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EDITORIAL

Quantitative Imaging to Illuminate Cardiovascular Risk in COPD—Progress, Context, and the Path Ahead

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Chronic obstructive pulmonary disease (COPD) is a systemic disorder whose clinical impact extends well beyond the lung.[1] The coexistence of COPD and coronary heart disease (CHD) is both common and clinically consequential, yet early identification of CHD in patients with COPD remains challenging.[2,3] Dyspnea, chest discomfort, and exercise limitation can be readily attributed to pulmonary disease alone, delaying cardiovascular evaluation until late in the disease course. The clinical consequences of this diagnostic inertia are substantial: the majority of patients with severe COPD remain undiagnosed for CHD, and the combination of these conditions further worsens patient prognosis.[2,3] This diagnostic gap contributes to increased mortality and morbidity that might otherwise be preventable through timely intervention. Improving risk stratification in this population is therefore not merely an academic exercise but an urgent clinical necessity.

In this issue, Bian et al present a multicenter study that addresses this challenge through the development of a nomogram integrating clinical characteristics, radiomics features, and deep learning–derived image signatures extracted from routine chest CT scans.[4] The authors demonstrate that combining these complementary data sources yields superior discrimination for identifying COPD patients at increased risk of CHD compared with clinical variables or radiomics features alone. Importantly, model performance was rigorously evaluated across training, internal validation, and external validation cohorts drawn from two independent centers, lending confidence to its generalizability. The resulting nomogram is intuitive, visually interpretable, and conceptually compatible with clinical use—attributes essential for any tool intended to influence point-of-care decision-making.

The methodological strengths of this work merit particular attention. First, the study capitalizes on imaging that is already widely used in COPD management, yet typically underutilized for its systemic information.[4] By extracting numerous radiomics deep learning features from whole lung parenchyma on non-contrast chest CT, the authors demonstrate how routine diagnostic studies can be repurposed to yield insights into cardiovascular vulnerability. Radiomics enables high-dimensional quantification of parenchymal patterns beyond human visual assessment—subtle textural heterogeneities, morphological variations, and density distributions that may reflect underlying pathophysiologic processes.[5] Meanwhile, deep learning through ResNet50 architecture can identify complex phenotypic signatures embedded within pulmonary structure that correlate with systemic inflammation, endothelial dysfunction, and oxidative stress—pathways mechanistically linking COPD and CHD.[4]

Second, the authors appropriately employ feature selection strategies to avoid overfitting, including univariate filtering, correlation analysis to eliminate redundancy, and LASSO regression with 10-fold cross-validation. This methodological rigor is crucial in radiomics research, where the high dimensionality of extracted features can lead to models that perform well in training but fail to generalize. The final radiomics signature comprised 10 features, while the deep learning radiomics (DLR) signature included 9 features—a parsimonious approach that balances model complexity with interpretability.

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Third, the clinical model itself reflects thoughtful variable selection. Through univariate and multivariate logistic regression, the authors identify age, platelet distribution width, red blood cell distribution width, triglycerides, and GOLD grade as independent predictors—variables that are readily available in routine practice and biologically plausible given known relationships between inflammation, lipid metabolism, hematologic parameters, and cardiovascular risk. The integration of these clinical features with imaging signatures through logistic regression yields a combined model that captures both traditional cardiovascular risk factors and novel imaging biomarkers.

Finally, the external validation across a geographically and institutionally independent cohort strengthens the methodological rigor considerably. Too often, promising models validated only through internal resampling fail when confronted with real-world heterogeneity in patient populations, scanning protocols, and clinical workflows. The maintenance of discrimination in the external cohort, coupled with favorable calibration curves and decision curve analysis demonstrating clinical utility across probability thresholds, suggests that this nomogram may indeed have value beyond the development sample.

This study also fits into a wider conversation regarding disease prediction in COPD. A landmark systematic review and meta-analysis by Bellou et al, published in the BMJ, surveyed over 400 prognostic models in COPD and found that while many tools had been proposed, few had undergone adequate external validation and even fewer had established clinical impact.[6] That work underscored an important point: the goal is not merely to construct prognostic models with strong statistical performance, but to build tools that meaningfully influence patient care and outcomes. The present study advances the field by offering a validated model that incorporates imaging-based phenotyping—a dimension largely absent in most traditional COPD risk models described in the BMJ review—while also demonstrating its incremental value over clinical variables alone.

It is, however, equally important to approach these findings with appropriate caution and to acknowledge limitations that temper enthusiasm and define the agenda for future research. The study is retrospective, with the limitations inherent to such designs, including residual confounding, selection bias, and variability in clinical documentation.[7–9] While the authors used mean imputation for missing laboratory values, this approach can attenuate associations and reduce reliability, particularly for inflammatory biomarkers like C-reactive protein and procalcitonin that may be missing non-randomly in patients with milder disease or fewer acute exacerbations. The cohort is predominantly male, which limits generalizability given well-established sex differences in cardiovascular risk expression, symptom presentation, inflammatory profiles, and COPD disease trajectories. Women with COPD often demonstrate different phenotypes, including greater susceptibility to emphysema at lower smoking exposures and distinct patterns of comorbidity accumulation.

The imaging analysis, while innovative in its focus on parenchymal lung features, does not incorporate mediastinal structures, coronary artery calcium scoring, or aortic calcification—features directly relevant to CHD that are visible on the same non-contrast CT scans and well-established as cardiovascular risk markers. Coronary calcium scoring in particular has demonstrated robust prognostic value across diverse populations and could potentially enhance model performance if integrated with parenchymal radiomics. The exclusion of patients with soft plaque on coronary angiography or CT angiography is methodologically appropriate to avoid circular reasoning but may limit applicability to the full spectrum of COPD patients, some of whom already have documented but subclinical coronary disease.

Perhaps most importantly, although the model estimates risk effectively from a statistical standpoint, the study does not define how risk estimation should alter clinical decisions. At what probability threshold should a COPD patient be referred for stress testing, coronary CT angiography, or invasive angiography? Should high-risk patients identified by the nomogram receive more aggressive lipid-lowering therapy, antiplatelet agents, or intensified cardiovascular risk factor modification? Should they be enrolled in pulmonary rehabilitation programs with integrated cardiovascular monitoring? Prognostic information has value only insofar as it leads to actionable care pathways that improve outcomes.[9] Without clear linkage to clinical actions and demonstration that acting on the model's predictions improves patient outcomes—whether through prevention of cardiovascular events, reduction in COPD exacerbations, improved quality of life, or reduced mortality—the nomogram remains a sophisticated research tool rather than a clinical instrument.

The implications for future research are therefore clear and compelling. Prospective validation in an implementation study will be essential to determine whether using this model in clinical practice improves patient-centered outcomes such as time to CHD diagnosis, appropriateness of cardiovascular referral, initiation of preventive therapies, exacerbation

frequency, hospital admissions, major adverse cardiovascular events, and all-cause mortality. Such studies should also evaluate whether the model performs equitably across patient subgroups defined by sex, ethnicity, smoking status, and COPD severity, ensuring that predictive accuracy does not inadvertently exacerbate existing health disparities. Integration of cardiac and vascular imaging features—particularly coronary calcium scoring and aortic calcification—may enhance both performance and biological interpretability while adding minimal additional computational burden. Cost-effectiveness analyses will be needed to determine whether the incremental diagnostic yield justifies the computational infrastructure and clinical workflow modifications required for implementation.

Equally important are implementation science studies to understand how such a tool can be incorporated into real-world COPD care pathways.[9] Can automated analysis of chest CT scans provide risk estimates at the point of care, embedded within radiology reports or electronic health record systems? How do clinicians interpret and respond to nomogram-derived risk scores? What educational interventions and decision support tools are needed to ensure appropriate use? What are the barriers to adoption in resource-limited settings where advanced imaging is available but computational resources or informatics infrastructure may be constrained? The most meaningful test will not be whether the model predicts risk in a statistical sense, but whether identifying high-risk patients earlier enables intervention at a stage when disease trajectories can still be altered and outcomes meaningfully improved.[7–9]

Despite these necessary cautions, the contribution of Bian et al is substantial and represents an important advance in COPD research. It reflects a conceptual shift from descriptive disease staging based on spirometry alone toward predictive, individualized medicine that integrates multiple dimensions of patient phenotype. It illustrates how quantitative imaging can extend the value of routine diagnostic studies, offering insight into systemic disease processes and cardiometabolic risk rather than lung structure alone. It demonstrates the feasibility of combining handcrafted radiomics features with deep learning signatures in a clinically interpretable framework. And it reinforces the growing recognition that COPD is fundamentally a multisystem disease requiring multisystem models of care—models that account not only for airflow limitation but also for cardiovascular vulnerability, metabolic derangement, skeletal muscle dysfunction, and psychosocial burden.

The broader significance of this work lies in its potential to catalyze a shift in how we approach COPD management. Rather than waiting for cardiovascular events to declare themselves clinically, we might identify vulnerable patients earlier using tools already embedded in routine care. Rather than managing COPD and CHD as separate silos, we might integrate risk assessment and therapeutic decision-making across organ systems.[1] Rather than relying solely on traditional risk factors that are often shared between diseases, we might leverage imaging biomarkers that capture disease-specific biology and pathophysiology. This vision is not without challenges—technical, logistical, financial, and cultural—but it is increasingly within reach as computational tools mature and clinical workflows evolve.

If validated prospectively, integrated thoughtfully into clinical workflows with clear action thresholds, and implemented equitably across diverse patient populations, the nomogram proposed by Bian et al may represent a meaningful step toward earlier recognition of cardiovascular vulnerability in COPD—a step that has the potential to improve not only diagnostic precision but also, ultimately, patient outcomes. The journey from prediction to impact is long and requires continued rigor, transparency about limitations, and commitment to implementation science. But the first step—demonstrating that routine imaging contains actionable information about cardiovascular risk in COPD—has now been taken with methodological care and multicenter validation. The path ahead is clear: we must now determine whether this information, when acted upon, changes lives.

Disclosure

The author reports no conflicts of interest in this work.

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