PREVALENCE, RISK FACTORS AND CLINICAL SIGNIFICANCE OF ACUTE KIDNEY INJURY ASSOCIATED WITH CARDIOGENIC SHOCK

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Argomento: Funzione renale e metabolica in terapia intensiva

BACKGROUND

Acute Kidney Injury (AKI) is a well-known complication of Cardiogenic Shock (CS) due to both renal congestion and hypoperfusion. While many studies have evaluated the prevalence of AKI in patients with CS due to acute cardiac ischemia, there is a paucity of data describing non-ischemic CS cohorts. Aim of the present study was to analyze the prevalence, risk factors and clinical impact of AKI in patients with all-cause non-surgical CS.

METHODS

Retrospective study including adult patients consecutively admitted for CS in our ICU (2010-2018). Post-cardiotomy and post-transplant patients were excluded. AKI was defined according to KDIGO criteria. Clinical and biochemical variables were collected at admission and at 90 days. Differences between groups were assessed using Student's t or Fisher's exact test. Risk factors for AKI were evaluated by multiple regression analysis and predictors for mortality at 90 days were analyzed with Cox analysis.

RESULTS

Among 118 patients, etiologies of CS included: 63 decompensated chronic cardiomyopathies (53%), 15 myocarditis (13%), 28 acute coronary syndromes (24%) and12 (10%) other causes.

Table 1 shows patients' baseline characteristics. The prevalence of AKI in our cohort was 63% (10.2% stage-1, 17.8% stage-2, 35% stage-3).

Age, lactate, central venous pressure (CVP), systolic blood pressure, ejection fraction and a history of chronic kidney disease were included in the multiple regression analysis. CVP was identified as the only independent risk factor for AKI (OR 1.11, 95%CI 1.03-1.2, p=0.006).

Age, lactate, CVP and AKI were included in Cox analysis: lactate (HR 1.1, 95%CI 1-1.1, p=0.001) and AKI (OR 3, 95%CI 1.2-7.3, p=0.015) were independent predictors of 90-day mortality.

CONCLUSIONS

Our findings confirm that AKI is often associated with all-cause CS. Venous congestion might play a

crucial role in the development of AKI, whose impact on mortality is significant.

Characteristics	No AKI (n = 43)	AKI (n = 75)	p-value
History			
Age (years)	47 ± 19	50 ± 14	0.38
Female sex	14 (32%)	18 (24%)	0.39
Hypertension	13 (30%)	21 (28%)	0.83
Chronic kidney disease	10 (23%)	27 (36%)	0.21
Hemodynamics			
Systolic blood pressure (mmHg)	101 ± 18	95 ± 19	0.14
Mean blood pressure (mmHg)	69 ± 12	67 ± 14	0.42
Central venous pressure (mmHg)	8.8 ± 4.7	11.8 ± 6.1	0.008
Ejection fraction (%)	26 ± 13	23 ± 12	0.18
Cardiac index (L/min/m²)	2.04 ± 0.72	2.54 ± 1.06	0.20
Oligoanuria	19 (44%)	43 (57%)	0.23
Biochemistry			
Lactate (mmol/L)	3.43 ± 3.62	5.52 ± 4.75	0.01
Arterial pH	7.42 ± 0.11	7.37 ± 0.13	0.03
Creatinine (mg/dL)	1.08 ± 0.40	2.08 ± 1.05	0.0001
Alanine transaminase (U/L)	158 ± 309	376 ± 645	0.031
Bilirubine (mg/dL)	1.36 ± 1.73	1.48 ± 1.46	0.71