Chronic Respiratory Disease

Chronic Respiratory Disease
2016, Vol. 13(3) 209–210

DOI: 10.1177/1479972316652624
crd.sagepub.com
SAGE

Measuring cardiovascular risk in COPD; child's play or MENSA?

KJ Rothnie and JK Quint

People with Chronic Obstructive Pulmonary Disease (COPD) have greater morbidity and mortality than the general population, and the association between cardiovascular disease (CVD) and COPD is fairly well established, with up to one third of those with COPD dying from cardiovascular causes.[1,2] Although risk factors such as smoking are clearly shared between COPD and CVD, COPD is also considered by some to be an independent risk factor for CVD.[3]

In recent years, there has been a sizable reduction in cardiovascular mortality in the general population, achieved in roughly equal parts by improved primary prevention of vascular events and by secondary prevention.[4] However, there are still discrepancies in some patient subgroups and the same magnitude of reduction in mortality has not been seen among people with COPD. There are a few reasons for this: there is evidence that COPD patients may present atypically at the time of a myocardial infarction (MI); and that after MI they are managed differently compared to non-COPD patients.[5,6] In addition, prognostic risk scores, such as the Global Registry of Acute Coronary Events (GRACE) score, which is used to predict risk of death after a MI, does not perform as well in COPD patients as people without COPD.[7]

Why is prediction of cardiovascular risk important, and are the findings from secondary prevention also reflected in primary prevention of CVD? More aggressive primary prevention of CVD is targeted to those at higher risk and in spite of higher incidence of cardiovascular events in people with COPD, relatively less attention has been paid to prediction and primary prevention of cardiovascular risk in this group. This is potentially an extremely important place to target efforts; after all, 'prevention is better than cure'. Currently, it is unclear if risk prediction scores for CVD perform as well in COPD patients as those without COPD. Given that cardiovascular risk prediction scores are driven largely by age, and the excess risk of MI in those with COPD seems to disproportionately affect younger COPD patients[3] it would not be unreasonable to assume that performance is not as good, opening the door for investigation and potential use of more novel risk factors in scoring systems.

In this article, John et al.[8] assess the performance of existing and novel cardiovascular risk markers, including skin auto fluorescence, in people with COPD and compare them to a group of smoking controls who have no known respiratory disease or symptoms. As many believe that the increased risk of CVD in those with COPD is mediated through the consequences of increased systemic inflammation and oxidative stress, measures, such as skin auto fluorescence which purport to assess these factors, are an appealing potential option. Further, as pulse wave velocity (PWV) assesses a more proximal risk factor (aortic stiffness), it too seems a worthy candidate to better describe the increased risk of CVD for populations in whom the disease process may be accelerated, such as COPD.

One of the strengths of this study is the generalizability of COPD patients included in the study; COPD patients were not excluded at the outset for having coexistent ischaemic heart disease or diabetes mellitus thus truly representing a population of COPD patients typically seen in an outpatient clinic. However, in the analysis, only the subset of COPD patients and controls without ischaemic heart disease or diabetes mellitus were compared. Controls were recruited via databases, clinics and by advertisement;

Imperial College London, London, United Kingdom of Great Britain and Northern Ireland

Corresponding author:
JK Quint, Imperial College London, Emmanuel Kaye Building, Manresa Road, London, SW3 6LR, United Kingdom of Great Britain and Northern Ireland.
Email: j.quint@imperial.ac.uk

not entirely from the same pool as cases, potentially biasing comparisons, as there was more self-reporting of CVD in the COPD patients than in the controls.

The authors found that patients with COPD had both a significantly greater skin auto fluorescence and aortic stiffness (as measured by PWV) than controls with a smoking history. Although there was a greater prevalence of self-reported ischaemic heart disease in patients with COPD compared to the control group, the 10-year future cardiovascular risk score calculators did not significantly distinguish between the groups. Not only were cardiovascular risk scores not significantly higher in patients with COPD compared to controls, there were no significant differences between patients and controls for any of the individual components of the cardiovascular risk scores. This is perhaps perplexing and there are several possible reasons for these findings.

It is possible that lack of difference in risk scores arose due to patient selection. But if we assume that the COPD patients in this study were indeed at higher risk of cardiovascular events compared to people without COPD (as is the case generally), perhaps some of the more novel CVD risk factors may be able to better describe CVD risk in those with COPD. Certainly this study lays the groundwork for future studies of novel CVD risk factors in those with COPD. Perhaps we need to think about introducing new tests such as skin auto fluorescence and PWV into routine clinical care; but is this practical? An alternative approach would be to assess aspects of COPD itself, such as frequency of exacerbations or the severity of airflow obstruction when considering risk of cardiovascular events. Or, maybe simplification is better and COPD, like diabetes mellitus, should simply be accepted and included universally as a risk factor for CVD.

Whether what is ultimately derived resembles a more simplistic or complicated approach (child's play or MENSA), this cross sectional study provides food for thought. Longitudinal studies allowing comparison of expected and observed risk of cardiovascular events are needed, as are more studies involving long term disease-free cohorts and study of more specific and novel markers including some of those measured in this study. However, if alternative strategies ultimately do prove to be useful in longitudinal studies in detecting future cardiovascular risk in people with COPD, persuading General Practitioners or Cardiologists to use them may be as difficult as finding the markers themselves.

References

1. Sin DD, Anthonisen NR, Soriano JB, et al. Mortality in COPD: role of comorbidities. *EurRespir J* 2006; 28: 1245–1257.
2. Calverley PMA, Anderson JA, Celli B, et al. Salmeterol and fluticasone propionate and survival in chronic obstructive pulmonary disease. *N Engl J Med* 2007; 356: 775–789.
3. Feary JR, Rodrigues LC, Smith CJ, et al. Prevalence of major comorbidities in subjects with COPD and incidence of myocardial infarction and stroke: a comprehensive analysis using data from primary care. *Thorax* 2010; 65: 956–962.
4. Smolina K, Wright FL, Rayner M, et al. Determinants of the decline in mortality from acute myocardial infarction in England between 2002 and 2010: linked national database study. *BMJ* 2012; 344: d8059. DOI: 10.1136/bmj.d8059.
5. Rothnie KJ, Smeeth L, Herrett E, et al. Closing the mortality gap after a myocardial infarction in people with and without chronic obstructive pulmonary disease. *Heart*. 2015; 101: 1103–1110.
6. Quint JK, Herrett E, Bhaskaran K, et al. Effect of β blockers on mortality after myocardial infarction in adults with COPD: population based cohort study of UK electronic healthcare records. *BMJ*. 2013 Nov 22; 347: f6650. DOI: 10.1136/bmj.f6650.
7. Rothnie K, Smeeth L, Herrett E, et al. GRACE scores in COPD patients with acute coronary syndromes: Performance and impact on secondary prevention. *European Respiratory Journal*. 2015; 46.
8. John, et al. Traditional and emerging indicators of cardiovascular risk in chronic obstructive pulmonary disease. *Chronic Respiratory Disease*. 2016; 13: 247–255.