degree of pulmonary edema and pulmonary fibrosis with high feasibility and very high agreement over 4 levels of severity. Our results are consistent with those of a recently published paper by Brattain et al. (4), which underlines the need for a computerized diagnostic decision support system based on sono-graphic video processing to aid nonexpert users. The added value of our findings lies in the use of an artificial neural network pattern recognition procedure to create a new decision support system tool for processing and computer-aided analysis of LUS images. Moreover, we analyzed B-line videos derived from pulmonary congestion and pulmonary fibrosis, with very similar results between the 2 etiologies.

This software could be a robust approach for developing a portable device for the individualized and automatic detection of pulmonary interstitial edema or fibrosis, which could have a high impact on public health. We are moving toward pervasive healthcare systems in human-oriented environments. Because telemedicine is being increasingly used in many different scenarios for patient monitoring and specialist consultations (5), the development of operator-independent, computer-based systems to support clinical judgment contributes to promoting radical changes toward a patient-centric healthcare environment.

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Relation Between the SYNTAX Score and Culprit Vessel Vulnerability in Non-ST-Segment Elevation Acute Coronary Syndrome

Several studies have demonstrated that multivessel disease is an independent predictor of coronary plaque progression and recurrent acute coronary events (1). Angiographic-based SYNTAX (Synergy Between PCI With TAXUS and Cardiac Surgery) score (SS) has consistently been shown to be an independent predictor of major adverse cardiac events, mortality, or both at follow-up ranging between 1 and 5 years (2,3). Several reasons may account for the increased risk of major adverse cardiac events in patients with high SS, including a higher number of obstructive plaques, more complex lesions, and a larger necrotic core. The aim of this study was to investigate the relation between SS and culprit vessel morphology as evaluated by optical coherence tomography (OCT) in patients with non-ST-segment elevation acute coronary syndrome (NSTEMI).

The study group comprised 144 patients with NSTEMI who underwent percutaneous coronary intervention (PCI) to treat de novo culprit lesions with clinically significant stenosis in the native coronary artery in whom OCT was performed before intervention. The culprit lesion was identified on the basis of coronary angiography, electrocardiography, and transthoracic echocardiography. Non-culprit plaques were defined as independent plaques and had to be situated at least 5 mm from the stent edge in treated or nontreated vessels. A total of 81 nonculprit plaques in 81 patients met these criteria. The SS was evaluated by 2 core laboratory analysts (Cardiocore, Yokohama, Japan), who looked together at the angiograms.

The patients were divided into tertiles according to the SS at baseline: a low SS group, 48 patients with an SS of <9 (range 1 to 8); an intermediate SS group, 47 patients with an SS of ≥9 to <16 (range 9 to 15); and a high SS group, 49 patients with an SS of ≥16 (range 16.0 to 46.5). Intraobserver and interobserver variability yielded acceptable concordance for the tertile of SS (kappa = 0.87 and 0.78, respectively). Angiographic diameter stenosis of the non-culprit plaques in the high SS group was significantly higher compared with that in the intermediate and low SS groups (45% vs. 35% vs. 36%; p = 0.02). The high SS group had a significantly lower minimum fibrous cap thickness (FCT) in the culprit lesion...
compared with the intermediate SS group and low SS group (60 [50 to 63] μm vs. 70 [57 to 93] μm vs. 77 [57 to 140] μm; p = 0.001). Moreover, the minimum FCT in the nonculprit lesion was significantly lower in the high SS group than in the intermediate and low SS groups (63 [102 to 210] μm; p = 0.001). The frequencies of lipid-rich plaque (90% vs. 85% vs. 68%; p = 0.02), thin cap fibroatheroma (73% vs. 40% vs. 40%; p = 0.001), and plaque rupture in the culprit lesion (63% vs. 47% vs. 31%; p = 0.007) and of multiple plaque ruptures in the culprit vessel (24% vs. 9% vs. 2%; p = 0.002) were significantly higher in the high SS group than in the intermediate and low SS groups. Figures 1A and 1B demonstrate the representative case in the high SYNTAX group (SS 23) and the low SYNTAX group (SS 7).

Our results suggest that patients with high SS may have heightened plaque vulnerability in culprit as well as nonculprit lesions. Several studies have shown that SS can predict the risk of adverse events in patients with coronary artery disease (2) as well as those with NSTEACS who undergo PCI. In accordance with the concept that plaque instability is a pan-coronary process, a high SS might reflect more vulnerable plaque morphology in both culprit and nonculprit lesions. Heightened vulnerability of nonculprit plaques in patients with high SS might thus provoke fatal or nonfatal coronary events even after successful revascularization for culprit lesions. Our results are supported by the findings of Genereux et al. (4), who showed that SS after PCI (residual SS) was at least as strong a predictor of subsequent ischemic events as SS calculated before PCI. Their findings and our results are considered to highlight the importance of complete revascularization in patients with high SS. In the present study, 83% (119 patients) had a low SS (<23) according to the original SYNTAX trial criteria (3). It is possible that culprit and nonculprit lesion vulnerability may have been even greater in intermediate and high SS patients according to the original cutoff values. Finally, a limitation of the present study is that to date, there have been no prospective studies correlating the OCT findings that we used to assess plaque vulnerability with future plaque rupture or adverse cardiac events.

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Letters to the Editor

We read with great interest the recent paper by Friedman et al. (1). The authors have endeavored to highlight the importance of early recognition of risk factors for cardiovascular and cerebrovascular disease and early management of these risk factors. However, we would like to highlight a factor worth considering.

Risk factors are no doubt important in the development of subclinical disease; however, we cannot treat every patient with risk factors, nor can we perform imaging on everyone with risk factors. This is just not practical.

However, there is growing evidence (2,3) that there is a strong correlation between subclinical cardiac dysfunction and subclinical brain infarcts, for example, as detected by Russo et al. (4) in the landmark CABL (Cardiovascular Abnormalities and Brain Lesions) trial through speckle tracking echocardiography. Therefore, imaging of the heart would be much more cost-effective in detecting subtle and pre-clinical changes. Nevertheless, it is an excellent systematic review that elucidates the importance of being pre-emptive in the management of any disease entity, hence reminding us, “get it before it metabolizes you.”

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REPLY: Asymptomatic Cardiovascular Risk Assessment: The Road Less Traveled

We thank Dr. Rai and colleagues for their interest in our paper (1). We agree with them on the importance of these risk factors in the development of subclinical disease. These risk factors need to be addressed in accordance with the American Heart Association/American College of Cardiology guidelines to reduce cardiovascular risk (2), and should be applicable to adults both with and without existing cardiovascular disease. In our review, we have gathered evidence from the published studies that the subjects without symptomatic cardiovascular, cerebrovascular, or peripheral vascular disease, but harboring the same risk factors for development of cardiovascular disease, also experience structural and functional brain imaging changes with cognitive consequences. Given that a greater number of persons in our aging population are projected to develop mild forms of vascular cognitive impairment and cardiovascular disease, it is important, through continued imaging investigations, to gain a better understanding of the mechanisms underlying their mutual development. Such research endeavors may lead to enhanced early interventions to reverse or halt their progression and improve clinical outcomes. We have not suggested that we undertake brain imaging in every patient harboring a vascular risk factor (1).

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