CLINICAL RESEARCH

Initial hospital pulse pressure and cardiovascular outcomes in acute coronary syndrome

Pression pulsée à la phase initiale d’une hospitalisation et événements cardiovasculaires au décours d’un syndrome coronaire aigu

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Summary

Background. — The association between admission pulse pressure (PP) and cardiovascular outcomes in acute coronary syndrome (ACS) is not well defined.

Aim. — To explore the prognostic value of initial PP in ST-segment elevation myocardial infarction (STEMI) and non-ST elevation ACS (NSTE-ACS).

Methods. — Over a 5-month period in 2007, 6704 consecutive patients with ACS were categorized into five groups according to initial PP: P1, PP \( \leq 0 \); P2, PP 31—40; P3, PP 41—50; P4, PP 51—60; P5, PP > 60 mmHg. Patient characteristics and in-hospital outcomes were analysed.

Results. — Mean PP was lower in men versus women (55 \( \pm \) 19 vs. 61 \( \pm \) 22), young versus old (53 \( \pm \) 17 vs. 59 \( \pm \) 21), STEMI vs. NSTE-ACS (51 \( \pm \) 18 vs. 60 \( \pm \) 18) and patients who died versus survived (46 \( \pm \) 22 vs. 57 \( \pm \) 19 mmHg) (\( P < 0.001 \) for all). Most patients with low PP had a high Global Registry of Acute Coronary Events risk score. Compared with P5, crude odds ratios (ORs) (95% confidence intervals) for death were: P1, 9 (5.78—13.35); P2, 3 (1.71—4.06); P3, 2.6 (1.54—4.34); P4, 1.9 (1.15—3.26).

KEYWORDS
Pulse pressure; Acute coronary syndrome; Mortality

Abbreviations: ACS, acute coronary syndrome; BP, blood pressure; CI, confidence interval; DBP, diastolic blood pressure; HF, heart failure; MAP, mean arterial pressure; ML, myocardial infarction; NS-T ACS, non-ST elevation acute coronary syndrome; PP, pulse pressure; SBP, systolic blood pressure; SD, standard deviation; STEMI, segment elevation myocardial infarction.

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Arterial blood pressure (BP) variables that are easily obtainable at the bedside include systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP) and pulse pressure (PP). Although these BP variables are of clinical importance in many cardiovascular disorders, the best predictor to consider in clinical practice is not well defined [1]. PP arises as a consequence of the episodic nature of cardiac contraction and the properties of the arterial circulation; it reflects stiffness of the aorta and large arteries, and pulse wave velocity [2,3]. Recently, PP has been identified as a more powerful prognostic predictor of cardiovascular events than SBP and DBP in selected populations [2—10]. Data from the population-based Framingham Heart Study showed that neither SBP nor DBP was superior to high PP in predicting coronary heart disease risk in middle-aged and elderly patients [6]. However, most of these findings were extracted from studies that evaluated the impact of PP in non-acute cardiovascular situations. Therefore, data on the prognostic influence of the initial hospital recording of the pulsatile components of the arterial BP wave (i.e., PP) in acute coronary events is lacking. The only available reports on PP after acute myocardial infarction (MI) were provided for a particular subgroup of high-risk patients, namely those with impaired left ventricular function [11—13]. The present study explores data from the Gulf Registry of Acute Coronary Events (Gulf RACE) to assess whether low versus high PP measured on admission plays an additional prognostic role in patients presenting with ST-segment elevation MI (STEMI) and non-ST elevation acute coronary syndrome (NSTE-ACS).
Methods

The data are derived from a prospective, multicentre, observational study of the Gulf RACE. In 2007 and for 5 months, the Gulf RACE investigators recruited 6704 consecutive acute coronary syndrome (ACS) patients from 64 hospitals in six Middle Eastern countries (Bahrain, Kuwait, Qatar, Oman, United Arab Emirates and Yemen). The study received ethical approval from the institutional ethical bodies in all participating countries. The rationale and details of the Gulf RACE have been described previously [14–16]. All participating centres were committed by written consent to include every consecutive patient with ACS. All patients gave informed consent to participate and care was taken to ensure data anonymity. Data were collected on record forms by the treating physicians. Completed data sheets were sent to the central data processing centre for uniform monitoring and registration. The primary outcome was in-hospital mortality rate. The secondary outcomes included recurrent myocardial ischaemia, heart failure (HF) and stroke.

Brachial PP was used as a marker of the pulsatile component of BP. A single BP measurement was taken with a sphygmomanometer at the time of presentation in the supine position; PP was calculated as the difference between SBP and DBP; MAP was calculated as two-thirds DBP plus one-third SBP. To explore the relationship between in-hospital outcome and arterial pressure indices, BP values were analysed as continuous and categorical variables. In the latter case, BP values were grouped in 10-mmHg classes according to the following cut-off values: P1, ≤ 30; P2, 31–40; P3, 41–50; P4, 51–60; P5, > 60 mmHg [11].

Briefly, diagnosis of the different types of ACS and definitions of data variables were based on the American College of Cardiology clinical data standard [14]. For the purpose of this report, ST-segment elevation MI and left bundle branch block MI were grouped together and called STEMI; non-ST-segment elevation MI and unstable angina were grouped together and called NSTE-ACS. An attempt was made to include everyone with a final diagnosis of ACS and there were no exclusion criteria [15–17].

Statistical analysis

Data are presented as proportions, medians or means ± standard deviations (SDs) as appropriate. Differences in categorical variables between respective comparison groups were analysed using the chi-square test. The continuous variables were analysed using one-way analysis of variance. The multivariable model for the predictors of the in-hospital outcomes included the following potential covariates: age, sex, diabetes mellitus, hypertension, ejection fraction, troponin T, aspirin, clopidogrel, glycoprotein inhibitors, beta-blockers and angiotensin-converting enzyme inhibitors. To assess the linear relationship among some or all of the independent variables in the regression model, collinearity was tested by calculating the correlation coefficient among the BP variables. Owing to collinearity between BP variables, only PP and SBP variables were taken into the multivariable analysis. All p values were two-sided tailed. p values < 0.05 were considered significant. In a subanalysis, PP patients were divided into two groups (≤ 30 vs. > 30 mmHg) for comparing the mortality rate in each group, stratified by the admission Killip class. All data analyses were carried out using the Statistical Package for Social Sciences, version 14 (SPSS Inc., Cary, NC, USA).

Results

Among 6704 patients who presented with ACS, data were successfully completed for 6638 patients. The mean PP, SBP, DBP and MAP were 56.5 ± 20, 139.8 ± 31, 83.5 ± 18 and 102 ± 20, respectively. Mean PP was lower in men compared with in women (55 ± 19 vs. 61 ± 22, P < 0.001) and in younger age compared with older age (53 ± 17 vs. 59 ± 21, P < 0.001). Table 1 describes patients’ characteristics in different PP subgroups. Among all PP groups, cardiovascular risk factors were less frequent in patients presenting with initial low PP, except for smoking and renal impairment. Patients with low PP were characterized by a higher resting heart rate and higher Killip class on admission. In comparison with high PP, patients in the P1 group presented far more frequently with STEMI (56% vs. 27%, P = 0.001), a higher level of peak troponin T (23 ± 61 vs. 14 ± 53, P = 0.005) and a left ventricular ejection fraction < 40% (36% vs. 19%, P = 0.001). Patients in the P1 group were less likely to receive evidence-based medications and were less likely to undergo coronary angiography.

Tables 2 and 3 show the clinical presentation and hospital outcomes in patients with NSTE-ACS and STEMI, stratified by the initial PP values.

Correlation coefficient analysis showed a significant linear relationship among the BP variables i.e. PP and MAP (r = 0.54, 95% confidence interval [CI] 0.51–0.55), SBP and PP (r = 0.81, 95% CI 0.80–0.83), DBP and PP (r = 0.24, 95% CI 0.22–0.30) and MAP and DBP (r = 0.95, 95% CI 0.94–0.96) (P < 0.001 for all).

Logistic regression multivariable analysis for in-hospital outcomes showed that an initial low PP was associated with a worse in-hospital outcome than high PP. The mean PP in patients who died after admission was lower than in those who survived (46 ± 22 vs. 57 ± 19 mmHg, P < 0.001). In comparison with the P5 subgroup, lower PP subgroups had a significant association with higher mortality: P1, OR 9, 95% CI 5.78–13.35; P2, OR 3, 95% CI 1.71–4.06; P3, OR 1.5, 95% CI 1.01–2.49. After adjustment, low PP was associated with high rates of mortality (OR 7.5 [3.77–14.72]) and stroke (OR 4.5 [1.20–18.88]) in ACS, a high rate of recurrent ischaemia in NSTE-ACS (OR 2.8 [1.52–5.22]), and a high rate of HF in STEMI (OR 2.1 [1.18–3.76]). Low SBP was associated with similar worse outcomes, although SBP was uniquely associated with a high rate of HF in NSTE-ACS and a high stroke rate in STEMI (adjusted ORs 3 [1.57–5.92] and 14 [2.25–86.01], respectively) (Table 4 and Fig. 1).

The mortality rate in the PP subgroups varied according to the admission Killip class (Fig. 2). Even in the absence of HF (i.e. Killip I); low PP was associated with greater mortality compared with high PP. There was a significant main effect for Killip class (P < 0.001) and PP (P < 0.001) on mortality; the interaction between the two variables was significant (P for interaction = 0.001). Fig. 3 shows that apart from the P5 subgroup, women had a significant higher crude mortality rate when compared with that for men (P1, 19%
Table 1 Clinical profiles, management and in-hospital outcomes in acute coronary syndrome patients.

<table>
<thead>
<tr>
<th></th>
<th>PP ≤ 30</th>
<th>PP 31–40</th>
<th>PP 41–50</th>
<th>PP 51–60</th>
<th>PP &gt; 60</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number (%)</strong></td>
<td>533 (8)</td>
<td>1244 (19)</td>
<td>1550 (23)</td>
<td>1241 (19)</td>
<td>2070 (31)</td>
<td>0.001</td>
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<tr>
<td><strong>Demographics</strong></td>
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<tr>
<td>Age</td>
<td>57 ± 14</td>
<td>54 ± 12</td>
<td>54 ± 12</td>
<td>56 ± 12</td>
<td>59 ± 12</td>
<td>0.001</td>
</tr>
<tr>
<td>Women (%)</td>
<td>24.0</td>
<td>18.8</td>
<td>20.4</td>
<td>22.8</td>
<td>31.7</td>
<td>0.001</td>
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<td><strong>Medical history</strong></td>
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<tr>
<td>Diabetes (%)</td>
<td>35.6</td>
<td>32.9</td>
<td>36.5</td>
<td>40.8</td>
<td>50.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>38.6</td>
<td>36.5</td>
<td>41.8</td>
<td>50.3</td>
<td>67.7</td>
<td>0.001</td>
</tr>
<tr>
<td>Dyslipidaemia (%)</td>
<td>23.8</td>
<td>26.2</td>
<td>30.4</td>
<td>32.4</td>
<td>38.7</td>
<td>0.004</td>
</tr>
<tr>
<td>Smokers (%)</td>
<td>41.5</td>
<td>44.9</td>
<td>41.5</td>
<td>39.1</td>
<td>29.8</td>
<td>0.001</td>
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<tr>
<td>Renal impairment (%)</td>
<td></td>
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<tr>
<td>Prior CAD (%)</td>
<td>46.7</td>
<td>40.3</td>
<td>43.5</td>
<td>45.7</td>
<td>50.6</td>
<td>0.001</td>
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<td><strong>Clinical characteristics</strong></td>
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<tr>
<td>Peak troponin (ng/mL)</td>
<td>23 ± 61</td>
<td>21 ± 52</td>
<td>16 ± 45</td>
<td>16 ± 48</td>
<td>14 ± 53</td>
<td>0.005</td>
</tr>
<tr>
<td>Heart rate</td>
<td>88 ± 31</td>
<td>84 ± 23</td>
<td>85 ± 22</td>
<td>86 ± 22</td>
<td>87 ± 22</td>
<td>0.002</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>97 ± 23</td>
<td>118 ± 17</td>
<td>131 ± 15</td>
<td>143 ± 19</td>
<td>169 ± 26</td>
<td>0.001</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>72 ± 22</td>
<td>79 ± 16</td>
<td>82 ± 15</td>
<td>86 ± 17</td>
<td>89 ± 20</td>
<td>0.001</td>
</tr>
<tr>
<td>MAP</td>
<td>81 ± 21</td>
<td>92 ± 16</td>
<td>99 ± 16</td>
<td>105 ± 19</td>
<td>117 ± 21</td>
<td>0.001</td>
</tr>
<tr>
<td>Body mass index</td>
<td>27 ± 5</td>
<td>27 ± 5</td>
<td>27 ± 5</td>
<td>28 ± 5</td>
<td>28 ± 6</td>
<td>0.009</td>
</tr>
<tr>
<td>Symptoms &gt; 12 hours (%)</td>
<td>47.5</td>
<td>34.2</td>
<td>29.3</td>
<td>25.8</td>
<td>25.0</td>
<td>0.001</td>
</tr>
<tr>
<td>Killip &gt; class 1 (%)</td>
<td></td>
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<tr>
<td>No typical angina (%)</td>
<td></td>
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<tr>
<td>LVEF &lt; 40% (%)</td>
<td>36.3</td>
<td>24.3</td>
<td>20.8</td>
<td>21.7</td>
<td>19.3</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Discharge diagnosis</strong></td>
<td></td>
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<tr>
<td>NSTE-ACS (%)</td>
<td>44.2</td>
<td>48.6</td>
<td>59.8</td>
<td>63.3</td>
<td>72.8</td>
<td>0.001</td>
</tr>
<tr>
<td>STEMI (%)</td>
<td>55.8</td>
<td>51.4</td>
<td>40.2</td>
<td>36.7</td>
<td>27.2</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Hospital treatments</strong></td>
<td></td>
<td></td>
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<tr>
<td>Aspirin (%)</td>
<td>97.1</td>
<td>97.5</td>
<td>98.3</td>
<td>98.2</td>
<td>98.1</td>
<td>0.32</td>
</tr>
<tr>
<td>Clopidogrel (%)</td>
<td>48.3</td>
<td>55.0</td>
<td>56.4</td>
<td>53.9</td>
<td>52.6</td>
<td>0.01</td>
</tr>
<tr>
<td>GP IIb/IIIa inhibitor (%)</td>
<td>5.9</td>
<td>9.2</td>
<td>12.1</td>
<td>10.2</td>
<td>11.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Beta-blockers (%)</td>
<td>48.1</td>
<td>62.2</td>
<td>69.4</td>
<td>68.9</td>
<td>66.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Statins (%)</td>
<td>73.8</td>
<td>77.2</td>
<td>82.8</td>
<td>81.7</td>
<td>83.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Heparin (%)</td>
<td>91.9</td>
<td>93.2</td>
<td>93.7</td>
<td>93.8</td>
<td>91.5</td>
<td>0.05</td>
</tr>
<tr>
<td>ACE Inhibitors (%)</td>
<td>53.8</td>
<td>66.1</td>
<td>67.3</td>
<td>70.3</td>
<td>75.7</td>
<td>0.001</td>
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<tr>
<td>Coronary angiogram (%)</td>
<td>13.3</td>
<td>16.1</td>
<td>19.4</td>
<td>19.5</td>
<td>20.6</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Hospital outcomes</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Recurrent ischaemia (%)</td>
<td>11.6</td>
<td>11.2</td>
<td>8.1</td>
<td>8.3</td>
<td>8.0</td>
<td>0.002</td>
</tr>
<tr>
<td>Heart failure (%)</td>
<td>25.9</td>
<td>17.2</td>
<td>12.1</td>
<td>15.4</td>
<td>16.9</td>
<td>0.001</td>
</tr>
<tr>
<td>Cardiogenic shock (%)</td>
<td>20.9</td>
<td>6.6</td>
<td>3.2</td>
<td>2.4</td>
<td>2.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Stroke (%)</td>
<td>2.2</td>
<td>0.6</td>
<td>0.4</td>
<td>0.5</td>
<td>0.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Mortality (%)</td>
<td>14.7</td>
<td>4.5</td>
<td>2.7</td>
<td>1.7</td>
<td>1.7</td>
<td>0.001</td>
</tr>
</tbody>
</table>

All continuous variables are presented as mean ± standard deviation. ACE: angiotensin-converting enzyme; BP: blood pressure; CAD: coronary artery disease; GP: glycoprotein; LVEF: left ventricular ejection fraction; MAP: mean arterial pressure; NSTE-ACS: non-ST-segment elevation acute coronary syndrome; PP: pulse pressure; STEMI: ST-segment elevation myocardial infarction.

vs. 11%; P2, 8.1% vs. 3.5%; P3, 4.7% vs. 2.1%; P4, 3.2% vs. 1%).

**Discussion**

The present study reported on the prognostic implications of initial low PP in patients presenting with ACS in a multinational, multicentre study. There are many key findings in this study. Firstly, in ACS, all BP variables were significantly correlated. Secondly, low PP was an independent predictor of stroke and mortality in overall ACS. Although PP was not superior to SBP, only low PP was an independent predictor of recurrent ischaemia in NSTE-ACS patients. Moreover, even in the absence of HF, low PP was a predictor of in-hospital mortality in ACS patients. Thirdly, women with initial low PP had a greater crude mortality rate compared with their counterpart men (Fig. 3). There was a significant
Table 2 Clinical profiles and in-hospital outcomes according to pulse pressure at presentation in ST-segment elevation myocardial infarction patients.

<table>
<thead>
<tr>
<th>PP ≤ 30</th>
<th>PP 31–40</th>
<th>PP 41–50</th>
<th>PP 51–60</th>
<th>PP &gt; 60</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>299 (12)</td>
<td>639 (25)</td>
<td>623 (24)</td>
<td>454 (17)</td>
<td>563 (22)</td>
</tr>
</tbody>
</table>

**Demographics**
- Age: 56 ± 14, 53 ± 12, 53 ± 11, 54 ± 12, 56 ± 12 (P = 0.001)
- Women (%): 20, 11, 10, 15, 17 (P = 0.001)

**Clinical characteristics**
- Diabetes (%): 35, 26, 32, 29, 40 (P = 0.001)
- Hypertension (%): 29, 26, 28, 35, 51 (P = 0.001)
- Dyslipidaemia (%): 15, 16.5, 17.5, 16, 22 (P = 0.04)
- Smokers (%): 48, 58, 56, 52.5, 45 (P = 0.001)
- Peak troponin (ng/mL): 42 ± 82, 40 ± 70, 35 ± 68, 36 ± 71, 42 ± 88 (P = 0.66)
- Heart rate: 84 ± 29, 83 ± 21, 85 ± 22, 86 ± 22, 86 ± 21 (P = 0.20)
- Systolic BP: 96 ± 26, 118 ± 18, 131 ± 17, 145 ± 19, 168 ± 26 (P = 0.001)
- Diastolic BP: 70 ± 22, 79 ± 18, 83 ± 16, 88 ± 19, 91 ± 20 (P = 0.001)
- MAP: 79 ± 23, 93 ± 18, 99 ± 16, 107 ± 19, 117 ± 21 (P = 0.001)
- Body mass index: 26 ± 5, 27 ± 5, 27 ± 5, 27 ± 5, 27 ± 5 (P = 0.09)
- Symptoms > 12 hours (%): 48, 34, 29, 26, 24 (P = 0.001)
- Killip > class 1 (%): 21.5, 7.5, 5, 5, 8 (P = 0.001)
- No typical angina: 18, 11, 10, 9, 14 (P = 0.001)
- Low GRACE risk score: 19, 35, 48, 55, 56 (P = 0.001)
- High GRACE risk score: 56, 30, 20, 19, 18 (P = 0.001)
- LVEF < 40%: 35, 22, 21, 25, 22 (P = 0.001)

**Hospital outcomes**
- Re-ischaemia (%): 9, 12, 6.4, 8, 10 (P = 0.01)
- Heart failure (%): 27, 19, 13, 17, 14 (P = 0.001)
- Cardiogenic shock (%): 28, 9.5, 5, 6, 6 (P = 0.001)
- Stroke (%): 3.5, 1.1, 0.5, 0.7, 1.4 (P = 0.001)
- Mortality (%): 20.5, 6, 3.5, 3, 4 (P = 0.001)

All continuous variables are presented as mean ± standard deviation. BP: blood pressure; GRACE: Global Registry of Acute Coronary Events; LVEF: left ventricular ejection fraction; MAP: mean arterial pressure; PP: pulse pressure.

**Figure 1.** Clinical predictors of hospital mortality according to initial pulse pressure (PP). ACE: angiotensin-converting enzyme; LV: left ventricular.
Table 3  Clinical profiles and in-hospital outcomes according to pulse pressure at presentation in non-ST-segment elevation acute coronary syndrome patients.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Number (%)</th>
<th>PP ≤ 30</th>
<th>PP 31–40</th>
<th>PP 41–50</th>
<th>PP 51–60</th>
<th>PP &gt; 60</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td>57 ± 14</td>
<td>56 ± 13</td>
<td>55 ± 12</td>
<td>57 ± 12</td>
<td>60 ± 12</td>
<td>0.001</td>
</tr>
<tr>
<td>Women (%)</td>
<td></td>
<td>29</td>
<td>27</td>
<td>27</td>
<td>27</td>
<td>37</td>
<td>0.001</td>
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<tr>
<td>Clinical characteristics</td>
<td></td>
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<tr>
<td>Diabetes (%)</td>
<td></td>
<td>36</td>
<td>40</td>
<td>39.5</td>
<td>47.5</td>
<td>55</td>
<td>0.001</td>
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<tr>
<td>Hypertension (%)</td>
<td></td>
<td>51</td>
<td>47</td>
<td>51</td>
<td>59</td>
<td>74</td>
<td>0.001</td>
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<td>Dyslipidaemia (%)</td>
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<td>36.5</td>
<td>39</td>
<td>42</td>
<td>45</td>
<td>0.001</td>
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<tr>
<td>Smokers (%)</td>
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<td>31</td>
<td>32</td>
<td>31</td>
<td>24</td>
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<td>Peak troponin (ng/mL)</td>
<td></td>
<td>5 ± 12</td>
<td>4 ± 10</td>
<td>4 ± 12</td>
<td>4 ± 21</td>
<td>4 ± 23</td>
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<tr>
<td>Heart rate</td>
<td></td>
<td>93 ± 32</td>
<td>85 ± 25</td>
<td>84 ± 22</td>
<td>86 ± 21</td>
<td>88 ± 22</td>
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<tr>
<td>Systolic BP</td>
<td></td>
<td>100 ± 20</td>
<td>117 ± 15</td>
<td>130 ± 16</td>
<td>142 ± 16</td>
<td>169 ± 26</td>
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<tr>
<td>Diastolic BP</td>
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<td>75 ± 19</td>
<td>78 ± 15</td>
<td>82 ± 14</td>
<td>85 ± 15</td>
<td>88 ± 19</td>
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<td>MAP</td>
<td></td>
<td>83 ± 18</td>
<td>91 ± 15</td>
<td>98 ± 14</td>
<td>104 ± 16</td>
<td>115 ± 20</td>
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<td>Body mass index</td>
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<td>27 ± 5</td>
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<td>28 ± 6</td>
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<td>0.009</td>
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<td>Symptoms &gt; 12 hours (%)</td>
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<td>25</td>
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<td>60</td>
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<td>Killip &gt; class 1 (%)</td>
<td></td>
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<td>19.5</td>
<td>17</td>
<td>19</td>
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<td>21.5</td>
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<tr>
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<td>39</td>
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<td>40</td>
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<td>29</td>
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<td>LVEF &lt; 40%</td>
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<td>39</td>
<td>28</td>
<td>21</td>
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<td>Hospital outcomes</td>
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<td>Re-ischaemia (%)</td>
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<td>11</td>
<td>9</td>
<td>8</td>
<td>7</td>
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<tr>
<td>Heart failure (%)</td>
<td></td>
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<td>15</td>
<td>11</td>
<td>14</td>
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<td>Cardiogenic shock (%)</td>
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<td>3.5</td>
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<td>0.001</td>
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<tr>
<td>Stroke (%)</td>
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<td>0.4</td>
<td>0.2</td>
<td>0.3</td>
<td>0.4</td>
<td>0.6</td>
<td>0.68</td>
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<tr>
<td>Mortality (%)</td>
<td></td>
<td>7</td>
<td>3</td>
<td>2</td>
<td>0.8</td>
<td>0.9</td>
<td>0.001</td>
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All continuous variables are presented as mean ± standard deviation. BP: blood pressure; GRACE: Global Registry of Acute Coronary Events; LVEF: left ventricular ejection fraction; MAP: mean arterial pressure; PP: pulse pressure.

Main effect for female sex (P < 0.001) and PP (P < 0.001) on mortality; the interaction between the two variables was significant (P interaction = 0.001). Fourthly, patients with initial low PP were less likely to receive evidence-based therapy, including coronary intervention. Fifthly, initial low PP inversely correlated with GRACE risk score. Furthermore, we assumed that poor prognosis in low PP groups could not be related only to low SBP and that low or narrow PP may be of clinical and prognostic value in patients with apparently normal SBP (Fig. 2). The present study also showed that low SBP was associated with similar worse outcomes; however, SBP was uniquely associated with a high HF rate in NSTE-ACS patients and stroke rate in STEMI patients (Table 4).

These findings may provoke debate, as most previous studies have suggested that high rather than low PP has a poor prognosis [11–13]. However, previous studies were not carried out on all the ACS subtypes and did not rely on measuring PP on admission. Moreover, in most of these studies, PP was measured either at discharge or weeks after MI and evaluated post-discharge rather than in-hospital outcome [1—3, 8, 9, 11–13, 18–30]. A previous study reported that in patients with ischaemic HF, high PP might be reflected in the atherosclerotic burden while low PP may be reflected in left ventricular dysfunction [27]. Low PP exclusively affected inhospital outcomes when it was measured on admission in patients presenting with acute stress situations, while high PP had worse long-term outcome and high PP was measured in stable chronic cases [1, 21, 25, 28, 29]. Despite the fact that high PP may exacerbate myocardial ischaemia as a result of increased afterload and reduced coronary perfusion, low PP may indicate low stroke volume, early sign of cardiogenic shock in the acute cardiac events and may be related to increased concentrations of natriuretic peptides [3, 27, 31]. Petrie et al. studied 1959 patients within the first 21 days post MI and reported that only in high Killip classes (II–IV) did a low PP independently predict cardiovascular mortality [26]. The current study expanded this finding and demonstrated that low initial PP was associated with higher mortality in Killip classes I and II. Interestingly, low PP was associated with higher troponin values and resting heart rate when compared with high PP groups. The latter two markers may partly explain the poor prognosis in our study.
Table 4  Unadjusted and adjusted predictors of hospital outcomes according to type of acute coronary syndrome.

<table>
<thead>
<tr>
<th></th>
<th>Recurrent ischaemia</th>
<th>Heart failure</th>
<th>Stroke</th>
<th>Mortality</th>
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<tr>
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<td>Unadjusted</td>
<td>Adjusted\a</td>
<td>Unadjusted</td>
<td>Adjusted\a</td>
</tr>
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<td>ACS</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>PP</td>
<td>1.5 (1.11—1.99)</td>
<td>1.3 (0.81—2.23)</td>
<td>1.8 (1.42—2.18)</td>
<td>1.5 (0.98—2.19)</td>
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<tr>
<td>SBP</td>
<td>1.6 (1.18—2.27)</td>
<td>1.2 (0.65—2.14)</td>
<td>4.3 (3.42—1.59)</td>
<td>3.3 (2.17—4.97)</td>
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<td>NSTE-ACS</td>
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<tr>
<td>PP</td>
<td>1.8 (1.26—2.66)</td>
<td>2.8 (1.52—5.22)</td>
<td>1.8 (1.38—2.52)</td>
<td>1.2 (0.69—2.22)</td>
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<tr>
<td>SBP</td>
<td>1.9 (1.17—3.12)</td>
<td>2 (0.87—4.48)</td>
<td>3.3 (2.28—4.75)</td>
<td>3 (1.57—5.92)</td>
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<tr>
<td>STEMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP</td>
<td>0.95 (0.61—1.49)</td>
<td>0.4 (0.15—1.09)</td>
<td>2 (1.45—2.73)</td>
<td>2.1 (1.18—3.76)</td>
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<tr>
<td>SBP</td>
<td>1.5 (0.93—2.26)</td>
<td>0.7 (0.30—1.85)</td>
<td>5 (3.81—6.91)</td>
<td>3.4 (1.96—5.97)</td>
</tr>
</tbody>
</table>

Data are presented as odds ratios (95% confidence intervals). ACS: acute coronary syndrome; NSTE-ACS: non-ST-segment elevation ACS; PP: pulse pressure ≤ 30 mmHg; SBP: systolic blood pressure < 100 mmHg; STEMI: ST-segment elevation myocardial infarction.

\( ^a \) Adjusted for age, sex, diabetes, hypertension, aspirin, clopidogrel, beta-blockers, angiotensin-converting enzyme inhibitors, glycoprotein inhibitors, troponin T and ejection fraction.
Conclusions

In ACS patients, arterial blood pressure variables were significantly correlated and had varying prognostic values. Although PP was not superior to SBP, low PP was of prognostic value in patients presenting with STEMI and NSTE-ACS—even in those with nearly normal SBP. Low PP was an independent predictor for stroke and mortality in overall ACS. When compared with SBP, low PP was an independent predictor for recurrent ischaemia in NSTE-ACS patients while low SBP was independent predictor for HF in NSTE-ACS patients and stroke in STEMI patients. PP is a simple measure that could be a part of risk scoring; however, prospective studies are needed to support our findings and to set an appropriate management plan for these patients.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

Acknowledgements

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References

[26] Petrie CJ, Robertson M, Voors AA, et al. A low pulse pressure predicts mortality in patients with left ventricular dysfunction post myocardial infarction, but only in those with signs


