recommend statin treatment may results in CVD events in 6th or 7th decade of life.[3,6,7]

However, one of the most frequent causes for the lack of implementation of adequate statin treatment in primary CVD prevention is the rigid adherence to the guideline recommendations by the medical practitioners and their attitude towards statin therapy.[8] At the educational meeting on the prevention of atherosclerotic CVD entitled: "*A new paradigm in atherosclerotic CVD prevention*", held for general practitioners (GPs) in September 2019 in Split, Croatia, out of 286 surveyed GPs, 32% of respondents pointed out dyslipidemia as the most important atherosclerotic risk factor, 8% smoking, 8% hypertension, 4% obesity, while 48% of GPs considered all CVD risk factors equally significant. At the same time, 6.5% of the surveyed GPs believe that LDL-C values <50 mg/dl achieved in young age and maintained throughout life may decrease the incidence of CVD slightly, 54.5% significantly, 16.4% in full and 22.6% only if other CVD risk factors were corrected. Nevertheless, when prescribing statins in primary prevention, as much as 78% of the GPs are guided by the current patient's absolute risk rather than by the long-term risk and they are not ready to recommend statin therapy to the apparently healthy young and middle-aged people with LDL-C value around or slightly above 116 mg/dl. At the same time, 42.9% of the GPs do not prescribe statin therapy to elderly in fear of its potential side-effects, and 39.3% due to the restrictive rules of insurance companies. Finally, only 16% of the GPs knew their own LDL-C values and none of them were taking statin as a measure of primary CVD prevention!

Therefore, one of the most important measures for improving primary CVD prevention is to educate medical professionals, especially GPs, that statin treatment should start in young or early middle age, thereby focusing on preventing atherosclerotic plaques rather than on decreasing the risk of CVD events, with the target LDL-C values as low as possible, preferably <50 mg/dl.[3,6] These LDL-C levels prevent or completely regress early atherosclerotic plaques, even in persons with additional CVD risk factors.[3,6] If medical professionals accept the paradigm - *if atherosclerotic plaques are prevented, atherosclerotic consequences do not occur* -[3] only a very small number of middle-aged people will not use an inexpensive, efficacious and safe statin therapy, and the dilemma about starting statin treatment as a measure of primary CVD prevention in elderly individuals will no longer exist. As the most important result of these preventive measures, the vast majority of population will be spared of potentially life-threatening and disabling CVD events and/or percutaneous or surgical coronary interventions.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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The Right Ventricle in COVID-19 Patients

The acute coronavirus disease 2019 (COVID-19), due to Severe Respiratory Syndrome Coronavirus-2 causes an inflammatory state with cytokine release.[1] Heart function may be affected, particularly in patients presenting with comorbidities and previous cardiovascular disease.[2] In the meantime, the COVID-19 causes lung injury and acute respiratory distress syndrome (ARDS), a frequent complication in critically ill patients.[3] The ARDS management requires specific lung ventilation strategies using recruiting maneuvers, prone position, high level of positive end-expiratory pressure and low tidal volumes. However, in intensive care unit, ARDS may be associated with hemodynamic instability and right ventricular (RV) dysfunction. The RV, in comparison with the left ventricle, discloses a thin wall with high dispensibility and acts normally in a low resistance system, in spontaneous ventilation. The afterload of the RV is determined by the pulmonary vascular resistance. In ARDS, the presence mechanical ventilation reduces the venous return, due the positive intrathoracic pressure. Also, in this situation, the RV afterload increases, secondary to the increase of the transpulmonary pressure and of the pulmonary vascular resistance and the vasoconstriction related to hypoxemia and hypercapnia.[4] The onset of RV dysfunction and RV dilation (Figure 1) may also affect the left ventricular function and loading. This is explained by ventricular interdependence and paradoxical septum. In this case, a reduced cardiac output occurs, depressing the systemic perfusion and exposing the patient to RV ischemia. In addition, COVID-19 patients disclose a hyper coagulate state avec a significant incidence of pulmonary thrombotic

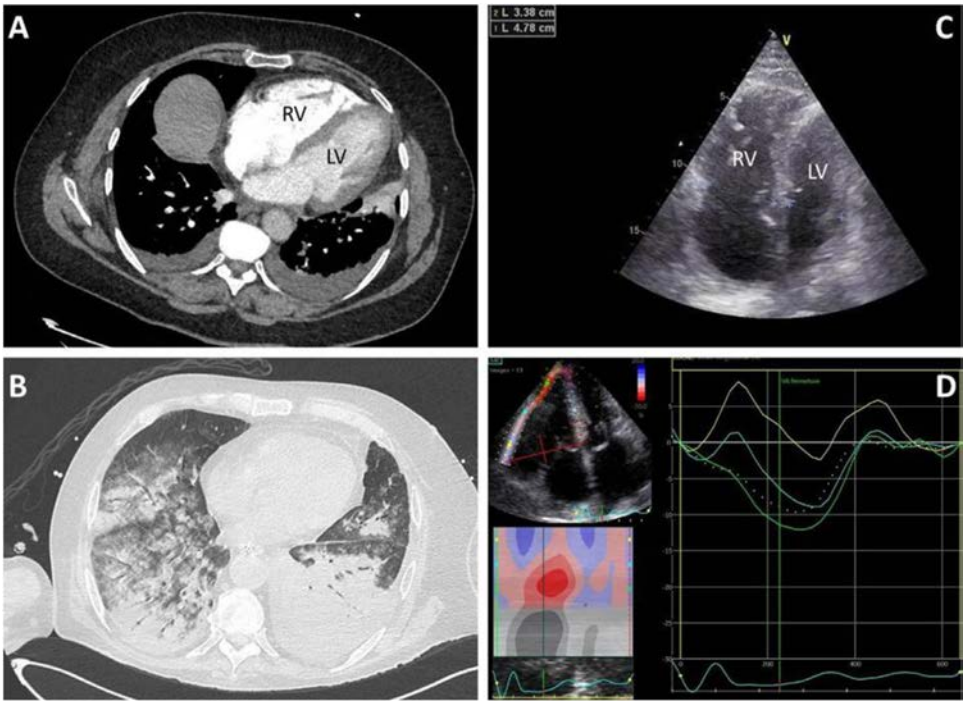

Figure 1. COVID-19 pattern and acute respiratory distress syndrome. (*A*) Right ventricular dilation on computed tomography. (*B*) Severe pulmonary involvement detected by computed tomography. (*C*) Echocardiographic right ventricular dilation (apical 4-chamber view). (*D*) Depressed longitudinal right ventricular strain.

complications, aggravated the RV afterload.[5] The onset of right ventricular dysfunction and RV dilatation (acute cor pulmonale) is associated with excess mortality in ARDS.[4] The acute cor pulmonale is classically associated with the following parameters: the driving pressure, the PaCO2 and the PaO2/FiO2 ratio.[4] Finally, the RV function, measured by the tricuspid annular plane systolic excursion, the right ventricular fractional area change and the right ventricular longitudinal strain, is a significant predictive factor of mortality in COVID-19 infected patients.[6] In this context, in addition with the left ventricular function analysis, it is of importance to check and assess the right ventricle in COVD-19 infected patients, particularly in critical situation.

Conflict of Interest

The authors have no conflicts of interest to declare.

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Reply to "Non-vitamin K antagonist oral anticoagulants for atrial fibrillation in obese patients"

We read with interest the article by Wang et al.[1] It reads "It affects 1 in 4 adults >40 years." This statement may be misunderstood easily. Does it mean prevalence or incidence or lifetime risk? Is not clear. The lifetime risk of atrial fibrillation >40 years is 25%, not the prevalence or incidence.[2]

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