Meta-analysis of admission hyperglycaemia in acute myocardial infarction patients treated with primary angioplasty: a cause or a marker of mortality?

Kuljit Singh1,2*, Benjamin Hibbert1, Balwinder Singh3, Kristin Carson2, Manuja Premaratne1, Michel Le May1, Aun-Yeong Chong1, Margaret Arstall4, and Derek So1

1University of Ottawa Heart Institute, Ottawa, ON, Canada K1Y 1J7; 2Basil Hetzel Institute, University of Adelaide, Adelaide, SA 5000, Australia; 3Department of Clinical Neurosciences, University of North Dakota School of Medicine & Health Sciences, Fargo, ND, USA; and 4Department of Cardiology, Lyell McEwin Hospital, University of Adelaide, Adelaide, SA 5000, Australia

Received 31 January 2015; revised 21 April 2015; accepted 21 April 2015; online publish-ahead-of-print 29 April 2015

Aims

Admission hyperglycaemia (AH) has been associated with worse outcomes in acute myocardial infarction (AMI). In the current review, we evaluated the impact of primary angioplasty (pPCI) on mortality in AMI patients with AH. Our second aim was to evaluate if AH is a marker of baseline risk or an independent predictor of mortality.

Methods and results

A comprehensive search of four major databases was performed. We included original research studies reporting data on mortality in AMI patients with AH (mean plasma glucose >156 mg/dL/8.7 mmol) and euglycaemia who were treated with pPCI. Of 481 citations, 12 studies were included in the analysis. Admission hyperglycaemia was associated with a higher 30-day [risk ratio (RR) 4.30, \( P < 0.0001 \)] and 1- to 3-year mortality (RR 2.26, \( P < 0.0001 \)). As well, AH was more prevalent in women and in patients with an increasing number of cardiac risk factors or angiographic predictors of mortality, such as previous AMI (RR 0.89, \( P = 0.01 \)), multivessel coronary disease (RR 0.72, \( P < 0.0001 \)), and involvement of left anterior descending artery (RR 0.92, \( P < 0.0001 \)). Moreover, patients with AH had larger infarcts (higher creatine kinase-MB; \( P = 0.004 \)) and more frequent ventricular arrhythmias (\( P = 0.002 \)).

Conclusion

Despite rapid revascularization and treatment of hyperglycaemia, patients with AH continue to have a higher mortality. Admission hyperglycaemia occurs more commonly in patients who have traditional predictors of worse outcomes—specifically prior infarction, anterior wall infarctions, and multivessel disease. Likely, AH is a predictor of rather than a bona fide therapeutic target in AMI.

Keywords

Mortality • Admission hyperglycaemia • Acute myocardial infarction

Introduction

Admission hyperglycaemia (AH) is a common finding among patients diagnosed with acute myocardial infarction (AMI) and is an independent predictor of short- and long-term morbidity.1,2 A meta-analysis nearly 15 years ago demonstrated an association between AH and in-hospital mortality.3 However, the management of AMI in the last 15 years had undergone significant changes. The predominant use of primary angioplasty (pPCI) over thrombolysis, reduction in the door to balloon time, and use of newer anti-platelet therapy have improved survival.4–7 However, AH is still thought to identify patients at an increased risk of mortality in this era of rapid reperfusion.

In an attempt to abrogate the increased mortality from AH, coronary care units have implemented insulin protocols to normalize glucose levels during AMI.8 However, treatment with insulin itself has shown mixed results.9 Data from small pilot studies, moderate-sized clinical

* Corresponding author. Tel: +1 613 276 4884; Fax: +1 613 276 4919; Email: kjaulakh@gmail.com

Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2015. For permissions please email: journals.permissions@oup.com
Admission hyperglycaemia in myocardial infarction

trials, and a meta-analysis suggested benefits of treatment with insulin.\textsuperscript{8,10} However, subsequent larger randomized control trials have failed to confirm improved survival.\textsuperscript{9,11} Similar to the impact of AH on mortality, the role of treatment with insulin in the context of contemporary revascularization remains unclear.

Thus, in the current study, we sought to conduct a systematic review to evaluate the 30-day and 1- to 3-year mortality in ST segment myocardial infarction (STEMI)/AMI patients treated with pPCI that presented with AH. We also sought to evaluate if AH is simply a marker of poor prognosis or a causal agent by examining the association of AH with prior established markers of adverse outcome such as previous infarct, presence of multivessel coronary artery disease, and anterior wall myocardial infarction.

Methods

Our systematic review and meta-analysis are reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement guidelines.\textsuperscript{12}

Study eligibility

We included comparative studies of any design (randomized control trials, cohort, case control, and cross-sectional). We only included studies where pPCI was the mode of reperfusion of AMI. Studies recruiting patients with unstable angina, non-ST-elevation MI, or where mode of revascularization was thrombolysis, were excluded. Eligible studies had to report mortality in AMI patients diagnosed with and without AH. Furthermore, only studies with >100 patients were considered eligible. Inclusion was restricted to publications in the English language or when translation of the foreign language was provided or could be easily achieved with Google translator. When data were reported from overlapping study samples (e.g. multiple publications from the same group), the most recent study or one with the highest number of patients was included in the analysis. Single case reports, editorials, and previous systematic reviews were not included.

A separate search was conducted to find studies reporting mortality and comparing use of intensive vs. standard use of insulin in AMI patients with AH. However, unlike the initial search, we included studies reporting AMI with AH irrespective of the mode of revascularization. Only studies comparing strict vs. standard blood sugar control using intensive or standard insulin protocols were included in the analysis. We included comparative studies of any design (randomized control trials, cohort, case control, and cross-sectional) and inclusion was limited to studies in English language.

Data sources and search strategy

A comprehensive search strategy was designed and a thorough computer-based search was performed using OVID MEDLINE, EMBASE, Google Scholar, and PubMed databases. No time limit to start date was applied and the search was conducted up to 31 July 2014. We reviewed both the identified manuscripts and references cited or citing indexed studies.

Study selection and data extraction

Two reviewers (K.S.) and (B.S.) screened all the titles and abstracts independently. This was followed by the full-text review of the selected articles by the same reviewers. We then extracted the data from selected studies using a standardized, pilot-tested extraction template. The following data were extracted: study characteristics (author, year of publication, country, study design, study population, number of participants, and objective of the study), participant characteristics (age and gender), clinical characteristics, cardiac risk factors, information on mortality (30-day and long-term during follow-up), and angiographic findings.

Quality assessment

We assessed the quality of the included studies using a subset of Tooth et al., the article titled ‘Quality of Reporting of Observational Longitudinal Research’, including only the 33 quality domains relevant to meta-analysis of the observational studies. We assessed the biases using classification of ‘low risk of bias’ when data for criterion were reported, ‘high risk of bias’ when data were not reported, and ‘unclear risk of bias’ when data for criterion were not relevant to the study design. Consensus or involvement of a third reviewer resolved disagreement between reviewers for classification.

Subset analysis

A subgroup analysis was performed in case high heterogeneity, among studies, was found during analysis. Subgroup analysis was performed according to study design, blood sugar level used to define hyperglycaemia (above and below 190 mg/dL, 10.6 mmol/L), and whether it was a single-centre or multicentre study.

Statistical analysis

Continuous variables were reported as means ± standard deviation (SD), whereas skewed data were described as medians ± interquartile range. The measure for estimating the common effect across the included studies was the risk ratio (RR) for binary outcomes and the mean difference for continuous outcomes. When the outcome was rare, we used odds ratio (OR) instead of RR. Heterogeneity between studies was assessed by combination of I\textsuperscript{2} statistic, Cochran’s Q test, and observation of the data for each outcome. Subgroup analysis was performed to find the cause of heterogeneity where I\textsuperscript{2} > 60%. The random-effects model was used in case of significant heterogeneity and the fixed-effects model was used when heterogeneity was low. If formal meta-analysis was not possible due to skewed distribution of the number of patients between each study, we disregarded the individual studies and used data as if obtained from a single study. A significant interaction between variables was considered when P ≤ 0.05. All calculations were performed using the meta-analysis software, Review Manager Version 5.2 Cochrane Collaboration.

Results

Literature identification

The literature search yielded 478 citations (Figure 1). An additional three citations were found through review of references and citing articles. Abstracts were reviewed for 481 short-listed citations and 151 articles were chosen for full-text review. Of the 151 full-text manuscripts reviewed for eligibility, 38 reported mortality in an AH group with 12 studies involving patients treated with pPCI.\textsuperscript{1,14–23} These 12 studies fulfilled all inclusion and exclusion criteria and were thus included in the final analysis.

Clinical characteristics

Among the 12 studies, 6 were prospective and the remainder had a retrospective design. The sample size ranged from 252 to 4698, with a total of 20 573 patients. All the patients included in the analysis had ST-elevation myocardial infarction. Mean age of patients diagnosed with AMI was 63 years (± 4.5 years). Among the whole cohort of patients, 65% were men and 23% had known diabetes
before developing AMI. The information of new diagnosis of diabetes post AMI was not available. The threshold blood sugar level (BSL) for diagnosis of hyperglycaemia varied among the studies. The average BSL separating AH from euglycaemia (EG) was 156 mg/dL (95% CI 134–156 mg/dL). The number of patients diagnosed with AH was 6938 (45%), and 12 549 (55%) of patients were labelled as EG patients. Among the patients diagnosed with AH, only 38% had a diagnosis of diabetes. The cardiac risk factors and details of the studies are described in Table 1 and 2.

**Mortality**

There were 724 deaths in the first 30 days, of which 512 occurred in the AH group. The 30-day mortality in patients diagnosed with AH group was significantly higher than that of the EG group (RR 4.30, 95% CI 3.64–5.09, P < 0.0001, I² = 5%, Figure 2). Of the 12 studies, 3 studies enrolled exclusively non-diabetic patients with AH. We performed a separate analysis of these three studies and found a markedly higher mortality in non-diabetic patients presenting with AH (RR 4.75, 95% CI 2.64–8.55, P < 0.0001, I² = 62%, Figure 3). Similarly, the
<table>
<thead>
<tr>
<th>No.</th>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Patient no.</th>
<th>BSL mg/dL (mmol/L)</th>
<th>Patients with AH</th>
<th>Hypertension numbers</th>
<th>High cholesterol numbers</th>
<th>Smoking numbers</th>
<th>Women numbers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Non-DM</td>
<td>DM</td>
<td>EG</td>
<td>AH</td>
<td>EG</td>
</tr>
<tr>
<td>1</td>
<td>Ekmekci</td>
<td>2013</td>
<td>Italy</td>
<td>677</td>
<td>168 (9.3)</td>
<td>174</td>
<td>46</td>
<td>257</td>
<td>133</td>
<td>118</td>
</tr>
<tr>
<td>2</td>
<td>Planer</td>
<td>2013</td>
<td>USA</td>
<td>3405</td>
<td>156 (8.6)</td>
<td>704</td>
<td>437</td>
<td>1144</td>
<td>671</td>
<td>934</td>
</tr>
<tr>
<td>3</td>
<td>Hoebers</td>
<td>2011</td>
<td>Netherlands</td>
<td>1646</td>
<td>140 (7.7)</td>
<td>725</td>
<td>174</td>
<td>227</td>
<td>285</td>
<td>158</td>
</tr>
<tr>
<td>4</td>
<td>Timmer</td>
<td>2011</td>
<td>Netherlands</td>
<td>4698</td>
<td>145 (8)</td>
<td>NA</td>
<td>NA</td>
<td>654</td>
<td>709</td>
<td>432</td>
</tr>
<tr>
<td>5</td>
<td>Marenzi</td>
<td>2010</td>
<td>Italy</td>
<td>780</td>
<td>190 (11)</td>
<td>74</td>
<td>74</td>
<td>303</td>
<td>73</td>
<td>281</td>
</tr>
<tr>
<td>6</td>
<td>Ergelen</td>
<td>2010</td>
<td>Turkey</td>
<td>2482</td>
<td>200 (11)</td>
<td>64</td>
<td>341</td>
<td>772</td>
<td>200</td>
<td>704</td>
</tr>
<tr>
<td>7</td>
<td>Lazzari</td>
<td>2009</td>
<td>Italy</td>
<td>252</td>
<td>140 (7.7)</td>
<td>101</td>
<td>0</td>
<td>65</td>
<td>54</td>
<td>58</td>
</tr>
<tr>
<td>8</td>
<td>Usami</td>
<td>2009</td>
<td>Japan</td>
<td>2433</td>
<td>190 (11)</td>
<td>231</td>
<td>0</td>
<td>1188</td>
<td>119</td>
<td>919</td>
</tr>
<tr>
<td>9</td>
<td>Gasior</td>
<td>2008</td>
<td>Poland</td>
<td>1310</td>
<td>140 (7.7)</td>
<td>378</td>
<td>289</td>
<td>260</td>
<td>431</td>
<td>336</td>
</tr>
<tr>
<td>10</td>
<td>Worthley</td>
<td>2007</td>
<td>Canada</td>
<td>980</td>
<td>140 (7.7)</td>
<td>345</td>
<td>133</td>
<td>199</td>
<td>221</td>
<td>NA</td>
</tr>
<tr>
<td>11</td>
<td>Lavi</td>
<td>2008</td>
<td>Israel</td>
<td>431</td>
<td>126 (7)</td>
<td>119</td>
<td>88</td>
<td>74</td>
<td>79</td>
<td>75</td>
</tr>
<tr>
<td>12</td>
<td>Chen</td>
<td>2014</td>
<td>Taiwan</td>
<td>959</td>
<td>140 (7.7)</td>
<td>271</td>
<td>271</td>
<td>157</td>
<td>189</td>
<td>93</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td></td>
<td></td>
<td>20053</td>
<td></td>
<td>3061</td>
<td>185</td>
<td>3500</td>
<td>3164</td>
<td>4108</td>
</tr>
</tbody>
</table>

BSL, blood sugar level; AH, admission hyperglycaemia; EG, euglycaemia; DM, diabetes mellitus.
number of deaths in the long-term follow-up stayed higher in the AH group (RR 2.26, 95% CI 1.73–2.94, \( P < 0.0001 \), \( I^2 = 63\% \), Figure 4). Notably, there was significant heterogeneity in the long-term mortality studies. Accordingly, we performed a subgroup analysis according to the length of follow-up (1 vs. 3 years) and found heterogeneity to be lower among studies reporting follow-up of a year.

**Treatment strategy for hyperglycaemia**

Two studies compared the treatment of hyperglycaemia with a strict and standard regimen of insulin in AMI patients treated with pPCI. These studies randomized patients with AH to intense insulin treatment vs. standard treatment following pPCI. Significant differences in inclusion criteria between the studies precluded a formal meta-analysis. Both of these studies were small and failed to show benefits of aggressive hyperglycaemia management.

**AH is an independent predictor or just another marker of high morbidity/mortality?**

Analysis was performed to see if clinical and angiographic predictors related to poorer prognosis [gender, previous history of AMI, multi-vessel coronary artery disease, and left anterior descending artery (LAD) infarction] were more common in the AH subgroup. Men tended to have relatively lower incidence of AH during AMI (OR 0.56, 95% CI 0.53–0.60, \( P < 0.0001 \), \( I^2 = 95\% \), see Supplementary material online, Figure S1). Furthermore, patients with AH were more likely to have suffered a previous MI (RR 0.89, 95% CI 0.81–0.98, \( P = 0.01 \), see Supplementary material online, Figure S2), have multi-vessel coronary disease (RR 0.72, 95% CI 0.61–0.85, \( P = 0.0001 \), see Supplementary material online, Figure S3), and had a higher incidence of LAD infarcts (RR 0.92, 95% CI 0.89–0.95, \( P < 0.0001 \), see Supplementary material online, Figure S4). There was no difference between TIMI III flow in the culprit artery post pPCI in the AH and EG cohort (\( P = 0.17 \), see Supplementary material online, Figure S5). Finally, AH was associated with a larger infarct size in five studies (\( n = 5157 \)) that reported peak creatine kinase-MB (mean difference 47.45, 95% CI 15.31–79.59, \( P = 0.004 \), \( I^2 = 91\% \), see Supplementary material online, Figure S6), and a higher number of ventricular arrhythmias (RR 2.74, 95% CI 1.44–5.22, \( P = 0.002 \), see Supplementary material online, Figure S7). Retrospective, prospective, single-centre and multi-centre studies showed similar heterogeneity.

**Quality assessment**

Overall, the study quality was good with 82% of all studies reporting a low risk of bias (see Supplementary material online, Figures S8 and S9). Eight criteria which were not reported included justification of the
number of included participants, reasons for non-inclusion of subjects, reporting of confounders, accounting for confounders in the analyses, accounting for missing data in the analyses, impact of bias assessed qualitatively, comparison of consenters with non-consenters, reasons for non-consent, and impact of bias assessed quantitatively.

Discussion

Admission hyperglycaemia in a contemporary era of rapid revascularization demonstrated a strong association with mortality and this association was maintained for both short- and long-term follow-up. Importantly, our study highlights that using pPCI did not abrogate the mortality impact of AH when compared with patients with EG. Notably, AMI patients who develop AH tend to have more cardiac risk factors such as hypertension, high cholesterol, smoking, or diabetes (Table 1). Not only that, these patients had relatively unfavourable coronary anatomy with severe coronary vessel disease, higher frequency of LAD occlusion, and higher rates of previous myocardial infarction. All these factors contributed to larger infarct size and higher events of ventricular arrhythmias in the AH subgroup.

Independent of the presence of diabetes, AH is a common finding among patients presenting with AMI. Indeed, reported incidence varies from 10 to 40% depending on the definition of hyperglycaemia used—with numerous studies identifying an association with poor outcome. However, the impact of AH with modern therapy, specifically pPCI, was previously not evaluated; thus, we performed an updated analysis and found that AH remains a strong predictor of mortality. Short-term mortality in patients with AH was nearly four times that of EG patients. One important finding of our analysis was that the survival difference between AH and EG patients remained significantly persistent even at long-term follow-up. These findings echo the higher mortality reported in the thrombolytic data, suggesting that despite a reduction in overall mortality, an AH associated gap is persistent. One possible explanation of this observation could be the presence of undiagnosed diabetes leading to advanced coronary artery disease—although none of the studies presented long-term follow-up on the development of diabetes mellitus. Longitudinal studies assessing the metabolic profiles of patients presenting with AH are needed to determine, if indeed, this hypothesis is correct.

Secondarily, we evaluated whether the clinical risk profile or angiographic findings differed between AH and EG patients. Our analysis suggested that AH occurs in patients who end up having larger infarcts and ventricular arrhythmias because of the presence of multivessel disease, anterior wall infarction, and/or a history of prior myocardial infarction—all established markers of increased risk. Thus, the presence of AH in this cohort is unlikely a major contributor to increased mortality, but rather a harbinger of increased infarct size and more extensive coronary disease. AH in such cases can be considered as a marker of worse outcomes similar to other non-traditional predictors such as leucocytes and C-reactive protein.

It has been suggested that AH can impart detrimental effects at the cellular level leading to increased myocardial injury. Hyperglycaemia abolishes ischaemic pre-conditioning by modulating the interactions between G1 proteins, adenosine receptors, nitric oxide, and KATP channels.

Figure 4 A subgroup analysis showing the presence of higher number of death in the admission hyperglycaemia group during long-term follow-up.
Indeed, in the NICE SUGAR trial, the conventional trial demonstrated higher mortality when BSL was kept under BSL under 110 mg/dL (6.1 mmol/L), findings of the NICE SUGAR (8 mmol/L). Studies of less stringent targets in patients with arm had improved outcomes at a more modest BSL of 145 mg/dL Berghe tion of how stringent glucose target need to be. While van den trials of critically ill patients demonstrated higher rates of death in the various trials of aggressive glycaemic control. More recently, inflammation and a reduction in differentiation of endothelial progenitor cells with tighter glycaemic control.6,37 However, in our analysis, studies of tighter glycaemic control did not translate into improved outcomes leaving an evidence gap between the potentially beneficial effects seen in preclinical models and the findings in clinical trials.

Nonetheless, the potential beneficial effects of treating hyperglycaemia have led some centres to treat AH with insulin—although no major associations have formalized glycaemic targets in guidelines. This practice has been adopted from the mixed results from clinical trials.

Nonetheless, the potential beneficial effects of treating hyperglycaemia have led some centres to treat AH with insulin—although no major associations have formalized glycaemic targets in guidelines. This practice has been adopted from the mixed results from clinical trials.

In conclusion, AH remains a strong predictor of short- and long-term mortality in the pPCI era. It occurs more commonly in patients who have traditional predictors of worse outcomes—specifically prior infarction, anterior wall infarctions, and multivessel disease. Likely, AH is a predictor of rather than a bona fide therapeutic target in AMI.

<table>
<thead>
<tr>
<th>No.</th>
<th>Author</th>
<th>Study design</th>
<th>Patient no.</th>
<th>BSL cutoff mg/dL (mmol/L)</th>
<th>Known diabetics</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Planer</td>
<td>Prospective</td>
<td>3405</td>
<td>156 (8.6)</td>
<td>566</td>
<td>AH was noted to be an independent predictor of short- and long-term mortality in both diabetic and non-diabetic patients.</td>
</tr>
<tr>
<td>2</td>
<td>Hoebers</td>
<td>Prospective</td>
<td>1646</td>
<td>140 (7.7)</td>
<td>209</td>
<td>In patients with or without diabetes, AH was an independent predictor of early but not late mortality.</td>
</tr>
<tr>
<td>3</td>
<td>Lazzeri</td>
<td>Prospective</td>
<td>252</td>
<td>140 (7.7)</td>
<td>None</td>
<td>Peak glycaemia was an independent risk factor of mortality. Mortality was highest with BSL &gt; 180 mg/dL and intermediate with BSL between 140 and 180 mg/dL.</td>
</tr>
<tr>
<td>4</td>
<td>Usami</td>
<td>Prospective</td>
<td>2433</td>
<td>198 (11)</td>
<td>None</td>
<td>AH was associated with higher 30-day mortality. Intracoronary thrombectomy at the time of pPCI reduced 30-day mortality in patients with AH.</td>
</tr>
<tr>
<td>5</td>
<td>Marenzi</td>
<td>Prospective</td>
<td>780</td>
<td>198 (11)</td>
<td>109</td>
<td>AH was found to be an independent predictor of contrast-induced nephropathy and worse in-hospital outcomes.</td>
</tr>
<tr>
<td>6</td>
<td>Lavi</td>
<td>Prospective</td>
<td>431</td>
<td>126 (7)</td>
<td>88</td>
<td>AH was present in 35% of the non-diabetic patients. Furthermore, 1-year mortality was significantly higher in non-diabetic patients with AH. Non-diabetic patients without AH had the best outcomes.</td>
</tr>
<tr>
<td>7</td>
<td>Ergelen</td>
<td>Retrospective</td>
<td>2482</td>
<td>200 (11.1)</td>
<td>612</td>
<td>Non-diabetic patients with AH carry the highest risk for early mortality, whereas diabetic patients AH predicted long-term mortality in STEMI patients.</td>
</tr>
<tr>
<td>8</td>
<td>Elmekci</td>
<td>Retrospective</td>
<td>627</td>
<td>168 (9.3)</td>
<td>122</td>
<td>This study only considered elderly patients (&gt; 65 years). AH was associated with higher in-hospital and long-term mortality. AH was also associated with higher MACE.</td>
</tr>
<tr>
<td>9</td>
<td>Timmer</td>
<td>Retrospective</td>
<td>4176</td>
<td>145 (8)</td>
<td>None</td>
<td>Elevated HbA1c levels were generally associated with increased long-term mortality regardless of admission glucose level. On the other hand, AH was associated with short-term mortality.</td>
</tr>
<tr>
<td>10</td>
<td>Chen</td>
<td>Retrospective</td>
<td>1035</td>
<td>140 (7.7)</td>
<td>306</td>
<td>AH was associated with higher in-hospital mortality and morbidity. Long-term mortality was also higher in patients presenting with AH. BSL &gt; 190 was able to predict worse in-hospital outcomes.</td>
</tr>
<tr>
<td>11</td>
<td>Worthley</td>
<td>Retrospective</td>
<td>980</td>
<td>140 (7.7)</td>
<td>152</td>
<td>AH was an independent predictor of in-hospital mortality in STEMI patients treated with pPCI. On the other hand, in-hospital mortality was similar between diabetic and non-diabetic patients.</td>
</tr>
<tr>
<td>12</td>
<td>Gasior</td>
<td>Retrospective</td>
<td>1310</td>
<td>140 (7.7)</td>
<td>352</td>
<td>One-year mortality was highest in the non-diabetic patients with AH.</td>
</tr>
</tbody>
</table>

AH: admission hyperglycaemia; BSL: blood sugar level; MACE: major adverse cardiovascular events; pPCI: primary angioplasty.


