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Which observations from the complete blood cell count predict mortality for hospitalized patients?

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Abstract

Background—Information on the prognostic utility of the admission complete blood count (CBC) and differential count is lacking.

Objective—To identify independent predictors of mortality from the varied number and morphology of cells in the complete blood count defined as a hemogram, automated five cell differential count and manual differential count.

Design—Retrospective cohort study and chart review.

Setting—Wishard Memorial Hospital, a large urban primary care hospital.

Patients—46,522 adult inpatients admitted over ten years to Wishard Memorial Hospital from January 1993 through December of 2002.

Intervention—None

Measurements—30-day mortality measured from day of admission as determined by electronic medical records and Indiana State Death records.

Results—Controlling for age and gender, the multivariable regression model identified three strong independent predictors of 30-day mortality: Presence of nucleated RBCs, burr cells, or absolute lymphocytosis was associated with a three-fold increased risk of mortality at 30 days. Nucleated RBCs were associated with a 25.5% 30-day mortality rate across a range of diagnoses, excluding sickle cell disease and obstetrical patients in which NRBCs were not associated with increased mortality. Burr cells were associated with a 27.3% mortality rate and found most commonly in patients with renal failure or liver failure. Absolute lymphocytosis predicted a poor outcome in trauma and CNS injury patients.

Conclusions—In patients admitted to the hospital, presence of nucleated RBCs, burr cells, or absolute lymphocytosis at admission is each associated with a three-fold increase in risk of 30-day mortality.

Keywords

Diagnostic Decision Making; Laboratory Testing; Electronic Medical Record

Introduction

The complete blood count (CBC) bundles the automated hemogram, a five-cell automated differential count and a reflex manual differential count (when required by protocol) and is

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one of the most frequently ordered admission laboratory tests. In practice, it is a routine ingredient of all hospital admission orders – physicians order a hemogram either alone or as part of a complete blood count for 98% of our medical/surgical admissions and the same is true at most institutions.[1] We know that the white blood cell count and hematocrit from the automated hemogram do predict disease severity and mortality risk.[2345] For example, elevated WBC counts predict a worse prognosis in patients with cancer or coronary artery disease,[67] and anemia predicts increased mortality risk in patients with heart failure.[89] Further, these two tests provide direct management guidance in common circumstances, e.g., bleeding and infection.

The CBC describes the number and morphology of over 40 different cell types, from acanthocytosis to vacuolated white blood cells. Disagreement exists regarding the clinical significance of many of these observations[10111213]. And only a few components of the manual differential, e.g., nucleated red blood cells (NRBCs) and lymphocytosis, have been quantitatively evaluated to determine their prognostic significance[14151617]. But these two observations have not been examined to determine their independent contributions to mortality predictions when taken in conjunction with their accompanying CBC observations. Which of the numerous cell types and numbers in the commonly ordered CBC, indicate that a patient is at high risk of mortality? Here we report an inpatient study that uses univariate and multivariate analyses of admission CBCs to predict 30-day mortality to answer that question.

Methods

Subjects and protocol

The Institutional Review Board of Indiana University, Purdue University, Indianapolis approved this study. We included adult (≥ 18 years old) patients admitted to Wishard Hospital between January 1, 1993 and December 31, 2002 with two exceptions: prisoners (for IRB reasons) and obstetrical patients, because their 30-day mortality is very close to zero (0.07% in our institution). Wishard Hospital is a large urban hospital serving a diverse but predominantly inner-city population from Indianapolis. If a patient was admitted more than once during the ten years of observation, we included only the first admission in the analysis in order to assure statistical independence of the observations. We extracted data from the Regenstrief Medical Record System (RMRS), a comprehensive medical records system carrying demographic data, vital signs, diagnoses, results of clinical tests, and pharmacy information from all inpatient, emergency department, and outpatient encounter sites.[18]

We obtained the admission and discharge ICD9 codes and DRG codes to assess the disease patterns associated with individual CBC abnormalities. We obtained these codes from routine hospital case abstractions performed by Wishard Hospital's medical records department using (NCoder+, Quadramed). We identified obstetrical patients via the DRGs 370–384 to exclude them. We calculated a Charlson comorbidity Index[19] for each patient as a marker of coexisting conditions using the ICD9 and CPT codes per the Charlson algorithm.

Outcomes

The primary outcome was 30-day mortality counted from the date of admission. We used information from the hospital record (inpatient deaths) and the Indiana State death tapes to determine the death dates on all patients. Patients were matched to the Indiana death tapes by an algorithm using name, social security number, date of birth, and gender.[20]

Hemogram and differential count test methods

The hemogram, differential counts and blood smear exams results that we included in this study all came from Wishard Hospital's laboratory. During this study, the hospital used only two cell counters: the Coulter STK-S and the Gen-S automated blood analyzer (Beckman Coulter, Brea, California) to produce hemogram and automated blood differential counts. Both instruments provided an automated differential count for five cell types: neutrophils, lymphocytes, monocytes, basophils and eosinophils. The latter machine also produced platelet counts and reticulocyte counts, but during the study period these counts were not routinely reported to physicians unless ordered specifically, so we have not included them in the analyses. The laboratory reflexively performed one hundred cell manual differential counts and blood smear exams when abnormalities were observed in the automated measures according to the College of American Pathologists (CAP) criteria. Both of the automated blood analyzers used the same automated CAP criteria for deciding when to add a manual differential count and blood smear analysis, and these criteria were constant during the course of the study. This protocol predicts manual differential abnormalities with high sensitivity, missing less than 1% of important findings in a manual differential.[21] When the CAP criteria did not require a manual differential count and blood smear exam, we assumed that those counts that are unique to the manual count, e.g., blast cell count, were zero and that all blood smear morphology abnormalities were absent.

Laboratories may report white blood cells as absolute counts (e.g., number of cells/mm 3) and/or percents. We converted all counts reported as percents to absolute numbers (e.g., WBC count × 1,000 × cell type % / 100). For absolute counts that have both a high and a low range, such as white blood cell (WBC) count, we constructed two binary variables. WBC-low takes on a value of one when the value of the WBC was below the lower limit of normal, and zero otherwise. WBC-high takes on a value of one if the WBC is above the upper normal limits, and zero otherwise. In the case of numeric variables such as NRBCs or blasts for which the presence of any cells on the manual differential count is abnormal, we constructed binary variables with zero meaning the cell type was absent and one meaning the count was one or above.

The presence of many observations in the manual differential count and smear assessment (e.g., burr cells) is reported in qualitative terms, such as "occasional," "few," "increased," "present," and their absence as "none seen," "unremarkable," or "no mention." We dichotomized all such results into present or absent for analysis purposes.

Statistical analysis

For all original variables, we plotted cell counts against 30-day mortality to graphically display the univariate association with mortality risk. To screen the effects of these 45 binary CBC variables univariately, we used each of them as the sole independent variable in a logistic regression model with 30-day mortality as the dependent variable.

The simultaneous effects of the 45 CBC measures on mortality were investigated using multiple logistic regression models, always controlling for age (in years, as a continuous variable) and gender (dichotomous) of the patients. Two approaches were taken to handle the large number of predictors in the model. First, we formed subgroups of predictors based on clinical judgment (e.g., the subgroup of "bands," Dohle bodies, and toxic granules associated with infections) and ran logistic regression for each subgroup to choose the significant predictors from the various subgroups to fit an overall prediction model of 30-day mortality. The results were verified using a second approach that did not depend on subjective judgment. Both backwards and forwards stepwise variable selection procedures were used to choose the subset of significant predictors ($p < 0.005$) of 30-day mortality in

logistic regression, again controlling for age and gender. To be sure that the predictive power of the models was not decreased by the categorization of the continuous variables, we also ran models that included the continuous variables as potential predictors. We used the c-statistic as a measure of the goodness-of-fit of the models. We included the Charlson Index and the ten most common admission diagnoses in our model to control for presence of comorbidities and prime reason for admission, respectively.

We performed the analysis using SAS software 8.02 (SAS Institute, Inc., Cary, NC).

Chart Review

For each of the independent predictors of 30-day mortality that were both statistically significant and had very high relative risk (>2.5), one author (AK) took a random sample of 100–200 patients with positive values for this predictor and reviewed the dictated discharge summary in order to asses the clinical correlates of these findings.

Results

During the 10 years from January 1993 through December 2002, physicians admitted 46,522 unique eligible patients. The patients averaged two admissions during the study period, for a total of 94,582 admissions. The overall 30-day mortality for these admissions was 3.4%. Automated hemograms (white blood cell count, hemoglobin, red cell count and red blood cell indices) were perfomed on 45,709 (98%) of these patients within one day of their admission. Seventy-seven percent (35,692) had a complete blood count that included an automated differential count and a reflex manual count and smear when required for by the CAP protocol, as well as an automated hemogram. The patients with an admission CBC with differential count had a 30-day mortality rate of 4%, slightly higher than patients who had only a hemogram. The Charlson score for the CBC with differential count patients was 0.83, indicating a lower level compared to a national average closer to one.[22] Table 1 shows the demographics of this study population.

Predictors of 30-day Mortality

We examined the univariate effect of age, gender, and the 45 CBC variables (Table 2) on 30-day mortality. Most of these variables showed a significant (P <.0001) effect on mortality. Only a few abnormalities, e.g., WBC low (< 5000/μL), basophilia (> 200/μL), eosinophilia (>450/μL), showed absolutely no relation to 30-day mortality. Increasing age and male sex were associated with increased mortality. Of the 45 CBC variables, 29 were strong (P < .0001) univariate predictors of mortality and had odds ratios greater than 2.5. Eight variables had univariate odds ratios (O.R.) greater than 4: toxic granules, Dohle bodies, smudge cells, Promyelocyte, Myelocytes, Metamyelocytes, NRBCs, and burr cells. All but two of these variables are a white blood cell observation.

All of the statistical approaches resulted in essentially the same model for predicting mortality. Table 3 shows that age, gender and 13 of the CBC variables were retained in the final model of dichotomous variables using backward and forward selection. Presence of lymphocytosis, burr cells, and NRBCs were the greatest independent predictors of mortality with odds ratios greater than 2.5. Only one variable, presence of sickle cells, predicted a *reduced* mortality rate (with an odds ratio well below 1).

The c-statistic (the ratio of the area under the ROC curve to the whole area, which reflects the overall predictive power of the final model), was about 0.80 by any approach, which compares favorably with previous prediction models[45]. Allowing continuous measures of CBC in the model did not increase the predictive power. Inclusion of the Charlson Index and the top ten admission diagnoses did not significantly change the prediction model, although

two admission diagnoses, chest pain and acute but ill-defined cerebrovascular disease, emerged as independent predictors of 30 day mortality, with odds ratios of 0.314 and 2.033 respectively at a P value < 0.0001.

Chart Review

Of 200 cases with NRBCs, the leading probable causes for this finding was severe hypoxia (average A-a gradient = 326 mmHg), acute anemia (average hgb = 6.1 gm/dL) or sickle cell anemia. Other diseases associated with NRBCs were infection/sepsis, HIV, solid tumors (breast/lung/colon/prostate), and leukemia or multiple myeloma. Presence of even a single NRBC at admission correlated with a 25.5% mortality rate. Of note, patients with sickle cell disease had NRBCs on 30–40% of hospital admissions, and had moderate anemia (hgb = 8.7 gm/dL), but had no excess mortality risk. Indeed, the 49 patients with sickle cells and NRBCs at admission had a 30-day mortality of 0%.

For most of the NRBC cases reviewed the patients exhibited overt signs of severe disease, e.g., shock, respiratory failure, or severe trauma, in addition to presence of NRBCs. However, in two of these cases the NRBCs were the only strong signals of disease severity. Both had NRBCs present on the day of discharge and were readmitted within three days *in extremis* and died. One was readmitted in fulminant septic shock, likely from a bacterial peritonitis or urinary tract infection, and the other was readmitted in shock, likely from decompensated heart failure.

Univariately, burr cells at admission correlated with a 27.3% mortality rate. A review of 100 random cases with burr cells revealed a pattern of associated diseases, i.e., acute renal failure, liver failure, and congestive heart failure different from that of the NRBC. There was little overlap in the presence of burr cells and NRBCs, but the 12% who had burr cells *and* NRBCs had a high mortality rate (57%).

Absolute lymphocytosis was associated with an 8.6% mortality rate. While the univariate risk is not as high as for NRBCs or burr cell admissions, lymphocytosis was much more common (8.5%) and within the logistic model the presence of lymphocytosis explained more of the Chi square statistic than any other variable except age. Indeed, lymphocytosis was a stronger predictor of 30-day mortality than high WBCs or anemia. Chart review of 200 cases with lymphocytosis showed a preponderance of cases with large physiologic stressors, e.g., traumatic tissue injury (surgery), or cerebrovascular injury. In one subset, half of patients (50.9% of 53 patients) who underwent craniotomy for trauma and had absolute lymphocytosis at admission died, compared with 20.8% of 101 patients admitted for the same diagnosis without absolute lymphocytosis.

Discussion

Some investigators have incorporated selected CBC measures, e.g., white blood cell count and hemoglobin/hematocrit into multivariable models that predict mortality or rehospitalizations.[781023] However, CBC reports can include a spectrum of more than 40 distinct counts and morphologic findings. Our study is the first to take into account all of the different variables in the complete blood count and differential to determine elements that independently predict high risk of mortality.

In addition to age and gender, our multivariable analysis of the 45 CBC variables found 13 independent predictors of mortality. Five were observations about white blood cells: absolute leukocytosis, high band form cell count, presence of metamyelocytes, presence of toxic granules, and absolute lymphocytosis. Eight were observations about red blood cells: high hematocrit and low hematocrit, high MCV and presence of macrocytes, high red cell

distribution width, presence of NRBCs, presence of burr cells and the presence of sickle cells. Since controlling for severity of illness by Charlson comorbidity scores did not significantly change the model—the CBC abnormalities among the predictors of mortality did not simply reflect how sick the patients were. Including the ten most common admission diagnoses did not significantly attenuate our reported odds ratios suggesting that the CBC predictors did not merely reflect the primary reason for admission. Interestingly, however, admission for chest pain did correlate with a greatly reduced risk of thirty day mortality, which may be reflective of physicians' low threshold for admitting patients with this complaint. Admission for acute but ill-defined cerebrovascular disease independently predicted a two fold increased risk of 30 day mortality.

What is the message to physicians from this analysis? Physicians order CBCs commonly, and may rely on quick heuristics to sift through the myriad findings in CBC reports. Our analysis focuses physicians' attention on high-impact findings in the CBC. We assume that physicians already regard low hematocrit, high hematocrit (a sign of fluid loss and/or chronic hypoxia), high WBCs, high band cells and the presence of metamyeloctes (left shift) as important prognostic indicators. These abnormal findings are routinely mentioned at morning report and in physician's notes.

Physicians, however, may not appreciate the importance of the other CBC findings that were predictive of mortality on our analysis. Macrocytosis and high RDW (indicating an abnormally wide distribution of red cell sizes) have not previously been reported as predictors of mortality. And though other studies have suggested that bands are not predictors of mortality[12], band cells *were* an important prognostic indicator in our study with an O.R =1.59, approaching the odds ratio for leukocytosis and anemia.

The most impressive predictors of mortality were burr cells, NRBCs and absolute lymphocytosis. These three had the highest multivariate odds ratios of any CBC finding: from 2.8 to 3.2. Taken univariately, the first two were associated with mortality rates eight to ten times the mortality of the average admitted patient. The literature carries anecdotal reports about burr cells being associated with an ominous prognoses[24,25,26], and more robust statistical analyses demonstrating that NRBCs are associated with increased mortality[15]. Lymphocytosis has also been reported as a mortality risk in patients with trauma and emergency medical conditions[16,17]. Our analysis shows that, indeed, all three of these findings are strong *independent* predictors of mortality.

The presence of sickle cells was also a strong predictor, but in the opposite direction. Patients with sickle cells in their smear had a mortality risk one third that of non-sickle cell patients. This does not indicate a protective effect. Rather, these patients are typically young patients admitted for pain control and other non-life-threatening conditions. In sickle cell patients, presence of NRBCs appears to be a finding intrinsic to the disease without the same mortality implications as in other patients in our study.

The overall logistic model including age, gender and admission CBC variables had a respectable c-statistic = 0.80, for predicting 30-day mortality. This compares well with other multivariable models. For example, the APACHE II score used to predict in hospital mortality for critical care patients has c-statistics that range from 0.78 to 0.86[42,72,28]. The APACHE score uses the worst value from the first two days of the admission for some of its predictors so it cannot provide as early a warning signal as the admission CBC and it requires significantly more data collection. The inclusion of more CBC findings in the APACHE model might increase its predictive accuracy.

Our multivariate analysis was based on very large patient samples using data collected through routine clinical care. However, our study has a number of limitations. The analysis

was done at only one institution and the exact logistic regression model may not apply to different institutions with different case mixes and laboratory procedures. Our institution's reported 30 day mortality rate of 3.4% was lower than the 4.6–11.9% reported in studies of patients admitted to general ward services[29,30,31], but this may be accounted for by the lower than average Charlson comorbidity scores in our study population. Our risk adjustment by Charlson comorbidity scores may not be as precise as risk adjustment tailored for our particular institution.[32] Our 30 day mortality rate was calculated using State Death Tapes, so we would miss patients that die outside of the State, although we believe this rarely happens. We developed predictive equations on the basis of 30-day mortality so we cannot comment on whether the CBC elements predict mortality beyond 30 days. For most variables, we analyzed only on a threshold of high or low, or present or absent. Increasing degrees of abnormality may further increase the predictive power of some variables. Finally, the CBC is only one of many tests and clinical findings; it may be that some of these other variables would displace some CBC variables and/or improve the overall predictive power at the time of admission laboratories. In this initial study, we describe the prognostic implication of the CBC across a wide range of diagnoses. Future work will focus on the predictive power of commonly gathered variables in more specific conditions (e.g. low white blood cell count in sepsis).

Physicians generally have an intuitive sense for identifying patients who are seriously ill and at high mortality risk[33], and adjust their diagnostic and therapeutic efforts accordingly. Our analysis highlights the value that certain observations in the CBC, notably burr cells, NRBCs, and absolute lymphocytosis, add to physicians' assessments of mortality risk. Even after adjustment for age, gender, comorbidities, common admission diagnoses, and other variables in the CBC, presence of these findings predicts a three-fold increase in 30-day mortality. Identifying the "red flags" within this ubiquitous test can make the difference in premature discharge or inappropriate triage of patients. Busy physicians can choose from a wide selection of ever-improving diagnostic tests, yet the workhorse CBC can serve as a simple and early identifier of patients with a poor prognosis.

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Table 1

Characteristics of 35,692 unique patients with a CBC and automated differential count.

Characteristic	Value
Average Age (y)	46.2 ± 17.7
Average LOS (d)	6.5 ± 8.1
Male (%)	55.4
Race	
White (%)	52.9
Black (%)	43.4
Other (%)	3.7
Average Charlson Index	0.83 ± 1.5
Most Common Admission Diagnoses (ICD9)	Chest Pain
	Pneumonia, Organism Unspecified
	Other Symptoms Involving Abd or Pelvis
	Unspecified Heart Failure
	Intermediate Coronary Syndrome
	Unspecified Hemorrhage of GI Tract
	Acute but Ill-defined Cerebrovascular Disease
	Diseases of Pancreas
	Cellulitis and Abscess of Leg except Foot
	Convulsions

Table 2

Univariate risk of 30-day mortality in patients with an admission CBC and automated differential count.

		Number (%)	Odds Ratio	P value
	Age (years, ≥18)	35688 (100)	1.039	<0.0001
	Gender (male)	19788 (55.4)	1.420	<0.0001
Hemogram	WBC > 12,000	11124 (31.2)	2.049	<0.0001
	WBC < 5,000	2176 (6.1)	0.938	0.5765
	Hematocrit (> 54)	212 (0.6)	2.633	<0.0001
	Hematocrit (< 37)	8687 (24.3)	2.359	<0.0001
	MCV (> 94)	6552 (18.4)	1.584	<0.0001
	MCV (< 80)	2815 (7.9)	1.258	0.0121
	High RDW (>14.5)	9478 (26.6)	2.647	<0.0001
	High MCH (>32)	5308 (14.9)	1.367	<0.0001
	Low MCH (<26)	2064 (5.8)	1.392	0.0011
	High MCHC (>36)	28 (0.1)	3.964	0.0109
	Low MCHC (<32)	738 (2.1)	2.190	<0.0001
Automated Differential Count	Neutrophilia (>7700)	10578 (37.8)	1.601	<0.0001
	Neutropenia (<1500)	469 (1.3)	2.831	<0.0001
	Basophilia (>200)	1137 (3.2)	1.362	0.0215
	Eosinophilia (>450)	1529 (4.3)	1.074	0.5788
	Monocytosis (>800)	10066 (28.2)	1.262	<0.0001
	Lymphocytosis (>4000)	3046 (8.5)	2.495	<0.0001
Manual Differential Count	Blast Cells (y/n)	31 (0.1)	1.638	00.5001
	Myelocyte (y/n)	215 (0.6)	8.231	<0.0001
	Promyelocyte (y/n)	25 (0.1)	13.429	<0.0001
	Metamyeloctye (y/n)	905 (2.5)	5.798	<0.0001
	Atypical Lymphocyte (y/n)	1303 (3.7)	1.881	<0.0001
	Hypersegmented Neutrophils (y/n)	141 (0.4)	3.061	<0.0001
	Microcytes (y/n)	3452 (9.7)	2.578	<0.0001
	Macrocytes (y/n)	3475 (9.7)	3.282	<0.0001
	Hypochromic RBCs (y/n)	2252 (6.3)	2.290	<0.0001
	Basophilic Stippling (y/n)	273 (0.8)	3.553	<0.0001
	Target Cells (y/n)	1140 (3.2)	2.866	<0.0001
	Polychromasia (y/n)	1675 (4.7)	3.622	<0.0001
	Toxic Granules (y/n)	1063 (3.0)	4.021	<0.0001
	Dohle Bodies (y/n)	524 (1.5)	4.821	<0.0001
	Ovalocytes (y/n)	1555 (4.4)	2.558	<0.0001
	Spherocytes (y/n)	465 (1.3)	3.132	<0.0001
	Schistocytes (y/n)	1484 (4.2)	3.150	<0.0001
	Sickle Cells (y/n)	62 (0.2)	0.389	0.3490
	Howell-Jolly Bodies (y/n)	71 (0.2)	3.025	0.0033

		Number (%)	Odds Ratio	P value
	Pappenheimer Bodies (y/n)	67 (0.2)	2.344	0.0468
	Burr Cells (y/n)	253 (0.7)	9.297	<0.0001
	Teardrop Cells (y/n)	538 (1.5)	2.150	<0.0001
	Vacuolated Cells (y/n)	897 (2.5)	3.667	<0.0001
	Giant Platelets (y/n)	781 (2.2)	3.102	<0.0001
	Smudge Cells (y/n)	50 (0.1)	5.237	<0.0001
	Cleaved Cells (y/n)	8 (0.0)	3.393	0.2533
	Band Forms (y/n)	7594 (21.3)	2.964	<0.0001
	NRBCs (y/n)	467 (1.3)	8.756	<0.0001

Table 3

Multivariate model of statistically significant (P < 0.005) predictors of 30 day mortality from the CBC and automated differential count pared by stepwise backwards selection.

Parameter	Odds Ratio	Confidence Interval	P Value
Age (years)	1.040	1.037–1.043	<0.0001
Gender (male)	1.965	1.746–2.213	<0.0001
WBC > 12,000	1.701	1.508–1.919	<0.0001
Hematocrit (> 54)	2.331	1.438–3.780	<0.0006
Hematocrit (< 37)	1.714	1.514–1.941	<0.0001
MCV (> 94)	1.352	1.186–1.543	<0.0001
High RDW (>14.5)	1.463	1.291–1.658	<0.0001
Lymphocytosis (>4000)	2.848	2.435–3.332	<0.0001
Metamyeloctye (y/n)	2.074	1.666–2.581	<0.0001
Macrocytes (y/n)	1.317	1.127–1.539	<0.0005
Toxic Granules (y/n)	1.494	1.200–1.859	0.0003
Sickle Cells (y/n)	0.039	0.005–0.292	0.0016
Burr Cells (y/n)	3.254	2.347–4.513	<0.0001
Band Forms (y/n)	1.586	1.386–1.814	<0.0001
NRBCs (y/n)	2.906	2.240–3.770	<0.0001