ACUTE KIDNEY INJURY FOLLOWING CORONARY ANGIOGRAPHY AT A CARDIOLOGY ICU IN A DEVELOPING COUNTRY: INCIDENCE, RISK FACTORS AND OUTCOME

Thilini Kalara Wickramasuriya[1], Nuwan Ranawaka[2]
[1]Senior Clinical Fellow, King's College Hospital, London, UK
[2]Consultant Intensivist, Cardiology Unit, National Hospital of Sri Lanka

Presenter - Thilini Kalara Wickramasuriya - MBBS (Col), MD Anaesthesia (Col)
Flat 11A, 81 Hanover Park, Peckham, London. SE15 5HD
thiliniwick@yahoo.com

Introduction

Coronary angiography and percutaneous interventions utilize iodinated radio-contrast media to give visual imaging of vessels and cardiac chambers. Regardless of current advancements in relevant imaging techniques and usage of contrast media, iodinated contrast still constitute a risk for contrast-induced acute kidney injury (CI-AKI) for patients undergoing coronary angiography, etc. This prominent iatrogenic complication, which affects prognosis and health care costs both, has been found to present in many patients with hospital acquired AKI.

According to Kidney Disease Improving Global Outcomes (KDIGO) 2012 Acute Kidney Injury guideline, contrast-induced acute kidney injury (CI-AKI) is categorized as an increase in serum creatinine of ≥ 0.5 mg/dl, ≥ 44 µmol/l or a 25% increase from baseline value assessed at 48 hours after a radiological procedure. The Society of Urogenital Radiology uses the same definition, with a change in assessment of creatinine changes. They consider serum creatinine increase occurring within 3 days after intravascular administration of contrast media without any other possible reason.[1]

In newly hospitalized patients due to AKI related problems, CI-AKI has been found to be responsible for 11% of the cases becoming the third most common cause.[2] Even though the risk for CI-AKI in patients with normal renal functions (even in patients with Diabetes) has been found to be low at around 1% to 2%,[3] the incidence can go as high as 25% for patients with renal problems or for patients that pose multiple complications like combination of CKD and diabetes, congestive heart failure, usage of nephrotoxic drugs and also advance age.[4]

According to studies, patients that develop CI-AKI have shown to possess a greater risk for death and also have shown prone to longer hospitalisation durations and immediate or late, cardiovascular complications.

The Cardiac Angiography in Renally Impaired Patients (CARE) study, a multicenter, large, prospective, double-blind RCT of patients who had severe to moderate chronic kidney disease and were undergoing cardiac angiography with or without percutaneous interventions also has shown that the adjusted incidence rate ratio for adverse events was twice as high in those with CI-AKI.[5]

Objectives

The objectives of this study were to,

1. Assess the incidence of CI-AKI in patients admitted to cardiac ICU of National Hospital of Sri Lanka (NHSL) following coronary angiography and/or percutaneous interventions
2. Discover other risk factors for the development of CI-AKI
3. Assess the incidence and outcome of patients who require renal replacement therapy

Methodology

All the patients admitted to Cardiology ICU NHSL following coronary angiography with or without percutaneous coronary intervention over a period of two weeks were included into the study. This was a retrospective observational study.

Ethical clearance has been taken from the NHSL ethical committee, also permission was obtained from Consultant Intensivist and all the Consultant Cardiologists at the Cardiology ICU NHSL to be carried out this study.

All the procedures performed with iso-osmolar contrast were taken into the study. CI-AKI was defined according to the serum creatinine component of the KDIGO criteria.

Data were analysed using SPSS software version 24.

Results

58 patients admitted to cardiology ICU NHSL following coronary angiography with or without percutaneous coronary intervention over a period of two weeks were audited. Out of these patients, 40 were males and 18 were females.

The incidence of CI-AKI was 17.24%. The incidence according to sex is shown in Figure 1 below. There is no statistically significant difference in the incidence of CI-AKI between male and female sex (Z = 0.674).

Figure 1 - Incidence according to sex

Total population

Patients with AKI

The incid[...] o age group distribu[...]own in Figure 2 [...]ne on the incidence[...] as this was not t[...] audit. But it is evid[...]ease in incidence o[...]f age.
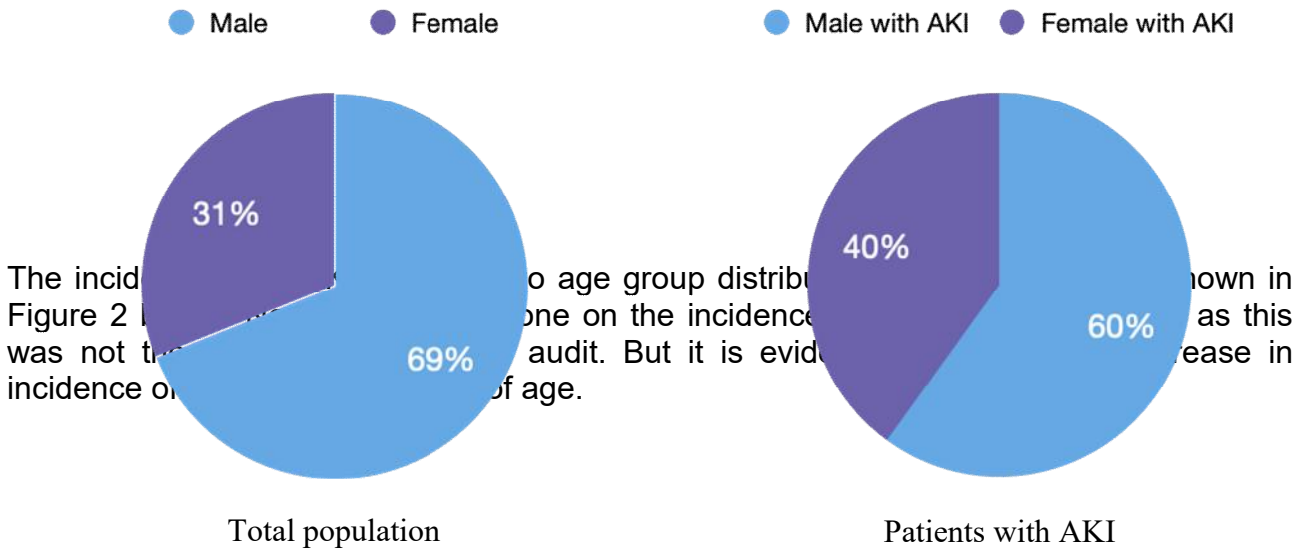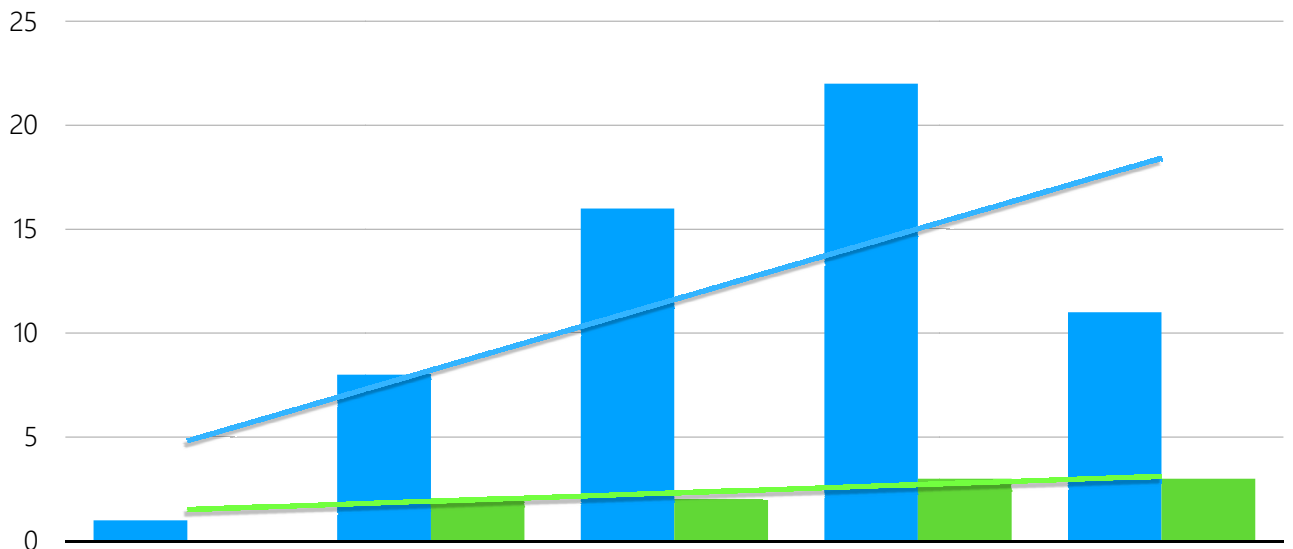
Figure 2 - Age group distribution

The incidence of identified risk factors for the development of CI-AKI were shown in Figure 3 below. The most common identified risk factors were hypertension (53.84%), diabetes (38.46 %), dyslipedemia (38.46 %), smoking (30.76 %) and Ischemic Heart Disease (15.38%).

Even though hypertension was present in more than half of the patients who developed CI-AKI, it was not shown to be associated with development of CI-AKI in a statistically significant way (Test statistic: $z = 1.581$, Critical value @ 5%: $C=1.96$, $P=0.114$).

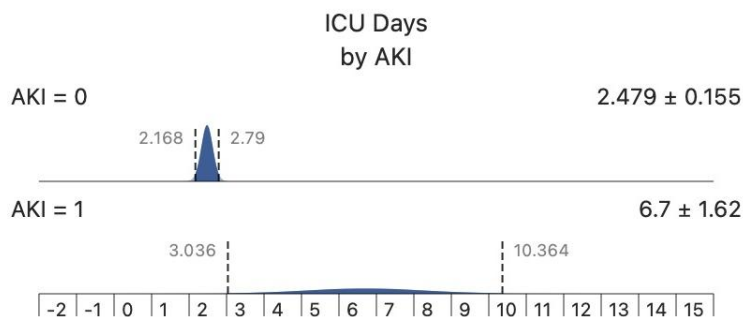Figure 3 - Incidence of identified risk factors as a percentage
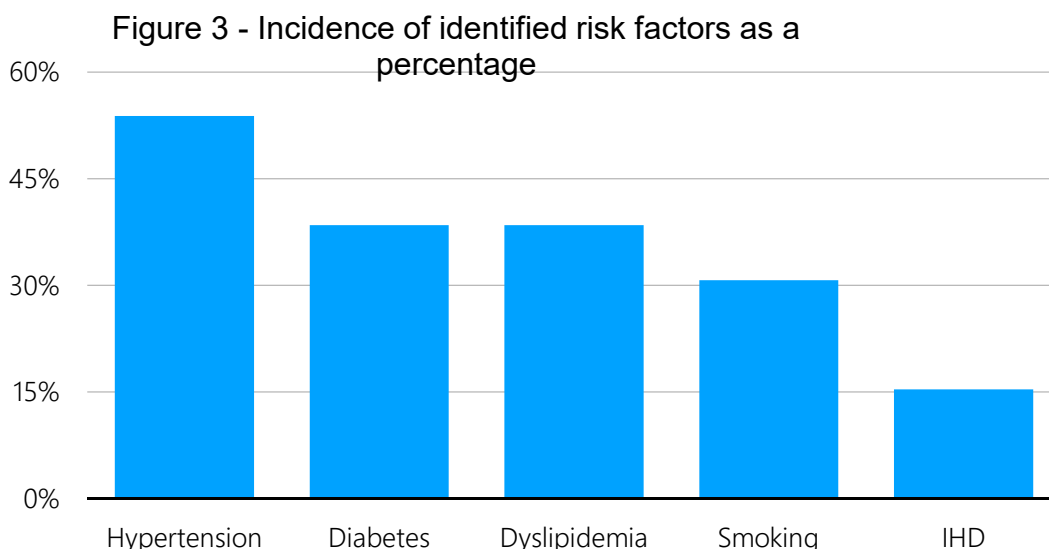
According to the study, the most common identified nephrotoxic drugs to be associated with CI-AKI were metformin (30%), ACEI (30%) and diuretics (10%). It is shown in Figure 4 below in comparison to total study population who were on nephrotoxic drugs.

Figure 4 - Usage of nephrotoxic drugs
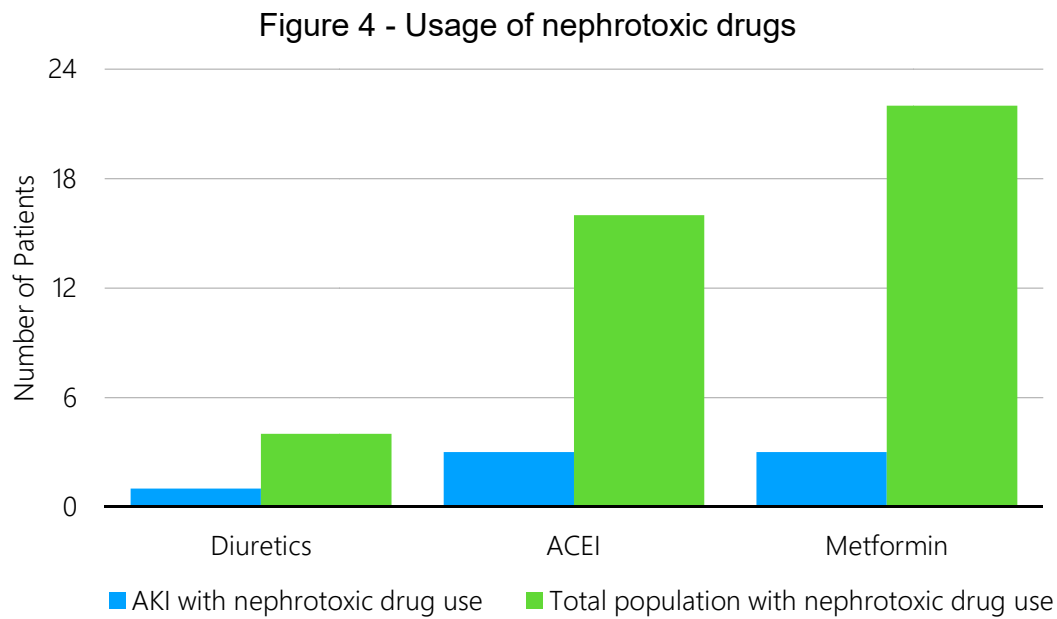
It is evident that metformin and ACEI have an equal chance of associated risk of developing CI-AKI. NSAIDs and gentamicin are also known nephrotoxic drugs.[6] Unfortunately, there were no patients who took these drugs in the study population.

The average length of intensive care stay was 6.7 days for the patients who developed CI-AKI, whereas it was 2.48 days for others who did not develop CI-AKI. This was statistically significant (χ^2 = 18.981, P value <0.001).

ICU Days
by AKI

AKI = 0   2.479 ± 0.155

2.168   2.79

AKI = 1   6.7 ± 1.62

3.036   10.364

-2 -1 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15

Out of all the studied patients, none of the patients required non-invasive ventilation via CPAP but 8.6% required invasive ventilation as shown in Figure 5. This was 30% for the patients who did develop CI-AKI whereas 4.16% for the patients who did not develop CI-AKI as shown in Figure 6. Patients with CI-AKI needing invasive ventilation was statistically significant (Test statistic Z= 2.684, Critical Value at 5% - c = 1.96, P value 0.008).

Figure 5 - Distribution of mechanical ventilation

2: 8.6% (5)

0: 91.4% (53)

AKI = 0

95.8%

AKI = 1

70%   30%
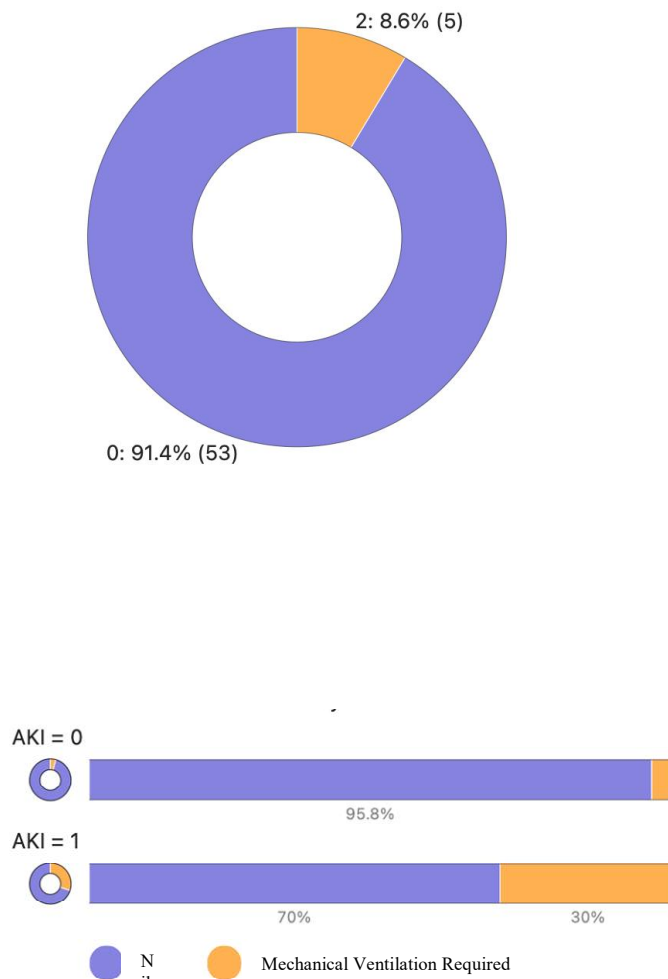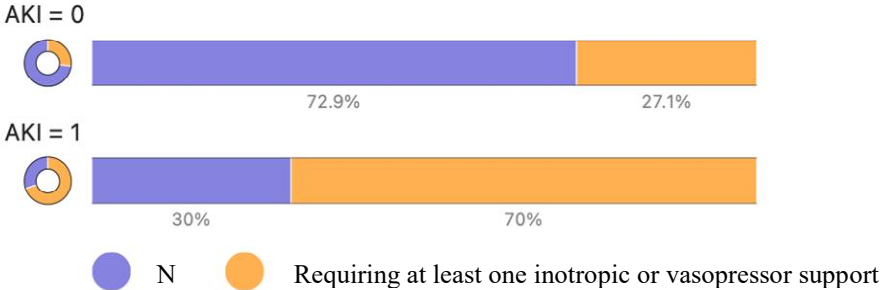
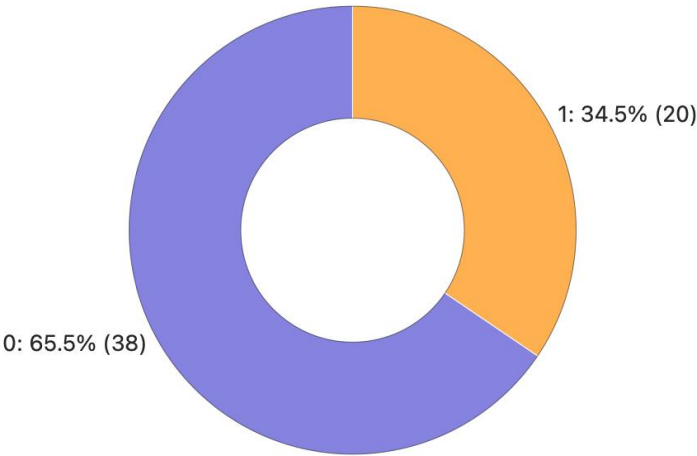N   Mechanical Ventilation Required
:1

Figure 6 - Graphical representation of requirement for mechanical ventilation between patients who developed AKI vs who did not

Out of the studied population, 34.48% of patients required inotropic or vasopressor support and it was 70% for the patients who developed CI-AKI with statistical significance of Z=2.597, Critical Value at 5% - C = 1.96 and P value 0.009. This is shown in Figure 7.

1: 34.5% (20)

0: 65.5% (38)

AKI = 0

72.9%   27.1%

AKI = 1

30%   70%

● N   ● Requiring at least one inotropic or vasopressor support

The usage of different inotropic or vasopressor agents are shown below in Figure 8.

Figure 7 - Distribution of patients requiring at least one inotropic or vasopressor support

40% of patients who developed CI-AKI died during the ICU stay, whereas it was 10.3% for the patients who did not develop CI-AKI. Figure 9. This was statistically significant with z=3.385, Critical Value at 5% - C=1.96 and a P value < 0.001.
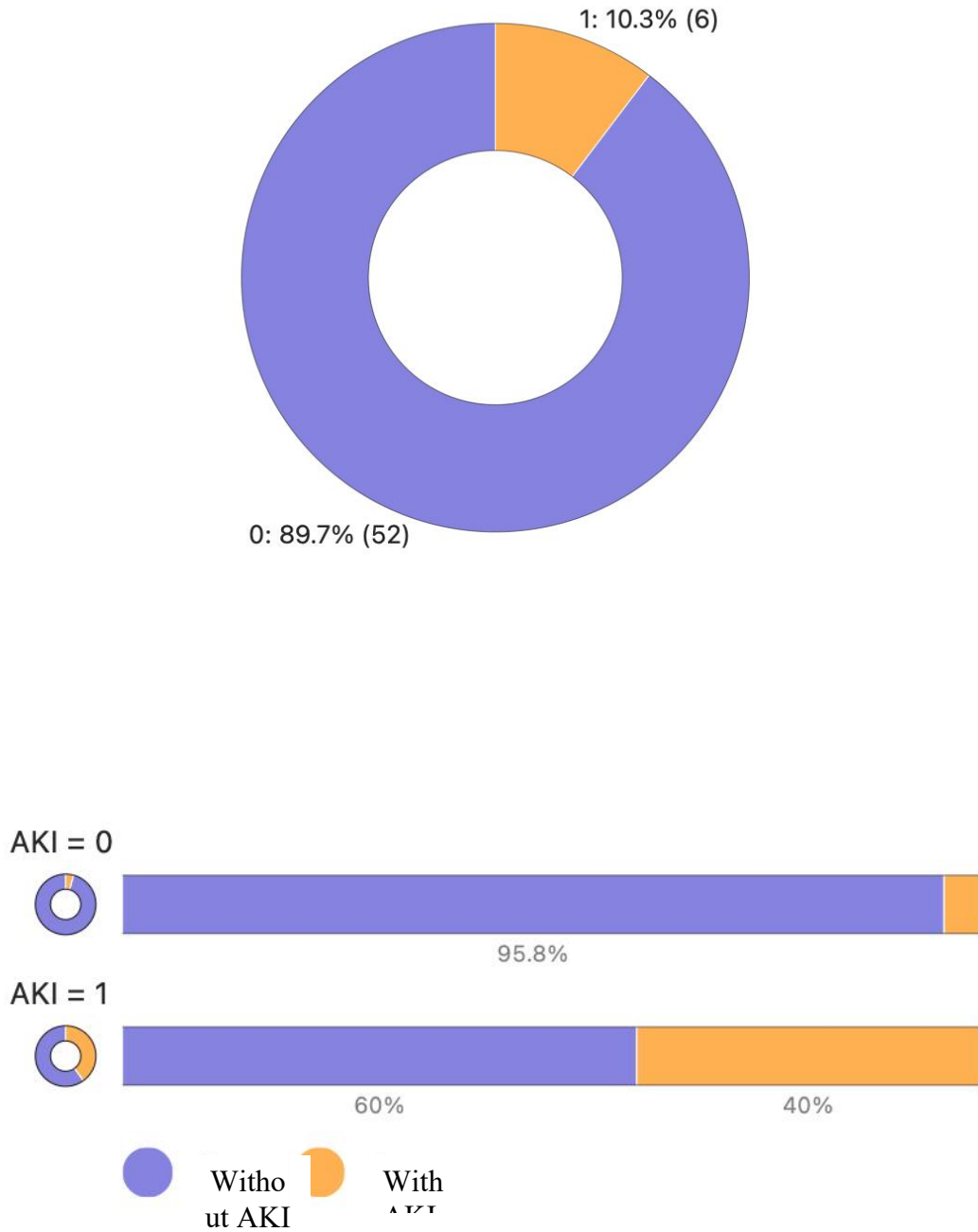

Figure 9 - Incidence of

Out of the patients who developed CI-AKI, 20% needed renal replacement therapy (RRT). The mortality rate of the patients who required RRT were 100%.

Summary

According to the study, the incidence of CI-AKI following coronary angiography and/or percutaneous interventions were 17.24%. The incidence was increasing as the age of the population increases, but there were no statistically significant difference in the incidence between the sex of the patients.

The most common identified risk factors for the development of CI-AKI were hypertension, diabetes, dyslipedemia and smoking. The most common identified nephrotoxic drugs to be associated with CI-AKI were metformin and ACEI. Even though it is well known that above risk factors are associated with increased incidence of AKI, it did not show any statistically significant increased risk of developing CI-AKI in this population.

There was a statistically significant increased duration of intensive care stay in patients who developed CI-AKI with the mean duration of 6.7 days.

Patients who developed CI-AKI had a statistically significant increased incidence of needing invasive mechanical ventilation and inotropic and/or vasopressor support.

Patients with CI-AKI had an increased incidence of in hospital death. This was almost 100% for the patients who required the RRT.

Conclusion

CI-AKI is associated with increased incidence of morbidity and in hospital mortality ultimately leading to increased health care costs. Early identification of patients who are at increased risk of developing CI-AKI and prompt and optimal management can reduce these adverse outcomes.

Recommendations

Organising teaching sessions for the medical and nursing staff working in the cardiac catheterisation laboratory and the cardiac ICU on CI-AKI.

Introduction of checklist to identify patients with high risk of CI-AKI upon admission to ICU.

Ensure renal function tests are monitored immediately upon admission to cardiac ICU on patients who do not have a pre-procedure test result and also to repeat the tests 48 hours after the procedure in high risk patients.

Departmental policy on early involvement of renal specialists in the management of patients who develop CI-AKI.

Re-audit after implementation of the above measures.

References

1. Thomsen HS, Morcos SK. Contrast media and the kidney: European Society of Urogenital Radiology (ESUR) guidelines. Br J Radiol 2003; 76: 513-518

2. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. Am J Kidney Dis 2002; 39: 930-936

3. Berns AS. Nephrotoxicity of contrast media. Kidney Int 1989; 36: 730-740

4. Rudnick MR, Goldfarb S, Tumlin J. Contrast-induced nephropathy: is the picture any clearer? Clin J Am Soc Nephrol 2008; 3: 261-262

5. Solomon RJ, Natarajan MK, Doucet S, et al. Cardiac Angiography in Renally Impaired Patients (CARE) study: a randomized double-blind trial of contrast-induced nephropathy in patients with chronic kidney disease. Circulation 2007; 115: 3189-3196

6. Everson M, Sukcharoen Kand Milner Q. Contrast-associated acute kidney injury. BJA Education, 20(12): 417-423 (2020)