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ORIGINAL ARTICLE

Hyperglycaemia in hospital inpatients: still a sticky situation

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Abstract

Background: Diabetes diagnosis is delayed 4–7 years and 50% are undiagnosed. Forty percent of hospitalized patients with any blood glucose level (BGL) ≥10 mmol/L have diabetes 3 months post-discharge, yet less than 5% are detected in hospital. We review identification of, and responses to, hyperglycaemia in inpatients at a teaching hospital.

Methods: The world's largest retrospective review of medical records for inpatients with venous BGL ≥11.1 mmol/L without known diabetes over 12 months (2005–2006). The primary outcome was recognition of hyperglycaemia; secondary outcomes were treatment and documentation of follow up. Logistic regression was performed with variables including BGL, admitting team, length of stay and endocrine team review.

Results: Of 10 973 people screened, 162 were eligible. The median age was 58 years and BGL 13.3 mmol/L, with increased mortality and length of stay. Hyperglycaemia was noted as definitely in 26%, maybe in 24% and definitely not in 50%. Forty percent of patients were treated in hospital and 19% on discharge. Follow up was documented for 24%. A higher BGL and review by the endocrine team were strongly associated with clinical recognition on uni- and multivariate analyses. However, where an endocrine review was sought for non-hyperglycaemia reasons, similar rates of non-recognition occurred.

Conclusion: Despite evidence for improved inpatient outcomes when treated, and high short-term progression to frank diabetes, inpatient hyperglycaemia remains frequently missed. In-hospital recognition is cheap, and vital for the implementation of activities to improve outcomes and prevent progression and complications. Changes to systems for checking pathology results, medical officer education and inpatient screening guidelines are indicated.

Introduction

Diabetes mellitus affects one million Australians aged 25 years and over, of whom 50% remain undiagnosed.[1] It was the sixth leading cause of death in Australia in 2002.[2] Population screening is not cost-effective from a healthy cohort,[3] but is recommended for at-risk groups.[4] The hyperglycaemic inpatient population is an at-risk group not currently included in screening recommendations, despite an Australian study demonstrating over three-quarters of those with one or more inpatient blood glucose level (BGL) ≥10 mmol/L to have either diabetes or impaired glucose metabolism after discharge.[5]

In-hospital hyperglycaemia is a marker of poor outcome, particularly for patients without prior history of diabetes for whom each 1 mmol/L rise in fasting plasma glucose is correlated with a 33% increase in mortality.[6] Controversy remains over the direction of association, whether improvements after insulin treatment are due to normalization of glucose levels or the presence of insulin

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itself,[7] and over ideal target BGL. Despite this, the American Diabetes Association[8] and American College of Clinical Endocrinology[9] agree that inpatient hyperglycaemia is common, detrimental, and improved control can decrease mortality, illness complications, hospital stay and healthcare costs.[10]

Identification of hyperglycaemic inpatients allows both implementation of acute treatment for glycaemic control, and follow up to detect impaired glucose metabolism (for which treatment with lifestyle modification or oral therapies may reduce progression to diabetes[11]) or lessen complications.[12]

To our knowledge, only two past studies examine identification of patients without known diabetes admitted to hospital with diabetic range[13] BGL (random BGL ≥ 11.1 mmol/L or fasting BGL ≥ 7.0 mmol/L). Both suggest patients are not recognized, treated, or formally screened for diabetes[14,15] with up to two-thirds not recognized to have hyperglycaemia at all.

No data exist for the Australian population. Also previous studies have been too small to examine factors associated with recognition of hyperglycaemia or documentation of follow up. We focus on inpatients with hyperglycaemia without a known history of diabetes mellitus as they have worse acute hospital outcomes, and risk long-term complications from unrecognized diabetes.

Materials and methods

Laboratory blood glucose readings from venous samples at a 320-bed inner-city teaching hospital were extracted from the pathology database from 1 May 2005 to 30 April 2006. Medical records for patients with BGL 11.1 mmol/L and above were reviewed.

Glucose was measured on venous plasma samples on a routine analyser using an Olympus hexokinase glucose method (Integrated Sciences, Chatswood, NSW, Australia).

The primary outcome was recognition of hyperglycaemia by medical staff. Hyperglycaemia was considered definitely noted if there was documentation in the progress notes of 'hyperglycaemia', 'diabetes', or a BGL with annotation to indicate abnormality, for example, underlining, circling or arrows. A BGL value without annotation was considered 'possibly' noted.

Secondary outcomes included documentation of a follow-up plan (in progress notes or discharge summary) and treatment. Treatment included in-hospital and/or discharge prescription of oral hypoglycaemic agents, insulin or both.

Other information collected included BGL, gender, age, admitting team, length of stay (LOS), intensive care unit (ICU) admission, whether or not the final venous glucose level during the admission was ≤7.8 mmol/L, and recorded assessment by an endocrine team member. Admitting team categories were: cardiology/cardiothoracic surgery (who have strong evidence for benefits of glycaemic control[8] and screen for hyperglycaemia), medical excluding cardiology, surgical excluding cardiothoracic surgery, and emergency medicine (whose focus on acute care might mean follow up was less rigorous). One investigator (K. T.) coded all files. This project was not subject to a formal Ethics and Research Committee application, but permission to review notes for audit was granted.

Statistics were carried out using SPSS software (v17.0, SPSS, Chicago, IL, USA). Descriptive statistics were tested using the z statistic. Univariate results are reported as odds ratios with significance determined using the χ^2 test and, for ordered groups, the Mantel–Haenszel χ^2 was used to determine if the trend was directional. Logistic regression was used in the multivariate analysis. The final multivariate model was tested by the Hosmer–Lemeshow test.

Results

A total of 20 976 consecutive BGL was carried out on 10 973 persons in 12 months to 30 April 2006 (Fig. 1). Of these, 766 persons (7%) had a BGL ≥ 11.1 mmol/L. From these, 162 (32%) of the hyperglycaemic inpatients had no history of diabetes and formed the study group. Twenty-six per cent of these patients were definitely noted to be hyperglycaemic (new diagnosis of diabetes for 9% plus another 17%) and 24% were 'possibly' noted (BGL recorded without annotation). However, for most (50%) hyperglycaemia was not noted.

Median age was 57.6 years (range 21–94); 44% were women; median BGL was 13.3 mmol/L (range 11.1–39.5). Most (50%) were admitted under a medical team (excluding cardiology), and 22% under cardiology/cardiothoracic surgery. Thirty-six per cent had been admitted to the ICU during their admission. The median LOS was 9 days (mean 18 days, 95% confidence interval (CI) 14.1–21.5 days), with 16 staying 45 days or longer. This compares with median 1 day (mean 3 days) for the entire hospital. The longer mean LOS did not differ according to admitting team: medicine (not cardiology) 18.2 versus 3.6 days, surgical (not cardiothoracic) 14.4 versus 5.0 days, cardiology/cardiothoracic surgery 13.2 versus 4.3 days, and emergency medicine 2.1 versus 1.0 days. The mortality rate was also higher at 9% (95% CI 4.8–14.1%), compared with 3.8% in the general hospital inpatient population.

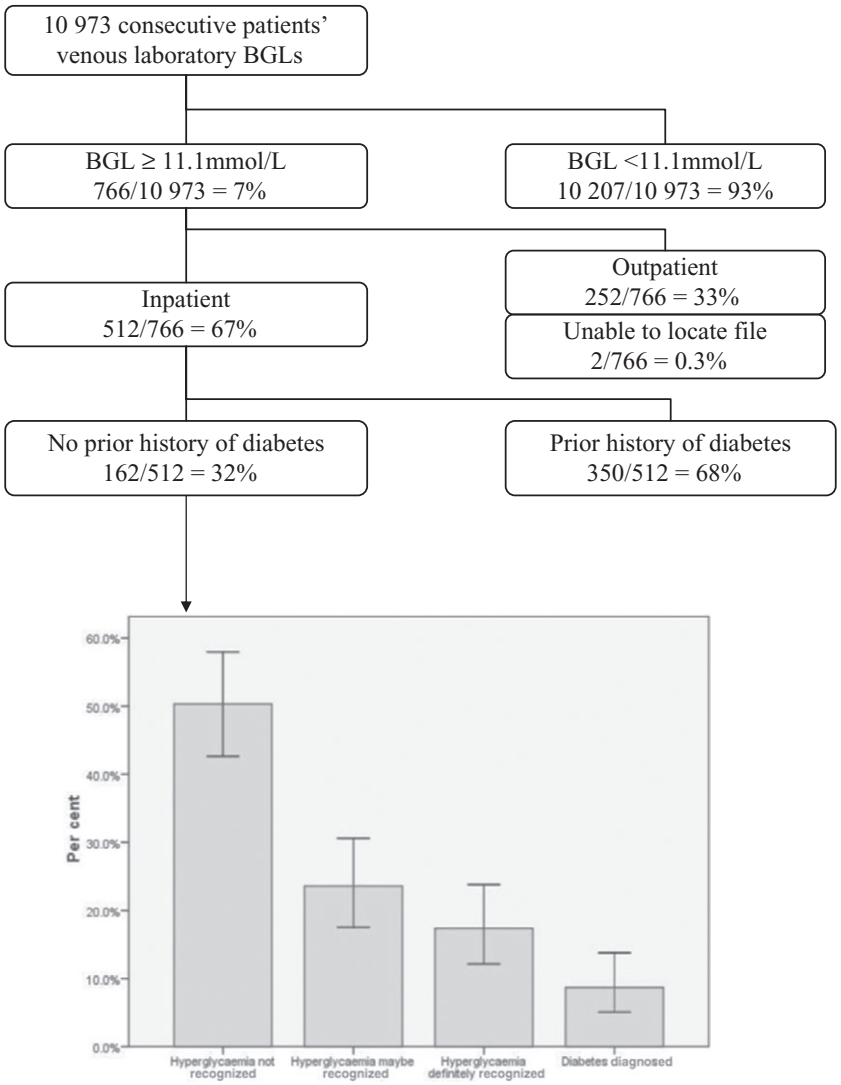

Figure 1 Recognition of hyperglycaemia in inpatients. BGL, blood glucose level.

Excluding those who died during admission (15, 9%) or had incorrectly coded data (one patient), 24% of the remaining 147 patients had follow-up documentation. Sixty-five (40%) patients commenced treatment for diabetes in hospital, 75% with insulin therapy (Table 1), and mostly through infusion or sliding scale. Where data were available, only 19% were discharged receiving treatment, mostly oral agents.

Data were examined to determine factors associated with detection of hyperglycaemia and documentation of a follow-up plan.

For those definitely noted to have hyperglycaemia (Table 2), recognition was associated on univariate analysis with higher BGL level and with endocrine team review. Each 1 mmol/L rise in BGL was associated with a 1.2-fold increased odds of recognition (95% CI 1.1–1.3, $P < 0.001$). The multivariate analysis showed similar but stronger associations, with variables included in the final model being admission to ICU ($P = 0.003$), endocrine team review ($P < 0.001$), higher BGL ($P < 0.001$), having a final venous BGL of the admission ≤7.8 mmol/L

Table 1 Treatment of recognized hyperglycaemia in patients without a previous diagnosis of diabetes

	In hospital No. (%)	On discharge No. (%)
Treated (total)	65 (40)	19 (19)
Oral agents	3 (2)	10 (10)
Insulin	48 (30)	6 (6)
Both oral agents and insulin	14 (9)	3 (3)
Not treated	96 (60)	79 (81)
Total	161†	98‡

†Missing = 1. ‡Missing = 64.

Table 2 Adjusted (univariate) and unadjusted (multivariate) OR for definite recognition of hyperglycaemia, according to different study variables, in patients without a previous diagnosis of diabetes

	Variable	Recognized (*n*)		Univariate analysis‡		Multivariate analysis†	
		Yes	No	OR	95% CI	OR	95% CI
Length of stay (days)	0–2	8	32	1.0			
	3–4	4	16	1.0	0.3–3.8		
	5–10	8	27	1.2	0.4–3.6		
	11–20	9	18	2.0	0.7–6.1		
	>20	13	26	2.0	0.7–5.6		
Further admissions	0	30	87	1.0			
	≥1	12	32	1.1	0.5–2.4		
Age groups (years)	≤40	7	18	1.0			
	40–49	6	26	0.6	0.2–2.1	0.8	0.1–5.6
	50–59	8	25	0.8	0.3–2.7	0.8	0.1–6.3
	60–69	8	16	1.3	0.3–9.3	7.5	1.1–52.5
	70–79	8	15	1.4	0.4–4.7	11.4	1.7–78.0
	≥80	6	19	0.7	0.8–0.2	7.2	1.0–50.4
Gender	Female	20	51	1.0			
	Male	22	68	1.2	0.6–2.5		
Admitting team	Medical	24	57	1.0			
	Surgical	7	19	0.9	0.3–2.4		
	Emergency medicine	3	16	0.4	0.1–1.7		
	Cardiology/cardiothoracic	8	27	0.7	0.3–1.8		
ICU admission	No	30	72	1.0			
	Yes	12	47	0.6	0.3–1.3	0.2	0.1–0.5
Endocrine team review§	No	22	109	1.0			
	Yes	19	8	11.7*	4.6–30.3	20.5	5.0–84.5
BGL level	Each 1 mmol/L rise	NA	NA	1.2*	1.1–1.3	1.3	1.1–1.4
Last plasma BGL before discharge	>7.8 mmol/L	28	94	1.0			
	≤7.8 mmol/L	14	25	1.9	0.9–4.1	3.9	1.3–11.7

$P < 0.001$ on univariate analysis. †The multivariate model contained all the variables for which multivariate results are listed, including ICU ($P = 0.003$), review by the endocrine team ($P < 0.001$), blood glucose level ($P < 0.001$), having a BGL ≤ 7.8 mmol/L before discharge ($P = 0.014$), and age ($P = 0.036$). ‡The univariate analysis not statistically significant except those marked with ''. §Missing = 4. 95% CI, 95% confidence interval; BGL, blood glucose level; ICU, intensive care unit; OR, odds ratio.

($P = 0.014$), and age ($P = 0.036$). All increased the odds of recognition, except for ICU admission (odds ratio (OR) 0.2, 95% CI 0.1–0.5). There were no statistically significant interactions between BGL and endocrine review or between BGL and admitting team.

Factors statistically significantly associated with follow-up documentation on uni- and multivariate analyses were: review by endocrine team, higher BGL, and having hyperglycaemia definitely noted. Additionally, ICU admission was inversely associated with follow up on multivariate analysis (OR 0.2, 95% CI 0.4–0.6). There was no statistically significant association between having a final venous BGL for the admission ≤7.8 mmol/L with follow-up documentation, even when the potentially confounding effect of treatment was controlled for (data not shown).

For nine hyperglycaemic patients without previously known diabetes, an endocrine review was sought for reasons other than hyperglycaemia (including panhypopituitarism, Addison's disease, diabetes insipidus, osteoporosis, bilateral phaeochromocytoma, hyperthyroidism and hyponatraemia). The endocrine team contributed to definite recognition of hyperglycaemia in four cases, possible recognition in one, and four had no recognition of hyperglycaemia. The initial BGL had only been ordered by the endocrine team in one of the nine cases.

Discussion

It has been over a decade since published data suggested that, if followed, most inpatients with hyperglycaemia (BGL ≥ 10 mmol/L) have an underlying dysglycaemic predisposition, as opposed to a temporary hyperglycaemia.[5] The impetus to detect inpatient hyperglycaemia has increased as data suggest hyperglycaemic inpatients have worse hospital outcomes, which treatment could improve. Despite controversy regarding target BGL and

mechanism of benefits of treatment, there can be no controversy that these patients should be followed after discharge.

In our series 7% of all patients had a BGL ≥ 11.1 mmol/L, a cut-off based on diagnostic random BGL[13] (using a cut-off of 10.0 mmol/L, a further 156 patients or 20% would be included). Approximately one-third (32%) of all hyperglycaemic inpatients had no prior history of diabetes, of whom 50% had no record that hyperglycaemia was noted, nor the BGL result checked. Only 9% were formally diagnosed with diabetes, compared with 5%[14] and 2%[15] in previous data.

The higher mortality seen in the hyperglycaemic patients may be contributed to by routine BGL testing in all cardiorespiratory arrest and cerebrovascular accidents. The longer average length of admission is partially explained by a high number of post-transplant patients. During the study period, 118 solid organ and haematological transplants were carried out, of which 13 were in our cohort (0.6% vs 8% respectively). Regardless, it cannot be excluded that hyperglycaemia itself contributed to mortality and LOS.

Poor recognition of hyperglycaemia may have been a consequence of either the pathology results not being reviewed or, if reviewed, lack of knowledge preventing appropriate action. At our institution, BGL are carried out only on request by a medical officer with random levels ≥ 7.8 mmol/L and fasting levels ≥ 5.5 mmol/L reported with an asterisk. Inadvertent omission of result checking is consistent with the Quality in Australian Health Care Study conclusion that 16% of adverse events are due to 'the failure to synthesise, decide and/or act on available information' whereas lack of knowledge accounts for only 1.1%.[16]

Additionally, contrary to our hypothesis, teams having evidence for benefit from glycaemic control in hospital (cardiothoracic surgery, cardiology, ICU) showed no better recognition than others. The endocrine team was equally poor at recognition of hyperglycaemia (50%), although a follow-up plan was more likely once they were notified of it.

Yet several factors do point to knowledge deficits. These include unwillingness to treat, use of sliding scale insulin, and poor rates of recognition of, and follow up for, the 'lower' levels of hyperglycaemia which some may consider to be 'stress' (implying temporary) hyperglycaemia.

Treatment commenced in only 40%, many insulin-treated patients being treated exclusively with sliding scale insulin. When this ceased they generally did not commence other therapy. At best sliding scale insulin is ineffectual, at worst it results in large excursions in BGL with poor control.[17]

Perhaps it has not been disseminated that evidence shows 'stress' hyperglycaemia is associated with worse outcomes than diabetes,[10] occurs in those susceptible to insulin deficiency[18] and usually reflects a chronic dysglycaemic predisposition when followed up with oral glucose tolerance testing (OGTT). For instance, diabetes and impaired glucose tolerance (IGT) were diagnosed after discharge in 40% and 35%, respectively, of hyperglycaemic (10 mmol/L or above) Australian surgical inpatients at 3 months' follow up;[5] and 16% and 38%, respectively, of hyperglycaemic (fasting BGL ≥ 7.0 mmol/L) Australian acute coronary patients at 4 weeks' follow up.[19] Therefore, even if transient 'stress' hyperglycaemia exists, in the majority hyperglycaemia indicates permanently impaired glucose metabolism.

Interestingly, having a final venous BGL ≤ 7.8 mmol/L before discharge increased the odds of recognition, but had no impact on follow up. The likely explanation is that repeated measurements were ordered when hyperglycaemia was recognized. However, these measurements did not change follow-up behaviour.

Regardless of the perceived aetiology of hyperglycaemia, treatment is recommended,[20] and follow up is indicated regardless of BGL on discharge. Despite suggestions, HbA1c still does not replace formal OGTT in screening for diabetes[21] or IGT.

Strategies for improvement include prompts on pathology results (e.g. 'follow up for diabetes is indicated'), revision of national screening guidelines and, most importantly, education of both staff and patients. As resident medical officers order and check blood results they are the logical target group for education, and have been shown to be responsive to training courses in diabetes management.[22] Additionally, implementation of a management plan is most safely undertaken by a trained multidisciplinary hospital diabetes team, including an endocrine team, dietetics, nursing and discharge planning.

Conclusion

Our study represents the largest audit published to date of the response to hyperglycaemic inpatients. Although controversies still remain over glycaemic targets and mechanisms of benefits of in-hospital insulin treatment, it cannot be denied that these patients are at risk of diabetes and require follow up. We cannot afford to delay strategies to improve recognition of hyperglycaemia. This would allow implementation of a co-ordinated management plan that could improve immediate, as well as long-term outcomes of diabetes.

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Supporting information

Additional supporting information may be found in the online version of this article.

Table S1 Statistically significant univariate and multivariate associations of variables with documentation of follow-up plan (all $P < 0.001$, except ICU $P = 0.007$).

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