Myocardial injury in patients hospitalized for SARS-CoV19: a maker or a marker of prognosis?

C. Montalto[1], S. Ghio[2], M. Pagnesi[3], A. Cappelletti[3], L. Baldetti[3], E. Baldi[2], C. Lombardi[4], L. Lupi[4], M. Metra[4], S. Perlini[1], L. Oltrona-Visconti[2]

[1]University of Pavia, Pavia, Italy; [2]Policlinic Foundation San Matteo IRCCS, Division of Cardiology, Pavia, Italy; [3]IRCCS San Raffaele Hospital, Milan, Italy; [4]Civil Hospital of Brescia, Brescia, Italy

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Background: Since the beginning of the coronavirus disease 2019 (COVID-19) pandemic, literature data are progressively accumulating, attesting to the possible prognostic role of cardiac troponins in patients who need hospitalization because of COVID-19 infection.
Purpose: To assess whether myocardial injury (measured by high sensitivity troponins) is an independent cause of disease severity and prognosis.
Methods: We performed a patient-level metanalysis (PROSPERO ID: CRD42020213209) in unselected patients hospitalized because of COVID-19 infection in whom the severity of respiratory failure was also evaluated at admission. To allow for comparison, troponin values were normalized to their threshold levels to obtain a normalized troponin (nTn) value which was used as a continuous variable in all analysis.
Results: A total of 722 patients were included in the analysis. Of note, patients who had elevated troponins at hospital admission had a significantly lower oxygenation status than those with normal nTn (PaO2/FiO2 232±215 vs. 276±124 mmHg/%; p<0.001). On the contrary, those with cardiovascular comorbidities had similar PaO2/FiO2 but higher nTn than those without (5.6817 vs. 2.1110 ng/mL; p=0.002).
After a median follow-up of 14 days, 180 deaths were observed. At multivariable regression analysis, age, male sex, moderate-severe renal dysfunction (eGFR <30 mL/min/m^2) and lower PaO2/FiO2, were independent predictor of death (igure 1). The restricted cubic spline curves in Figure 2A and 2B show the hazard ratios (HRs) and 95% confidence interval for death according to nTn and PaO2/FiO2 levels as continuous variables. A linear increase in the HR is observed with lower PaO2/FiO2 values below the normal value of 300. On the contrary, the nTn spline curve is near-flat with large confidence interval for values above the normality thresholds.
Conclusion: In patients hospitalized for COVID-19, mortality is mainly driven by gender, age and respiratory failure while myocardial damage is not an independent predictor of worse survival when respiratory function is accounted for.

Age	1.083	1.061	1.105	< 0.0001
Male sex	2.049	1.319	3.184	0.0014
eGFR < 30 mL/min	2.108	1.237	3.594	0.0061
PO2/FiO2*	1.110	1.206	1.022	0.0133
Diabetes mellitus	1.472	0.988	2.193	0.0573
normalized hs-Tn	1.014	0.996	1.032	0.1232
Cancer	0.692	0.429	1.115	0.1303
Cardiovascular comorbidities	1.168	0.800	1.706	0.4217
Anemia	1.069	0.728	1.569	0.7350

*** per each 50 mmHg/% decrease**

Figure 1

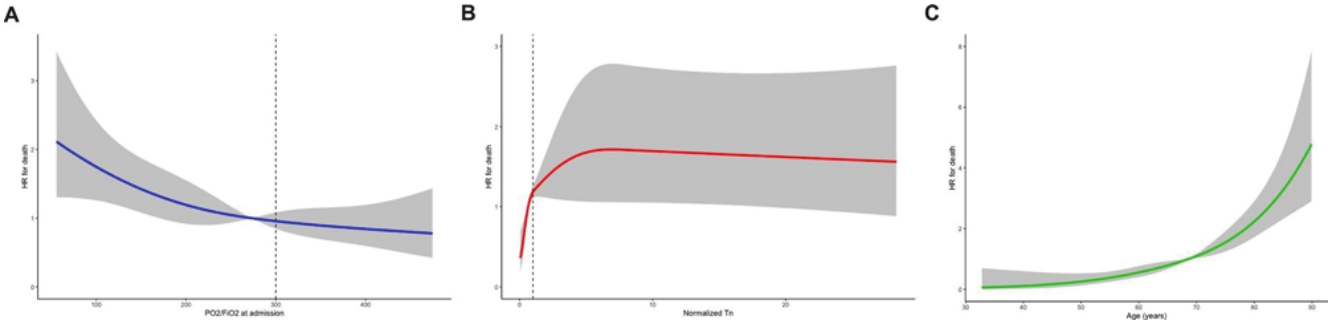

Figure 2