Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Compared with nonresponders, CRT responders showed improvement in septal CW and WW, whereas the lateral wall demonstrated a significant decrease in CW and increase in WW (**Table 1**). Method of MW evaluation allows to detect different patterns of segmental changes in CW and WW between CRT responders and nonresponders with significant correction of unbalanced MW distribution in CRT responders, which can be observed already in the first days after CRT implantation.

In this retrospective study, we demonstrated differences in baseline segmental MW indices between CRT nonresponders and responders, the latter being characterized by larger WW of the septum and larger CW of the lateral wall. Importance of contractile reserve for CRT response has been previously reported (2). On multivariate analysis baseline CW of the lateral wall was independently associated with CRT response. Although the presented OR may indicate lack of discriminative value in clinical practice, similar findings for MW indices OR were reported in other studies (3). Considering the complexity of mechanisms involved in CRT response, these data suggest that CW of the lateral wall is 1 of the contributors to the reverse remodeling and should be further investigated in larger studies.

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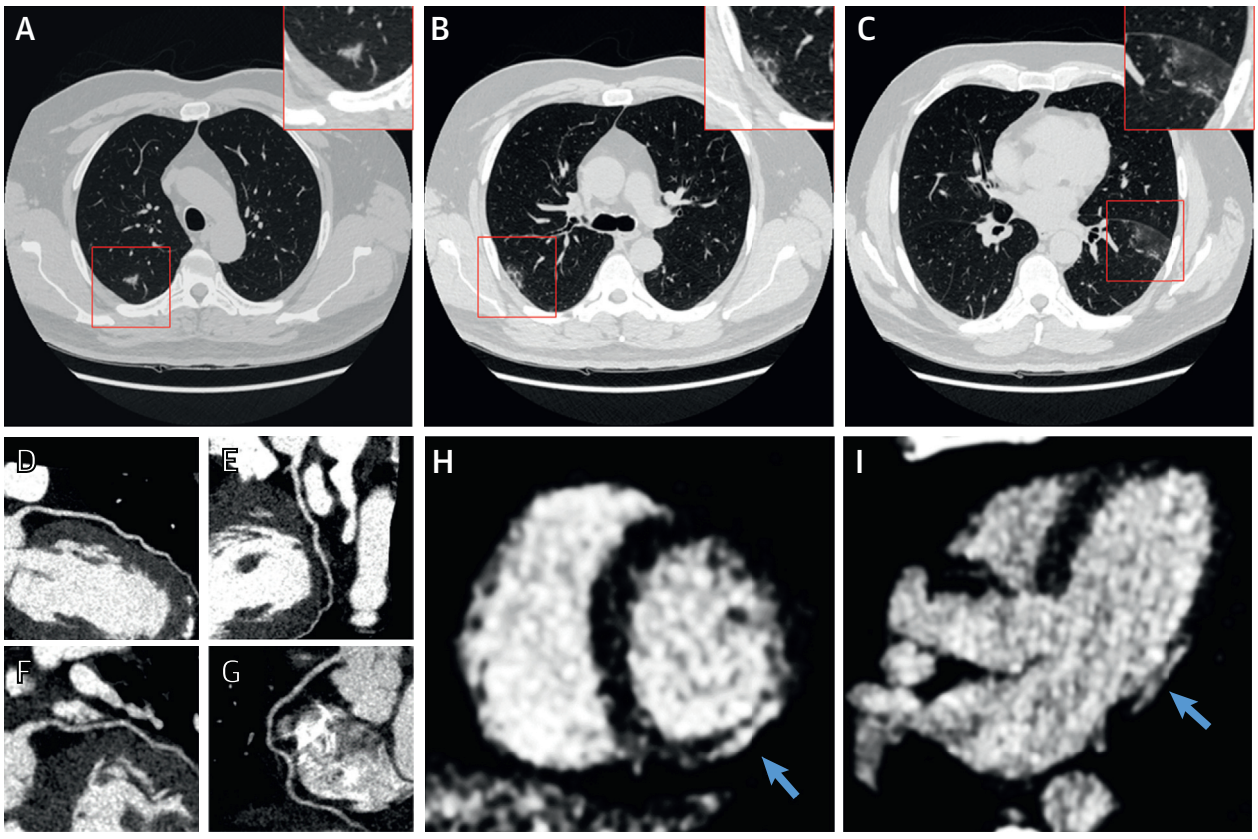
LETTERS TO THE EDITOR

"Quadruple Rule-Out" With Computed Tomography in a COVID-19 Patient With Equivocal Acute Coronary Syndrome Presentation

A 59-year-old man from an endemic area for coronavirus disease-2019 (COVID-19) in northern Italy presented to the hospital with dyspnea and chest pain. On physical examination, his temperature was 36.9°C and electrocardiogram showed ST-segment elevation in leads V_2 to V_3. The blood tests showed leukopenia (3,800 cells/μl) and troponin I increase (140 ng/l). Chest X-ray showed no pathological findings. Because of the COVID-19 emergency, a large-scale hub-and-spoke model was developed in the Lombardy region to select dedicated cardiology centers for management of acute coronary syndromes (ACS) to support other general hospitals that were converted to treat only patients with COVID-19 (1). In agreement with this model, the patient received a nasopharyngeal swab that tested positive for SARS-CoV-2 and subsequently underwent invasive coronary angiography (ICA) in a dedicated catheterization laboratory. ICA demonstrated normal coronary arteries with a diagnosis of myocardial injury with nonobstructive coronary artery disease. Although cardiac magnetic resonance (CMR) was considered for further evaluation, it was not performed because of equipment and room cleaning and disinfection issues. The day after admission, to evaluate for lung infection, pulmonary embolism, and myocardial injury, as suspected by biomarker elevation, a modified scan protocol including a nonenhanced acquisition followed by electrocardiogram-triggered contrast-enhanced computed tomography (CT) with delayed

FIGURE 1 Modified Scan Protocol to Evaluate for Lung Infection, Pulmonary Embolism, and Myocardial Injury



Pre-contrast computed tomography showing scattered patchy ground-glass opacities in the peripheral areas of both lungs with associated consolidation (**A to C**, with **red boxes** magnifying lung abnormalities). Coronary computed tomography angiography showing absence of coronary artery disease in the left anterior descending artery **(D)**, left circumflex **(E)**, marginal branch **(F)**, and right coronary artery **(G)**. Delayed postcontrast scan showing a large subepicardial area of hyperdensity in the basal-mid inferolateral wall of the left ventricle **(H and I, blue arrows)** suggesting the presence of myocarditis.

acquisition was performed. The pre-contrast CT showed scattered patchy ground-glass opacities in the peripheral area of the lungs (Figures 1A to 1C), while the contrast-enhanced scan confirmed the absence of both pulmonary embolism and coronary artery disease (Figures 1D to 1G). Finally, the delayed post-contrast scan showed a large subepicardial area of hyperdensity in the basal-mid inferolateral wall of the left ventricle (Figures 1H and 1I), suggesting the presence of myocarditis in addition to early-stage interstitial pneumonia. The patient was medically treated with a progressive clinical improvement.

The Lombardy region in northern Italy is one of the areas with the highest number of patients with COVID-19. Comparing the period between February 15 and March 20, 2020, with the same period last year, we observed a significant increase of ACS with no obstructive coronary artery disease (27% vs. 4%; $p < 0.01$). Recent reports suggest that acute cardiac injury is present in approximately 7% of patients with COVID-19 and may be related to myocarditis. Although CMR is known to be highly effective in identifying the underlying diagnosis beyond myocardial infarction with nonobstructive coronary arteries, some concerns related to equipment and room disinfection should be considered in patients with COVID-19. Recently, cardiac CT (CCT) has shown the ability to detect myocardial damage (2,3) through late iodine-enhanced scans. A lesson from this case is that in these patients with equivocal ACS presentation who are hemodynamically stable, CCT could be considered to rule out coronary artery disease as an alternative to ICA. Moreover, additional information to rule out interstitial pneumonia, pulmonary

embolism, and myocarditis could be obtained with dedicated protocol acquisition in a new concept of "quadruple rule out." However, the potential renal injury related to the higher amount of contrast agent required should be carefully evaluated on a case-by-case basis. To the best of our knowledge, this is the first report showing the use of CCT as part of a comprehensive cardiopulmonary assessment during the COVID-19 pandemic, thus, potentially minimizing exposure to healthcare providers and personal protective equipment utilization.

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Heart–COVID-19 Lung Interactions

More Common Cause for Right Ventricular Dysfunction Than We Thought

We read with great interest the paper by Argulian et al. (1), describing the prevalence of right ventricular (RV) dilation among 105 patients with coronavirus disease-2019 (COVID-19). Thirty-one patients were mechanically ventilated, and 32 patients (31%) presented with RV enlargement. The authors (1), however, do not refer on the proportion of mechanically ventilated patients with RV dilation. Mechanical ventilation, vasoactive medication use, and RV enlargement were significantly associated with increased mortality; however, all 3 are interrelated.

Heart-lung interactions may be pronounced in COVID-19 pathophysiology under mechanical ventilation. When lung compliance is relatively normal, as has been reported in COVID-19 lung (2), a substantial amount of the alveolar pressure is transmitted to the pleural pressure. Increased positive end-expiratory pressure (PEEP) levels may induce dead space ventilation and compression of the pulmonary vasculature, leading to increased pulmonary vascular resistance (PVR) and right heart dilation and dysfunction (3). Increased PVRs may be exacerbated in the setting of nonmassive pulmonary embolism (COVID-19 has been related to lung thrombotic events). This could be the case in the 5 patients with confirmed pulmonary embolism (computed tomography angiography) in the study by Argulian et al. (1). Moreover, relatively high PEEP, according to current guidelines (4), in a nonrecruitable lung with almost normal compliance (as in COVID-19) may significantly increase pleural pressure and have a detrimental impact on hemodynamics by deteriorating venous return (5). The effects are exaggerated when the patients are relatively hypovolemic in the initial phase after intubation (fever in the preceding days); restricted fluid resuscitation is also indicated in acute respiratory distress syndrome to keep the lung "dry" in an effort to improve oxygenation and avoid intubation (4).

Increased PVRs and a reduction in venous return decrease the cardiac output. The first reaction to correct hypotension, in an intensive care unit setting, is vasopressor initiation. At the tissue level, hypovolemia and vasoconstriction induce hypoperfusion and end-organ damage, which can lead to multiorgan dysfunction syndrome and an unfavorable outcome. Our rationale is that RV dilatation and dysfunction, vasopressors, and mechanical ventilation are interchangeably related in patients with COVID-19. Argulian et al. (1) nicely report that all 3 factors are associated with increased mortality. It would be informative if the authors could provide data on: 1) RV dimensions before and after initiation of mechanical ventilation; 2) PEEP levels in patients with and without RV enlargement; and 3) possible associations between RV dimensions and PEEP levels. Of note, in Figure 1, both patients are mechanically ventilated.