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Original Article

Lung function and outcomes in emergency medical admissions

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ABSTRACT

Background: We examine the ability of pre-existing measures of Forced Expiratory Volume in 1 s (FEV1), and Diffusion Capacity for Carbon Monoxide (DLCO) to determine the subsequent 30-day mortality outcome following unselected acute medical admission.

Methods: Between 2002 and 2017, we studied all emergency medical admissions (106,586 episodes in 54,928 patients) of whom 8071 were classified as respiratory. We employed logisitic multiple variable regression models to evaluate the ability of FEV1 or DLCO to predict the 30-day hospital mortality outcome.

Results: The 30-day hospital episode mortality outcome demonstrated curvilinear relationships to the underlying FEV1 or DLCO values; adjusted for major outcome predictors, a higher FEV1 – OR 0.85 (95% CI: 0.82, 0.89) or DLCO OR 0.76 (95% CI: 0.73, 0.79) values predicted survival. The range of predicted mortalities was from 3.3% (95% CI: 2.5, 4.0) to 23.5% (95% CI: 20.8, 26.2); the FEV1 (Model1) and DLCO (Model2) outcome prediction was essentially equivalent (Chi2 = 2.9: $p = 0.08$).

Conclusion: The 30-day mortality outcome was clearly related to the pre-admission FEV1 and DLCO value. The outcome relationship was curvilinear. Either parameter appears a useful tool to explore hospital outcomes. Previously suggested cut-points are likely an artefact and not supported by these data.

1. Introduction

Pulmonary function tests (PFTS) are an important tool in the assessment and clinical surveillance of patients with respiratory conditions including Chronic Obstructive Pulmonary Disease (COPD) and interstitial lung diseases. Spirometric measurements including Forced Expiratory Volume in 1 s (FEV1) and Forced Vital Capacity (FVC) yield information relating to large and small airways, while Diffusion Capacity for Carbon Monoxide (DLCO) is a valuable measure of alveolar efficiency reflecting pathology of the pulmonary parenchyma and capillary bed [1].

Several predictors of mortality in COPD have been identified. These include FEV1, age, body mass index (BMI), dyspnoea at rest, exercise capacity, and exacerbation frequency [2]. Multidimensional indices have shown a better survival prediction than the FEV1 (%) alone [3]. Baseline individual pulmonary function parameters (FEV1, FVC, or DLCO) all predict mortality in Interstitial Pulmonary Fibrosis [4]. The Composite Physiologic Index (CPI) which adjusts for the coexistence of emphysema with fibrosis and incorporates FEV1, FVC and DLCO into the equation is also a useful prognostic tool [5].

The literature mapping PFT's and hospital outcomes is less well developed. Studies have suggested that reduced FEV1 strongly predicts increased length of stay and in-hospital mortality following cardiac surgery. Hence, one could improve risk stratification of complex patients undergoing cardiac surgery [6]. Pulmonary Function Tests particularly DLCO, prior to cardiac surgery, predicted post-operative outcomes [7]. However, in heart failure patients, pulmonary function tests including FEV1 and DLCO did not predict mortality in continuous flow Left Ventricular Assist Device (LVAD) implantation [8]. In the oncology arena DLCO appeared more sensitive to detect a deterioration in lung function during therapy with Paclitaxel and Carboplatin [9]. Post-operative cardiopulmonary complications were not related to DLCO or preoperative lung function variables in patients undergoing pneumonectomy [10]. More generally, the application of threshold values for FEV1 have been said to better predict outcomes [11], and dyspnoea scores have been proposed as a better methodology than objective FEV1 values in asthma exacerbations [12].

Many factors influence hospital mortality outcomes. If important predictors such as Acute Illness Severity Score [13,14], the Charlson Comorbidity Index [15], the Chronic Disabling Score [16] and Sepsis

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Status [17] are not determined then spurious association may be derived.

We have a large pulmonary function test (PFT) database from our lung function laboratory; many of these patients (with pre-admission pulmonary function test) have had an emergency medical admission and we were interested as to whether values of the FEV1 and DLCO would be predictive of outcomes in emergency admissions. Therefore we have examined data on acute unselected emergency medical admissions between 2002 and 2017 to explore the relationship between FEV1, DLCO and 30-day-in-hospital mortality.

2. Methods

2.1. Background

St James's Hospital, Dublin serves as a secondary care centre for emergency admissions in a catchment area with a population of 270,000 adults. All emergency medical admissions were admitted from the Emergency Department to an Acute Medical Admission Unit, the operation and outcome of which have been described elsewhere [18,19]. As a city centre hospital, St James's admits persons resident elsewhere but working in the capital in addition to visitors to Dublin who became acutely ill. The number of emergency medical admissions resident in the catchment area was 74.5%; this compares with a figure of 59% for Emergency Depatrment (ED) presentations where the social influences on emergency department visitations on two London hospitals have been examined [20].

2.2. Data collection

An anonymous patient database was employed, collating core information of clinical episodes from the Patient Administration System (PAS), the national hospital in-patient enquiry (HIPE) scheme, the patient electronic record, the emergency room and laboratory systems. HIPE is a national database of coded discharge summaries from acute public hospitals in Ireland [21,22]. International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) has been used for both diagnosis and procedure coding from 1990 to 2005 with ICD-10-CM used since then. Data included parameters such as the unique hospital number, admitting consultant, date of birth, gender, area of residence, principal and up to nine additional secondary diagnoses, principal and up to nine additional secondary procedures, and admission and discharge dates. Additional information cross-linked and automatically uploaded to the database includes physiological, haematological and biochemical parameters.

This study had no interventional component, used anonymised routinely collected data, complied with data protection legislation and was undertaken with the approval of hospital authorities; hence did not require approval from our institutional ethics committee.

2.3. Measurement of FEV1 and DLCO

Spirometric parameters (FVC and FEV1) and the carbon monoxide diffusing capacity of the lung (DLCO) were measured using the VMax Encore system (Vyaire Medical, 26,125 North Riverwoods Blvd, Mettawa, IL 60045, USA). All assessments were carried out by Respiratory Physiology staff to European Respiratory Society (ERS) standards [23]. Predicted values were calculated using European Community of Coal and Steel (ECCS) reference values [24,25].

2.4. Multi-morbidity instrument

Hospital HIPE codes [21,22] were interrogated to construct a measure of multi-morbidity. To devise the score, we searched ICD9 hospital episode discharge codes (back-mapping ICD10 codes to ICD9 as appropriate) based on the definition proposed by the US Department of Health and Human Services for chronic physical or mental health disorders, that limit people 'in activities that they generally would be expected to be able to perform'. These ICD codes were similar to those proposed by the Canadian group for multi-morbidity [26] and the work of Quan [27,28]; they were grouped by system into the following ten groups: (i) cardiovascular, (ii) respiratory, (iii) neurological, (iv) gastrointestinal, (v) diabetes, (vi) renal, (vii) neoplastic disease, (viii) others (including rheumatological disabilities), (ix) ventilatory assistance required and (x) transfusion requirement. We have previously detailed these ICD9 codes as a supplement Table [29]. In addition, we searched other hospital databases for evidence of diabetes (Diamond database), FEV1 $<$ 2 l (data pulmonary function laboratory), troponin status (high sensitivity troponin $>$ 25 ng/l), low albumin ($<$ 35 G/dl) or haemoglobin levels ($<$ 10 G/dl) and chronic renal insufficiency - Modification of Diet in Renal Disease study (MDRD) equation $<$ 60 ml/min*1.73 m^2 [30,31]. The 'morbidity score' for each individual's clinical episode during the study was weighted by its relative importance against the 30-day mortality outcome in the multiple variable regression analysis.

2.5. Statistical methods

Descriptive statistics were calculated for background demographic data, including means/standard deviations (SD), medians/inter-quartile ranges (IQR), or percentages. Comparisons between categorical variables and mortality were made using chi-square tests.

We employed a logistic model with robust estimate to allow for repeated admissions; the correlation matrix thereby reflected the average dependence among the specified correlated observations [13]. Logistic regression analysis identified potential mortality predictors and then tested those that proved to be significant univariate predictors ($p < 0.01$ by Wald test). For the 30-day hospital mortality outcome, we entered predictor variables in a multiple variable logistic model that included age, Acute Illness Severity Score [13,14], the Charlson Comorbidity Index [15], the Chronic Disabling Score [16] and Sepsis Status [17] and Morbidity Score.

We used the margins command in Stata 15 to estimate and interpret adjusted predictions for sub-groups, while controlling for other variables such as time, using computations of average marginal effects. Margins are statistics calculated from predictions of a previously fitted model at fixed values of some covariates and averaging or otherwise over the remaining covariates. In the multiple variable model (logistic), we adjusted univariate estimates of effect, using the previously described outcome predictor variables. The model parameters were stored; post-estimation intra-model and cross-model hypotheses could thereby be tested.

Adjusted odds ratios (OR) and 95% confidence intervals (CI) were calculated for those predictors that significantly entered the model ($p < 0.10$). Statistical significance at $P < 0.05$ was assumed throughout. Stata v.15 (Stata Corporation, College Station, Texas) statistical software was used for analysis.

3. Results

3.1. Patient demographics

During the 16-year study period (2002–2017), there were a total of 106,586 episodes of medical emergencies in 54,928 unique patients admitted through the Emergency Department. These episodes represented all emergency medical admissions, including patients admitted directly into the Intensive Care Unit or High Dependency Unit, respectively. The proportion of males was 48.5%. The median length of stay (LOS) was 4.3 days (IQR: 1.7, 8.9). The median age was 58.9 yr. (IQR: 38.2, 76.3) with the upper 10% boundary at 85.0 yr. There were 8071 individuals classified as respiratory with a respective total of 25,274 episodes; respiratory patients/admissions therefore although

only 14.7% of admitted patients accounted for 23.7% of all emergency medical episodes over that time period.

The demographic characteristics (Table 1) is outlined with a division of non-respiratory vs. respiratory and tabulated (to allow group comparisons) by Acute Illness Severity [13,14], Charlson Co-Morbidity Index [15], Morbidity Score and Sepsis status [17]. Respiratory admissions were somewhat older 68.0 yr. (IQR: 55.7, 77.0) vs. 60.3 yr. (39.3, 78.0) and had a longer hospital stay 6.0 day (2.9, 10.7) vs. 4.8 day (1.9, 9.2). The episode mortality appeared lower due to more frequent admissions (2.9% vs. 5.0%); however if one considers only per patient analysis (last episode if > 1) then the mortality appeared quite similar for non-respiratory and respiratory - 11.0 vs 10.9% (respectively). The categories of Acute Illness Severity, Charlson co-Morbidity and overall Morbidity scores demonstrated higher frequency of severity grades or scores for respiratory compared with non-respiratory admissions (Table 1).

Overall, the median weighted morbidity score was 6.5 (IQR 3.8, 8.8 points) with 90% and 99% values at 11.1 and 16.4 points respectively. The area under the Receiver Operating Characteristics (AUROC) curve for this morbidity score was 0.83 (95% CI: 0.83, 0.84) – it predicted a broad range of 30-day hospital mortality outcomes from at 6 points 2.3% (95% CI: 2.2%, 2.5%), to 10 points 7.9% (95% CI: 7.7%, 8.2%), to 14 points 23.1% (95% CI: 22.1%, 24.1%) and finally at 18 points 49.9% (95% CI: 47.5%, 52.2%) (Table 1).

3.2. Relationship of FEV1 to 30-day mortality outcomes (Fig. 1)

The relationship between 30-day hospital episode mortality outcome and underlying FEV1 value was curvilinear (Fig. 1); adjusted for other outcome predictors of Acute Illness Severity, Charlson Co-Morbidity Score, Chronic Disabling and Sepsis Status, a higher FEV1 predicted survival – OR 0.85 (95% CI: 0.82, 0.89). 7987 patients were identified with FEV1 measurements on record. At a cut-point of FEV1 < 0.77 l, the predicted per patient 30-day hospital mortality was 17.9 (95% CI: 15.9, 19.9), an FEV1 < 1.521 predicted mortality of 11.6 (95% CI: 10.7, 12.4) whilst those with the best function FEV1 > 2.81 l predicted mortality of 6.2 (95% CI: 5.0, 7.4).

Table I
Characteristics of Emergency Medical Admissions by Respiratory Status.

	Others (N = 72957)	Respiratory (N = 22904)	p-value
Age (yr.)			
Median (Q1, Q3)	60.3 (39.3, 78.0)	68.0 (55.7, 77.0)	< 0.001
Length stay (day)			
Median (Q1, Q3)	4.8 (1.9, 9.2)	6.0 (2.9, 10.7)	< 0.001
Gender			
Male	36070 (49.4%)	10781 (47.1%)	< 0.001
Female	36887 (50.6%)	12123 (52.9%)	
30-day hospital mortality			
Alive	69302 (95.0%)	22239 (97.1%)	< 0.001
Dead	3655 (5.0%)	665 (2.9%)	
Illness severity			
1	2768 (4.2%)	161 (0.8%)	< 0.001
2	5924 (9.0%)	586 (2.8%)	
3	9114 (13.9%)	1751 (8.2%)	
4	10980 (16.7%)	3560 (16.7%)	
5	11904 (18.1%)	5062 (23.8%)	
6	24956 (38.0%)	10178 (47.8%)	
Charlson index			
0	37871 (52.0%)	6131 (26.8%)	< 0.001
1	17116 (23.5%)	8767 (38.3%)	
2	17840 (24.5%)	7965 (34.8%)	
Morbidity score			
< 6	16472 (36.4%)	6125 (32.5%)	< 0.001
≥6 < 8	13527 (29.9%)	4700 (24.9%)	
≥8 < 12	12758 (28.2%)	6323 (33.5%)	
≥12 < 20	2537 (5.6%)	1719 (9.1%)	
Sepsis group			
1	55740 (76.4%)	17608 (76.9%)	< 0.001
2	14608 (20.0%)	4646 (20.3%)	
3	2609 (3.6%)	650 (2.8%)	

LOS: length of stay, IQR: Inter-Quartile.

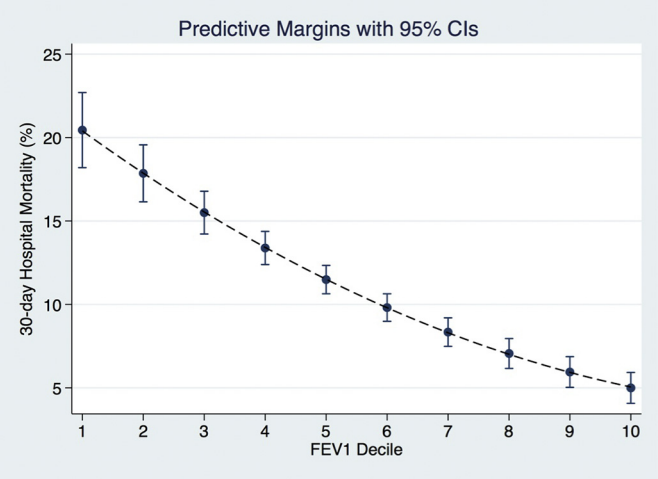

Fig. 1. The relationship between 30-day hospital episode mortality outcome and underlying FEV1 value was curvilinear. The mortality outcome, plotted against deciles of FEV1 (cut-points 0.77, 0.95, 1.14, 1.33, 1.52, 1.75, 2.0, 2.32 and 2.81 l), was adjusted in the model for Acute Illness Severity, Charlson Co-Morbidity Score, Chronic Disabling and Sepsis Status.

3.3. Relationship of DLCO to 30-day mortality outcomes (Fig. 2)

The relationship between 30-day hospital episode mortality outcome and underlying DLCO value was curvilinear (Fig. 2); adjusted for other outcome predictors of Acute Illness Severity, Charlson Co-Morbidity Score, Chronic Disabling and Sepsis Status, a higher DLCO predicted survival – OR 0.76 (95% CI: 0.73, 0.79). 6523 patients were identified with DLCO measurements on record. At a cut-point of DLCO < 7.4, the predicted per patient 30-day hospital mortality was 23.5 (95% CI: 20.8, 26.2), and DLCO < 13.5 predicted mortality of 10.6 (95% CI: 9.7, 11.6) whilst those with the best function DLCO > 21.9 predicted mortality of 3.3 (95% CI: 2.5, 4.0). The mortality outcome between the FEV1 (Model1) and DLCO (Model2) to predict 30-day

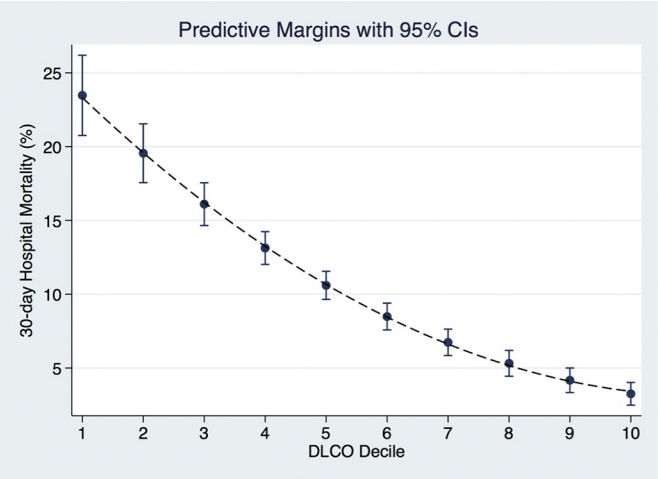

Fig. 2. The relationship between 30-day hospital episode mortality outcome and underlying DLCO value was curvilinear. The mortality outcome, plotted against deciles of DLCO (cut-points 7.4, 9.2, 10.7, 12.1, 13.5, 14.9, 16.6, 18.9 and 21.9 ml/min/mmHg) was adjusted in the model for Acute Illness Severity, Charlson Co-Morbidity Score, Chronic Disabling and Sepsis Status.

mortality outcome was essentially equivalent (Chi2 = 2.9: $p = 0.08$).

4. Discussion

These data show that as lung function declines, 30 day mortality outcomes progressively increase. Both function parameters independently predicted worse outcomes, after adjustment for Acute Illness Severity and Co-Morbidities/Case Complexity; moreover, the prediction of mortality outcomes, related to the decile of the parameter distribution, was equivalent. The graded nature of the response for each variable and the smooth transition across deciles of function would suggest the possibility that threshold effects utilized in disease severity categorisation systems are potentially artefactual.

We utilized a large database of 106586 admissions in 54928 patients. The analysis was opportunistic and sought, in patients who had respiratory function tests undertaken at some point prior to an emergency medical hospital admission, to determine respiratory function outcome relationships. We identified 7987 and 6523 patients with existing pulmonary function tests (FEV1 and DLCO values respectively); it is likely the size of this database together with the ability to adjust for known outcome predictors may explain why there was such a clear cut relationship. Thus, suggestions that DLCO could predict operative outcomes of cardiac surgery [7] or detect lung damage during oncological therapy [9] appear well founded.

Neither FEV1 or DLCO showed any particular age effect. Similar curvilinear relationships between pulmonary function and mortality were shown in older and younger patient groups (data not shown). This is consistent with previous observations that elderly patients referred to a hospital-based pulmonary function test lab can be expected to achieve spirometry and DLCO values comparable to younger adult patients [32].

Our data is consistent with observations that FEV1 is an important predictor of mortality in COPD [33]. Survival following an acute exacerbation is inversely proportional to baseline FEV1 [34]. It is possible that airflow obstruction and ventilatory impairment are associated with higher risk of colonization by aggressive bacteria causing exacerbation [33]. The prevalence of Pseudomonas aeruginosa and Haemophilus influenzae has been shown to correlate with the greatest degree of functional impairment, as measured by FEV1 [34].

People with COPD have an increased prevalence of lung cancer, myopathy, osteoporosis, anaemia, coronary artery disease, anxiety, and depression [35]. Epidemiological studies have shown that smokers with reduced FEV1 have considerably higher all cause morbidity and mortality than smokers with normal lung function [36]. Reduced pulmonary function has also been shown to predict morbidity and premature death from non-respiratory causes. A reduced FEV1, for example, is a strong marker for coronary artery disease [36,37], and stroke [38]. Among smokers of similar smoking exposure, reduced FEV1 is associated with three to four-fold increase in cardiovascular mortality [36]. Additionally, FEV1 values in the bottom quartile have been associated with a doubling of mortality risk in patients with dementia [39].

The relationship between pulmonary function and levels of various systemic inflammatory markers demonstrate an inverse linear relationship between FEV1 and C-Reactive Protein (CRP) [35]. A baseline CRP value of > 3 mg/l in this population has been found to predict COPD related hospitalizations and death [35]. Patients with stable COPD also maintain increased levels of cytokines like Interleukin 6 (IL-6) and Tumour Necrosis Factor Alpha (TNFα) [40]. Whether the correlation with multisystem morbidity represents separate manifestations of a systemic inflammatory process is a matter of debate.

Our hospital catchment area contains many high deprivation small areas [41]. Low socio-economic status (SES) may be determined from different periodic National Census measures including income, education, occupation or housing. Different pulmonary diseases including chronic obstructive pulmonary disease (COPD), asthma, and pulmonary hypertension have been linked to low SES [42]. Low SES influences both the admission rate incidence and 30-day in-hospital mortality [43] of respiratory admissions. The level of air pollutants on the day of admission also influenced the 30-day mortality outcome in patients from the same catchement area [44]; a finding in keeping with studies linking atmospheric pollution with respiratory morbidity [45]. Admissions from this deprived subgroup are over represented with respiratory disease admissions and are more likely to require ventilation during the admitted episode. SES seems to be an important albeit under recognized contributor to pulmonary disease. There was a significant negative correlation between lung function (primarily FEV1 and FVC) and SES that persisted even after adjusting for smoking status, occupational exposures, and race [46].

The fact that this study was conducted in a single inner city centre with high rates of social deprivation may hence represent a study limitation. Patients in our catchement area have a substantially higher prevalence of alcohol and drug abuse compared to other centres. While our analysis corrects for multimorbidity and illness severity at presentation, our demographic may present a host of intangible factors affecting the inpatient clinical trajectory of many patients including poor physiological reserve at younger ages, delirium and withdrawal syndromes. This may limit the generalizability of our results.

5. Conclusion

We have shown that after correcting for other important predictor variables (particularly illness acuity and complexity/comorbidity) that FEV1 and DLCO predict 30-day mortality outcomes. Both FEV1 and DLCO predict mortality equivalently. This sustained curvilinear relationship with all-cause mortality across the pulmonary function decile range is a novel finding. These results support the well documented links between curtailment of pulmonary function and multi system disease as well mortality following an acute exacerbation of COPD specifically. Our findings also support the idea that pulmonary function may be reflective of a systemic maladaptive biological environment linking pulmonary disease with systemic multimorbidity and death.

Conflict of interest statement

The authors declare that they have no conflict of interest regarding this publication.

Declarations of interest

None.

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