Impact of dexamethasone on cardiac injury in critically ill COVID-19 patients

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Aims/Background: Severe COVID-19 pneumonia requiring intensive care treatment remains a clinical challenge to date. Dexamethasone was reported as a promising treatment option, leading to a reduction of mortality rates in severe COVID-19 disease as well as ventilator-dependent days. However, the effect of dexamethasone treatment on cardiovascular outcomes including cardiac injury monitored by cardiac enzymes remains largely elusive.

Methods: For this study, we retrospectively screened 224 consecutive COVID-19 patients between 4/2020 and 1/2021 in three Europeen Hospitals. To avoid bias effects of further applied COVID-19 specific medications including tacilizumab, remdesevir and sarilumab, 46 patients treated with at least one of these substances were excluded from further analyses. In total 178 critically ill COVID-19 patients requiring intensive care treatment and mechanical ventilation were recruited. 113 patients (63.5%) were treated with dexamethasone for a median duration of 10 days (IQR 9–10). 65 patients (36.5%) constituted the non-dexamethasone group. The assessment of cardiac injury was based on cardiac enzymes.

Results: Baseline charactaristics shown in Tab. 1. While peak inflammatory markers seemed to be reduced by dexamethasone treatment (CRP and a trend towards decrease of interleukin 6 levels (CRP maximum level: median: 20 ng/mL (IQR 12–28) vs. 22 ng/mL (IQR 14–37), p=0.043; IL-6 maximum level: median: 192 pg/mL (IQR 78–533) vs. 708 pg/mL (550–885), p=0.085), in the dexamethasone Group also shown a significant reduction in peak troponine levels as shown in Figure 1. CK and CK-MB do not differ significantly by Dexamethasone application. Of note, no significant changes in baseline characteristics were observed between the dexamethasone and non-dexamethasone group (Table 1).

Conclusion: In severe COVID-19, antiinflammatory effects of dexamethasone treatment could be associated with a significant reduction in myocardial injury. Further studies should further evaluate whether Dexamethasone effects directly myocardial involvement in COVID 19.

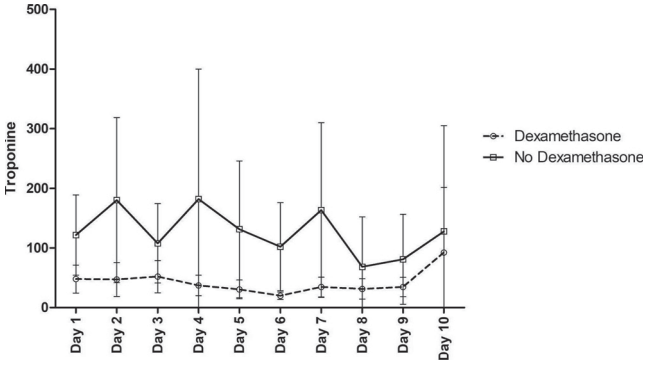

Figure 1. Dynamics of hs-troponine

	Dexamethasone (n= 113)		No dexamethasone (n= 65)		
Baseline characteristics	**median**	**IQR**	**median**	**IQR**	**p-value**
Age (years)	66	59-78	64	56-76	0.313
BMI (kg/m²)	29	26-33	27	25-31	0.099
	%	**n**	**%**	**n**	**p-value**
Male sex	72.6	82	72.3	47	0.970
Diabetes mellitus	36.3	41	29.2	19	0.411
Arterial hypertension	66.4	75	53.8	35	0.111
History of smoking	29.2	33	21.5	14	0.294
Coronary artery disease	20.4	23	16.9	11	0.693
Peripheral artery disease	6.2	7	6.2	4	0.991
Atrial fibrillation	17.7	20	10.8	7	0.279
Heart failure	15.0	17	9.2	6	0.355
Obstructive lung disease	23.0	26	13.8	9	0.172
Structural lung disease	8.0	9	4.6	3	0.540
Malignancy	7.1	8	9.2	6	0.773
History of thromboembolism	12.4	14	6.2	4	0.209
Therapeutic anticoagulation during ICU stay	74.3	84	49.2	32	0.001

Table 1. Baseline characteristics