Eosinopenia an inflammatory marker of adverse clinical outcomes in patients presenting with acute myocardial infarction

Authors:
M Alkhalil\textsuperscript{1}, AK Kearney\textsuperscript{1}, MH Hegarty\textsuperscript{1}, CS Stewart\textsuperscript{1}, PD Devlin\textsuperscript{1}, CO Owens\textsuperscript{1}, MS Spence\textsuperscript{1}, \textsuperscript{1}Belfast Health and Social Care Trust - Belfast - United Kingdom of Great Britain & Northern Ireland,

Topic(s):
ST-Elevation Myocardial Infarction (STEMI)

Citation:

Background:
Inflammation is an indicator of worse clinical outcomes following acute myocardial infarction. Eosinopenia was identified as a surrogate of inflammation in sepsis and obstructive airway disease. Whether this readily-available marker has any impact on long term outcomes following ST-segment elevation myocardial infarction (STEMI) is yet to be determined.

Purpose:
We sought to study the incidence and relationship between eosinopenia and infarct severity and whether low eosinophil had impact on clinical outcomes following STEMI.

Methods:
606 consecutive STEMI patients undergoing primary PCI from a large volume single centre were enrolled. Low eosinophil count was defined as <40 cells/ml from samples within 2-hours post reperfusion. Primary endpoint was defined as composite of death, MI, stroke, unplanned revascularisation, re-admission for heart failure over 3.5 years follow up.

Results:
65% of patients had eosinopenia. Patients in the low eosinophil group had larger infarct size as measured by troponin value [2934 vs. 1177ng/L, P<0.001] and left ventricle (LV) systolic function on echocardiography [48% vs. 50%, P=0.029]. There was a modest correlation between eosinophil count and both troponin (r= -0.25, P<0.001) and ejection fraction (r= 0.10, P=0.017). The primary endpoint was higher in eosinopenic patients (28.8% vs. 20.4%, HR 1.49, 95%CI 1.05 to 2.13, P=0.023) (Figure). The difference was mainly driven from higher percentage of unplanned revascularisations (8.2% versus 2.9%, P= 0.012) (Table). Low eosinophil count was an independent predictor of adverse cardiovascular events, beyond infarct severity, in elderly, non-diabetic patients (HR 2.04, 95%CI 1.04 to 4.01, P=0.038).

Conclusions:
Eosinopenia is a readily-available marker which was associated with a larger infarcts and worse clinical outcomes over long term follow up.

<table>
<thead>
<tr>
<th>Clinical characteristics</th>
<th>Low eosinophil</th>
<th>Normal eosinophil</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long term clinical events</td>
<td>28.8% (112)</td>
<td>20.4% (42)</td>
<td>0.026</td>
</tr>
<tr>
<td>Long term mortality</td>
<td>14.1% (55)</td>
<td>11.1% (23)</td>
<td>0.31</td>
</tr>
</tbody>
</table>
Abstract: Eosinopenia an inflammatory marker of adverse clinical outcomes in patients presenting with acute myocardial infarction

Authors: M Alkhalil 1, AK Kearney 1, MH Hegarty 1, CS Stewart 1, PD Devlin 1, CO Owens 1, MS Spence 1

1 Belfast Health and Social Care Trust – Belfast – United Kingdom of Great Britain & Northern Ireland

Background: Inflammation is an indicator of worse clinical outcomes following acute myocardial infarction. Eosinopenia was identified as a surrogate of inflammation in sepsis and obstructive airway disease. Whether this readily-available marker has any impact on long term outcomes following ST-segment elevation myocardial infarction (STEMI) is yet to be determined.

Purpose: We sought to study the incidence and relationship between eosinopenia and infarct severity and whether low eosinophil had impact on clinical outcomes following STEMI.

Methods: 606 consecutive STEMI patients undergoing primary PCI from a large volume single centre were enrolled. Low eosinophil count was defined as <40 cells/ml from samples within 2–hours post reperfusion. Primary endpoint was defined as composite of death, MI, stroke, unplanned revascularisation, re-admission for heart failure over 3.5 years follow up.

Results: 65% of patients had eosinopenia. Patients in the low eosinophil group had larger infarct size as measured by troponin value [2934 vs. 1177ng/L, P<0.001] and left ventricle (LV) systolic function on echocardiography [48% vs. 50%, P=0.029]. There was a modest correlation between eosinophil count and both troponin (r=–0.25, P<0.001) and ejection fraction (r= 0.10, P=0.017). The primary endpoint was higher in eosinopenic patients (28.8% vs. 20.4%, HR 1.49, 95%CI 1.05 to 2.13, P=0.023) (Figure). The difference was mainly driven from higher percentage of unplanned revascularisations (8.2% versus 2.9%, P= 0.012) (Table). Low eosinophil count was an independent predictor of adverse cardiovascular events, beyond infarct severity, in elderly, non-diabetic patients (HR 2.04, 95%CI 1.04 to 4.01, P=0.038).

Conclusions: Eosinopenia is a readily-available marker which was associated with a larger infarcts and worse clinical outcomes over long term follow up.

<table>
<thead>
<tr>
<th>Long term MI</th>
<th>6.9% (27)</th>
<th>4.9% (10)</th>
<th>0.32</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long term unplanned revascularisation</td>
<td>8.2% (32)</td>
<td>2.9% (6)</td>
<td>0.012</td>
</tr>
<tr>
<td>Long term re-admission CCF</td>
<td>6.7% (26)</td>
<td>4.9% (10)</td>
<td>0.37</td>
</tr>
<tr>
<td>Long term stroke</td>
<td>2.6% (10)</td>
<td>1% (2)</td>
<td>0.19</td>
</tr>
</tbody>
</table>