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Ventriculo-arterial interplay in acute pulmonary edema: relationship with the ejection fraction and the clinical outcome

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Ventriculo-arterial coupling (VAC) represents a comprehensive expression of the mechanical efficiency and performance of the ventriculo-vascular system. It is defined as the ratio between the arterial elastance (Ea) and the end-systolic ventricular elastance (EES) and it has potential clinical applicability in different settings. The interaction between the ventricle and the aorta in the setting of acute heart failure has been insufficiently investigated.

We sought to assess the VAC in patients with acute pulmonary edema (PE) and to establish its relationship with the ejection fraction (EF) and clinical outcome.

We included 120 consecutive patients (mean age 74 ± 12 years, 61 men) admitted for acute PE, with either preserved or reduced EF. The control group consisted of 50 subjects (mean age 40 ± 13 years, 35 men) with no previous cardiac history. All patients underwent standard echocardiography on admission and we assessed the VAC non-invasively. We followed the patients for a composite endpoint of death, recurrent PE and acute coronary syndrome (ACS) for a month after hospitalisation.

The VAC was significantly impaired in the acute PE group: 1.05 ± 0.49 vs. 0.84 ± 0.16 ($p < 0.001$). In the study group, 59 patients (49%) had preserved EF (mean EF $55 \pm 8\%$) and 61 patients (51%) had reduced EF (mean EF $28 \pm 7\%$, $p < 0.001$). Subgroup analysis in the study group showed that the VAC was more impaired in patients with low EF (1.29 ± 0.56) vs. preserved

EF (0.79 ± 0.20 , $p < 0.001$). VAC had a moderate negative correlation with the EF in the study group, both for low EF patients ($r = -0.31$, $p = 0.01$) and preserved EF patients ($r = -0.30$, $p = 0.02$).

14 patients (12%) in the study group had at least one in-hospital major cardiovascular event (MACE): in the low EF subgroup, there were 7 recurrent PE (11.5%) and 1 death (1.6%), while in the preserved EF subgroup, there were 5 recurrent PE (8.5%) and 1 ACS (1.7%). There was no significant difference in VAC between patients with in-hospital MACE and MACE-free patients ($p = 0.55$ for low EF subgroup, $p = 0.59$ for preserved EF subgroup). 10 patients (8.3%) in the study group had at least one MACE in the first month after discharge: in the low EF subgroup, there were 4 recurrent PE (6.6%) and 1 death (1.6%), while in the preserved EF subgroup, there were 2 deaths (3.4%) and 3 recurrent PE (5.1%). VAC was more impaired in low EF patients with MACE at 1 month (2.27 ± 0.85) vs. low EF patients MACE-free at 1 month (1.21 ± 0.44 , $p = 0.04$). No differences in VAC were noticed for the preserved EF subgroup ($p = 0.97$).

Ventriculo-vascular interaction is decoupled in acute PE, with VAC being more impaired when the EF is reduced. Furthermore, for patients with acute PE and low EF, VAC was worse in those who suffered a MACE at 30 days. This suggests the prognostic value of VAC in acute PE and it highlights the importance of integrating this easy-to-obtain parameter in the echocardiographic evaluation of acute heart failure patients.