Dexamethasone improves cardiovascular outcomes in critically ill COVID-19, a real world scenario multicenter analysis

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Funding Acknowledgement: Type of funding sources: None.

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. However, the effect of dexamethasone treatment on cardiac injury and pulmonary embolism remains largely elusive.

Methods: In total 178 critically ill COVID-19 patients requiring intensive care treatment and mechanical ventilation were recruited in three European medical centres and included in the present retrospective study. 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 control group.

Results: While peak inflammatory markers were reduced by dexamethasone treatment, the therapy also led to a significant reduction in peak troponin levels (231% vs. 700% indicated as relative to cut off value, p=0.001). Similar, dexamethasone resulted in significantly decreased peak D-Dimer levels (2.16 mg/l vs. 6.14mg/l, p=0.002) reflected by a significant reduction in pulmonary embolism rate (4.4% vs. 20.0%, p=0.001). The antithrombotic effect of dexamethasone treatment was also evident in the presence of therapeutic anticoagulation (pulmonary embolism rate: 6% vs. 34.4%, p<0.001). Of note, no significant changes in baseline characteristics were observed between the dexamethasone and non-dexamethasone group. **Conclusion:** In severe COVID-19, antiinflammatory effects of dexamethasone treatment seem to be associated with a significant reduction in myocardial injury. Similar, a significant decrease in pulmonary embolism, independent of anticoagulation, was evident, emphasizing the beneficial effect of dexamethasone treatment in severe COVID-19.

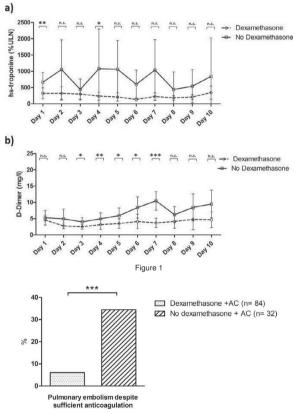


Figure 2