INTRODUCTION

Acute kidney injury (AKI) is an important cause of mortality and morbidity in the community. Contrast-related AKI can also be seen after contrast agent exposure which is known nephrotoxic. Although the pathophysiology is not clearly elucidated, it is thought that contrast nephropathy may occur due to hemodynamic changes such as direct tubular toxicity effect of the contrast agent, oxidative stress and renal vasoconstriction. Although the incidence of contrast-related AKI is less than 2% in the general population, the incidence is higher in patients with diabetes and chronic kidney disease (CKD).

Patients with AKI are known to have an increased risk factor for development of contrast induced nephropathy after contrast exposure when compared to patients with normal kidney function (1-3). However, the incidence of contrast nephropathy after contrast exposure with AKI is not clear (4). In this study, we aimed to investigate the incidence of contrast nephropathy and changes in renal function in patients undergoing coronary angiography with AKI.

MATERIALS and METHODS

Patient Population

The study was planned as a retrospective study and we evaluated the clinical outcome of patients who underwent isosomlar contrast angiography in the arterial phase with acute kidney injury between January 2014 and December 2017.

Results:

The number of AKI patients included in the study was 280 and the control group consisted of 478 participants. There was no statistically significant difference between two groups. (3.2% (n: 9) of patients with AKI contrast nephropathy vs 6.5% (n: 31) of patients with normal kidney function and contrast nephropathy (p> 0.05). On the other hand, there was statistically significant difference between two groups according to diabetes mellitus (patients with contrast nephropathy 26 (65%) had diabetes vs 14 (35%) participants had no diabetes). This difference was statistically significant (p <0.05).

Conclusion:

In this study, in patients who underwent emergency angiography with a preliminary diagnosis of acute coronary syndrome in the emergency department, we did not find an increased risk of acute renal injury and contrast-enhanced nephropathy compared to the patient population with normal kidney function.

Keywords: Acute kidney injury; contrast nephropathy; coronary angiography
nephropathy were included and following two definitions are used increase from baseline in serum creatinine (sCr) ≥0.3mg/dl or increase from baseline of 25% within 72 hours (5).

AKI was defined as an absolute increase in sCr, at least 0.3 mg/dL (26.5 μmol/L) within 48 hours or by a 50% increase in sCr from baseline within 7 days, or a urine volume of less than 0.5 mL/kg/h for at least 6 hours before contrast exposure (6).

Control group was defined as patients who have kidney function test normal before contrast exposure.

Diabetes Mellitus was defined as medical history of patients who used anti diabetic agents or HbA1c ≥6.5% (7).

Statistical Analysis
Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) for Windows 20 (IBM SPSS Inc., Chicago, IL) program. The normal distribution of the data was evaluated by Kolmogorov-Smirnov test. Numerical variables with normal distribution were shown as mean ± standard deviation and numerical variables with not normal distribution were shown as median (min-max). Categorical variables were shown as numbers and percentages. Continuous variables were compared with independent sample t-test, ANOVA or Mann Whitney U test, Kruskall Wallis H test where appropriate. Comparison of categorical variables was made with Chi-square test. P<0.05 values were considered statistically significant.

RESULTS
The number of AKI patients included in the study was 280 and the control group consisted of 478 participants. 116 (41.4%) patients in the AKI group were female and 166 (34.7%) patients of the control group were female. The mean eGFR of the AKI group was 43.6ml/min, while the eGFR of the control group was 60.4ml/min. eGFR difference was statistically significant when compared with patients with AKI and normal renal function (p <0.001). The median length of hospitalization was 5 day in patients with AKI and 4 day in the control group. This difference was not statistically significant (p> 0.05) (Table 1).

Table 1. Demographic and clinical data of patients with AKI and normal kidney function

<table>
<thead>
<tr>
<th></th>
<th>AKI (n=280)</th>
<th>Normal (n=478)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>73 (35-95)</td>
<td>74 (21-92)</td>
<td>0.182</td>
</tr>
<tr>
<td>Gender (female), n (%)</td>
<td>116 (41.4)</td>
<td>166 (34.7)</td>
<td>0.06</td>
</tr>
<tr>
<td>GFR(ml/min)</td>
<td>43.6±10.2</td>
<td>63.4±2.8</td>
<td>0.001*</td>
</tr>
<tr>
<td>Hospitilization(day)</td>
<td>5 (0-114)</td>
<td>4 (0-76)</td>
<td>0.088</td>
</tr>
<tr>
<td>Contrast Nephropathy n (%)</td>
<td>9 (3.2)</td>
<td>31 (6.5)</td>
<td>0.052</td>
</tr>
</tbody>
</table>

Table 2. The relationship between Contrast Nephropathy and Diabetes Mellitus

<table>
<thead>
<tr>
<th></th>
<th>DM (+)</th>
<th>DM (-)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contrast Nephropathy (+) (n=40)</td>
<td>26</td>
<td>14</td>
<td>*0.004</td>
</tr>
<tr>
<td>(65%) (35%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contrast Nephropathy (-) (n=718)</td>
<td>301</td>
<td>417</td>
<td></td>
</tr>
<tr>
<td>(41.9%) (58.1%)</td>
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DISCUSSION
Contrast nephropathy is one of the most common causes of AKI in clinical practice (8). This kidney injury can be irreversible and may progress CKD (9). The most important mechanism in the pathophysiology of contrast nephropathy is medullary hypoxia and direct renal tubular toxicity due to medullary vasoconstriction (10,11). Oxidative stress also plays an important role in the development of contrast nephropathy (12,13). In addition, contrast exposure causes increased viscosity in the circulation, which further exacerbate the medullary hypoxia (14).

The most important risk factor for the development of contrast nephropathy is known to be CKD and diabetes mellitus (15). In one study, the incidence of contrast nephropathy was higher in patients with CKD and diabetes than in patients with CKD (16). Similar to the studies in the literature, in our study, a significantly higher detection of diabetes patients in the development of contrast nephropathy indicates that diabetes is an important risk factor in the development of contrast nephropathy.

In addition, contrast exposure was found to be intra-arterial and the risk of contrast nephropathy was higher compared to intra venous exposure (17). One of the reason for this may be the higher density of the contrast agent in the kidney in the arterial phase (18). In our study although percutaneous transluminal coronary angioplasty (PTCA) was performed in the arterial phase, we found similar contrast nephropathy in patients with AKI when compared to the group with normal kidney function. Although the development of contrast nephropathy in patients with AKI is of clinical importance, we have shown in this study, it may not be worry about the development of contrast nephropathy in patients with AKI.
patients (9). In another report, 16% persistent renal damage was detected in patients who underwent PTCA and a significantly higher 5-year mortality rate was found (19). In another study, the incidence of new renal failure was found 0.9% within 6 months after PTCA. In this study, also a lower incidence of CKD was found in PTCAs with trans-radial catheter than femoral catheters (20). In another study, the incidence of contrast nephropathy was found to be less than 2%, but in patients with CKD and diabetes, the incidence can be as high as 50% (21). In our study, factors such as the presence of trans-radial catheters, use of isoosmolar contrast media, research population may be the reason for lower incidence of contrast nephropathy.

Another study showed that, the risk of contrast induced nephropathy has been exaggerated. Clinical adverse outcomes from cardiac catheterization and intervention is lower than expected side effects such as contrast nephropathy (22). Fluctuations in creatinine levels can be seen especially in hospitalized patients. Contrast nephropathy can be overdiagnosis due to many confounding factors (23). In our study, we included cases that were diagnosed with contrast nephropathy and were confirmed with nephrologist. As a result, the confounding factors were eliminated and the actual incidence of the contrast nephropathy was found similar with normal kidney function in the literature (24). We can speculated that, patient with AKI may not increase risk for contrast nephropathy.

CONCLUSION

Patients with risk factors such as diabetes and chronic kidney damage should be closely monitored for the development of contrast nephropathy. The incidence of contrast nephropathy with AKI was found to be lower than expected.

This report has some limitations. This study was performed retrospectively. Before hospitalization, the current treatments for patients are unknown. The etiology of AKI has not been clarified by prerenal, renal and postrenal diseases. However, studies have shown that kidney damage is an independent risk factor despite of etiology (25). This study will help to clarify this issue because it is difficult to make a prospective study of incidence of contrast nephropathy in patients with AKI in the literature.

Competing Interests: The authors declare that they have no competing interest.

Financial Disclosure: There are no financial supports.

Ethical Approval: The study was approved in accordance with the patient rights regulation and approved by the Ankara Numune Education and Research Hospital Scientific Research Evaluation Commission on the date of 21/03/2018 with the decision number 1856-2018.

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22. Katzberg RW, Newhouse JH. Intravenous contrast medium–induced nephrotoxicity: is the medical risk really as great as we have come to believe? Radiology 2010;256:21-8.

