Final results of BIRCOV trial
(ARB, ACEI, DRi in COVID-19)

Abstract. Background. The question of the possible effect of the inhibitors of the renin-angiotensin system (iRAS) on hypertensive subjects who fell ill with COVID-19 has been discussed in the literature. SARS-CoV-2 is well-known to use an angiotensin-converting enzyme 2 receptors facilitating virus entry into host cells. There are three possible mechanisms of angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARB) effect in COVID-19 in clinical practice: with worsening, neutral, or helpful function. Considering the different mechanisms of blood pressure reduction by iRAS, one can expect differences in people with COVID-19 receiving these drugs. The purpose of the BIRCOV study is to pinpoint possible clinical and laboratory differences in hypertensive people who received iRAS and suffered from coronavirus infection. Materials and methods. Patient-Oriented Evidence that Matters (POEM) intervention was designed as an open prospective randomized two medical centers trial in subjects suffering from COVID-19 who have been receiving iRAS, either ACEI, ARB, or direct renin inhibitor (DRi) as basic antihypertensive therapy. One hundred and twenty people with stage 1–2 hypertension have been screened; 108 subjects were enrolled in the BIRCOV study. COVID-19 was confirmed by a PCR test; the disease follow-up was divided into 2 periods: up to 12 weeks and up to 24 weeks. The primary outcome measure was as follows: blood pressure (BP) was known one week before COVID-19 onset and was measured during the disease on weeks 2, 4, 12, 24. The secondary outcome measures were clinical features. Subanalysis in patients with chronic kidney disease (CKD) was performed. Results. All patients were randomized into 3 groups who received: ACEi — 42 (39 %), ARB — 35 (32 %), or DRi — 31 (29 %). The BIRCOV trial documented the trend of BP lowering in the first two weeks of the COVID-19 disease with its gradual return to baseline values up to the 12th week. Twenty-three (21 %) patients have withdrawn medicine for up to 2 weeks due to severe hypotension. However, the BP values after COVID-19 in most subjects remained lower than the baseline ones for 4 weeks. The use of ACE inhibitors significantly increased the risk of withdrawal compared to DRi (RR 1.648; 95% CI 0.772–3.519; NNT 7.0) and ARB (RR 13.023; 95% CI 1.815–93.426; NNT 2.9) due to COVID-19. The synchronous decline of estimated glomerular filtration rate (eGFR) and systolic BP was more pronounced in CKD patients. The greatest decrease in eGFR was noted in people who have been taking ACEi. The drop in eGFR ranged from 23 % in CKD stage 1 to 45 % in CKD stage 4. Two people required short-term dialysis. The analysis of secondary outcome points demonstrated that in 23 % of people without preceding albuminuria it developed in the A2 range. During 12 weeks of observation, 81 % of patients had spontaneous albuminuria reduction. Post-COVID-19 (above 12 weeks) albuminuria remained in 19 % of patients, 90 % of them had a history of CKD. Patients with preceding CKD had an increase in albuminuria in 78 % of cases, and its return to the baseline was observed only in 24 % of patients by the 12th week and in 49 % of individuals in 24 weeks. Conclusions. People with stage 1–2 hypertension who are receiving chronic iRAS and suffer from COVID-19 may develop hypotension with ACE inhibitors. COVID-19 leads to transient albuminuria and decreased glomerular filtration rate, which is especially dangerous for people with CKD. Keywords: renin-angiotensin system inhibitors; angiotensin receptor blockers; angiotensin-converting enzyme inhibitors; direct renin inhibitor; COVID-19; BIRCOV trial
Background

Current international guidelines suggest continuing the usage of antihypertensive drugs, in particular inhibitors of the renin-angiotensin-aldosterone system, in hypertensive people who become ill with COVID-19 [1] with no differences between the five classes of antihypertensive agents [2].

It is well-known that the SARS-CoV-2 uses an angiotensin-converting enzyme 2 (ACE2) receptor and furin to enter the cell [3–5]. So if ACE2 levels are higher or lower in some hypertensive subjects, then the severity of disease and blood pressure (BP) level might be different [6]. It is natural to assume that SARS-CoV-2 can affect the state of the inhibitors of the renin-angiotensin system (iRAS).

Although most studies do not point out a negative effect of the virus on blood pressure levels, there is information about the different effects of various RAS inhibitors. Mandeep R. et al. (2020) found some differences between angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) [7]. Probably, there also could be a difference for direct renin inhibitor (DRi) [8].

In this regard, in March 2020, we initiated a study which was aimed to pinpoint possible clinical and laboratory differences in hypertensive people who received iRAS and suffered from coronavirus infection.

Materials and methods

The BIRCOV trial (ARB, ACEI, DRi in COVID-19) is registered in ClinicalTrials.gov (NCT04364984, https://clinicaltrials.gov/ct2/show/NCT04364984). The study began on April 1, 2020; the primary completion was achieved on July 24, 2021, and final results were available on August 1, 2021.

Study protocol. Patient-Oriented Evidence that Matters (POEM) [10] intervention was designed as an open prospective randomized two medical centers trial in subjects suffering from COVID-19 who have been receiving iRAS, either ACEi, ARB, or DRi as basic antihypertensive therapy.

One hundred and twenty people with stage 1–2 hypertension were screened.

Study population: patients with proved COVID-19 and preliminary stage 1–2 hypertension receiving iRAS at the onset of COVID-19 were being observed for 24 weeks.

Sampling method: non-probability sample. Minimum age: 18 years; maximum age: 90 years.

Sex: all.

Inclusion criteria: hypertension, stage 1–2.

Exclusion criteria: hypertension, stage 3, heart failure (NYHA) 3–4.

COVID-19 was confirmed by a PCR test, the disease follow-up was divided into 2 periods: up to 12 weeks and up to 24 weeks.

Primary outcome measure: BP was known one week before COVID-19 and was measured during the disease course on weeks 2, 4, 12, 24. Secondary outcome measures: the number of patients with fever (above 37.2 °C) up to 3 weeks after COVID-19 onset; the number of patients with cough (time frame: 12 weeks); the number of patients with throat pain (time frame: 2 weeks); the number of patients with diarrhoea (time frame: 2 weeks), and the number of patients who needed hospital admission and intensive care unit (time frame: 24 weeks).

Informed consent form — optional under 42 CFR Part 11, all patients gave their verbal consent to submit personal data.

The BIRCOV trial included subanalysis of patients with chronic kidney disease (CKD) — kidney arm, with the primary endpoints: BP and estimated glomerular filtration rate (eGFR) measuring and the secondary endpoint — albuminuria grade (Fig. 1).

Hydration status was elevated by the method of Ivanova M.D. et al. [10]. Statistical evaluation of the research results was carried out in the package of medical statistics [11]. The risk of progression to kidney failure requiring dialysis or transplantation (using the Kidney Failure Risk Equation [12]) was calculated for all patients of kidney arm on weeks 2, 4, 12, and 24 from COVID-19 onset.

Results

One hundred and twenty outpatient subjects were screened; 112 were enrolled; 108 (96 %) completed the study (4 died); 60 (56 %) males and 48 (44 %) females; mean age 55.0 ± 1.12 years old (18–87; coefficient of variation 0.210514, coefficient of asymmetry –0.261873). Among hypertensive patients, 35 (32 %) had stage 1 hypertension, 73 (68 %) had stage 2. Eighty-three (77 %) subjects had CKD, ranging from 1 to 4 stages: CKD 1 — 23 (27 %), CKD 2 — 46 (56 %), CKD 3 — 10 (12 %), CKD 4 — 4 (5 %).

All patients were randomized into 3 groups who received: ACEi — 42 (39 %), ARB — 35 (32 %), or DRi — 31 (29 %). Eighty-four (78 %) patients had combined iRAS with calcium channel blockers and diuretics, 17 (16 %) combined iRAS with B-blockers, 7 (6 %) received iRAS monotherapy.

Clinical features arm

Primary outcome measure: blood pressure

The reason for the prescription of iRAS and its combination with other antihypertensive agents was the presence of hypertension itself. At the beginning of the trial, 35 patients had stage 1 hypertension, 77 — stage 2. Four patients (2 males, 2 females) died within the first 2 months from the COVID-19 onset. Among 108 hypertensive persons who finished the trial, 35 (32 %) previously had stage 1 hypertension, 73 (68 %) had stage 2 before getting prescribed drugs. Thus, a week before the development of COVID-19, the mean blood pressure was 137.0 ± 0.9 / 83.0 ± 0.6 mm Hg (coefficient of variation 0.067728, coefficient of asymmetry 1.029771). The dynamics in blood pressure by control points are shown in Table 1. Table 1 presents the baseline BP values with a follow-up of 2, 4, 12, and 24 weeks in ACEi, ARB and DRi groups.

The BP changes did not have significant statistical differences between the chosen medicine one week before enrolment. However, we had a clear documented trend of BP lowering in the first two weeks of the COVID-19 disease (Fig. 2) with its gradual return to baseline values up to the 12th week. Twenty-three (21 %) patients discontinued a
The data obtained indicated that the use of ACE inhibitors significantly increases the risk of withdrawal compared to DRI (RR 1.648; 95% CI 0.772–3.519; NNT 7.0) and ARB (RR 13.023; 95% CI 1.815–93.426; NNT 2.9) due to COVID-19.

No less interesting was the restoration of normotensiveness after the onset of coronavirus infection. It turned out that in the group of those taking DRI, after 4 weeks, there were practically no significant differences from the baseline BP values, and after 12 weeks, the consequences of hypotension were eliminated. On the contrary, in people who took ACE inhibitors, lower blood pressure values were still maintained in the post-COVID period.

The Table 2 shows the secondary outcomes measure:

- the number of patients with fever (above 37.2°C) up to 3 weeks,
- the number of patients with cough (time frame: 12 weeks),
- the number of patients with throat pain (time frame: 2 weeks),
- the number of patients with diarrhoea (time frame: 2 weeks),
- the number of patients who needed hospitalization and intensive care unit (time frame: 24 weeks).

The data in Table 2 characterizes the course of COVID-19 in...
the BIRKOV study. The analysis of clinical symptoms did not reveal any dependence on the type of antihypertensive therapy with iRAS. The mortality rate was 3.7%. Two of the patients received ACEi and two received ARB. The absolute risk for ARB compared to DRi was 0.057, for ACEi versus DRi — 0.048. Thus, the absolute risk of death in people with COVID-19 receiving ARB was higher than in people taking ACE inhibitors, despite the presence of more severe hypotension in the first 4 weeks from COVID-19 onset.

**Kidney arm**

Table 3 represents the baseline eGFR values with a follow-up of weeks 2, 4, 12, and 24 in ACEi, ARB, and DRi groups.

The synchronous decline of eGFR and systolic BP was more pronounced in CKD patients. The greatest decrease in eGFR was noted in people who took ACEi, weeks 0–24; the correlation coefficient (r) is 0.815; the relationship between the studied features is direct; the tightness (strength) of the relationship according to the Chaddock scale is high, the number of degrees of freedom (f) is 3; the Student’s t-test is 2.432; although the dependence of the features was statistically insignificant (p = 0.135563).

The individual analysis demonstrated that eGFR decline correlated directly with the advancement of CKD. The drop in eGFR ranged from 23% in CKD 1 to 45% in CKD stage 4. Two people required short-term dialysis.

**Figure 2. Systolic and diastolic blood pressure values 2 weeks after the onset of COVID-19**

**Table 1. The baseline BP values in dynamics by the weeks and groups of antihypertensive treatment**

<table>
<thead>
<tr>
<th>Drug/Week</th>
<th>−1</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>12</th>
<th>24</th>
<th>P value −1–0</th>
<th>P value 0–2</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi, n = 42</td>
<td>138.0 ± 1.1 / 83.0 ± 1.2</td>
<td>126.0 ± 1.2 / 77.0 ± 0.7</td>
<td>104.0 ± 0.9 / 68.0 ± 0.6</td>
<td>114.0 ± 1.1 / 72.0 ± 0.7</td>
<td>128.0 ± 1.2 / 77.0 ± 1.0</td>
<td>137.0 ± 1.2 / 81.0 ± 1.2</td>
<td>P ≤ 0.01</td>
<td>P ≤ 0.01</td>
</tr>
<tr>
<td>ARB, n = 35</td>
<td>136.0 ± 1.1 / 82.0 ± 1.2</td>
<td>130.0 ± 1.0 / 78.0 ± 0.7</td>
<td>131.0 ± 1.0 / 77.0 ± 0.6</td>
<td>133.0 ± 1.0 / 78.0 ± 0.6</td>
<td>135.0 ± 1.1 / 79.0 ± 0.9</td>
<td>137.0 ± 1.2 / 82.0 ± 1.2</td>
<td>P = 0.02</td>
<td>P ≤ 0.01</td>
</tr>
<tr>
<td>DRi, n = 31</td>
<td>134.0 ± 1.4 / 82.0 ± 1.2</td>
<td>127.0 ± 1.2 / 79.0 ± 0.6</td>
<td>115.0 ± 0.9 / 70.0 ± 0.6</td>
<td>121.0 ± 0.9 / 74.0 ± 0.6</td>
<td>125.0 ± 1.0 / 79.0 ± 0.8</td>
<td>129.0 ± 1.2 / 80.0 ± 1.2</td>
<td>P ≤ 0.01</td>
<td>P ≤ 0.01</td>
</tr>
</tbody>
</table>

**Table 2. Data on the secondary outcome measure, N (%)**

<table>
<thead>
<tr>
<th>Patients</th>
<th>onset</th>
<th>2 weeks</th>
<th>3 weeks</th>
<th>4 weeks</th>
<th>12 weeks</th>
<th>24 weeks</th>
<th>Relative risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>with fever (above 37.2 °C)</td>
<td>101 (90)</td>
<td>–</td>
<td>12 (11)</td>
<td>–</td>
<td>0</td>
<td>0</td>
<td>Onset — 3 weeks: RR 8.417; 95% CI 4.926–14.382; NNT 1.213</td>
</tr>
<tr>
<td>with cough</td>
<td>87 (78)</td>
<td>78 (70)</td>
<td>–</td>
<td>–</td>
<td>3 (3)</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>with throat pain</td>
<td>56 (50)</td>
<td>1 (1)</td>
<td>–</td>
<td>–</td>
<td>0</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>with diarrhoea</td>
<td>8 (7)</td>
<td>0</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>who needed hospitalization and intensive care unit</td>
<td>4 (3.5)</td>
<td>18 (16)</td>
<td>4 (4)</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
<td>Onset — 2 weeks: RR 0.222; 95% CI 0.078–0.635; NNT 7.714</td>
</tr>
<tr>
<td>who died</td>
<td>–</td>
<td>–</td>
<td>3 (2.6)</td>
<td>1 (1)</td>
<td>0</td>
<td>0</td>
<td>3 to 4 weeks: RR 3.00; 95% CI 0.317–28.390; NNT 54.00</td>
</tr>
</tbody>
</table>
Seventy-eight percent of patients with preceding CKD had an increase in albuminuria and its return to the baseline values was observed only in 24% of patients by the 12th week and in 49% in 24 weeks.

An albumin/creatinine ratio was available in 24 patients with CKD. A two- and five-year prognosis of the risk of developing end-stage renal failure was calculated for them (Tables 4, 5).

The post-COVID-19 syndrome was presented by the development of albuminuria in patients that were previously clear of it, and worsening albuminuria in patients that had already had it.

Discussion
ACE inhibitors form their effect through the ACE1 receptors, while the SARS-CoV-2 uses the ACE2 receptors [13]. Sequential metabolism of angiotensin I–9, then angiotensin 1–7 goes two ways: 1) acting as an agonist through the Mas-1 receptors leading to vasodilation, 2) acting as an antagonist of the angiotensin AT1 receptor, enhancing vasodilation [14].

Thus, the SARS-CoV-2 seems similar in its mechanism of action to ARB that explains hypotension in the acute period of coronavirus infection. A double block of ACE inhibitors and ARB (the SARS-CoV-2) is accompanied by the largest blood pressure decrease, a double block of DRI and ARB (the SARS-CoV-2) is characterized by a smaller decrease in pressure, and a double block of ARB + ARB (the SARS-CoV-2) has practically no effect on blood pressure. This is associated with a trend that increases the risk of death in people with COVID-19 who are taking ARB as an antihypertensive agent [15].

Jordana B. Cohen et al. (2021) presented three possible mechanisms of the effect of RAS inhibitors, one of which, in our opinion, was shown in the results of the BIRKOV study [15]. At the same time, a small triple-blind study has shown no reduction in blood pressure. Perhaps, staying in intensive care units and hospitals does not allow us to depict the features that were established by us on an outpatient basis [16].

The second most important result of the BIRCOV study was a transient decrease in renal function by eGFR for healthy people and quite pronounced for people with CKD, accompanied by an increase in albuminuria. These data are in good agreement with the known ones, claiming higher morbidity and mortality in patients with CKD [17, 18].

The BIRKOV study supplements the available data on the absence of a negative effect of RAS inhibitors on the COVID-19 course [20]. Further studies are required for profound discovery of the characteristics of the course of COVID-19 infection in people with concomitant diseases, including hypertension and CKD [19].

Conclusions
COVID-19 has been shown to induce hypotension in outpatients if they receive an ACE inhibitor for hypertension. The working hypothesis indicates DRI as the safest antihypertensive treatment drug in 24-weeks follow-up observation with the least volatility of BP and mortality.

The nature of BP reduction in people with stage 1–2 hypertension, taking iRAS allows comparing the effect of SARS-CoV-2 with the action similar to ARB, i.e. in people taking ACE inhibitors, the effect of BP reduction was comparable to the double block of iRAS: ACE inhibitors + ARB.

The synchronous decline of eGFR and systolic BP was more pronounced in CKD patients. The greatest decrease in eGFR was noted in people who took ACEi and in CKD 4. Not all patients with CKD had a return to baseline albuminuria and renal function after COVID-19.

Table 3. The changes in eGFR (ml/min/1.73 m²) in patients by the weeks and groups of antihypertensive treatment

<table>
<thead>
<tr>
<th>Drug</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>12</th>
<th>24</th>
<th>P value 0–2</th>
<th>P value 0–4</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi, n = 42</td>
<td>69.0 ± 1.7</td>
<td>52.0 ± 1.1</td>
<td>51.0 ± 0.9</td>
<td>58.0 ± 2.0</td>
<td>68.0 ± 1.9</td>
<td>P ≤ 0.01</td>
<td>P ≤ 0.01</td>
</tr>
<tr>
<td>ARB, n = 35</td>
<td>72.0 ± 1.7</td>
<td>70.0 ± 1.8</td>
<td>73.0 ± 1.5</td>
<td>70.0 ± 1.6</td>
<td>71.0 ± 1.8</td>
<td>Not reliable</td>
<td></td>
</tr>
<tr>
<td>DRI, n = 31</td>
<td>71.0 ± 1.8</td>
<td>70.0 ± 1.6</td>
<td>69.0 ± 1.5</td>
<td>72.0 ± 1.7</td>
<td>70.0 ± 1.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4. Risk of progression to kidney failure requiring dialysis or transplantation (using the Kidney Failure Risk Equation) in men (n = 14), %

<table>
<thead>
<tr>
<th>Control check/risks</th>
<th>2 weeks</th>
<th>12 weeks</th>
<th>24 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over 2 years</td>
<td>0.1</td>
<td>0.1</td>
<td>0</td>
</tr>
<tr>
<td>Over 5 years</td>
<td>0.4</td>
<td>0.3</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Table 5. Risk of progression to kidney failure requiring dialysis or transplantation (using the Kidney Failure Risk Equation) in women (n = 10), %

<table>
<thead>
<tr>
<th>Control check/risks</th>
<th>2 weeks</th>
<th>12 weeks</th>
<th>24 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over 2 years</td>
<td>0.1</td>
<td>0.1</td>
<td>0</td>
</tr>
<tr>
<td>Over 5 years</td>
<td>0.3</td>
<td>0.2</td>
<td>0</td>
</tr>
</tbody>
</table>
У літературі дискутується питання...


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Conflicts of interests.

Authors declare the absence of any conflicts of interests and their own financial interest that might be construed to influence the results or interpretation of their manuscript.

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Підсумкові результати дослідження BIRCOV (БРА, ІАПФ, ПІР при COVID-19)

Резюме. Актуальність. У літературі дискутується питання про можливий вплив інгібіторів рені-ангіотензин-альдосеронової системи (іАПФ) на стан людей з гіпертензією, які захворіли на COVID-19. Основою для такої дискусії є використання інгібіторів рені-ангіотензинперетворюючого ферменту 2 (ПІР) для проникнення в клітину. Три можливих механізми взаємодії іПАС з коронавірусом можуть бути реалізовані в клінічній практиці: ініціація релейної реакції, неінтралітна або той, що допомагає організму чинити опір COVID-19. З огляду на різні механізми зниження тиску інгібіторами ПААС можна очікувати різних результатів в стані людей з COVID-19, які отримують названі препарати. Метою дослідження було вивчення клінічних особливостей і лабораторних показників у пацієнтів із гіпертензією 1–2-го ступеня, які отримували іАПФ або блокатори рецепторів антагоністів (БРА), або прямі інгібітори реніну (ПІР) як основну антигіпертензивну терапію. Було обстежено 120 людей з гіперто-
Матеріали і методы.

1. Іванов Д.Д.

1. Напрямок медичної навчальної установи.

1. Видання: журнал, номер, рік.

1. URL: http://kidneys.zaslavsky.com.ua

1. Якість дослідження: оригінальна стаття / Original Article.

1. Назва дослідження: BIRCOV (БРА, ИАПФ, ПИР при COVID-19)

1. Ключові слова: інгібітори ренін-ангіотензин-альдостеронової системи, інгібітори антагоністів відділу II, інсульт.

1. Основна мета дослідження: вивчення впливу інгібіторів ренін-ангіотензин-альдостеронової системи на стан пациєнтів з хронічною ішемічною болісю серцевої нирки.

1. Матеріал для аналізу: 120 осіб з хронічною ішемічною болісю серцевої нирки.

1. Методи: опис аналізу.

1. Результати: зменшення рівня швидкості клубочкової фільтрації.

1. Заключення: інгібітори ренін-ангіотензин-альдостеронової системи можуть бути рекомендовані для лікування патології серця та нирок.

1. Список літератури:

1. Іванов Д.Д., Іванова М.Д., Крестанелло Т.

1. Оригінальна стаття: BIRCOV (БРА, ИАПФ, ПИР при COVID-19).

1. URL: http://kidneys.zaslavsky.com.ua

1. Заголовок: Результати дослідження BIRCOV (БРА, ИАПФ, ПИР при COVID-19)

1. Актуальність: відсутня

1. Резюме: Стаття дискутує вплив інгібіторів ренін-ангіотензин-альдостеронової системи на стан пациєнтів з хронічною ішемічною болісю серцевої нирки.

1. Ключові слова: інгібітори ренін-ангіотензин-альдостеронової системи, інгібітори антагоністів відділу II, інсульт.

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1. Матеріал для аналізу: 120 осіб з хронічною ішемічною болісю серцевої нирки.

1. Методи: опис аналізу.

1. Результати: зменшення рівня швидкості клубочкової фільтрації.

1. Заключення: інгібітори ренін-ангіотензин-альдостеронової системи можуть бути рекомендовані для лікування патології серця та нирок.

1. Список літератури:

1. Іванов Д.Д., Іванова М.Д., Крестанелло Т.

1. Оригінальна стаття: BIRCOV (БРА, ИАПФ, ПИР при COVID-19).

1. URL: http://kidneys.zaslavsky.com.ua

1. Заголовок: Результати дослідження BIRCOV (БРА, ИАПФ, ПИР при COVID-19)

1. Актуальність: відсутня

1. Резюме: Стаття дискутує вплив інгібіторів ренін-ангіотензин-альдостеронової системи на стан пациентів з хронічною ішемічною болісю серцевої нирки.

1. Ключові слова: інгібітори ренін-ангіотензин-альдостеронової системи, інгібітори антагоністів відділу II, інсульт.

1. Основна мета дослідження: вивчення впливу інгібіторів ренін-ангіотензин-альдостеронової системи на стан пациентів з хронічною ішемічною болісю серцевої нирки.

1. Матеріал для аналізу: 120 осіб з хронічною ішемічною болісю серцевої нирки.

1. Методи: опис аналізу.

1. Результати: зменшення рівня швидкості клубочкової фільтрації.

1. Заключення: інгібітори ренін-ангіотензин-альдостеронової системи можуть бути рекомендовані для лікування патології серця та нирок.

1. Список літератури:

1. Іванов Д.Д., Іванова М.Д., Крестанелло Т.

1. Оригінальна стаття: BIRCOV (БРА, ИАПФ, ПИР при COVID-19).

1. URL: http://kidneys.zaslavsky.com.ua

1. Заголовок: Результати дослідження BIRCOV (БРА, ИАПФ, ПИР при COVID-19)

1. Актуальність: відсутня

1. Резюме: Стаття дискутує вплив інгібіторів ренін-ангіотензин-альдостеронової системи на стан пациентів з хронічною ішемічною болісю серцевої нирки.

1. Ключові слова: інгібітори ренін-ангіотензин-альдостеронової системи, інгібітори антагоністів відділу II, інсульт.

1. Основна мета дослідження: вивчення впливу інгібіторів ренін-ангіотензин-альдостеронової системи на стан пациентів з хронічною ішемічною болісю серцевої нирки.

1. Матеріал для аналізу: 120 осіб з хронічною ішемічною болісю серцевої нирки.

1. Методи: опис аналізу.

1. Результати: зменшення рівня швидкості клубочкової фільтрації.

1. Заключення: інгібітори ренін-ангіотензин-альдостеронової системи можуть бути рекомендовані для лікування патології серця та нирок.

1. Список літератури: