ESC
European Society
of Cardiology

466 The role of pulmonary act in the patient with SARS-CoV-2 infection

Annita Bava[1], Domenica Zema[1], Concetta Zito[2], and Francesco Antonio Benedetto[1]
[1]Grande Ospedale Metropolitano, 'Bianchi Melacrino Morelli' di Reggio, Calabria, Italy, and [2]Policlinico Universitario 'G. Martino' di Messina, Italy

Already from the first data in China in early 2020 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 Great Metropolitan Hospital of Reggio Calabria. The mean age was 63.6 ± 16.5 years. 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 to look for signs of left ventricular dysfunction and/or repercussions of lung disease on the right

sections. 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 exitus and the need for intensive care (ICU). For this purpose, the enrolled patients were divided into two subpopulations: those with better prognosis and those with poorer prognosis (ICU/exitus). We then analysed the reciprocal correlation of each of the investigated parameters and searched for the presence of echocardiographic signs of repercussion on the right sections of the pulmonary pathology. Among the patients with the poorest prognosis, 81.2% were hypertensive, 12.5% diabetic, 25% dyslipidaemic. Comparing the two subpopulations analysed, it emerged that patients with the worst prognosis were known hypertensive (P 0.02). Longer QTc intervals were associated with higher levels of CRP ($P < 0.0001$) and PCT (P 0.005). All markers of cardiovascular damage had significantly higher values in the most critically ill patients (P 0.001 for D-dimer, $P < 0.001$ for baseline and peak Troponin, P 0.001 for CK-MB, P 0.007 for BNP) and similar behaviour had indices of inflammation ($P < 0.001$ for PCR and IL-6). Patients with poorer prognosis had significantly lower lung AcT values (P 0.002), which correlated with higher D-dimer levels (P 0.01) and more complicated hospital stays (P 0.02). 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 (P 0.009) and higher IL-6 levels (P 0.004). 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, as an expression of a greater pulmonary microthrombotic burden, and a poorer prognosis, in the presence of PAPs basically 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.