P118 THE ROLE OF PULMONARY ACT IN THE PATIENT WITH SARS-COV-2 INFECTION

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Already from the first data in China it emerged that patients with cardiovascular comorbidities had an increased risk of contracting SARS-CoV-2 infection and a more unfavourable clinical course. From March to May 2020, 85 patients affected by COVID-19 were enrolled, hospitalized at the Hospital of Reggio Calabria. All patients underwent anamnesis, clinical evaluation, chest CT, ECG and measurement of markers of cardiovascular damage (Troponin I, CK-MB, LDH, D-dimer, BNP) and of inflammation (PCR, IL-6, and PCT). Thirty-one patients underwent echocardiography. In particular, we evaluated parietal dimensions and thicknesses, biventricular function and transvalvular tricuspid and pulmonary flows and correlated the data obtained with ECG, radiological, clinical, and biohumoral parameters. The aim of our study was to evaluate the prognostic impact of cardiovascular involvement in COVID-19, investigating the effect of cardiovascular risk factors, levels of cardiovascular damage markers and newly emerging ECG and echocardiographic changes on a composite primary endpoint, consisting of the combination of death and the need for intensive care (ICU). The enrolled patients were divided into two subpopulations: those with better prognosis and those with poorer prognosis (ICU/exitus). We analysed the reciprocal correlation of each of the parameters and searched for the presence of echocardiographic signs of repercussion on the right sections of the pulmonary pathology. All markers of cardiovascular damage had significantly higher values in the most critically ill patients and similar behaviour had indices of inflammation. Patients with poorer prognosis had significantly lower lung AcT values, which correlated with higher D-dimer levels and more complicated hospital stavs. There were no statistically significant differences between PAPs, right ventricular size, TAPSE and pulmonary trunk diameter in the two subpopulations. Larger right ventricular diameters were associated with more dilated lung trunks and higher IL-6 levels. The most interesting data of our study is the behaviour of pulmonary AcT: lower values of AcT were associated with higher levels of D-dimer, expression of a greater pulmonary microthrombotic burden, and a poorer prognosis, in the presence of PAPs normal. The dynamic analysis of this parameter, which is easy to calculate in the patient's bed, can play a crucial role in the instrumental follow-up of patients hospitalized for SARS-CoV-2 infection.

Parametri bioumorali	Pazienti senza endpoint combinato al FU (N = 60)	Pazienti con endopint combinato al FU (N = 25)	p
D-dimero (ng/ml)	617 (I.R. 263- 2882)	2279 (I.R. 848- 6341)	0,001
Troponina I (basale, ng/L)	0,012 (I.R. 0,012- 0,012)	0,012 (I.R. 0,012- 0,034)	<0,001
Troponina I (picco, ng/L)	0,012 (I.R. 0,012- 0,018)	0,052 (I.R. 0,018- 0,557)	<0,001
CK-MB (U/L)	13 (I.R. 9-16)	18 (I.R. 13-29)	0,001
BNP (pg/ml)	247 (I.R. 45-582)	799 (I.R. 348- 3418)	0,007
PCR (picco, mg/L)	74 (I.R. 12-135)	153 (I.R. 127-197)	<0,001
IL-6 (pg/ml)	7,9 (I.R. 3,2-20,8)	527 (I.R. 72-5000)	<0,001

Tabella 17. Confronto dei dati bioumorali tra pazienti con e senza endpoint combinato (mediana e range interquartile). CK-MB: Creatinfosfochinasi-MB. BNP: Peptide Natriuretico Cerebrale. PCR: Proteina C Reattiva. IL-6: Interleuchina-6.

Parametri ecocardiografici	Pazienti senza endpoint combinato al FU (N = 60)	Pazienti con endpoint combinato al FU (N = 25)	p
Diametro basale VD (mm)	37,1±3,5	38,1±3,8	0,52
FE (%)	60±5,8	61±5	0,54
TAPSE (mm)	23,5±2,8	23,6±3,7	0,95
PAPs (mmHg)	27,1±9,5	32,7±7,5	0,17
AcT polmonare (msec)	116,7±17,7	93,1±11	0,002
Tronco polmonare (mm)	23±3,9	25,1±3,7	0,2
AcT/PAPs	4,5±1,2	3,5±1,5	0,13

 Tabella 16. Confronto dei parametri ecocardiografici tra pazienti con e senza endpoint combinato (media ± DS). FE: Frazione d'Eiezione. TAPSE: "Tricuspid Annular Plane Systolic Excursion". PAPs: Pressione arteriosa sistolica polmonare. AcT: Acceleration Time polmonare.