

POVIDONE-IODINE A POSSIBLE CAUSE OF CONTRAST INDUCED AKI? OUR CENTER ANNUAL REPORT

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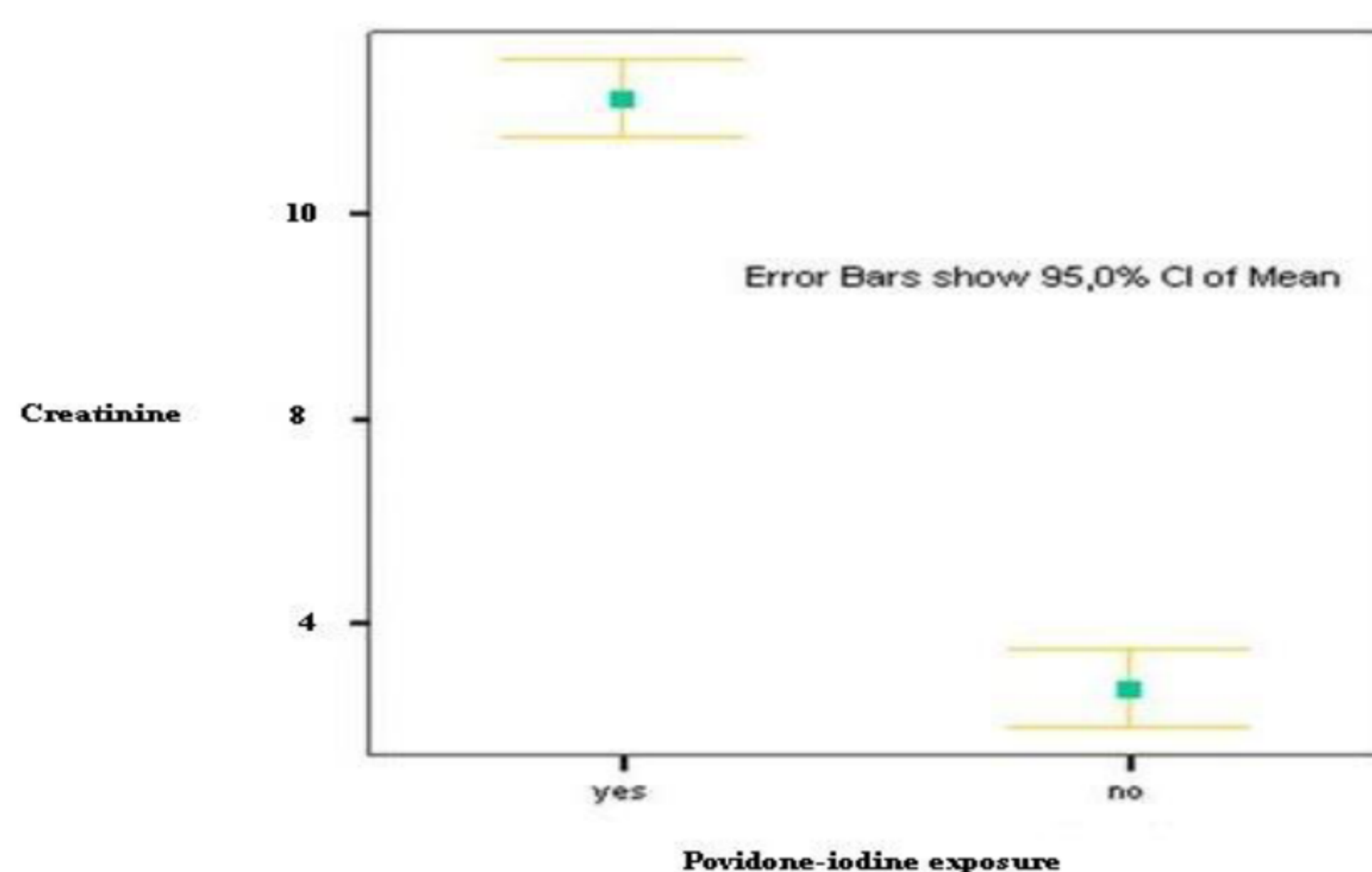
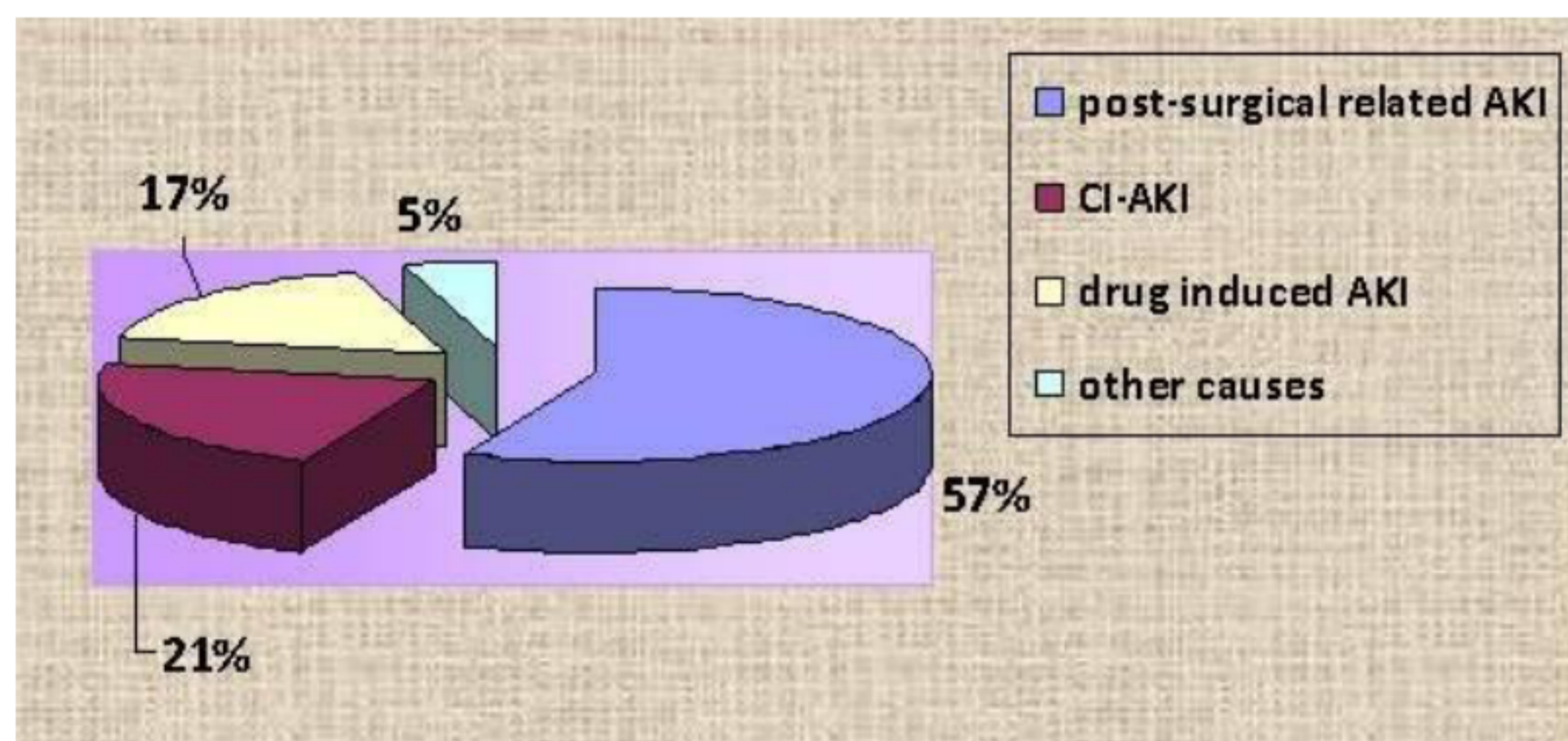
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OBJECTIVES

Nowadays, it is worldwide recognized that acute kidney injury (AKI) represents one of the most important and increasingly common death- and ESRD progression-related cause. Therefore, each year our Center assesses data regarding AKI possible etiology of hospitalized cases for a future better management and control

METHODS

In 2012, 75 patients were diagnosed with AKI and 68% ($n = 51$) – Group A – of them presented acute-on-chronic kidney disease and 32% – Group B – associated no known renal impairment. All patients were age and sex matched and routine lab test (e.g.: hemoglobin, BUN, creatinine, ESR, C-protein, uric acid, albumin levels, coagulation profile) were performed including eGFR measurement using Cockcroft-Gault formula. In the present report we focused on Group B patients and all data were statistically analyzed using SPSS 17.0 software.



RESULTS

Group B included 24 individuals with the following possible etiologies: 57% post-surgical related AKI causes, 21% contrast induced AKI (CI-AKI), 17% drug induced AKI and 5% other causes. The most interesting aspect we observed was regarding the CI-AKI cases. 4 of these patients, all women, presented CI-AKI following non-intravascular use of iodinated substances (povidone-iodine), who underwent hysteroscopy for diagnosis of primary sterility. 2.5 – 1 days after the procedure, the subjects were hospitalized due to an acute elevation in serum creatinine (Cr mean level = 9.32 ± 1.3 mg/dL; $p < 0.001$) and BUN (BUN mean level = 230.35 ± 5.7 mg/dL; $p < 0.001$) values associating oligoanuria, dizziness, nausea and vomiting.

In all cases, except standard AKI management, HD was required and after 10.3 ± 2.7 days of RRT, 3 of them returned to normal renal function and 1 patient remained on chronic HD program. There was a clear correlation between povidone-iodine exposure and increased nitrogen waste products values and consequently, acute renal failure ($p < 0.001 < \alpha = 0.05$; $\chi^2 = 33.523$; $df = 2$).

CONCLUSIONS

Our findings highlighted the possibility of CI-AKI even in non-intravascular administration situation and should raise the attention of the practitioners to consider this diagnosis when iodine substances, injected or not, were recently used. Further larger clinical trials are needed to confirm our results for assure a better management and control of AKI patients with related or not preexistent renal impairment.

