Long-term glycaemic control (HbA1c), not admission glucose, predicts hospital re-admission in diabetic patients

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ABSTRACT

Background
Diabetic patients are commonly hyperglycaemic on presentation. Admission hyperglycaemia is associated with adverse outcomes, particularly prolonged hospitalisation. Improving inpatient glycaemia may reduce length of hospital stay (LOS) in diabetic patients.

Aims
To determine whether in-hospital recognition and treatment of admission hyperglycaemia in diabetic patients is associated with reduced LOS.

Methods
Medical records were reviewed from 1 November 2011 to 31 May 2012 for 162 diabetic patients admitted with a blood glucose level (BGL) ≥11.1mmol/L. In-hospital outcomes were compared. Stepwise multiple regression was used to evaluate factors contributing to LOS.

Results
Compared to the untreated individuals (n=67), hyperglycaemia treatment (n=95) was associated with a longer LOS (median eight vs. four days, p<0.01), higher HbA1c (9.0 vs. 7.3 per cent, p<0.01), more infections (50 vs. 25 per cent, p<0.01), and more patients with follow-up plans (35 vs. 10 per cent, p<0.01). Higher HbA1c was significantly related to more follow-up (r=0.30, n=110, p<0.01) with a trend to lower re-admission in those with follow-up plans (r= -1.41, n=162, p=0.07).

Conclusion
Recognition and treatment of admission hyperglycaemia in diabetic patients was associated with longer LOS than if untreated. Contributory factors to LOS include: illness severity, infections, and higher HbA1c. Although follow-up plans were few (27 per cent) for diabetic patients with hyperglycaemia, it was significantly more likely in those with higher HbA1c. Diabetic patients’ complexities require timely multidisciplinary team involvement. Improved follow-up care, particularly for hospitalised diabetic patients identified to have chronically poor glycaemic control, may help prevent future diabetic patient re-admissions.

Key Words
hyperglycaemia, diabetes mellitus, glycaemic control, community management

What this study adds:
1. What is known about this subject?
Admission hyperglycaemia is a common occurrence in diabetic patients. Intensive treatment of admission hyperglycaemia remains controversial, and can cause serious adverse outcomes, prolonging hospitalisation.

2. What new information is offered in this study?
Correcting acute hyperglycaemia in diabetic patients is not ideal and may be harmful. Efforts should focus on
optimising follow-up diabetes care, particularly for those with elevated HbA1c.

3. What are the implications for research, policy, or practice?
Timely, multidisciplinary follow-up in diabetic patients with chronically poor glycaemic control should be made a priority. Improved follow-up may help prevent re-admissions amongst diabetic patients.

Background
Diabetes mellitus represents a significant economic burden on Australian health care.1,2 The country’s total annual healthcare costs have been estimated at AUD $3 billion for type 2 diabetes (2002) and $570 million for type 1 diabetes (2009), with half the expenses directed towards hospital services.1,2

People with diabetes are frequently admitted to hospital, and hyperglycaemia at admission is common.3 A higher admission blood glucose level (BGL) is an indicator of poor clinical outcome, with higher mortality, illness complications, infection risk, increased length of hospital stay (LOS), intensive care unit (ICU) admissions, and greater healthcare costs.4-8 Randomised interventional studies have demonstrated that improved glycaemic control can reduce morbidity and mortality.9-12 However, immediate attainment of intensive glycaemic control is controversial, with inconsistent results and sometimes serious adverse outcomes (i.e., severe hypoglycaemia and/or increased rates of mortality) accompanying very tight glycaemic control.13-19

Hospital admission provides an opportunity for evaluation of clinical status and glycaemic control in people with diabetes,3 and in particular for identifying individuals that require more intensive outpatient diabetes management. Inpatient multidisciplinary diabetes care teams have been shown to reduce LOS significantly in some situations, with potential health and economic benefits for both the patient and the community.20,21

To our knowledge, no study has yet investigated the relationship between LOS and inpatient hyperglycaemia management among people with diabetes in an Australian hospital. We aimed to determine whether, in people with known diabetes, recognition and treatment of admission hyperglycaemia is associated with shorter LOS.

Method
Venous BGLs from patients (≥18 years of age) admitted to the emergency department of a 350-bed tertiary referral hospital in Sydney, Australia, were extracted from the pathology database from 1 November 2011 to 31 May 2012 and measured on a Roche Modular analyser (Roche Diagnostics Australia, Castle Hill, Australia) using the hexokinase method.

Medical records of patients with an admission BGL ≥11.1mmol/L were reviewed. This cut-off level was chosen as it is consistent with the random BGL for diagnosis of diabetes mellitus.22 Ethics approval was granted by the St Vincent’s Hospital, Sydney (HREC LNR/12/SVH/58) and the University of Notre Dame Australia Human Research Ethics Committee (HREC012029S).

Patient information collected included age, sex, type, and duration of diabetes, comorbidities, pre-admission medications, treating specialty, ICU admission, documented in-hospital hypoglycaemia (defined as BGL<4.0mmol/L), and mortality. The primary outcome was LOS (days). Secondary outcomes included documented recognition of hyperglycaemia by medical staff as either definite (“hyperglycaemia” written in progress notes or BGL recorded with annotation—circling, underlining, or upward arrows) or possible (BGL recorded without annotation); average BGL in the first and last 24 hours of hospitalisation; HbA1c level (reported within six months prior to the patient’s admission date to hospital or during his/her hospital stay); presence and type of infections during hospitalisation; documented endocrinologist review for hyperglycaemia; presence and type of inpatient hyperglycaemia treatment, documented follow-up plan for diabetes care (recorded either in the progress notes or the discharge summary); and documented re-admission(s) into hospital. One investigator (TYC), not involved in patient care, collected and coded all data.

All patients with a diagnosis of diabetes mellitus prior to hospital admission were included in the study. A patient was classified as having prior history of diabetes if International Classification of Diseases Tenth Revision (ICD-10) codes E10-E14 were present in the medical files or if the word “diabetes” or abbreviations (T1DM, T2DM, IDDM, NIDDM) were written in the progress notes. Patients admitted more than once during the study period were assessed only on their first qualifying admission.

Patients were categorised into two groups: hyperglycaemia treated (HT) or untreated (HU). In-hospital treatment of
hyperglycaemia occurred if there was: (1) a change in dosage or type, or addition, of oral diabetes medication, insulin, or both to the pre-admission medication regimen; or (2) initiation of oral medication, insulin, or both in patients not taking diabetes medications pre-admission. No initiation or change of pre-admission diabetes medication was classified as “untreated”.

Statistical analyses
Statistical analyses were performed using SPSS version 22 (IBM SPSS, New York, NY). Baseline and in-hospital characteristics were compared with results expressed as medians with interquartile ranges (IQRs). Statistical significance was assessed using the Mann-Whitney U test for continuous variables and Fisher’s exact test for categorical variables, as variables were not normally distributed. Correlation between re-admission, follow-up, HbA1c level, and admission BGL were evaluated using Spearman’s rank correlation co-efficient. For all analyses \( p<0.05 \) was considered statistically significant.

To evaluate factors contributing to LOS, a stepwise multiple linear regression analysis was performed with the following variables: age, in-hospital hypoglycaemia, type of anti-hyperglycaemia treatment (i.e., insulin or oral hypoglycaemic agents [OHA]), in-hospital infections, admission BGL, and ICU admission. LOS data were log transformed prior to multiple regression analysis, due to positive skewing. Confidence intervals (CI) are expressed at 95%.

Results
Baseline characteristics
The hospital’s emergency department ordered 12,690 consecutive BGLs from 1 November 2011 to 31 May 2012, of which 7,827 persons had a first admission BGL (Figure 1). This encapsulated 84 per cent of separations during this time. Of these, 162 people admitted to hospital formed the study group. Eighty per cent were Caucasian, 61 per cent male, and the age range was 19 to 98 years. There were 95 in the HT group and 67 in the HU group (Figure 1).

The baseline characteristics of both groups are shown in Table 1. Compared to HU, patients in HT were significantly younger (median 70 vs. 75 years; \( p=0.03 \)), more commonly had type 1 diabetes (16 vs. 4.5 per cent, \( p=0.04 \)), and were taking insulin and glucocorticoids prior to admission (Table 1).

Of the 95 HT patients, 40 per cent were managed with short-term insulin interventions (supplemental insulin or insulin infusion) alone, and 50 per cent were treated with a combination of glucose-lowering interventions (i.e., insulin infusion, supplemental insulin, changes to pre-admission diabetes medication dosage, or addition of another diabetes medication). Those with an admission BGL >16mmol/L were more likely to be treated in the first 24 hours of admission (\( n=48, 51 \text{ per cent}; p<0.01 \), and with combination therapy (\( n=33, 35 \text{ per cent}; p<0.01 \)).

In-hospital outcomes
Data comparing in-hospital variables between HT and HU are shown in Table 2. Significantly more patients from the treated group were definitely noted to be hyperglycaemic compared to the untreated on admission (45 vs. 16 per cent, \( p<0.01 \)). This was 3.7 times more likely if the admission BGL was >16mmol/L (95% CI, 1.6–8.7, \( p<0.01 \)). In half (49 per cent) of the HU group, hyperglycaemia was not recognised (Table 2).

Interestingly, hyperglycaemia treatment was associated with a longer LOS than not receiving treatment (median eight vs. four days; \( p<0.01 \)). It should be noted that having diabetes itself is associated with a longer LOS overall, as illustrated by the shorter (median two days) LOS for the total patients admitted through the emergency department during the same seven-month period.

Infection during hospitalisation was 2.9 times more likely in HT than HU (95% CI, 1.5–5.7, \( p<0.01 \)), with skin and wound infections predominating (Table 2). Admission into ICU during hospitalisation was more common in HT compared with HU (20 vs. 1.5 per cent, \( p<0.01 \)).

While there was a significantly higher admission BGL and HbA1c level in patients HT compared to HU (Table 2), there was no difference in the average BGL 24 hours prior to discharge (\( p=0.12 \)). HT participants had a higher absolute difference and per cent reduction in BGL from admission to discharge compared to HU (both \( p<0.01 \)). No significant difference in hypoglycaemia incidence was found between groups (Table 2).

Although fewer than half (46 per cent) of the HT group were reviewed by an endocrinologist during hospitalisation, this was 18 times more likely than the HU group (95% CI, 5.4–63, \( p<0.01 \)). Where documented referral for endocrine review occurred, it was delayed by a median one day from the time of glucose measurement. Subset analysis between patients who were reviewed by the endocrinologist (\( n=47 \)) and those not (\( n=115 \)) identified higher admission BGL (median 19 vs. 14mmol/L, \( p<0.01 \)), higher HbA1c level
(median 10 vs. 7.8 per cent, *p*<0.01), higher likelihood of having follow-up plans (64 vs. 9.6 per cent, *p*<0.01), and longer LOS (median eight vs. four days, *p*<0.01).

**Outpatient outcomes**

Excluding those who died during admission (n=11, 6.8 per cent), only 27 per cent of the 151 patients had a documented follow-up plan for diabetes management after hospitalisation. Follow-up was more likely for HT patients (OR=4.6; 95% CI, 1.9–11; *p*<0.01) or those patients that received endocrine review (OR=17, 95% CI, 6.9–40, *p*<0.01). Hyperglycaemia treatment was also associated with a trend to fewer re-admissions in the short study time (18 vs. 30 per cent, *p*=0.09). The type of diabetes (type 1 or type 2) did not significantly impact hospital re-admission (27 vs. 16 per cent, *p*=0.46).

No statistically significant correlation was found between admission BGL and re-admission (*p*=0.11, *n*=162, *p*=0.17) or HbA1c level and re-admission (*p*=0.31, *n*=110, *p*=0.89). However, higher HbA1c was significantly related to more follow-up diabetes care (*p*=0.30, *n*=110, *p*<0.01) and a trend to lower re-admission was correlated in those diabetic patients with follow-up care plans (*p*=1.41, *n*=162, *p*=0.07).

**Stepwise multiple regression analysis of LOS**

Seven variables (age, in-hospital glycaemia, type of anti-hyperglycaemic treatment—insulin or OHA, in-hospital infections, admission BGL, and ICU admission) were entered into a stepwise multiple linear regression model, and four variables were retained (age, in-hospital glycaemia, inpatient insulin treatment, and in-hospital infections), which together explain 20 per cent of the variance in LOS ($R^2_{(adj)}=0.20$, *p*<0.01). Hypoglycaemia contributed 11 per cent, inpatient insulin treatment 5.6 per cent, and infections 2.3 per cent of the variance in LOS (Table 3).

**Discussion**

Emergency hospital admission provides an opportunity to assess and improve diabetes control. Despite this, almost half (49 per cent) of the HU group in our study did not have admission hyperglycaemia recognised and only 45 per cent of those treated (HT) were definitely recognised. Compared to the borderline high median admission BGL (13mmol/L) in the HU cohort, the significantly higher median admission BGL (17mmol/L) in the treated appears to be the trigger for recognition and inpatient treatment of admission hyperglycaemia. Furthermore, the presence of more intensive pre-admission diabetic intervention (i.e., on insulin) and hyperglycaemia-triggering medications (i.e., glucocorticoids) in our HT group may also trigger earlier recognition and treatment of admission hyperglycaemia by medical staff.

Hospital hyperglycaemia is an important marker of chronically poor glycaemic control. Participants with more marked hyperglycaemia (median admission BGL 17mmol/L, discharge BGL 10mmol/L) also had poor diabetes control (HbA1c 9.0 per cent), although similar discharge BGLs were achieved as in those with better, prior long-term glycaemic control (discharge BGL 9.9mmol/L, HbA1c 7.3 per cent). A longer LOS was also observed in this group, although the median LOS (eight vs. four days) for both HT and HU groups, respectively, were longer than the general hospital population (two days). Longer LOS for those with diabetes and/or hyperglycaemia are well documented and is multifactorial.

We identified age, hypoglycaemia, inpatient insulin treatment, and infection as contributors to 20 per cent of the variance. The effect of treatment in our study cohort is confounded by the known detrimental associations with hyperglycaemia itself. Hyperglycaemic patients are likely to be more sick, with higher ICU admissions, mortality, and infection rates, confirmed in our HT cohort.

The association between hypoglycaemia incidence and LOS has previously been described, where mild-to-moderate hypoglycaemia (BGL 2.3–3.9mmol/L) and severe hypoglycaemia (BGL≤2.2mmol/L) were associated with a 51 per cent and 133 per cent increase in LOS, respectively. It is possible that treatment of hyperglycaemia itself was associated with a longer LOS, or conversely, provided a longer timeframe to observe hypoglycaemia occurrence, as when corrected for LOS the number of observed hypoglycaemic events between groups was not statistically different. The higher rates of recorded hypoglycaemia may also reflect the common hospital use of supplemental insulin as sole corrective therapy, and also that patients receiving such therapy may undergo more frequent monitoring of glucose measurements.

Achieving target BGLs with intensive glycaemic control in an acute hospital setting can be harmful with adverse outcomes, such as severe hypoglycaemia and increased mortality risk. It is important to avoid hypoglycaemia in hospital patients, as prior hypoglycaemic exposure attenuates cardiovascular autonomic control (specifically baroreflex sensitivity and sympathetic nervous system response) to hypotensive stress, and this may be amplified in patients with diabetes. Furthermore, Pezzella et al. demonstrated that more liberal in-hospital glycaemic
control (6.7–10.0 mmol/L) in coronary artery bypass graft (CABG) patients with and without diabetes had similar long-term outcomes (i.e., survival and/or improved health-related quality of life) as those who had strict glycaemic control (5.0–6.6 mmol/L). Such work supports an aim in hospital to avoid hyperglycaemia, rather than to achieve tight glycaemic control.

Our findings emphasise that recognition of hyperglycaemia in hospitalised patients should help in identifying those with chronically poor glycaemic control (i.e., higher HbA1c levels) who would benefit from planned community intervention and for whom longer term follow-up is best. The American Diabetes Association (ADA) and the European Association of the Study of Diabetes (EASD) recommend a patient-centred approach to diabetes care, with continued monitoring of individualised glycaemic targets and response to glucose-lowering therapies in a community setting. Our study reveals an alarmingly low rate of diabetes team follow-up post-discharge (27 per cent), suggesting poor continuity in complex patient care.

The majority of follow-up diabetes care was significantly correlated with worsening chronic glycaemia. Timely (within seven days) and multidisciplinary outpatient follow-up care should be targeted at those patients with the highest clinical complexity (in our study, identified by worse prior glycaemia) and therefore highest underlying risk of re-admission. Although there was no correlation between chronic glycaemia and re-admissions, there was a trend towards fewer re-admissions in those diabetic patients who had follow-up care plans. Further studies evaluating the benefits of long-term follow-up (≥1 year) are clearly required, particularly assessing whether improvements in HbA1c levels led to beneficial outcomes (i.e., reduced hospital re-admissions, mortality, infection rate) in those diabetic patients who received appropriate follow-up care compared to those without.

Optimising diabetes care follow-up amongst poorly controlled glycaemic patients requires the provision of services that specifically address their needs. This necessitates a close collaboration between the diabetes teams at the local hospital and the community general practitioners (GPs). The establishment of Primary Health Networks (PHNs) in Australia, effective July 2015, may help to achieve this with the long-term aim of reducing unnecessary hospitalisations and re-admissions.

Identifying suboptimal glycaemic control in the inpatient setting could be initiated with dedicated diabetes teams; this has previously been shown to improve glycaemic control by increasing recognition rates and reducing delay in initiating therapy. Such review could be triggered by admission hyperglycaemia rather than a formal referral, which, when it occurred at all in our study, occurred at a median one day after admission. Furthermore, follow-up plans were 17 times more likely after endocrine consultation, with the median HbA1c level in the referred group being 10 per cent. Continuing communication between diabetes teams at local hospitals and GPs is essential for good long-term glycaemic control. Previous work has demonstrated the benefits of better long-term glycaemic control when GPs and diabetes teams communicated effectively and worked together to provide diabetes care.

In order to reduce the risk of hospital re-admission among a diabetic cohort with multiple comorbidities and elevated HbA1c levels, it is important that the PHNs provide a multidisciplinary, disease-specific service. Intensive, generalised primary care follow-up is not recommended, given prior data suggesting that this results in increased re-admissions within a medically vulnerable population (i.e., the elderly). Further prospective studies focusing on the duration and nature of follow-up care among chronically poor glycaemic patients should be studied.

There are limitations to our study. This was a retrospective, non-randomised study conducted in a single centre. Our capacity to definitively assess and comment on long-term outcomes for HT and HU cohorts was likely affected by the relatively short follow-up period. However, being an observational study, we could detect an unacceptably high “threshold” for recognition, treatment (and endocrine consultation) in regard to admission BGL.

**Conclusion**

This study has identified a longer LOS in diabetic patients treated for admission hyperglycaemia. While this may seem paradoxical, it is likely explained by factors suggesting such patients are unwell, with a higher infection rate, ICU admissions, and higher initial HbA1c levels. Results also show that adjustment to diabetes treatment produced a significantly higher absolute and per cent reduction from average admission to discharge BGLs, but with an apparently low percentage of follow-up diabetes care.

Although observational, our findings are consistent with previous randomised studies—that targeting tight glucose control in hospital may not be beneficial. We found that endocrine consultation and community follow-up in
chronically hyperglycaemic subjects was low but was correlated with elevated HbA1c levels, and there was a trend towards fewer re-admissions with follow-up diabetes care.

Given the findings of this work, a priority for PHNs is to provide an integrated multidisciplinary diabetes service for the at-risk diabetic population (with chronic glycaemia) with the possibility of further reducing hospital re-admissions. Further studies will determine whether the presence of PHNs and the priority for obligatory in-hospital endocrine assessment coupled with a community management plan in patients with known diabetes improves long-term outcomes.

References


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We would like to thank Dr Graham Jones, head of Chemical Pathology at St Vincent’s Hospital, Sydney, for his assistance with extraction of the admission venous BGLs from the hospital’s pathology database.

PEER REVIEW
Not commissioned. Externally peer reviewed.

CONFLICTS OF INTEREST
The authors declare that they have no competing interests.

ETHICS COMMITTEE APPROVAL
St Vincent’s Hospital Sydney HREC LNR/12/SVH/58
The University of Notre Dame Australia HREC 012029S
Figure 1: Schematic presentation of participant selection

7827 patients with first admission venous BGL
(1 November 2011 – 31 May 2012)

Admission venous BGL ≥ 11.1mmol/L
425/7827 (5.4%)

Admission venous BGL < 11.1mmol/L
7401/7827 (95%)

390 patient files reviewed
390/426 (92%)

36 patient files not found
36/426 (8.6%)

Prior diagnosis of DM
283/390 (73%)

No prior diagnosis of DM
101/390 (26%)

Missing notes
6/390 (1.5%)

Included
162/283 (57%)

Excluded
121/283 (43%)

Hyperglycaemia treated (HT)
95/162 (59%)

Hyperglycaemia untreated (HU)
67/162 (41%)

Discharged from ED
94/121 (78%)

Transferred to other hospitals
27/121 (22%)

BGL: Blood glucose level; DM: Diabetes mellitus; ED: Emergency Department
Table 1: Baseline study characteristics

<table>
<thead>
<tr>
<th></th>
<th>Hyperglycaemia Treated (HT)</th>
<th>Hyperglycaemia Untreated (HU)</th>
<th>( p ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( n=95^1 )</td>
<td>( n=67^2 )</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Age (years)</td>
<td>70 (54 – 79)</td>
<td>75 (61 – 86)</td>
<td>0.03*</td>
</tr>
<tr>
<td>Male</td>
<td>58</td>
<td>41</td>
<td>61%</td>
</tr>
<tr>
<td>Ethnicity</td>
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<td></td>
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</tr>
<tr>
<td>Caucasian</td>
<td>77</td>
<td>52</td>
<td>81%</td>
</tr>
<tr>
<td>Admitting team</td>
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</tr>
<tr>
<td>Medical</td>
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<td>48</td>
<td>68%</td>
</tr>
<tr>
<td>Surgical</td>
<td>28</td>
<td>17</td>
<td>30%</td>
</tr>
<tr>
<td>Psychiatry</td>
<td>2</td>
<td>2</td>
<td>2.1%</td>
</tr>
<tr>
<td>Co-morbidities</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Macrovacular</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IHD</td>
<td>36</td>
<td>26</td>
<td>39%</td>
</tr>
<tr>
<td>CVA</td>
<td>9</td>
<td>6</td>
<td>9.7%</td>
</tr>
<tr>
<td>PVD</td>
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<td>2</td>
<td>9.7%</td>
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<tr>
<td>Microvascular</td>
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</tr>
<tr>
<td>Neuropathy</td>
<td>17</td>
<td>11</td>
<td>18%</td>
</tr>
<tr>
<td>Nephropathy</td>
<td>17</td>
<td>11</td>
<td>18%</td>
</tr>
<tr>
<td>Retinopathy</td>
<td>9</td>
<td>3</td>
<td>9.7%</td>
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<tr>
<td>Other comorbidities</td>
<td>88</td>
<td>63</td>
<td>93%</td>
</tr>
<tr>
<td>Type of DM</td>
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<td>0.04*</td>
</tr>
<tr>
<td>Type 1</td>
<td>15</td>
<td>3</td>
<td>16%</td>
</tr>
<tr>
<td>Type 2</td>
<td>80</td>
<td>64</td>
<td>84%</td>
</tr>
<tr>
<td>Duration of DM (years)</td>
<td>16 (6–21)</td>
<td>12 (4–18.5)</td>
<td>0.09</td>
</tr>
<tr>
<td>Pre-admission treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifestyle only</td>
<td>7</td>
<td>4</td>
<td>7.4%</td>
</tr>
<tr>
<td>Oral agents</td>
<td>50</td>
<td>52</td>
<td>53%</td>
</tr>
<tr>
<td>Insulin</td>
<td>59</td>
<td>19</td>
<td>62%</td>
</tr>
<tr>
<td>Other medications</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ACE inhibitor</td>
<td>23</td>
<td>26</td>
<td>25%</td>
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<tr>
<td>ARB</td>
<td>28</td>
<td>17</td>
<td>30%</td>
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<tr>
<td>Statin</td>
<td>48</td>
<td>44</td>
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</tr>
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<td>Glucocorticoid</td>
<td>28</td>
<td>10</td>
<td>30%</td>
</tr>
<tr>
<td>Current smoker</td>
<td>17</td>
<td>8</td>
<td>20%</td>
</tr>
</tbody>
</table>

1 Numbers for some categories do not sum to 95 due to missing data.
2 Numbers for some categories do not sum to 67 due to missing data.
*Significant at \( p<0.05 \).

HT: Hyperglycaemia treatment; HU: Hyperglycaemia untreated; IHD: Ischaemic heart disease; CVA: Cerebrovascular accident; PVD: Peripheral vascular disease; DM: Diabetes mellitus; ACE: Angiotensin converting enzyme; ARB: Angiotensin receptor blocker.
Table 2: Comparison of in-hospital outcomes between HT and HU groups

<table>
<thead>
<tr>
<th>Category</th>
<th>Hyperglycaemia Treated (HT)</th>
<th>Hyperglycaemia Untreated (HU)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission BGL</td>
<td>17 (13–21)</td>
<td>13 (12–15)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Average BGL in first 24 hr</td>
<td>14 (12–16)</td>
<td>11 (9.5–12)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Average BGL in last 24 hr</td>
<td>10 (8.7–12)</td>
<td>9.9 (7.6–12)</td>
<td>0.12</td>
</tr>
<tr>
<td>24hr admission and discharge BGL difference</td>
<td>3.1 (1.0–5.8)</td>
<td>0.5 (0–2.9)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>% Reduction in admission to discharge BGL</td>
<td>23 (7.3–40)</td>
<td>5.0 (0–29)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>9.0 (7.8–11)</td>
<td>7.3 (6.6–8.4)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Admission hyperglycaemia recognition – n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not recognised</td>
<td>24 (25%)</td>
<td>33 (49%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Definitely recognised</td>
<td>43 (45%)</td>
<td>11 (16%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Possibly recognised</td>
<td>28 (30%)</td>
<td>23 (34%)</td>
<td>0.61</td>
</tr>
<tr>
<td>In-hospital hypoglycaemia – n (%)</td>
<td>19 (20%)</td>
<td>7 (10%)</td>
<td>0.13</td>
</tr>
<tr>
<td>Hypoglycaemia incidence (events per 100 person-days)</td>
<td>4.0 ± 9.0</td>
<td>3.0 ± 11</td>
<td>0.68</td>
</tr>
<tr>
<td>DKA on admission – n (%)</td>
<td>9 (9.5%)</td>
<td>0 (0.0%)</td>
<td>0.01*</td>
</tr>
<tr>
<td>In-hospital infections – n (%)</td>
<td>47 (50%)</td>
<td>17 (25%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Skin/Wound</td>
<td>14 (15%)</td>
<td>0 (0.0%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Sepsis/Bacteraemia</td>
<td>9 (9.5%)</td>
<td>6 (9.0%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Other</td>
<td>24 (25%)</td>
<td>11 (16%)</td>
<td>0.25</td>
</tr>
<tr>
<td>In-hospital mortality – n (%)</td>
<td>9 (9.5%)</td>
<td>2 (3.0%)</td>
<td>0.13</td>
</tr>
<tr>
<td>ICU admission – n (%)</td>
<td>19 (20%)</td>
<td>1 (1.5%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Endocrinology review – n (%)</td>
<td>44 (46%)</td>
<td>3 (4.5%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Length of stay (days)</td>
<td>8.0 (3–15)</td>
<td>4.0 (2–8)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Diabetes follow-up – n (%)</td>
<td>33 (35%)</td>
<td>7 (10%)</td>
<td>&lt;0.01*</td>
</tr>
<tr>
<td>Re-admissions – n (%)</td>
<td>17 (18%)</td>
<td>20 (30%)</td>
<td>0.09</td>
</tr>
</tbody>
</table>

1 Numbers for some categories do not sum to 95 due to missing data.
2 Numbers for some categories do not sum to 67 due to missing data.
3 Compared using Welch’s t-test; reported as mean ± SD.
4 Other infections include pulmonary (pneumonia), urinary tract (urinary tract infections, pyelonephritis), or gastrointestinal (gastroenteritis).
*Significant at p<0.05.

HT: Hyperglycaemia treated; HU: Hyperglycaemia untreated; BGL: Blood glucose level; hr: hours; HbA1c: Glycosylated haemoglobin; DKA: Diabetic ketoacidosis; ICU: Intensive care unit.
Table 3: Stepwise multiple regression analysis evaluating factors contributing to LOS\(^1\) (n=160)

<table>
<thead>
<tr>
<th>Model variables</th>
<th>R(^2)</th>
<th>Adjusted R(^2), (^2)</th>
<th>R(^2) Change</th>
<th>p value for R(^2) change</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital hypoglycaemia</td>
<td>0.106</td>
<td>&lt;0.01*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inpatient Insulin treatment</td>
<td>0.056</td>
<td>&lt;0.01*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.034</td>
<td>&lt;0.01*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In-hospital infection</td>
<td>0.023</td>
<td>&lt;0.01*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^1\)Dependent variable: LOS (log transformed due to positive skewing).

\(^2\)Variables such as in-hospital hypoglycaemia, inpatient insulin treatment, in-hospital infection, and age were included in the final model.

\(^3\)Adjusted for age, in-hospital hypoglycaemia, type of anti-hyperglycaemic treatment—insulin or oral hypoglycaemic agents, in-hospital infections, admission blood glucose level, and intensive care unit admission.

*p<0.05 indicates the change in R\(^2\) is statistically significant.

LOS: Length of hospital stay