where the incidence can be as high as 14 cases per 1000 people. Community-acquired pneumonia is generally mild but can potentially lead to dangerous complications in elderly and immunocompromised patients, requiring hospitalization in up to 50% of cases and with a mortality rate of 0.7 per 1000 people annually. Acute kidney injury is a serious complication, occurring in up to 52.5% of cases of severe community-acquired pneumonia.

THE AIM of the study was to assess the renal function in young patients (18–44 years) with severe community-acquired pneumonia and without a history of underlying chronic diseases.

METHODS: The renal function of 220 patients aged 18–44 years without preexisting chronic comorbidities who received treatment in the intensive care unit (ICU) for severe community-acquired pneumonia (SCAP) between 2011 and 2017 was evaluated.

RESULTS: Severe community-acquired pneumonia in young adults without chronic comorbidities was associated with a diastolic blood pressure below 60 mmHg in 146 patients (66.3%) and bilobar or multilobar lung involvement in 141 patients (64%). Acute kidney injury (AKI) complicated SCAP in 25.4% of cases. In SCAP, AKI was more frequent in viral-bacterial cases (31.91%) than in bacterial cases (20.8%) ($p < 0.05$). The frequency of AKI increased with SCAP severity, with AKI developing in 85.7% of patients within an average of 2.5 (2–3) days after initiation of mechanical ventilation ($p < 0.01$). SCAP complicated by AKI was associated with a significant increase in hospital stay, ICU stay, and mortality. The median hospital stay of patients with SCAP was 23 (18–30) days in those without AKI and 28 (20–43) days in those with AKI ($p < 0.01$). Additionally, the median ICU stay for patients with SCAP was 3 (2–4) days in those without AKI and 4 (3–7) days in those with AKI ($p = 0.001$). Mortality was significantly higher in SCAP patients with AKI (10.7%) than in those without AKI (0.6%) ($p < 0.01$). Complete renal function recovery was observed in 99.52% of surviving patients, with normal urine output and serum creatinine levels.

CONCLUSIONS: Despite advances in early diagnosis and modern pharmacotherapy, community-acquired pneumonia remains a clinically significant condition. Severe cases are characterized by respiratory failure and dysfunction of multiple organs and systems, including the kidneys. The high incidence of AKI in SCAP indicates the need for heightened awareness and early detection of its potential occurrence.

Keywords: community-acquired pneumonia; viral-bacterial pneumonia; respiratory failure; acute kidney injury; kidney failure; creatinine; glomerular filtration rate; urine output.

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Нарушения функции почек при внебольничной пневмонии тяжелого течения у лиц молодого возраста

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АННОТАЦИЯ

Введение. Пневмония остается серьезной проблемой в XXI веке из-за высокой распространенности и значительного числа летальных исходов, особенно среди взрослых, где заболеваемость может достигать 14 случаев на 1000 человек. Течение внебольничной пневмонии обычно легкое, но может привести к опасным осложнениям у пожилых и иммунодефицитных пациентов, требуя стационарного лечения до 50% случаев и с смертностью 0,7 на 1000 человек в год. Одним из серьезных осложнений ВП является острое повреждение почек (ОПП), которое может развиваться до 52,5 % случаев при тяжелой форме.

Цель — изучить функцию почек при внебольничной пневмонии тяжелого течения у пациентов молодого возраста (18–44 лет) без сопутствующих хронических заболеваний.

Метод. Исследована функция почек при внебольничной пневмонии тяжелого течения у 220 пациентов в возрасте от 18 до 44 лет без сопутствующих хронических заболеваний, получавших лечение в отделении реанимации и интенсивной терапии с 2011 по 2017 г.

Результаты. Выявлено, что у 146 (66,3 %) пациентов молодого возраста без сопутствующих хронических заболеваний тяжесть течения внебольничной пневмонии сопровождается низким диастолическим артериальным давлением (менее 60 мм рт. ст.), у 141 (64 %) больного — двух- или многодолевым поражением легочной ткани. Тяжелое течение внебольничной пневмонии осложняется острым повреждением почек в 25,4 % случаев. При вирусно-бактериальной этиологии внебольничной пневмонии тяжелого течения частота встречаемости острого повреждения почек составляет 31,91 %, при бактериальной этиологии — 20,8 % ($p < 0,05$). Острое повреждение почек имеет тенденцию к увеличению распространенности с увеличением тяжести течения внебольничной пневмонии. Острое повреждение почек развивается в 85,7 % случаев в среднем через 2,5 (2–3) дня после начала искусственной вентиляции легких ($p < 0,01$). Показано, что осложнение внебольничной пневмонии тяжелого течения в виде острого повреждения почек статистически значимо увеличивает продолжительность лечения в стационаре, в отделении реанимации и интенсивной терапии, а также летальность. Медиана пребывания больных в стационаре при внебольничной пневмонии тяжелого течения без острого повреждения почек составила 23 (18–30) дня, а с острым повреждением почек — 28 (20–43) дней ($p < 0,01$). Медиана длительности пребывания в отделении реанимации и интенсивной терапии больных, страдающих внебольничной пневмонией тяжелого течения без острого повреждения почек, составила 3 (2–4) дня, с острым повреждением почек — 4 (3–7) дня ($p = 0,001$). Летальность в группе больных, страдающих внебольничной пневмонией тяжелого течения с острым повреждением почек, составила 10,7 %, без острого повреждения почек — 0,6 % ($p < 0,01$). У 99,52 % выздоровевших пациентов наблюдалось полное восстановление функции почек с нормальным объемом выделяемой мочи и уровнем сывороточного креатинина.

Заключение. В целом, несмотря на своевременность диагностики и достижения современной фармакологии, внебольничная пневмония сохраняет актуальность и при тяжелом течении характеризуется не только дыхательной недостаточностью, но и нарушением функций других органов и систем, в том числе почек, а частая встречаемость острого повреждения почек при тяжелом течении внебольничной пневмонии должна обуславливать настороженность в отношении возможности его возникновения.

Ключевые слова: внебольничная пневмония; вирусно-бактериальная пневмония; дыхательная недостаточность; острое повреждение почек; почечная недостаточность; креатинин; скорость клубочковой фильтрации; объем мочи.

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BACKGROUND

In the 21st century, pneumonia remains a significant challenge for the healthcare system owing to its widespread prevalence across diverse populations. However, despite the advancements in modern antibacterial agents and respiratory support technologies, the mortality rate linked to community-acquired pneumonia (CAP) continues to be high. In adults, its incidence is up to 14 cases per 1000 people. The World Health Organization reports that pneumonia and other lower respiratory tract infections were linked to significant mortality in 2019. This group of diseases ranked fourth among the primary causes of death due to infectious diseases.[1]

According to official data, the average CAP incidence in Russia was 391.8 per 10,000 people between 2011 and 2018. However, the COVID-19 pandemic has altered these figures substantially. In 2020, the CAP incidence in Russia increased by 4.74 times compared to the pre-pandemic period, reaching 1856.2 per 10,000 people. The statistics included SARS-CoV-2-caused pneumonia cases. The incidence rate was 2.93 times higher in 2021 than it was prior to the pandemic, at 1148.4 per 10,000 persons. SARS-CoV-2-caused pneumonia has been counted individually from 2021[1].

According to reports, the incidence of pneumonia ranges from 30‰ to 50‰ in conscripts and from 4‰ to 6‰ among contract servicemen—a significantly lower rate. Given its high prevalence, this indicates the relevance of examining the course of pneumonia in conscripts. A thorough understanding of severe community-acquired pneumonia (SCAP) in young populations is essential for enhancing therapeutic outcomes in military conscripts [2, 3].

Although CAP is typically not severe, older adults with concomitant respiratory or cardiovascular disease and patients with immunodeficiencies stand a risk of developing life-threatening complications. Up to 50% of patients require inpatient care, with the annual mortality rate reaching 0.7 per 1000 persons. Every fourth to fifth hospitalized patient requires treatment in the intensive care unit (ICU) [4].

CAP can be accompanied by potentially life-threatening complications, including pleurisy, empyema, acute respiratory distress syndrome, sepsis, disseminated intravascular coagulation, and acute kidney injury (AKI). All these complications pose substantial health risks and require comprehensive and timely treatment.

AKI is associated with impaired renal excretory function, leading to the accumulation of uremic toxins and water, hyperhydration and depression of the heart muscle, and combined (hydrostatic and non-hydrostatic) pulmonary edema. Mechanical ventilation exacerbates damage to the lungs, kidneys, and heart by triggering the release of inflammatory mediators, often requiring prolonged respiratory therapy with artificial ventilation in renal dysfunction patients. Thus, SCAP is characterized by a vicious circle of mutual damage to the lungs and kidneys [5].

AKI is a frequent complication of CAP. Research indicates that the probability of mortality in CAP and AKI patients, especially those on mechanical ventilation, increased dramatically [6], and CAP was complicated by AKI in 16%–25% of cases [7]. In SCAP patients, the incidence of AKI was up to 52.5% [8], surpassing 70% in those with influenza A (H1N1) virus-induced SCAP [9]. However, despite existing publications, data on renal function in young patients (18–44 years) with SCAP without chronic comorbidities remain limited.

This study aimed to evaluate renal function in young patients (18–44 years) with SCAP without chronic comorbidities.

Study objectives:

1. To evaluate the clinical characteristics of young patients with SCAP complicated by AKI.

2. To determine the AKI incidence in young patients with SCAP.

3. To determine the effect of mechanical ventilation on AKI development in young patients with SCAP.

4. To assess the influence of AKI on the length of hospital and ICU stay, as well as on mortality rates, in young patients with SCAP.

MATERIALS AND METHODS

This retrospective cohort observational controlled study included 220 patients aged 18–44 years who were diagnosed with SCAP and received treatment from 2011 to 2017 at the anesthesiology and reanimation unit and intensive care unit of Military Clinical Hospital No. 1586 (Podolsk).

Exclusion criteria: a history of acute and/or chronic kidney disease; diabetes mellitus; atherosclerosis; cancer; prior large vessel surgery; use of nephrotoxic drugs (aminoglycosides, amphotericin B); and a history of radiocontrast use, regardless of the duration of its use.

The study patients were divided into two groups: group 1 included 164 patients with SCAP without AKI; group 2 included 56 patients with SCAP and AKI.

Diagnosis and treatment were conducted in accordance with the current Russian clinical guidelines. Clinical severity and prognosis were assessed, and treatment options were

[1] The WHO published statistics on the leading causes of death and disability worldwide for the period 2000–2019. Available at: https://www.who.int/ru/news/item/09-12-2020-who-reveals-leading-causes-of-death-and-disability-worldwide-2000-2019 Date of access: December 18, 2024.

selected based on recommended clinical and laboratory indicators (criteria) as well as multifactorial scales: SMRT-CO (systolic blood pressure, multilobar infiltration, respiratory rate, tachycardia, confusion, oxygenation) [10], SOFA (sepsis-related sequential organ failure assessment) [11], CURB-65 and CRB-65 (confusion of consciousness, nitrogen content, respiratory rate, blood pressure, age 65 years or older) [12], and SIRS (systemic inflammatory reaction syndrome) [13].

The diagnosis, stage stratification, and AKI treatment were carried out based on current Russian [14] and international recommendations [15, 16] applicable at the time of diagnosis and treatment. AKI was defined according to the KDIGO (Kidney Disease: Improving Global Outcomes) system [16] as any of the following alterations in parameters: increase in serum creatinine (*SCr*) by ⩾0.3 mg/dL (⩾26.5 μmol/L) within 48 h; or increase in *SCr* to ⩾1.5 times baseline, which is known or presumed to have occurred within the prior seven days; or urine volume <0.5 mL/kg/h for 6 h.

In most cases, data pertaining to baseline *SCr* levels were missing; therefore, estimated baseline *SCr* (*ebSC*) levels were used [15]. The following formulas were employed for the calculations:

$$\Delta SCr \text{ (абс.)} = SCr - pSCr, \quad (1)$$

where Δ*SCr* (abs.) is the absolute change vs. the estimated baseline *SCr*, μmol/L;

$$\Delta SCr(\text{отн.}) = \frac{SCr}{pSCr'}, \quad (2)$$

where Δ*SCr* (rel.) is the relative change vs. the estimated baseline *SCr*.

The baseline *SCr* levels are presented in Table 1.

Following an AKI diagnosis, all patients received primary intensive nephroprotective drug therapy. In case of AKI progression, they underwent renal replacement therapy (RRT) in accordance with the current Russian and international guidelines. Nephroprotective drug therapy was administered based on established principles and aimed to alleviate hypoxia of the tubular epithelial cells.

Statistical analysis was performed using Excel 2013 of Microsoft Office 2013 and SPSS Statistics packages. Inter-group differences were evaluated using the nonparametric Mann–Whitney *U*-test. The Spearman rank correlation coefficient was used to evaluate the strength of the relationship between the parameters. Results are presented as *Me* (Q_{25}; Q_{75}), where *Me* is the median and Q_{25} and Q_{75} are the lower and upper quartiles, respectively. The Pearson χ^2-test was used to ascertain the correlation between the two categorical variables. Differences were considered statistically significant with p-value < 0.05.

The study was conducted with the approval of the Independent Ethics Committee at the F.P. Gaaz Moscow Medical and Social Institute (Protocol No 1 dated May 18, 2020).

RESULTS AND DISCUSSION

The study population comprised male SCAP patients aged 18 to 44 years with a median age of 20 years (19; 22), representing the typical demographic of individuals receiving treatment in military hospitals. Among patients without chronic comorbidities, SCAP was associated with low (less than 60 mm Hg) diastolic blood pressure (DBP) in 146 (66.3%) patients and with bilobar or multilobar lung tissue damage in 141 (64%) patients. While only 10.9% and 23.1% of patients in groups 1 and 2 had systolic blood pressure (SBP) below normal, respectively, 64% and 73.2% of patients in groups 1 and 2 exhibited DBP below normal.

In group 2, nearly all clinical and laboratory criteria of disease severity were observed in a greater number of patients and manifested with increased intensity. Hypotension was more prevalent and lung damage was more extensive in this group. Leukopenia was seen in 51.78%

Table 1. Estimated basal serum creatinine level, μmol/L (adapted from [15] R. Bellomo et al., 2004)
Таблица 1. Расчетный базальный уровень сывороточного креатинина, мкмоль/л (адаптировано из [15] R. Bellomo и соавт., 2004)

Age, years	Men (excluding black individuals)	Women (excluding black individuals)
20–24	115	88
25–29	106	88
30–39	106	80
40–54	97	80
55–65	97	71
> 65	88	71

of the patients, indicating a severe form of the disease. Thus, each patient exhibited one or more SCAP markers (Table 2).

The study revealed a weak direct correlation (Spearman) between CAP severity and AKI incidence using the SIRS scale (r = 0.192; p < 0.05). A moderate direct correlation was observed using the SOFA scale (r = 0.414; p < 0.05). Additionally, weak direct correlation was found using the SMRT-CO scale (r = 0.205; p < 0.05) and the CURB scale (r = 0.221; p < 0.05), while the CRB-65 scale showed a very weak positive correlation (r = 0.001; p < 0.05).

Out of 208 patients, 197 (89.5%) exhibited two or more SIRS criteria for pneumonia severity: ≥38°C (febrile temperature) or ≤36°C (hypothermia); heart rate ≥90/min (tachycardia); respiratory rate ≥20/min (tachypnea) or hyperventilation with blood carbon dioxide content ≤32 mm Hg; leukocyte count ≥12 × 10^9/L (leukocytosis) or ≤4 × 10^9/L (leukopenia) or >10% bands (left shift). The proportion of patients demonstrating two or more criteria was 69.6% and 34.7% in groups 2 and 1, respectively. Among the 208 patients assessed for SCAP severity using the SOFA score, 100 patients (45.4%) had a score of 2 or higher. Of these, 40% developed AKI. In contrast, only 11.11% of patients with SCAP and a SOFA score of 1 developed AKI. Compared to 38.4% of patients in group 1, 76.9% of patients in group 2 had a SOFA score of 2 or above. The pneumonia severity was also assessed using the SMRT-CO scale (n = 208), where patients with a score of 3 were diagnosed with AKI in 33.3% of cases, and those with a score of 4 were diagnosed with AKI in 43.3% of cases. Sixty-six percent of patients in group 1 had a score of 2, and 59.6% of patients in group 2 exhibited a score of three or higher. In patients with SCAP, the incidence of AKI increased in parallel with higher CURB scores (n = 213): AKI was identified in 20.9% of patients with a SCAP score of 0–1, in 31.25% with a score of 2, and in 75% with a score of 3 or more. In contrast to just 1.9% of patients in group 1, 16% of patients in group 2 had a score of three or higher. AKI developed in 29.16% of patients with a score of 0 and in all patients (100%) with a score of 3–4 on the CRB-65 scale (n = 213), according to Table 3.

If the results of microbiology and virology testing was negative, laboratory parameters (such as leukocyte count, changes in leukocyte differential, C-reactive protein,

Table 2. Clinical and laboratory parameters of pneumonia severity in the examined
Таблица 2. Клинические и лабораторные показатели тяжести пневмонии у обследуемых больных

Parameter	All patients		Group 1		Group 2		χ^2	p
	n	%	n	%	n	%		
Respiration rate > 30/min	12	5.45	6	3.65	6	10.7	4.030	0.045
Blood oxygen saturation (pulse oximetry) SpO_2 < 90%	34	15.45	22	13.4	12	21.4	2.052	0.15
SBP < 90 mm Hg	31	14.09	18	10.9	13	23.1	5.165	0.02
DBP < 60 mm Hg	146	66.36	105	64.0	41	73.2	1.579	0.20
Bilobar or multilobar lung damage	141	64.09	99	60.36	42	75	3.885	0.04
Extrapulmonary site of infection (brain abscess, meningitis, etc.)	24	10.90	19	11.58	5	8.9	0.303	0.58
Anuria	4	1.81	–	–	4	7.14	–	–
Leukopenia < 4×10^9/L	74	33.63	45	27.4	29	51.78	11.085	< 0.001
Arterial blood oxygen saturation SpO_2 < 90%*	11	18.33	2	4.4	9	50	19.39	< 0.001
Arterial oxygen tension PO_2 < 60 mm Hg*	19	31.66	10	22.2	9	50	5.263	0.022
Hemoglobin level < 100 g/L	22	10	13	7.90	9	16	3.077	0.080
Hematocrit < 30%	18	8.1	8	4.80	10	17.8	9.361	< 0.05
Acute renal failure (anuria, *SCr* > 0.18 mmol/L, urea > 15 mmol/L)	56	25.4	–	–	56	100	–	–

* A study of 60 patients with severe community-acquired pneumonia, 18 of whom had acute kidney injury.
* Исследование 60 больных, страдающих ВПТТ, из них у 18 было выявлено ОПП.

procalcitonin, etc.) were evaluated in conjunction with the clinical parameters. In all, 125 (56.82%) patients experienced bacterial SCAP, 94 (42.73%) patients had viral/bacterial SCAP, and 1 (0.45%) patient had viral SCAP (Table 4).

In patients with SCAP and AKI, viral/bacterial pneumonia was more prevalent than bacterial pneumonia. Clinically, viral/bacterial SCAP is more severe than bacterial SCAP. A SOFA score of two or higher was present in 44.8% of the bacterial pneumonia patients, compared to 52.17% of patients with viral/bacterial pneumonia. AKI complications were documented in 31.91% of patients with viral/bacterial SCAP and 20.8% of bacterial SCAP patients ($p < 0.05$).

Out of 220 patients diagnosed with SCAP, 56 (25.45%) experienced AKI, verified according to the KDIGO criteria. Twenty-two (10%) patients had stage 1 AKI, twenty-six (11.8%) had stage 2 AKI, and eight (3.6%) had stage 3 AKI.

In patients with viral/bacterial SCAP, AKI was detected in 31.9% of patients. Among these, 11 (11.7%) patients had stage 1 AKI, 16 (17%) had stage 2 AKI, and 3 (3.1%) had stage 3 AKI. In patients with bacterial SCAP, AKI was diagnosed in 20.8% of patients; among these, 11 (8%) patients had stage 1 AKI, 10 (8%) had stage 2 AKI, and 5 (4%) had stage 3 AKI.

Higher SCr levels and decreased urine volume, which are frequent in AKI, were noted in 26 (11.8%) and 39 (17.7%) of the SCAP patients, respectively. A total of 14 patients (6.4%) exhibited an increase in serum creatinine (SCr) levels of 1.5-fold or more: 6 patients (2.7%) had an increase

Table 3. Estimated severity of community-acquired pneumonia using SMRT-CO, SOFA, CURB, and CRB-65 scales
Таблица 3. Оценка тяжести внебольничной пневмонии по шкалам SMRT-CO, SOFA, CURB, CRB-65

Score	Group 1	Group 2	χ^2	*p*
SOFA (*n* = 208)				
0–1	96	12	23.111	< 0.01
2–6	58	34	12.578	< 0.00
7–20	2	6	11.093	< 0.00
SMRT-CO (*n* = 208)				
0	40	8	2.311	= 0.12
1	37	9	0.93	= 0.33
2	26	4	2.545	= 0.11
3	36	18	2.701	= 0.10
⩾ 4	17	13	6.284	= 0.01
CURB scale (*n* = 213)				
0–1	121	32	8.101	= 0.01
2	33	15	0.786	= 0.38
⩾3	3	9	15.569	< 0.01
CRB-65 scale (*n* = 213)				
0	51	21	0.464	= 0.50
1–2	106	31	2.659	= 0.10
3–4	0	4	11.429	= 0.00

Table 4. Etiology of severe community-acquired pneumonia in patients of both groups, abs. (%)
Таблица 4. Этиология внебольничной пневмонии тяжелого течения у больных обеих групп, абс. (%)

Pneumonia etiology	All patients	Group 1	Group 2	χ^2	*p*
Bacterial	125 (56.8)	99 (60.3)	26 (46.4)	3.305	0.07
Viral/bacterial	94 (42.7)	64 (39)	30 (53.5)	3.483	0.06
Viral	1 (0.45)	1 (0.6)	0	–	–

of 1.5–1.9-fold, 3 patients (1.36%) had an increase of 2–2.9-fold, and 5 patients (2.27%) had an increase of 3-fold or greater. Among 39 (17.7%) patients with reduced urine volume, 8 (3.6%) patients had a urine volume of 0.5 mL/kg/h for 6 to 12 h, 24 (10.9%) had a urine volume of 0.5 mL/kg/h or less for 12 h or less, and seven (3.1%) had a urine volume of 0.3 mL/kg/h or less for 24 h or more (oliguria) or anuria for 12 h or more.

Among the 56 patients with AKI, 9 (16%) exhibited two key criteria: oliguria/anuria and increased SCr levels. Overall, the renal function parameters in group 1 patients were within normal limits (Table 5).

In the SCAP patients, 11 (5%) underwent mechanical ventilation, two of them also received extracorporeal membrane oxygenation, and AKI was diagnosed in 7 (63.6%) patients. In groups 2 and 1, a total of seven patients (12.5%) and just four patients (2.4%) required mechanical ventilation, respectively. This difference in the frequency of mechanical ventilation was statistically significant ($\chi^2 = 8.896$; $p = 0.003$).

Of the 11 patients on mechanical ventilation, 7 (63.6%) developed AKI. Among these, 6 (85.7%) were diagnosed with AKI after an average of 2.3 days (*Me* = 2.5 [2; 3]) of being on mechanical ventilation, and only one patient was diagnosed with AKI before the onset of mechanical ventilation. The association between mechanical ventilation and AKI was statistically significant ($\chi^2 = 66.717$; $p < 0.001$).

Ten AKI patients received renal replacement therapy (RRT) (Table 6). Among them, six patients underwent RRT for stage 3 AKI, three for stage 2 AKI due to water and electrolyte imbalance (hyperhydration and hyperkalemia) and acid-base imbalance, and one patient for extrarenal indications. Unfavorable outcomes were observed in stage 3 AKI patients who received RTT: three patients experienced respiratory failure, two developed persistent multiple organ failure, and one had septic shock. Following timely RT, three patients demonstrated improvement in their water and electrolyte imbalance and acid-base imbalance. In one case, pulmonary embolism caused the patient to succumb despite receiving RRT.

In group 1, the duration of hospital stay was 11–96 days (*Me* = 23 [18–30]), including an ICU stay of 1–19 days (*Me* = 3 [2–4]). In group 2, the duration of hospital stay was 11–75 days (*Me* = 28 [20–43]), including an ICU stay of 1–19 days (*Me* = 4 [3–7]) (Table 7).

Mortality in group 2 was significantly higher than that in group 1 (10.7% and 0.6%, respectively) ($\chi^2 = 13.836$; $p < 0.01$). In group 2, all seven patients who succumbed had been diagnosed with viral/bacterial pneumonia.

At discharge, 98.2% of the group 2 patients had normal SCr levels and urine volume. During the 3-month follow-up, no indications of renal failure were seen, and the SCr levels of the single patient who had a SCr level of 133 μmol/L at discharge reverted to normal.

Therefore, we conducted a comprehensive review of the available literature on renal function in SCAP patients. Although numerous studies have investigated the clinical and laboratory markers of AKI, we were unable to identify any that specifically evaluate AKI as a complication of SCAP in young individuals without chronic comorbidities. It has been reported that AKI complicated SCAP in over 50% of cases. Even among patients with nonsevere CAP, AKI was rather prevalent, occurring in 16%–25% of patients. More than 70% of influenza A (H1N1) virus-associated SCAP cases are known to result in AKI. In older patients with long-term comorbidities, AKI is more prevalent. [5].

The prevalence of AKI in SCAP patients was found to be 25.4%, which is slightly lower than that reported in the literature. This discrepancy may be attributed to the fact that the study participants were young and had no underlying chronic comorbidities. CAP severity was linked to low DBP in 66.3% of the patients. Compared to group 1 (patients

Table 5. Renal function parameters of the examined patients, *Me* (Q_{25}; Q_{75})
Таблица 5. Показатели почечной функции обследуемых пациентов, *Me* (Q_{25}; Q_{75})

Parameter	All patients	Group 1	Group 2		
			stage 1	stage 2	stage 3
Urine volume, mL/kg/h	0.8 (0.59; 1.1)	0.9 (0.7; 1.2)	0.7 (0.4; 0.9)	0.4 (0.3; 0.4)	0 (0; 0.2)
SCr, μmol/L	102 (90; 116)	97 (87; 108.5)	144 (123; 157)	105 (95; 113)	382.5 (112; 486)
Absolute change in *SCr* against estimated baseline *SCr*, μmol/L	–13 (–24; 2)	–17 (–28; 6)	31 (11; 42)	9.5 (–18; 20)	276.5 (6; 371)
Relative change in *SCr* against estimated baseline *SCr*	0.88 (0.79; 1)	0.85 (0.75; 0.94)	1.2 (109; 1.4)	0.91 (0.82; 1.15)	3.64 (1.06; 4.22)

Table 6. Parameters of group 2 patients who received renal replacement therapy
Таблица 6. Показатели больных 2-й группы, получавших заместительную почечную терапию

Patient	AKI stage	SCr, μmol/L	Urine volume, mL/kg/h	GFR, L/min/1.73 m^2	Indications to RRT	RRT type	Treatment outcome	Cause of death
1	3	509	Anuria for ⩾12 h	11	Stage 3 AKI	CVVHF, CVVHDF	Death	Respiratory failure
2	3	490	Anuria for ⩾12 h	13	Stage 3 AKI	HDF	Death	Multiple organ failure
3	3	482	Anuria for ⩾12 h	14	Stage 3 AKI	HDF	Death	Multiple organ failure
4	3	375	Anuria for ⩾12 h	17	Stage 3 AKI	HD, HDF	Death	Septic shock
5	3	112	Anuria for ⩾12 h	83	Stage 3 AKI	HDF + UF	Death	Respiratory failure
6	3	173	Anuria for ⩾12 h	43	Stage 3 AKI	HDF	Death	Respiratory failure
7	0	116	0.5 mL/kg/h	70	Non-renal indications	HDF	Death	Pulmonary embolism
8	2	203	<0.5 mL/kg/h for ⩾12 h	37	Water and electrolyte imbalance and acid-base imbalance	HF	Recovery	–
9	2	133	<0.5 mL/kg/h for ⩾12 h	59	Water and electrolyte imbalance and acid-base imbalance	HF	Recovery	–
10	0	140	>0.5 mL/kg/h	57	Non-renal indications	CVVHF	Recovery	–

Note: GFR, glomerular filtration rate; CVVHF, continuous venovenous hemofiltration; CVVHF, continuous venovenous hemodiafiltration; HDF, hemodiafiltration; HD, hemodialysis; UF, ultrafiltration; HF, hemofiltration;.
Примечание: GFR — скорость клубочковой фильтрации; CVVHF — непрерывная вено-венозная гемофильтрация; CVVHF — непрерывная вено-венозная гемодиафильтрация; HDF — гемодиафильтрация; HD — гемодиализ; UF — ультрафильтрация; HF — гемофильтрация; .

Table 7. Duration of hospital stay and intensive care unit stay of the examined patients, *Me* (Q_{25}–Q_{75})
Таблица 7. Длительность госпитализации и нахождения обследуемых больных в отделении реанимации и интенсивной терапии (ОРИТ), *Me* (Q_{25}–Q_{75})

Показатель	All patients	Group 1	Group 2	Mann–Whitney *U*-test	*p*
Duration of hospital stay, median	25 (19–30)	23 (18–30)	28 (20–43)	2814,5	0,01
Duration of ICU stay, median	3 (2–5)	3 (2–4)	4 (3–7)	2384,5	0,00

with SCAP without AKI), hypotension was more severe in group 2 (patients with SCAP with AKI). In group 2, 73.2% of the patients demonstrated a DBP lower than normal limits, and 23.1% of the patients exhibited an SBP lower than normal limits. In group 1, DBP was lower than normal limits in 64% of patients, and SBP was lower than normal limits in 10.9% of patients. There was a statistically significant difference in DBP across the groups ($\chi 2$ = 5.165; p = 0.02).

Our results aligned with those of a study conducted by Zhi et al. [18], who examined AKI development in patients with severe sepsis of various origins. The study included 582 patients who underwent ICU treatment. Sepsis-associated AKI was observed in 315 patients. Arterial hypotension was more prevalent in AKI patients than in patients without AKI: mean SBP was 128.61 ± 25.95 mm Hg and 123.29 ± 52.53 mm Hg, respectively (p = 0.00). In groups 1 and 2, the DBP was 71.31 ± 15.52 mm Hg and 67.5 ± 18.33 mm Hg, respectively (p = 0.01).

The SOFA score for 119 (54%) patients was 2. SOFA scores in group 2 were higher than those in group 1. For scores 0–1

($p < 0.01$), χ^2 was 23.111; for scores 2–6 ($p < 0.01$), it was 12.578; and for scores 7–20 ($p < 0.01$), it was 11.093.

We confirmed a statistically significant correlation between CAP severity and an increased risk of AKI. Therefore, patients with more severe CAP had a higher risk of developing AKI. We discovered a moderate correlation between the SOFA score for SCAP severity and AKI incidence. In patients with SCAP, AKI developed in 11.11% of patients with a SOFA score of less than 2 and 40% of patients with a SOFA score of 2 or more. A SOFA score of 2 was recorded in 76.9% and 38.4% of patients in group 2 and group 1, respectively. Patients with SCAP and a high risk of requiring respiratory support and vasopressors (SMRT-CO score 3) experienced a higher AKI incidence (33.3%). For patients with an SMRT-CO score of 4 or higher, this risk rose to 43.3%.

With increasing CURB scores, SCAP patients were more likely to develop AKI. In patients with CURB scores of 0–1 and 2, respectively, AKI developed in 20.9% and 31.25% of cases. In patients with a score of 3 or more, AKI developed in 75% of patients. These results highlight the need for careful monitoring of disease severity in patients with SCAP to prevent AKI.

This study revealed that AKI was more prevalent in viral/bacterial SCAP patients. A total of 53.5% and 46.4% of patients with AKI exhibited viral/bacterial SCAP and bacterial SCAP, respectively ($p = 0.07$). In group 1, 39% of the patients had viral/bacterial SCAP, while 60.3% of the patients experienced bacterial SCAP ($p = 0.06$). Viral/bacterial SCAP was associated with a slightly higher risk of developing AKI (31.91%) compared to those with bacterial SCAP alone (20.8%) ($p < 0.05$). This emphasizes the significance of the differential diagnosis between viral/bacterial SCAP and bacterial SCAP for determining AKI risk and treatment correction.

Zhi et al. compared the severity of patients with or without AKI using the SOFA and APACHE II scores [18]. The outcomes were comparable for both groups. According to the authors, AKI patients experienced a higher severity of the condition as evidenced by the SOFA and APACHE II scores. The SOFA scores revealed statistically significant differences between the groups ($p = 0.001$). The group without AKI included 267 patients with a mean score of 5.1 ± 3.2, whereas the group with AKI included 315 patients with a mean score of 8.3 ± 3.7.

Our results are consistent with earlier research that demonstrated that mechanical ventilation was an independent risk factor for AKI development in critically ill patients. For example, Zhi et al. [18] detected a statistically significant association between mechanical ventilation and the risk of AKI development ($p < 0.01$). However, a prospective study by de Abreu et al. [19] did not confirm this association. The authors found no statistically significant association between non-invasive ventilation and AKI in a cohort of 100 patients with lung disease and severe respiratory failure (86 of them received mechanical ventilation, and 14 received non-invasive ventilation). Instead, they observed that an oxygenation index below 200 was an independent factor linked to AKI development. These inconsistent results highlighted the need for additional research to elucidate the contribution of mechanical ventilation in AKI development.

According to our data, the duration of hospitalization and ICU stays in group 2 were significantly longer than in group 1. All patients were transferred to the ICU based on the SCAP treatment guidelines, and AKI was diagnosed as early as possible by daily monitoring of SCr and urine volume. Strict control and correction of fluid balance to avoid hypovolemia, along with effective treatment of the underlying disease, prevented AKI, slowed down AKI progression, prevented chronic kidney disease (CKD) development, and mitigated mortality risk.

Significant differences in mortality rates were noted between patients with and without AKI: 10.7% and 0.6%, respectively ($\chi^2 = 13.836$; $p < 0.01$). In 6 out of 7 lethal outcomes, patients were diagnosed with stage 3 AKI. Our results were consistent with those of a study by de Abreu et al. [19], that involved 100 patients with respiratory disease receiving treatment in the intensive care unit. Our study demonstrated a significant difference in the mortality rates between patients with and without AKI: the mortality rate in AKI patients was almost 2.5 times higher than in those without AKI (62.8% vs. 27.6%; $p = 0.001$).

A study by Shum et al. [20] discovered that AKI patients were more likely to develop physiological and biochemical abnormalities and comorbidities. The ICU, hospital, and 90-day mortality rates were comparable in patients with or without AKI, although the rates increased with the AKI grades. We hypothesized that AKI etiology plays a key role in the frequency of fatal outcomes. Sepsis-related AKI mortality rates reached up to 32%, statistically substantially higher than prerenal AKI mortality rates from hypovolemia (19%; $p < 0.01$). However, the mortality rates among cardiogenic AKI patients were even higher (46%; $p < 0.001$).

A remote consequence of AKI may include CKD, which is associated with disability and increased mortality. According to different authors, 2%–33% of patients with a history of RRT needed RRT again in the later period [18, 19]; these variations may be attributed to AKI etiology.

With the exception of one case when the SCr at discharge was 133 mmol/L, we discovered that all patients with SCAP

and AKI demonstrate kidney function test results within normal ranges. But during the three-month follow-up, this patient's SCr also returned to normal levels.

CONCLUSION

SCAP was found to be complicated by AKI in more than 50% of the cases. Even among patients with nonsevere CAP, AKI developed in 16%–25% of patients. AKI was identified in more than 70% of influenza A (H1N1) virus-associated SCAP cases. AKI is more prevalent in older patients with chronic comorbidities. Although multiple studies of AKI are available, we found no studies specifically evaluating AKI as a complication of SCAP in young individuals without chronic comorbidities. Therefore, our results, as the first study in this population, can contribute to the existing body of knowledge on AKI.

In young patients (18–44 years) without chronic comorbidities, AKI occurred in 25.4% of SCAP cases and showed a moderate direct correlation with the underlying etiology of SCAP; the AKI incidence was 31.91% and 20.8% in patients with viral/bacterial SCAP and bacterial SCAP, respectively ($p < 0.05$). Additionally, there was a tendency for AKI to occur more frequently as SCAP severity increased. In patients on mechanical ventilation, AKI developed in 85.7% of cases and began, on average, 2.5 days (2; 3) after the initiation of mechanical ventilation ($p < 0.01$).

AKI occurring as a complication of SCAP was associated with a statistically significant increase in the duration of hospital stay, ICU stay, and mortality. The median duration of hospital stay was 23 (18–30) days in group 1 and 28 (20–43) days in group 2 ($p = 0.01$). The median duration of ICU stay was 3 (2–4) days in group 1 and 4 (3–7) days in group 2 ($p = 0.001$). The mortality rate was 10.7% in group 2 and 0.6% in group 1 ($p < 0.001$).

Complete recovery of renal function, evidenced by normal urine output and serum creatinine levels, was observed in 99.52% of recovered patients. At the 3-month follow-up, the only patient who was discharged with a high SCr level had this parameter returned to the normal levels.

Increasing physicians' awareness about AKI risk in SCAP patients, along with adherence to the treatment guidelines for the underlying disease (SCAP) and timely diagnosis and treatment of AKI, can prevent AKI development, slow down its progression, prevent the development of CKD, and reduce mortality. Timely assessment of renal function through daily monitoring of SCr and urine volume in SCAP patients enables an early diagnosis of AKI. This facilitates optimization of fluid management, reduction of fluid overload, and adjustment of the therapy for electrolyte disorders, including hyperkalemia, thereby preventing hypovolemia and extravascular fluid accumulation in the lungs. It also allows the initiation of nephroprotective therapy at an earlier stage, helping prevent the progression of kidney damage..

ADDITIONAL INFORMATION

Authors' contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

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