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Research article

Identifying older diabetic patients at risk of poor glycemic control

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Abstract

Background: Optimal glycemic control prevents the onset of diabetes complications. Identifying diabetic patients at risk of poor glycemic control could help promoting dedicated interventions. The purpose of this study was to identify predictors of poor short-term and long-term glycemic control in older diabetic in-patients.

Methods: A total of 1354 older diabetic in-patients consecutively enrolled in a multicenter study formed the training population (retrospective arm); 264 patients consecutively admitted to a ward of general medicine formed the testing population (prospective arm). Glycated hemoglobin (HbA1c) was measured on admission and one year after the discharge in the testing population. Independent correlates of a discharge glycemia ≥ 140 mg/dl in the training population were assessed by logistic regression analysis and a clinical prediction rule was developed. The ability of the prediction rule and that of admission HbA1c to predict discharge glycemia ≥ 140 mg/dl and HbA1c > 7% one year after discharge was assessed in the testing population.

Results: Selected admission variables (diastolic arterial pressure < 80 mmHg, glycemia = 143–218 mg/dl, glycemia > 218 mg/dl, history of insulinic or combined hypoglycemic therapy, Charlson's index > 2) were combined to obtain a score predicting a discharge fasting glycemia ≥ 140 mg/dl in the training population. A modified score was obtained by adding 1 if admission HbA1c exceeded 7.8%. The modified score was the best predictor of both discharge glycemia ≥ 140 mg/dl (sensitivity = 79%, specificity = 63%) and 1 year HbA1c > 7% (sensitivity = 72%, specificity = 71%) in the testing population.

Conclusion: A simple clinical prediction rule might help identify older diabetic in-patients at risk of both short and long term poor glycemic control.

Background

Optimal glycemic control has been proved to prevent the onset and/or to slow the progression of several complications of diabetes and, thus, to improve quality of life [1–4]. Guidelines have been proposed to optimize the management of outpatients with diabetes and to assess the quality of the achieved glycemic control [5–16]. Attention has not been paid to the quality of glycemic control in people admitted to acute care hospital. Indeed, glycemic levels during the hospital stay, i. e. during a few days, are expected to lack any effect on the risk of complications which is related to the quality of glycemic control through several years. However, diabetic in-patients might represent a convenient sample for verifying whether any relationship exists between admission clinical/laboratory characteristics and the quality of glycemic control achieved at discharge and one year later. Targeting patients at risk of poor long term glycemic control could help promoting strategies to improve the quality of diabetes care.

Older diabetics represent the ideal population to be targeted because logistic problems frequently limit their access to ambulatory health care facilities. Furthermore, standards of care have been reported to be quite poor for a considerable proportion of older Medicare diabetic patients [17].

The present study has two objectives: 1) to develop a clinical prediction rule targeting older patients at risk of being discharged in poor glycemic control; 2) to verify whether the same prognostic model can predict long term poor glycemic control, as expressed by HbA1c > 7% one year later.

Methods

Study design

The first part of this study aimed at developing a predictive score targeting subjects at risk of poor glycemic control at discharge in a population of diabetic patients enrolled in the GIFA (Gruppo Italiano di Farmacovigilanza nell'Anziano) study (retrospective arm of the study). The predictive score was, then, prospectively validated in a population of 264 diabetic in-patients consecutively admitted to a ward of general medicine. Accordingly, we will define "training" and "testing" population the ones in which the score was developed and validated, respectively [18]. The ability of both predictive score and admission HbA1c values to predict abnormal HbA1c one year after discharge was assessed in the testing population. The study design was approved by Ethical Committees of participating institutions. Patients gave their informed consent to participate in the study. The study design is summarized in figure 1 (see additional file: figure1.ppt).

Training population: data source

We used data collected by the GIFA in six non consecutive periods (May – June and September – October in the years 1993, 1995 and 1997). The design of the GIFA study has been described elsewhere [19,20]. The study aimed at assessing drug use at home and in the hospital by a standardized protocol. All patients admitted to participating centers (32 wards of Geriatrics and 37 wards of Internal Medicine) during the study period were recruited without exclusion criteria. A study physician completed a questionnaire on admission recording demographic and personal information (such as smoking habit, household composition), functional and cognitive status. Results of laboratory tests performed at admission and at the time of discharge were also collected. Serum glucose was assessed by the hexokinase method. The study physician updated the questionnaire daily with detailed information on the therapy prescribed and finally recorded discharge diagnoses and prescriptions.

Training population: selection criteria

The number of patients enrolled in the GIFA study in the period we considered was 14,037. We excluded persons younger than 65 years (n = 3,311) and those who died during the hospital stay (n = 751). Of the 9,982 remaining patients, 1,833 (18.4%) had a diagnosis of diabetes mellitus, identified by the codes 250.0 – 250.6; 250.7; 250.9; 337.1 – 357.2; 354.5; 355.9; 358.1; 362.0; 366.4; 443.8, 581.8, 582.8, 583.8 of the International Classification of Disease [21]. We then excluded those with missing data on glycemia or any other of the variables to be tested as potential correlates of the quality of achieved glycemic control (n = 479). These variables assess health status, type of hypoglycemic therapy prior to admission, comorbidity and severity of the metabolic derangement, as expressed by selected laboratory and clinical parameters. The 1,354 remaining patients formed the training population.

Training population: clinical measures

Height and weight were measured while the patient was fasting and wore light clothing. Weight of dehydrated or edematous patients was measured after the achievement of the euvolemic status. Then, Body Mass Index (BMI = weight/height^2) was computed.

We measured the performance on the Activities of Daily Living (ADLs: dressing, bathing, transferring, toileting, walking, eating) [22], and divided patients in the following three categories: independent in all ADLs, dependent on external help in 1 to 5 ADLs, dependent on external help in all ADLs.

Cognitive status was assessed by the Abbreviated Mental Test (AMT), which is a 10-item screening test for dementia

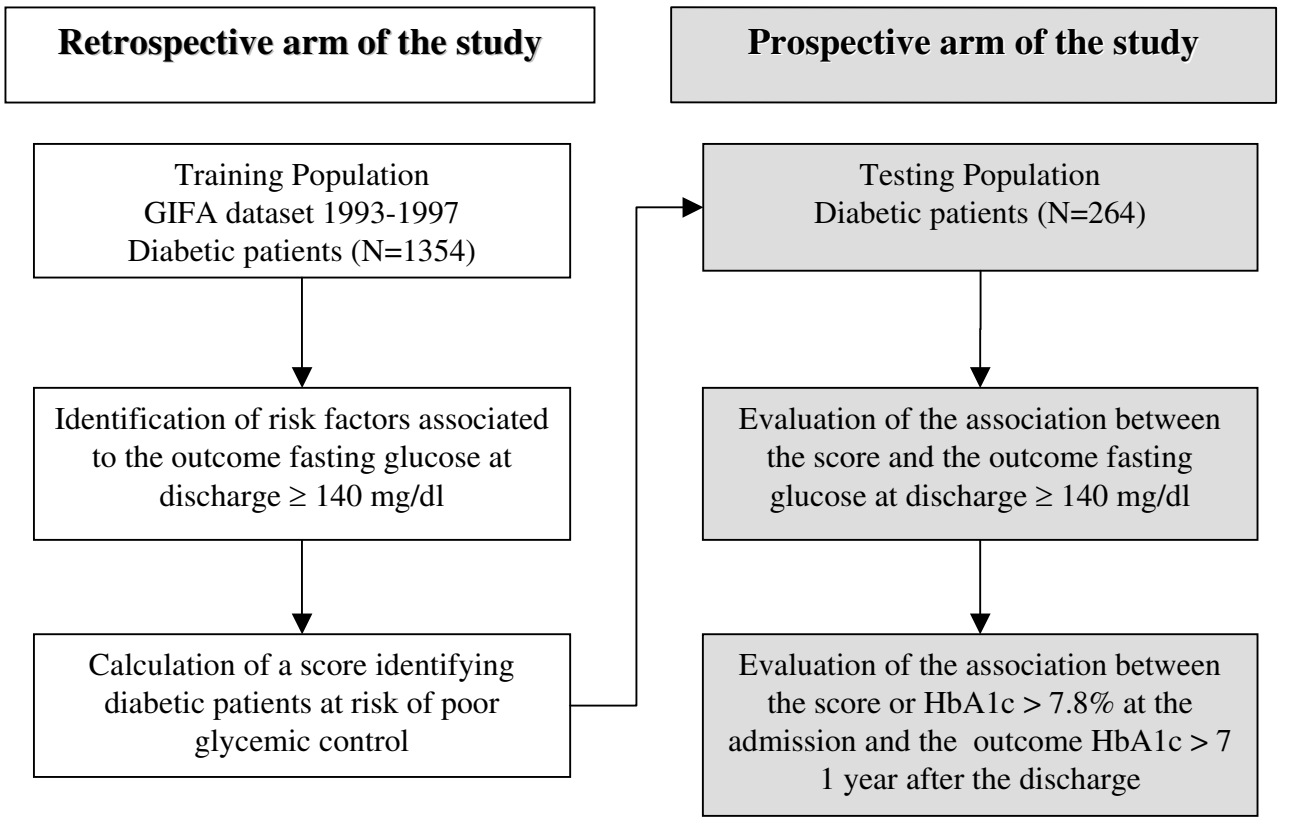

Figure 1
Design of the study.

validated in Italy [23,24]. We considered abnormal a score lower than 7. This cut-off level for AMT score has been reported to yield 100% sensitivity and 71% specificity with respect to the DSM III diagnosis criteria of dementia [24].

Comorbidity was measured according to Charlson et al. [25].

The hospital staff measured arterial blood pressure at the time of the patient's admission. The average of two consecutive measurements was considered. Heart rate was measured on an electrocardiographic strip.

On admission, the study physician requested the patients and/or, if necessary, their relatives or caregivers to display all containers for drugs taken during the last two weeks prior to admission or to recall the names of drugs. Also, the study physician recorded daily all the drugs prescribed during the hospital stay (including dosage), as well as those prescribed at discharge. Drugs were codified according to Anatomical, Therapeutic and Chemical codes, version 9th [26]. In the present study, we considered only oral hypoglycemic agents and insulin for analysis.

Testing population

It included 264 diabetic patients selected out of the 399 admitted to a ward of general medicine of an acute care hospital (University Hospital of Messina) in 1999–2000 to be matched to the training population for age, gender and education as well as for performance on AMT and ADL (prospective arm of the study). Discharged patients were seen by internists in the outpatient department every 3 to 6 months depending upon the individual needs.

Clinical characteristics and values of selected laboratory indexes were systematically collected as in the training population. HbA1c levels were measured by HPLC (Dia-

Table 1: Comparison among training population and patients excluded from the analysis because of missing data

	Training population (N = 1354)	Patients excluded (N = 479)	t	P
Age (yrs)	77.0 ± 6.7	77.9 ± 7.2	-2.524	0.012
Gender (males)	577 (42.6)	209 (43.6)		0.699
BMI (kg/m^2)	26.3 ± 4.8	25.6 ± 4.6	2.495	0.013
Systolic blood pressure (mmHg)	149 ± 26	149 ± 25	-0.038	0.969
Diastolic blood pressure (mmHg)	82 ± 14	82 ± 13	0.591	0.555
Education (yrs)	5.4 ± 3.3	5.0 ± 2.9	2.377	0.018
Type of ward				0.023
Geriatrics	961 (71.0)	343 (71.6)		
Medicine	366 (27.0)	135 (28.2)		
Others	27 (2.0)	1 (0.2)		
Charlson	2.5 ± 1.8	2.5 ± 1.6	0.878	0.380
AMT	6.9 ± 3.0	6.9 ± 3.1	0.308	0.758
ADL				0.821
Independent	661 (48.8)	234 (48.9)		
Assistance	292 (21.6)	98 (20.5)		
Dependent	390 (28.8)	144 (30.1)		
Hypoglycemic therapy prior to admission				0.002
Diet alone	659 (48.7)	263 (54.9)		
Oral agents alone	444 (32.8)	160 (33.4)		
Insulin alone	213 (15.7)	42 (8.8)		
Oral agents and insulin	38 (2.8)	14 (2.9)		
Plasma glucose (mg/dl)	198 ± 102	159 ± 78	7.459	0.001
Cholesterol (mg/dl)	189 ± 53	184 ± 51	1.658	0.098
Triglycerides (mg/dl)	162 ± 108	149 ± 98	2.020	0.044
White blood cells (cells/mm^3)	8576 ± 4337	8564 ± 6100	0.044	0.965
Serum sodium (mEq/l)	139 ± 4.5	139 ± 4.3	-0.354	0.723
Serum potassium (mEq/l)	4.3 ± 0.6	4.3 ± 0.6	2.275	0.023

Data are means ± SD or number of cases with percent in parentheses.

man DM BioRad, Segrate – Milano, Italy) both on admission and one year after the discharge. The latter measurement was made on 199 patients. Nineteen out of the remaining 65 patients were dead at the time of follow up, whereas 46 refused to participate or could not be tracked.

Data analysis

In order to define the profile of patients at risk of poor glycemic control, we considered socio-demographic data (age, sex, years of formal education), select laboratory findings (glycemia, cholesterol, triglycerides, sodium, potassium, creatinine, white cell count), BMI, Charlson's index, AMT and ADL scores. We categorized laboratory parameters using the tertiles of their distribution to make the comparisons more meaningful from a clinical point of view. We classified anti-diabetic therapy as follows: diet alone if no drugs were prescribed, oral hypoglycemic therapy alone, insulin alone, combined oral hypoglycemic therapy and insulin.

The glycemic control achieved at discharge was considered as inadequate if fasting glycemia exceeded 140 mg/dl [27]. This threshold was used because the study period antedated the revision of normal glycemic standards proposed in 1999 [28]. Admission variables univariately associated with the outcome glycemia ≥ 140 mg/dl at discharge were entered into a logistic regression analysis aimed at identifying independent correlates of the outcome [29]. Selected variables were excluded from the logistic regression to limit multicollinearity with admission glycemia. The analysis was repeated for patients admitted to the hospital after the introduction of the Diagnosis Related Payment (DRG) system as well as for those who were hospitalized primarily because of diabetes mellitus. This precautionary measure aimed at excluding that the DRG-related shortening of the stay and/or differences in the clinical importance of diabetes could affect the model predicting poor glycemic control at discharge.

We developed a score to target patients at risk of poor glycemic control. Variables recognized to be independently

Table 2: Demographic, historical, clinical and laboratory correlates of fasting plasma glucose ≥ 140 mg/dl at discharge in the training population

	Fasting plasma glucose at discharge			
	< 140	≥ 140	OR*	95% CI
Age (yrs)	76.8 ± 6.8	77.2 ± 6.7	1.01	0.99–1.02
Gender (males)	332 (44.9)	245 (39.8)	0.82	0.66–1.02
BMI (kg/m^2)	26.1 ± 4.8	26.7 ± 4.8	1.02	1.0–1.05
Systolic blood pressure (mmHg)				
<140	130 (17.6)	116 (18.9)	1.0	
140–160	370 (50.1)	299 (48.6)	0.89	0.66–1.19
>160	239 (32.3)	200 (32.5)	0.92	0.67–1.26
Diastolic blood pressure (mmHg)				
<80	101 (13.7)	126 (20.5)	1.0	
80–90	452 (61.2)	320 (52.0)	0.56	0.41–0.75
>90	186 (25.2)	169 (27.5)	0.71	0.51–0.99
Education (yrs)				
< 5	240 (32.5)	227 (36.9)	1.0	
5–8	349 (47.2)	261 (42.4)	0.82	0.64–1.05
> 8	91 (12.3)	57 (9.3)	0.70	0.47–1.04
Type of ward				
Geriatrics	532 (72.0)	429 (69.8)	1.0	
Medicine	195 (26.4)	171 (27.8)	1.09	0.86–1.39
Others	12 (1.6)	15 (2.4)	1.63	0.75–3.53
Charlson				
0–1	265 (35.9)	206 (33.5)	1.0	
2	193 (26.1)	136 (22.1)	0.92	0.69–1.23
3+	281 (38.0)	273 (44.4)	1.28	1.01–1.64
AMT				
≥ 7	483 (65.4)	386 (62.8)	1.0	
< 7	244 (33.0)	216 (35.1)	1.06	0.83–1.34
ADL				
Independent	365 (49.4)	296 (48.1)	1.0	
Assistance	175 (23.7)	117 (19.0)	0.80	0.60–1.06
Dependent	192 (26.0)	198 (32.2)	1.23	0.95–1.60
Hypoglycemic therapy prior to admission				
Diet alone	386 (52.2)	273 (44.4)	1.0	
Oral agents alone	253 (34.2)	191 (31.1)	1.05	0.83–1.35
Insulin alone	86 (11.6)	127 (20.7)	2.07	1.51–2.84
Oral agents and insulin	14 (1.9)	24 (3.9)	2.40	1.22–4.74
Plasma glucose (mg/dl)				
< 143	344 (46.5)	110 (17.9)	1.0	
143–218	233 (31.5)	218 (35.4)	2.90	2.18–3.85
> 218	162 (21.9)	287 (46.7)	5.50	4.12–7.34
Cholesterol (mg/dl)				
< 164	237 (32.1)	183 (29.8)	1.0	
164–208	245 (33.2)	185 (30.1)	0.96	0.73–1.26
> 208	212 (28.7)	209 (34.0)	1.24	0.93–1.63
Triglycerides (mg/dl)				
< 110	232 (31.4)	143 (23.3)	1.0	
110–168	217 (29.4)	175 (28.5)	1.31	0.98–1.75
> 168	185 (25.0)	193 (31.4)	1.67	1.24–2.23
White blood cells (cells/mm^3)				
< 6800	248 (33.6)	196 (31.9)	1.0	
6800–8970	262 (35.5)	186 (30.2)	0.90	0.69–1.17
> 8970	224 (30.3)	225 (36.6)	1.28	0.98–1.67

Table 2: Demographic, historical, clinical and laboratory correlates of fasting plasma glucose ≥ 140 mg/dl at discharge in the training population *(Continued)*

Serum sodium (mEq/l)				
< 138	185 (25.0)	210 (34.1)	1.0	
138–141	300 (40.6)	230 (37.4)	0.68	0.52–0.88
>141	242 (32.7)	169 (27.5)	0.61	0.46–0.80
Serum potassium (mEq/l)				
< 4.1	237 (32.1)	185 (30.1)	1.0	
4.1–4.6	294 (39.8)	260 (42.3)	1.13	0.88–1.46
>4.6	200 (27.1)	165 (26.8)	1.05	0.79–1.39

Data are means ± SD or number of cases with percent in parentheses. Data may not yeld 100% because of missing data. * Odds ratios adjusted for age and gender.

correlated with the outcome were assigned a score whose magnitude ranged between 0 and 5 and was directly proportional to the corresponding odds ratio. The final score for the individual patient was obtained by summing partial scores, i. e. those corresponding to predictors collected in that patient. The ability of the score to predict the outcome glycemia ≥ 140 mg/dl control at discharge and the best cut off value of the score were assessed by measuring the area under the receiving operating characteristic (ROC) curve in the testing population [30].

Statistical analysis was performed using SPSS software package version 10.0 (SPSS Inc, Chicago, IL, USA).

The predictive score was validated in the testing population. In this population, we calculated also the sensitivity and specificity of HbA1c in detecting the probability of poor glycemic control at discharge, using a cut off value of 7.8 %, which was empirically identified as the best cut off level. We obtained a modified score by adding 1 to the original score in patients with admission HbA1c > 7.8%. Finally, we compared the diagnostic accuracy of the three predictors (the predictive score, HbA1c >7.8% on admission, and the modified score) versus the outcome HbA1c > 7% one year after the discharge.

Results

The main characteristics of the training population and of patients excluded from the study because of incomplete data recording are reported in Table 1. Excluded patients were slightly older and had lower formal education and BMI, but comparable level of comorbidity, physical and cognitive performance. They were in better metabolic control, as reflected by lower admission serum glycemia and triglycerides and lesser use of insulin prior to admission (Table 1).

Patients enrolled in the training population were grouped according to whether their glycemia at discharge was or not inferior to 140 mg/dl (Table 2). Groups had comparable age, prevalence of males, educational level, nutritional status, cognitive and physical capabilities. Diabetes mellitus was the cause of hospitalization in 16.1% of patients discharged with glycemia < 140 mg/dl and 22.6% of patients discharged with glycemia >139 mg/dl. The allocation to a ward of Geriatrics or General Medicine was unrelated to the outcome glycemic control, whereas the use of insulin alone or with oral hypoglycemic agents prior to admission, diastolic hypotension on admission and a Charlson's index greater than 2 were more common among patients having fasting glycemia ≥ 140 mg/dl at discharge. The higher the glycemia on admission the greater the risk of not achieving a satisfactory glycemic control at discharge. Also hypertrygliceridemia and hyponatremia on admission were more prevalent among patients discharged with glycemia ≥ 140 mg/dl.

Logistic regression analysis identified five admission variables independently correlated with poor glycemic control at discharge: diastolic arterial pressure lower than 80 mmHg, glycemia = 143–218 mg/dl, glycemia > 218 mg/dl, use of insulin with or without oral hypoglycemic agents in the two weeks prior to admission, Charlson's index > 2 (Table 3). We did not include hypertrygliceridemia in the model because of the collinearity with admission hyperglycemia, which likely reflects the parallelism between serum levels of glucose and tryglicerides in decompensated diabetes. Logistic regression analyses limited to the DRG era (1995–1997) and to patients for whom diabetes was the cause of admission confirmed results achieved in the whole population.

In an attempt to interpret the relationship between lower diastolic pressure and poor metabolic control, we characterized hypotensive patients and found that a greater Charlson's index (2.8 ± 2.2 vs. 2.5 ± 1.7, $p < 0.005$) and lower cholesterol level (169 ± 50 vs. 193 ± 53 mg/dl, $p < .001$) were the hallmark of this subset.

Table 3: Results of logistic regression analysis having plasma glucose ≥ 140 mg/dl at discharge as the dependent variable in the training population

	Fasting plasma glucose at discharge			
	< 140	≥ 140	OR	95% CI
Age (years)	76.8 ± 6.8	77.2 ± 6.7	1.01	0.99–1.03
Gender (males)	332 (44.9)	245 (39.8)	0.88	0.70–1.12
Diastolic blood pressure < 80 mm Hg*	101 (13.7)	126 (20.5)	1.63	1.19–2.22
Plasma glucose on admission (mg/dl)				
<143	344 (46.5)	110 (17.9)	1.0	**** _ ****
143–218*	233 (31.5)	218 (35.4)	2.84	2.13–3.79
>218*	162 (21.9)	287 (46.7)	5.29	3.91–7.15
White blood cells > 8970 cells/mm^3	224 (30.3)	225 (36.6)	1.09	0.85–1.40
Serum sodium < 138 mEq/l	185 (25.0)	210 (34.1)	1.02	0.78–1.33
Insulin or combined hypoglycemic therapy prior to admission*	100 (13.5)	151 (24.6)	2.03	1.50–2.75
Charlson's Index > 2*	281 (38.0)	273 (44.4)	1.32	1.04–1.67

Data are means ± SD or number of cases with percent in parentheses. * Marks variables found to be independently correlated with the outcome.

A score predicting poor glycemic control was developed as follows: each independent correlate of the outcome was assigned a score whose magnitude was directly proportional to the corresponding odds ratio. The score for admission glycemia > 218 mg/dl was arbitrarily established to be 5, while scores of the remaining predictors were computed according to the ratio between odds ratios of glycemia > 218 mg/dl and that of individual predictors obtained by the logistic regression analysis and were approximated to the unit. For example, the score of glycemia = 143–218 was computed by solving the following proportion: 2.84 : 5.29 = x : 5 and was found to be 2.7, which was approximated to 3. Insulin therapy prior to admission and diastolic arterial pressure inferior to 80 mmHg scored 2, Charlson's index > 2 scored 1. For each patient a final score was obtained by summing scores of individual predictors. Thus, a subject who used insulin prior to admission, had admission glycemia = 268 mg/dl and Charlson's index = 3 scored 8, i. e. 2 + 5 + 1 = 8.

Selected indexes of diabetes severity and the distribution of the predictive score in the testing population are summarized in Table 4. The distribution of the score levels was used to compute the corresponding pairs of sensitivity and specificity values used for drawing the ROC curve (Figure 2) (see additional file: figure2.bmp). The area under the curve was 0.72, which is consistent with a good, but not excellent predictive model. The cut off level of the predictive score achieving the best discrimination of patients was 4 and had sensitivity of 76% and specificity of 60%. These figures were comparable with sensitivity (79%) and specificity (63%) achieved by the level 5 of the

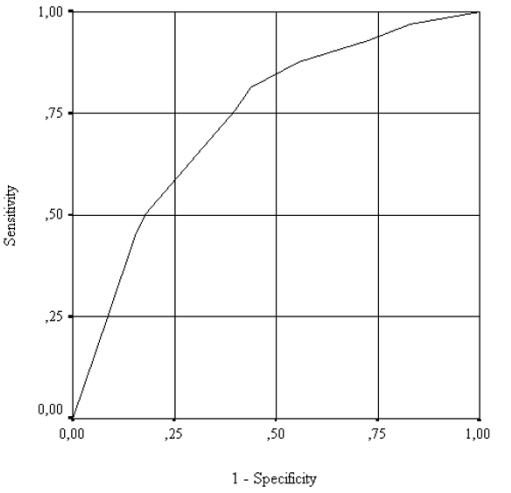

Figure 2
Receiving operating characteristic (ROC) curve of the predictive score versus the outcome poor glycemic control at discharge in the testing population.

In the testing population, HbA1c levels on admission and the predictive score were highly correlated (rho = 0.58, p < 0.001). Quartiles of HbA1c could predict the outcome glycemic control at discharge at least as effectively as the original and the modified predictive scores: HbA1c > 7.8 %, corresponding to the lower limit of the third quartile, had sensitivity of 85% and specificity of 60% versus the outcome poor glycemic control at discharge.

Table 4: Selected indexes of diabetes severity and distribution of the predictive score in the testing population *

	Fasting plasma glucose at discharge					
	< 140	≥ 140	B	SE(B)	OR**	95% CI
Plasma glucose at the admission (mg/dl)						
<143	75 (55.6)	28 (21.7)			1.0	
143–218	41 (30.4)	48 (37.2)	1.16	0.31	3.19	1.73–5.89
>218	19 (14.1)	53 (41.1)	2.05	0.35	7.76	3.89–15.5
Insulin or combined hypoglycemic therapy prior to admission	56 (41.5)	79 (61.2)	0.85	0.26	2.35	1.42–3.88
Score						
0	23 (17.0)	4 (3.1)			1.0	
1	14 (10.4)	5 (3.9)	0.73	0.76	2.08	0.46–9.28
2	23 (17.0)	7 (5.4)	0.65	0.70	1.91	0.48–7.55
3	16 (11.9)	8 (6.2)	1.19	0.70	3.29	0.83–13.0
4	5 (3.7)	7 (5.4)	2.13	0.81	8.45	1.74–41.1
5	30 (22.2)	33 (25.6)	1.92	0.60	6.81	2.09–22.2
6	3 (2.2)	7 (5.4)	2.80	0.90	16.5	2.84–95.6
7	9 (6.7)	25 (19.4)	2.92	0.68	18.5	4.92–69.9
≥ 8	12 (8.9)	33 (25.6)	2.93	0.65	18.8	5.26–67.2

* See the text, section "Results", for the method of computing the predictive score. **Adjusted for age and gender. Data in columns 2 and 3 are numbers (percentage).

Table 5: Sensitivity and specificity of the predictive score, HbA1c >7.8% at the admission and the modified score versus the outcome HbA1c >7% one year after the discharge in the testing population

	Score (level 4)	HbA1c >7.8% at admission	Modified score (level 5)
Sensitivity, %	66	69	72
Specificity, %	71	56	71
Positive predictive value, %	81	75	79
Negative predictive value, %	57	56	58

Sensitivity: True positives/(True positives + False negatives); Specificity: True negatives/(True negatives + false positives); Positive predictive value: True positives/(True positives + False positives); Negative predictive value: True negatives/(True negatives + False negatives).

When compared with the predictive score and HbA1c >7.8% on admission, the modified predictive score showed the best mix of sensitivity (72%) and specificity (71%) towards the outcome HbA1c > 7% one year after discharge (Table 5). It could target accurately diabetics at risk of long term poor metabolic control (positive predictive value: 79%), but not those likely to have their diabetes well compensated one year after the discharge (negative predictive value: 58%).

Discussion

This study demonstrates that over 50% of older diabetic patients are discharged from wards of general medicine and geriatrics of the acute care hospital in poor metabolic control and can be reliably identified by an admission value of HbA1c > 7.8 % or a simple clinical prediction rule. More importantly, these predictive tools effectively target patients exposed to a significant risk of poor long term glycemic control. The clinical prediction rule has greater specificity than HbA1c > 7.8% in predicting poor long term glycemic control, i.e. it outweighs HbA1c > 7.8% in

targeting subjects who will have their diabetes well controlled one year after being discharged.

Patients with more advanced diabetes, as reflected by the systematic use of insulin at home and/or glycemia>218 mg/dl on admission, were at special risk of being discharged in poor glycemic control. Hypotension on admission was another risk factor for poor glycemic control likely because of its association with polipathology. Indeed, physicians are expected to encounter greater difficulty in tailoring the hypoglycemic therapy to the needs of patients in more unstable conditions and to compensate them more cautiously.

The analysis limited to the 1995–1997 period excluded that the mechanism of the payment based upon the Diagnosis-Related Group could have unduly shortened the stay and, thus, prevented the achievement of optimal glycemic control. On the contrary, in the United States the introduction of the Diagnosis-Related Group payment system could have worsened the quality of care provided to diabetic patients [31]. These observations caution against generalizing the present results to other health systems.

Achieving a satisfactory metabolic control is the only measure proven to prevent diabetic microvascular and neuropathic complications [1–4]. Unfortunately, such an objective was missed in a consistent proportion of diabetic patients, as 65.8% of them had high HbA1c levels one year after discharge. The necessity of balancing the benefit of glycemic control towards the risk of hypoglycemia only to some extent justifies such a result. Indeed, the protective effect of optimal glycemic control towards selected diabetic complications and the ensuing improvement in quality of life largely outweigh the risk of hypoglycemic episodes [1–4]. Furthermore, sustained improvement in glycemic control results in significant health cost saving with a lag time of 1 or 2 years [32]. However, our population was older than that enrolled in the UKPDS trials and by Wagner et al. Older age is associated with reduced sensitivity to neurovegetative effects of hypoglycemia [33]. Furthermore, social factors such as lack of both formal and informal support caution against an overzealous treatment of diabetes, mainly in patients with cognitive impairment or defective vision. However, lack of data on older diabetic populations prevented us from choosing levels of optimal metabolic control different from those reported to protect from microvascular complications. In this perspective, HbA1c > 7% likely represents a convenient index of poor long term metabolic control, whereas glucose ≥ 140 mg/dl seems a questionable short term index of metabolic decompensation. Indeed, achieving the optimal glycemic control might not be the primary outcome of in-patient management if comorbidity and frailty coexist with diabetes.

The recent experience shows that a considerable lag time exists between the unequivocal demonstration and the increase in the use of important and even lifesaving drugs such as ace-inhibitors for congestive heart failure; nevertheless, rate of use still remains far from the optimal one [34,35]. It is likely that the optimization of hypoglycemic therapy will follow a similar time course. Indeed, the benefit of optimal glycemic control has been definitively proven in 1998 and further confirmed in 1999–2000. However, a pessimistic attitude towards diabetes rather than an insufficient knowledge of this disease seems to hinder the implementation of management guidelines by physicians, at least by primary care providers [36]. Furthermore the poor quality of documentation sent to the general practitioners after the patient's discharge from the hospital could contribute to worsen the subsequent management of diabetes [37].

Selected interventions such as a cooperative strategy between general practitioners and diabetologists or the organization of mini clinics by general practitioners or education programs can significantly improve metabolic control [38–41]. While these interventions are generically devoted to the average home-dwelling diabetic patients, present findings suggest that special attention should be paid to selected older patients discharged from the acute care hospital. These patients should benefit from a very careful supervision by the primary care providers and/or from programs aimed at optimizing the home management.

Limitations of our study deserve to be cited: 1) Fasting glycemia was the only measure of short term glycemic control. A glycemic profile would have provided a more complete and reliable estimate of the quality of therapy. However, morning hyperglycemia has been reported to be an uncommon consequence of nocturnal hypoglycemia in elderly diabetics on various therapeutic regimens [42]. Thus, it is likely that only a minority of patients classified as undertreated were exposed to an excess of hypoglycemic therapy at night. 2) Conclusions apply to older hospitalized patients and not to the general population. Thus, the proposed method aims at selecting diabetic patients amenable to special care programs after the discharge and, thus, optimizing the use of the few available economic resources. It has not been designed to target elderly homedwelling diabetics at risk of poor metabolic control; 3) The score was validated in a single General Medicine ward and not on multicenter basis. Thus, we cannot assume that discharge and therapeutic behaviors reflect those characterizing most of Geriatrics and General Medicine wards. This might limit the generalizability of results ; 4) Patients

were not systematically screened for diabetic complications. This prevented us from verifying whether the presence of complications affected the therapeutic approach or had prognostic implications.

Conclusions

The present study shows that most of the older patients admitted to an acute care hospital with a primary or second-listed diagnosis of diabetes mellitus are discharged in poor metabolic control and continue to have high HbA1c levels one year after the discharge. Patients at risk of poor long term metabolic control can be effectively targeted by a simple clinical prediction rule on admission. However, attempts should be made to improve the diagnostic accuracy of the proposed clinical prediction rule by including selected presently untested variables such as the presence and severity of diabetic complications. Finally, research is needed to verify whether the identified predictors can be conveniently used to select patients amenable to dedicated home care programs.

Competing interests

None.

Authors' contribution

RAI, AC, and CP participated in the design of the study, analysis of data and drafting of the manuscript. FC carried out the collection of data regarding the testing population, and contributed to the drafing and critical revision of the manuscript. LC, and BM participated in the analysis of data and drafting of the manuscript. FP participated in the drafting and critical revision of the manuscript. PC participated in the design and coordination of the study.

All authors read and approved the final manuscript.

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