



Retrospective Monocentric 10-Year Analysis Of Sepsis-Associated Acute Kidney Injury: Impact On Outcome, Dialysis Dose And Residual Renal Function

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Background

Acute kidney injury (AKI) is a common clinical problem in critically ill patients admitted to Intensive Care Units (ICU).

AKI is an independent predictor of poor outcome.

Sepsis represents the predominant cause of AKI in ICU patients.

Septic AKI is associated with microvascular dysfunction, multiple organ failures (MOF) and high mortality rates.

The mechanisms of sepsis-associated AKI and MOF are related to the presence of high levels of circulating plasmatic inflammatory mediators that correlate with mortality and lead to the intensification of the dialytic dose.

Aims of the study

The aims of this study were:

- 1) to evaluate the incidence of sepsis as cause of AKI in the period 2001-2010 in the monocentric experience of our 900-bed University Hospital;
- 2) to identify the impact of sepsis on outcome and on residual renal function of AKI patients;
- 3) to identify the impact of sepsis on the management of renal replacement therapy .

Methods

We analyzed all patients admitted to ICUs and treated by Renal Replacement Therapy (RRT) for AKI in the period 2001-2010. For all patients RIFLE, SOFA and ATN_ISS scores were calculated.

ATN_ISS: 0.032 (decade of life) - 0.086 (gender) - 0.109 (nephrotoxicity) + 0.109 (oliguria) + 0.116 (hypotension) + 0.122 (jaundice) + 0.150 (coma) - 0.154 (consciousness) + 0.182 (mechanical ventilation).

Diagnosis of sepsis/septic shock was performed according to published criteria.

Patients' outcome was assessed 28 days after ICU admission.

Statistical analysis was performed using the Hemer-Lemeshow test.

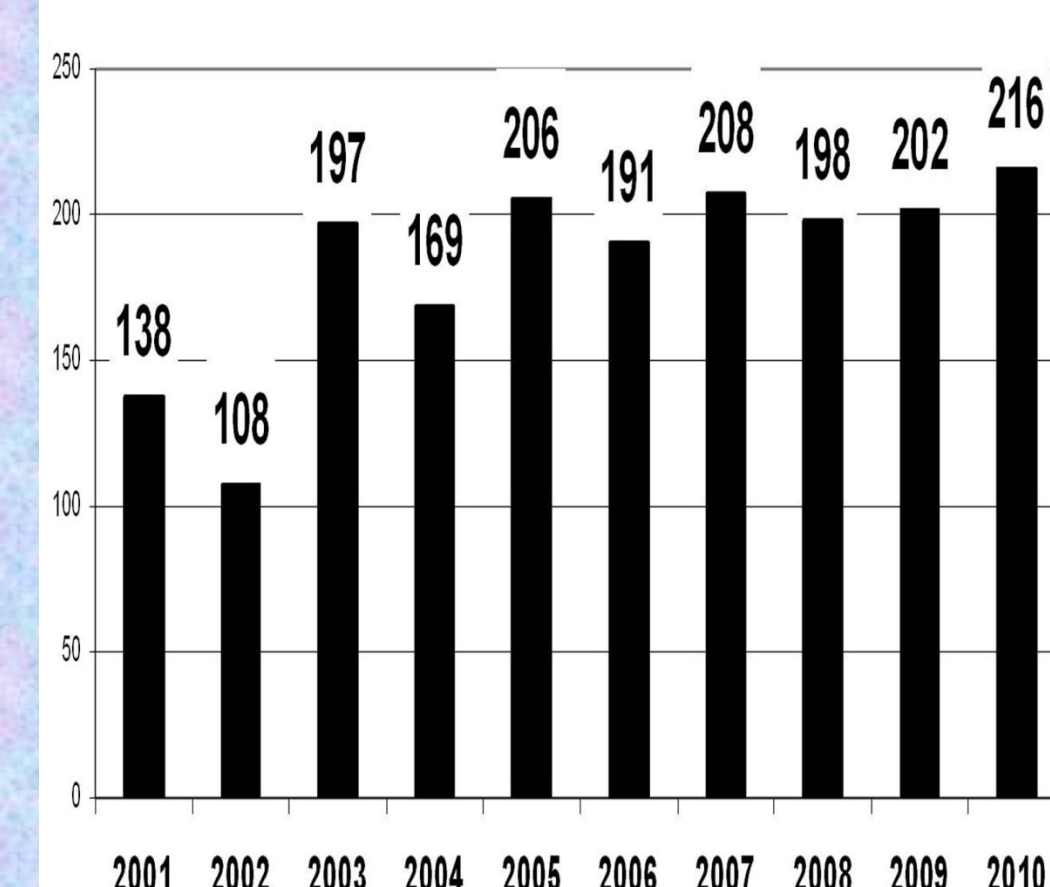


Figure 1. Number of AKI patients/year. Total: 1833 patients

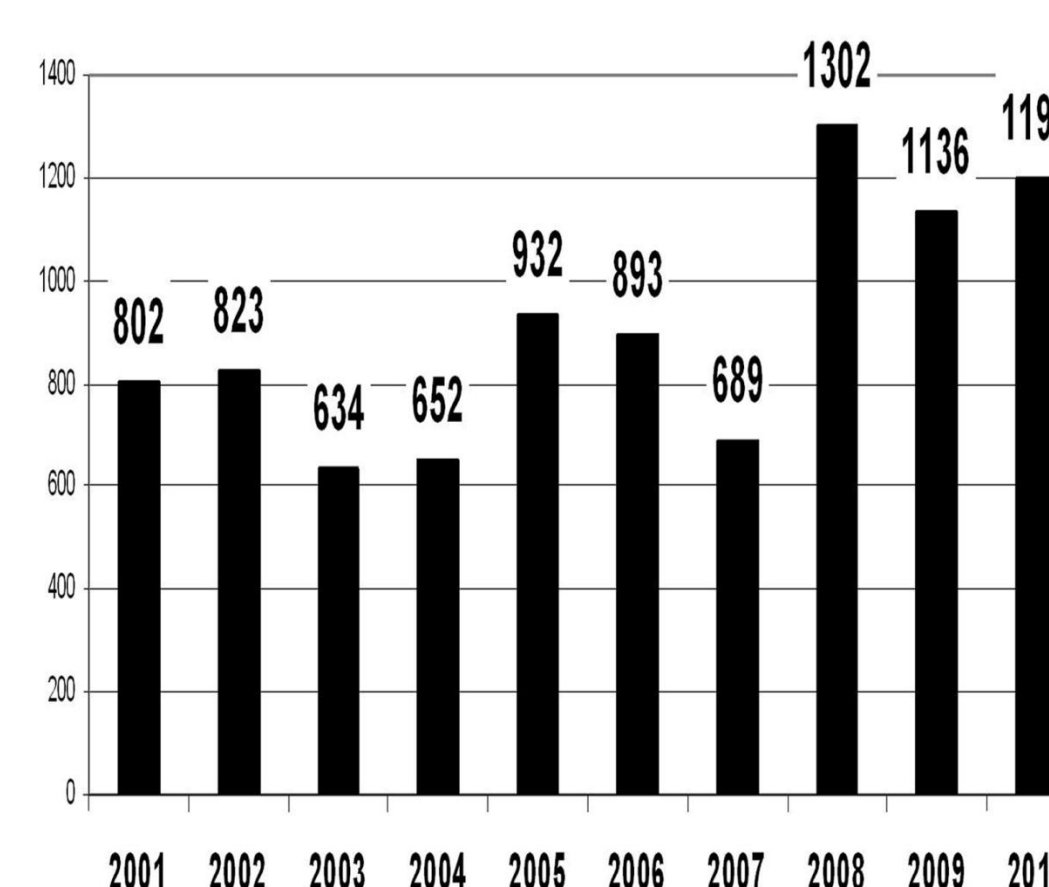


Figure 2. Number of RRT sessions/year. Total: 9061 RRT sessions

| | |
|--------------------------|-------------------|
| Age | 66.4 ± 11.5 years |
| Male | 64.7 % |
| Serum creatinine | 3.8 ± 1.9 mg/dl |
| Number of organ failures | 3.4 ± 1.38 |
| SOFA | 10.6±1.3 |
| ATN_ISS score | 0.738 ± 0.192 |

Figure 3. Characteristics of whole AKI population treated by RRT

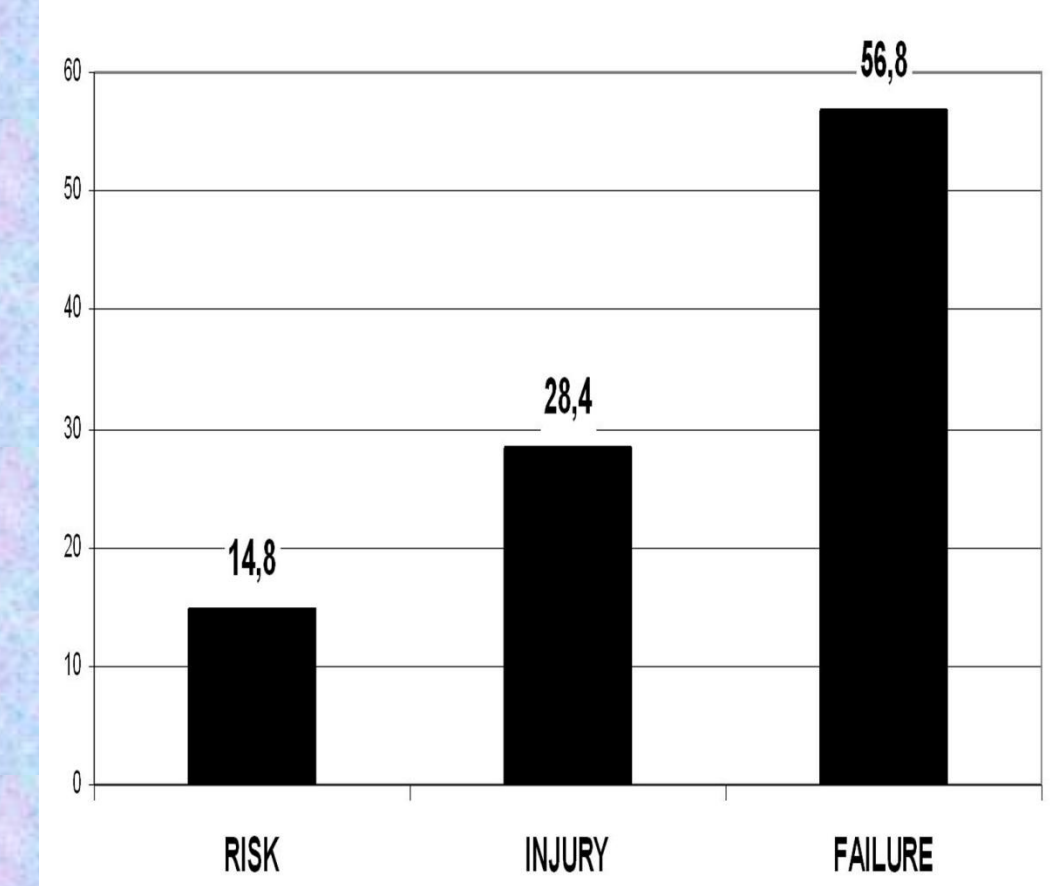


Figure 4. RIFLE criteria

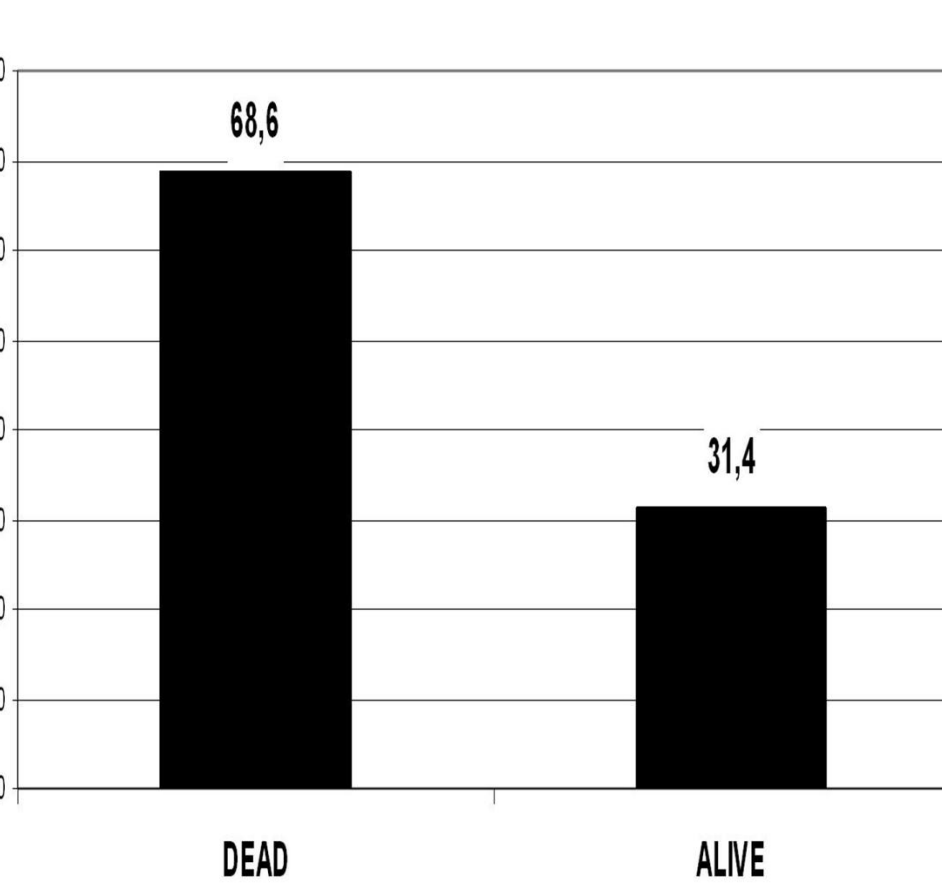


Figure 5. Percentage of mortality in AKI patients at day 28

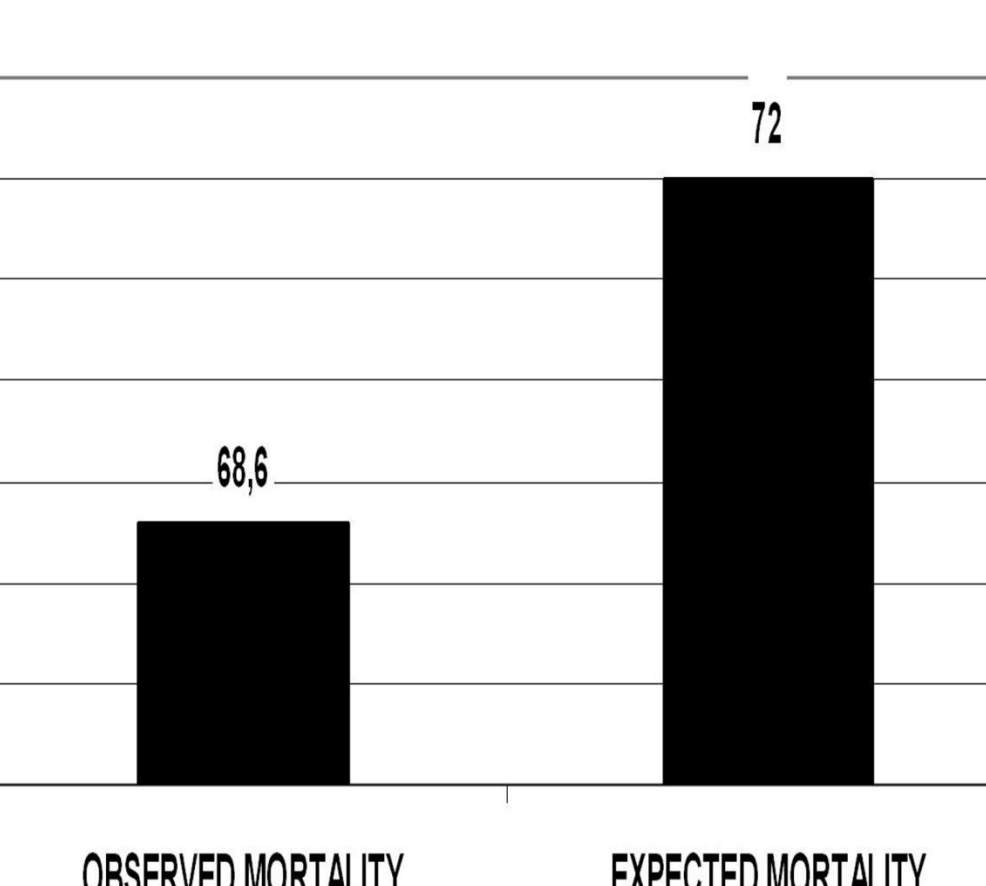


Figure 6. Percentage of observed vs. expected mortality in AKI patients at day 28

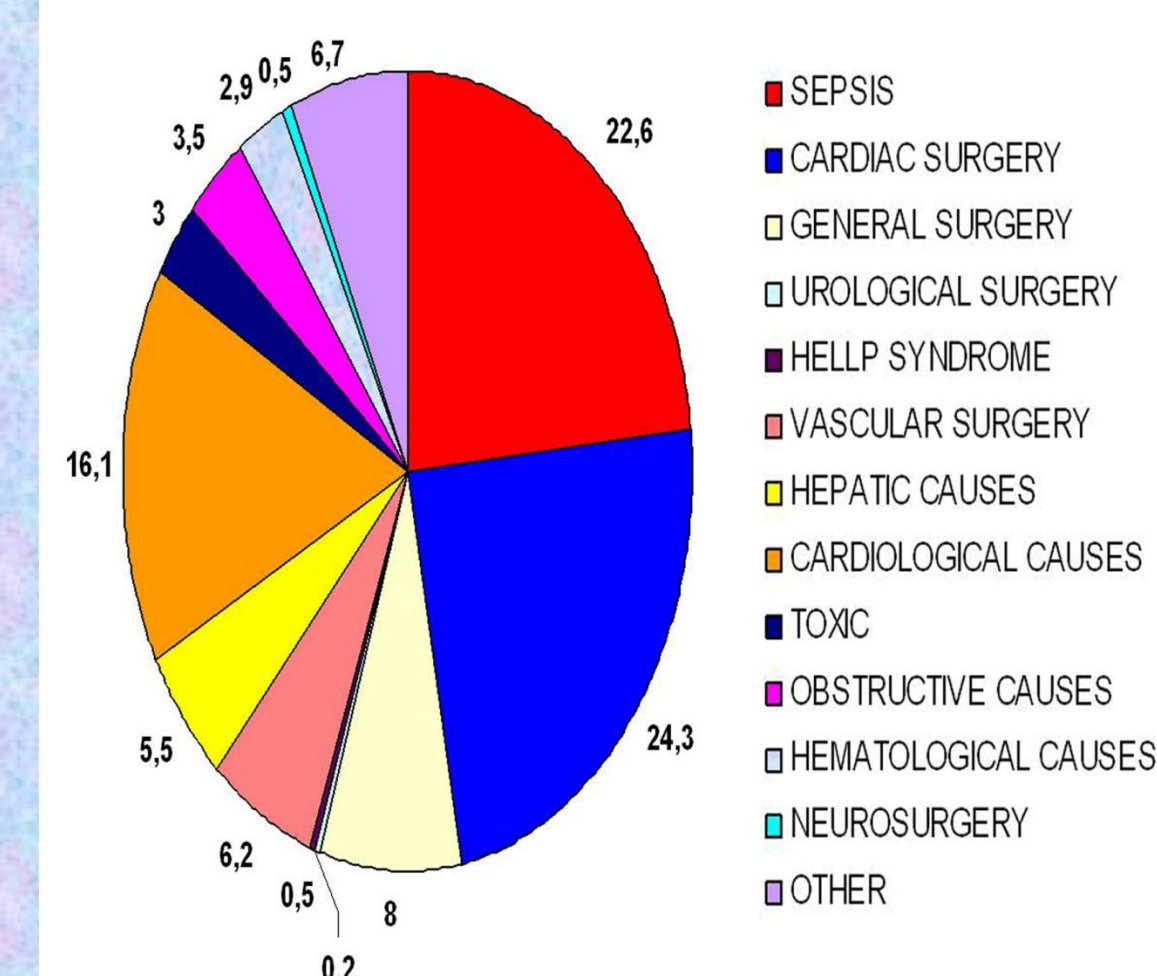


Figure 7. Cause of AKI - percentage

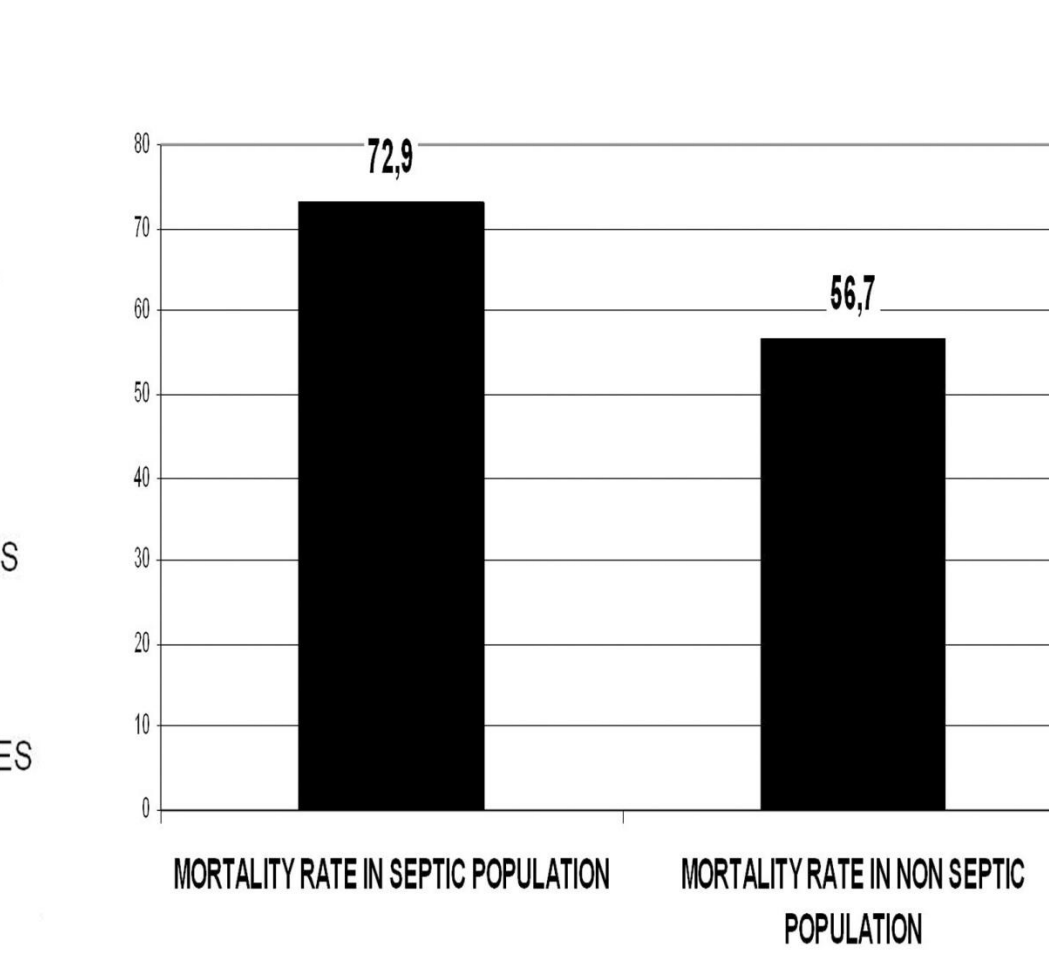


Figure 8. Percentage mortality in septic vs. non septic AKI populations

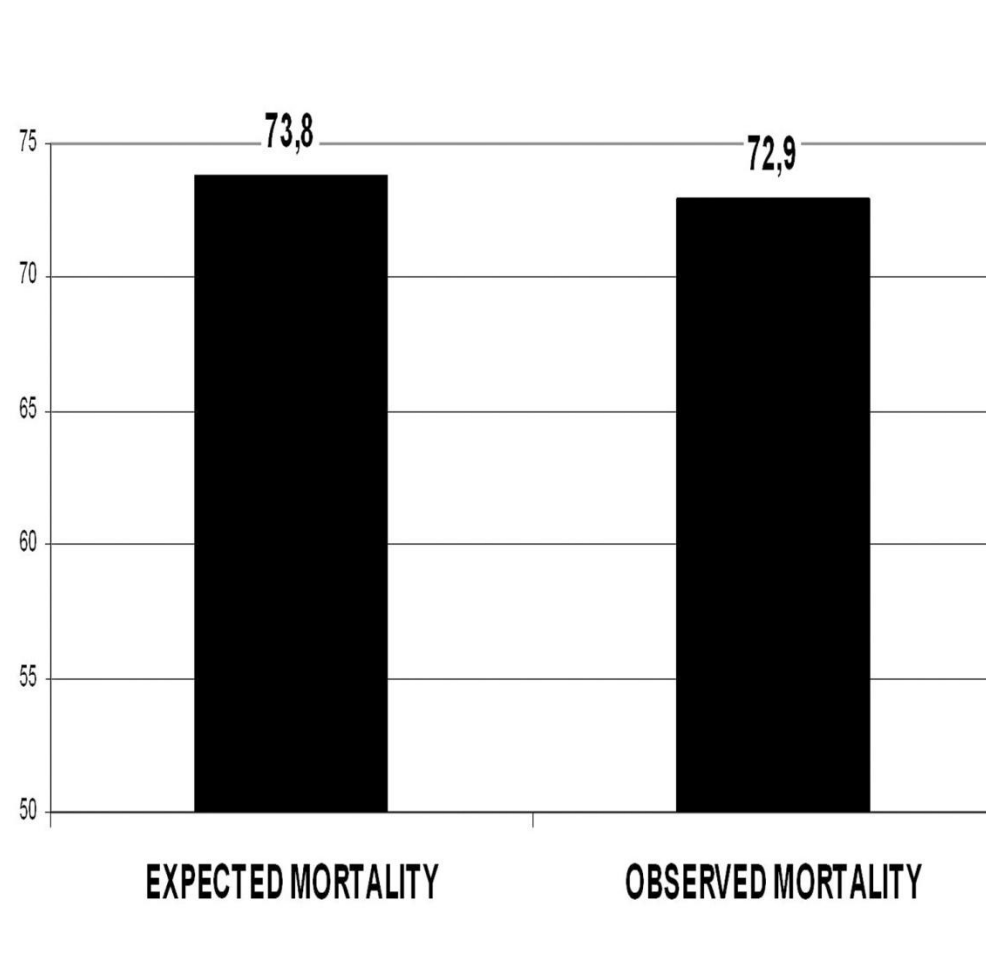


Figure 9. Percentage of observed vs. expected mortality in septic AKI population

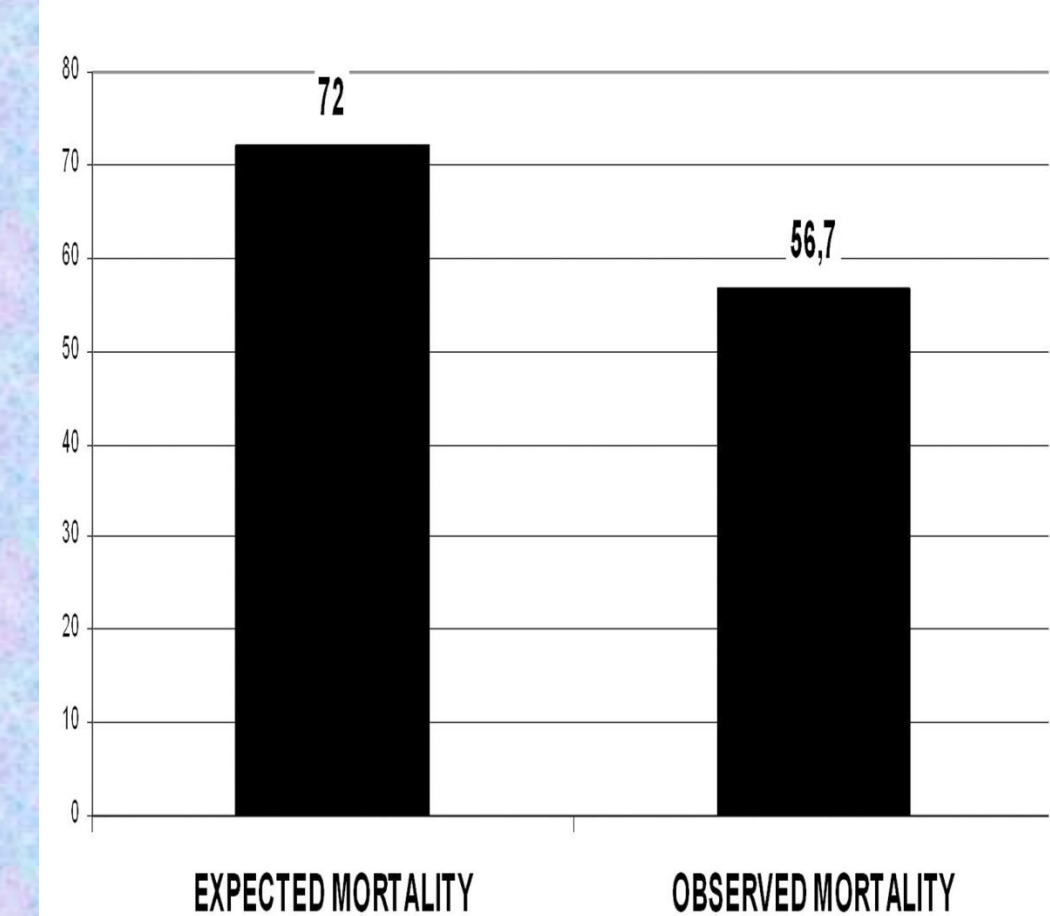


Figure 10. Percentage of observed vs. expected mortality in non septic AKI population

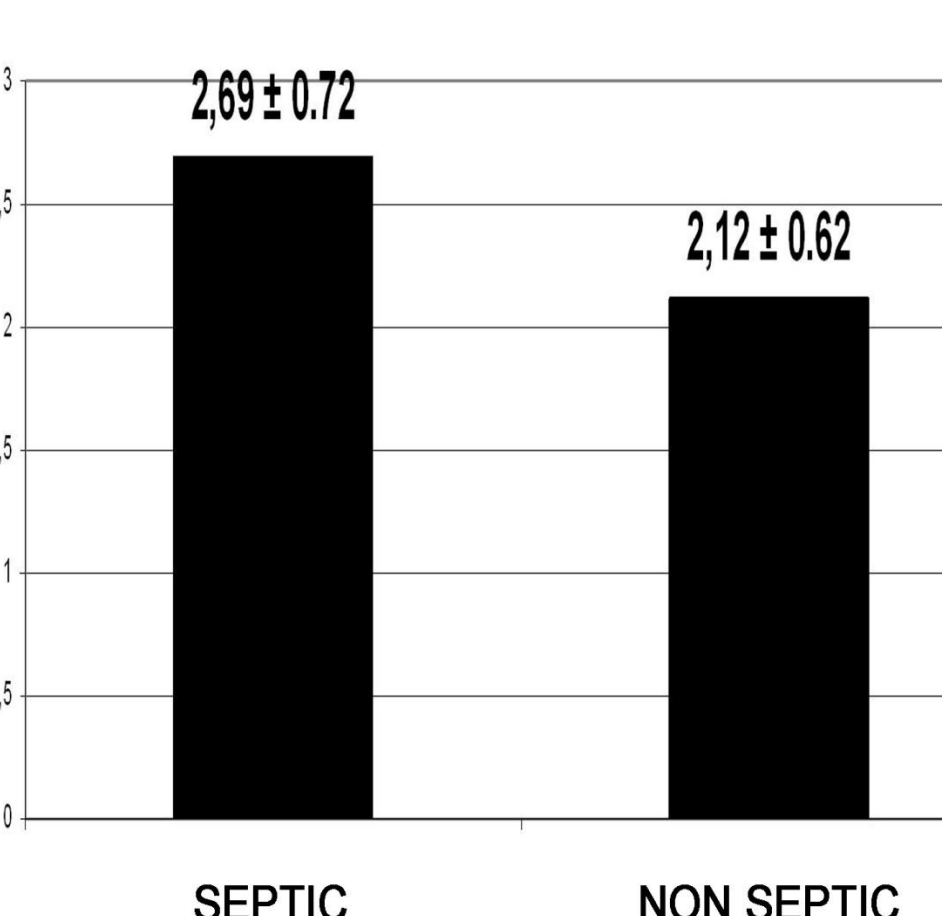


Figure 11. Serum creatinine at day 28 in septic vs. non septic population

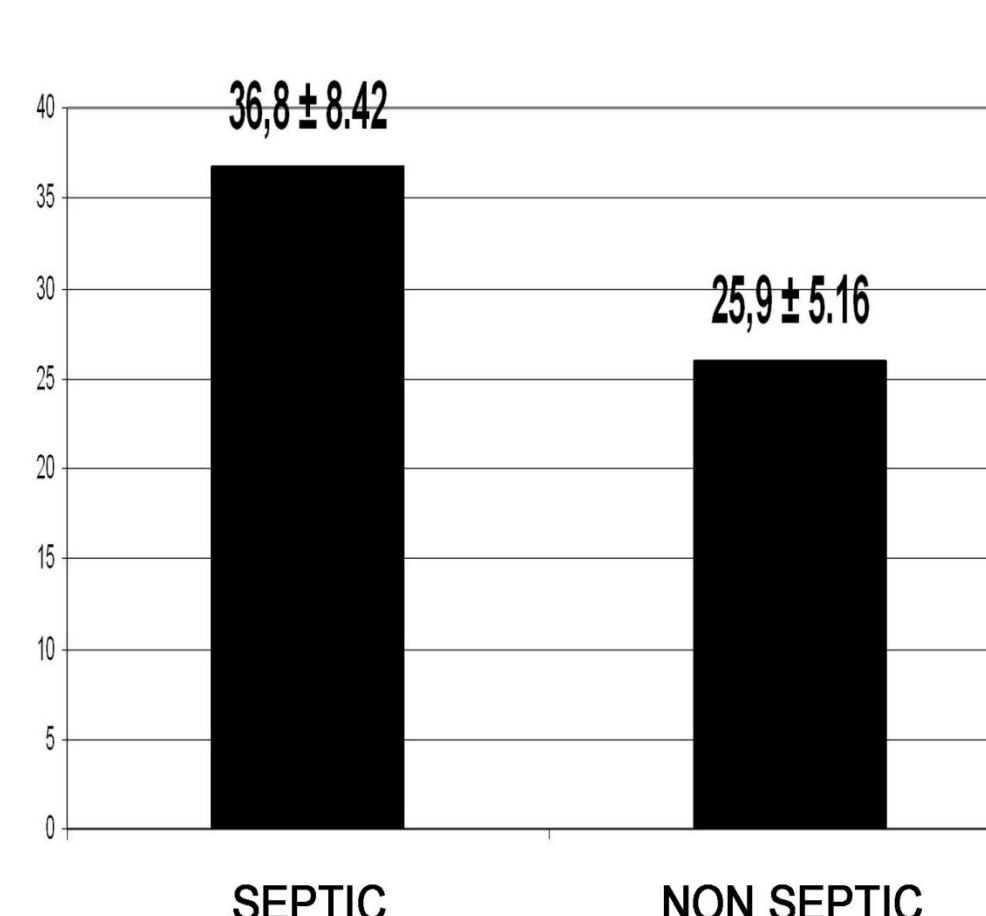


Figure 12. Administered dose (ml/Kg/h) in septic vs. non septic population

Results

We treated by RRT 1833 patients with AKI (Fig. 1) for a total of 9061 sessions performed (Fig. 2). Patients characteristics were: age 66.4±11.5 yrs; 64.7% males; serum creatinine at the start of RRT 3.8±1.9 mg/dl; number of organ failures: 3.4±1.3; SOFA 10.6±1.3; ATN_ISS 0.738±0.192 (Fig. 3).

In Fig. 4, we represented the distribution of AKI patients enrolled in the study according to RIFLE criteria: Failure 56.8%, Injury 28.4%, Risk 14.8%; At day 28 after admission in the study, the mortality in the whole AKI population was 1257/1833 (68.6%) (Fig. 5). The observed mortality was significantly lower than that expected mortality as assessed by the ATN_ISS score (72%), (p<0.05) (Fig. 6).

Sepsis and cardiac surgery were the prevalent causes of AKI: in particular, sepsis was responsible of 415/1833 cases of AKI (22.6%) (Fig. 7). We then distinguished 2 groups: the septic group (S) and the non-septic group (NS, 1418/1833: 77.4%). In the S group, mortality at day 28 was 302/415 (72.9%), whereas in NS group was 804/1418 (56.7%), (p<0.05) (Fig. 8).

In the S group, the expected mortality was 73.8%, whereas the observed mortality was 72.9% (p>0.05) (Fig. 9). By contrast, in the NS group, the expected mortality was 72%, whereas the observed mortality was 56.7% (p<0.05) (Fig. 10). These results confirmed the worse outcome of patients with sepsis-associated AKI.

In addition, in the surviving patients, serum creatinine at day 28 after admission in the study was 2.69±0.72 mg/dl in the S group and 2.12±0.62 mg/dl in the NS group (p>0.05) (Fig. 11). These data suggest that AKI represents a risk factor for the development of chronic kidney disease. Despite the lack of statistical significance, in comparison to the NS group, the S group showed a tendency toward a worse renal function.

Last, in a small cohort of patients (n=50; 27 septic, 23 non septic), we evaluated the administered convective dose. We found that administered dose was significantly higher in the S group (36.84±8.42 ml/Kg/h) than in the NS group (25.93±5.16 ml/Kg/h). These results suggest that the complex clinical picture of septic patients characterized by severe hemodynamic alterations, increased need of vasopressors and fluid overload often requires an intensification of the dialytic treatment.

Conclusions

Our retrospective analysis showed that:

- 1) sepsis was the most relevant cause of AKI;
- 2) mortality at day 28 in the S group was significantly higher than in the NS group;
- 3) in the NS group but not in the S group, the observed mortality was lower than the expected;
- 4) AKI leads to a possible progression toward chronic kidney disease in both groups;
- 5) administered dose in the S group was higher than in NS group, suggesting that S group is subjected to a more intense dialytic treatment with the aim to limit fluid overload and to remove circulating inflammatory mediators in a condition of severe hemodynamic instability.