EVALUATION OF PENTAZOCINE IN ACUTE MYOCARDIAL INFARCTION

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Abstract:

Background: The elevation or reduction of pain is a critical factor in the care of patients with acute myocardial infarction (AMI). For the treatment of pain of cardiac origin, the knowledge of haemodynamic action of opioid analgesics is a must.

Methodology: All age group of both sex, who were suffering from acute myocardial infarction (AMI) admitted in the intensive cardiac care unit as per the data available in medical record section.

Parameters for studied: Physiological parameters: Age and Sex, ECG parameters recorded after administration of opioids (Heart rate, Rhythm, Axis, PR interval, P wave, QRS complex, ST segment elevation/depression, T wave amplitude, Q waves) were recorded at 6 hrs, 12 hrs., 24 hrs after administration of pentazocine. Clinical status at the time of discharge from the hospital was noted.

Biochemical: Serum enzymes studies after administration of opioids included—Lactate Dehydrogenase, Creatinine Phosphokinase, – Serum Glutamate Oxaloacetate Transaminase (SGOT).

Result: Among the total number of patients 68% Pentazocine treated patients were males, while 32% Pentazocine patients were females. On comparison of ECG changes in Pentazocine treated patients at 6 hours, 12 hours, 24 hours and at discharge, the observations revealed persistent ECG changes, extension of infract, and regression patterns which were statistically highly significant Pentazocine treated patients. The study of clinical status of patients at discharge revealed that only 39.31% of Pentazocine treated patients became normal, while the clinical status of 40.17% of Pentazocine treated patients. This study revealed that the clinical status at discharge was statically highly significant.

Conclusion: The study can be concluded the pentazocine is contraindicated in patients with AMI as an analgesic has it deteriorate the ECG changes by its ability increase myocardial contractility, Heart rate and cardiac work.

Keywords: Pentazocine, Acute Myocardial Infarction, ECG

Introduction

Acute myocardial infarction (AMI) is one of the most common diseases of the modern era. The Mortality rate with AMI is approximately 30% with more than half of these deaths occurring before the individual gets emergency care. Although the
mortality rates after admission for AMI have declined, yet those who survive initial hospitalization die in the first year after AMI. Survival is markedly reduced in elderly patients over 65 years of age[1]. Relief of pain is a humanitarian issue and it has been said “To leave a person in avoidable pain and suffering should be regarded as a serious breach of fundamental human rights”[2].

In the emergency department, the goals for the management of patient with suspected AMI include control of cardiac pain, rapid identification of patients who are candidates for urgent reperfusion therapy[1]. The elevation or reduction of pain is a critical factor in the care of patients with Acute myocardial infarction[3].

For the treatment of pain of cardiac origin, the knowledge of haemodynamic action of opioid analgesics is a must[4].Parenterally administrated narcotic analgesics are a critically important part of therapy for the patients with acute myocardial ischemic syndromes. These agents are very effective and when used with appropriate caution and monitoring are also generally safe. They not only relieve sensation of pain but also relieve the effective and physiologic reaction to pain and thus reduce patient anxiety.

Pentazocine acts as a weak antagonist or a partial agonist at mu receptor while it is agonist at Kappa. In-patients with coronary artery disease Pentazocine administered intravenous elevates mean aortic pressure, left ventricular end diastolic pressure, mean pulmonary artery pressure (MPAP)[5], increased blood flow, contractile force, and causes an increase in cardiac work[6-12].

The agonist antagonist opioids such as Butorphanol, Nalbuphine, and Buprenorphine have largely replaced the hazardous Pentazocine, in the treatment of AMI (i.e. as analgesic)[13]. Among these Buprenorphine a long acting the Thebaine derivative is a powerful analgesic used to treat chest pain in patients with suspected AMI[14-15].

In view of the aforementioned beneficial pharmacological actions of Buprenorphine and detrimental adverse reactions of Pentazocine more particularly so in the setting of Acute myocardial infarction, it was thought prudent to evaluate the use of Pentazocine and Buprenorphine in the treatment of Acute myocardial infarction and either substantiate the already existing facts or to add to the existing knowledge regarding the use of these two drugs in Acute myocardial infarction. Keeping these goals in mind, this study was planned.

Material & Methods

Study design: Observation descriptive retrospective study
Ethical approval: The study was approved by the IEC of PIMS (DU)
Study place: Intensive cardiac care unit of Pravara Rural Hospital
Inclusion criteria: All age group of both sex, who were suffering from acute myocardial infarction (AMI) admitted in the intensive cardiac care unit and treated with Pentazocine opioid, data available in medical record section.
Exclusion criteria: Patients of Acute myocardial infarction who had complications like: Cardiogenic shock, Arrhythmias, Congestive cardiac failure before hospitalizations were not included in the study group.
Sample size: sample size was n = 117

Parameters for studied: [16]

Physiological Parameters

Age and Sex, ECG parameters recorded after administration of opioids (Heart rate, Rhythm, Axis, PR interval, P wave, QRS complex , ST segment elevation/ depression, T wave amplitude, Q waves) were recorded at 6 hrs, 12 hrs, 24 hrs after administration of opioids. Clinical status at the time of discharge from the hospital were noted

Biochemical

Serum enzymes studies after administration of opioids included– Lactate Dehydrogenase, Creatinine Phosphokinase, – Serum Glutamate Oxaloacetate Transaminase (SGOT).

Statistical Analysis

Statistical analysis was done by using ‘Z’ test. p < 0.05 was considered to be statistically significant.
Results

Fig 1: Age and sex wise distribution of patients under study

Fig 2: Sex wise distribution of patients under study

Table 1: Comparison of ECG parameters in Pentazocine and Buprenorphine treated patients of acute myocardial infarction (values recorded within 24 hrs of administration of Opioids).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pentazocine (n = 117)</th>
</tr>
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<tbody>
<tr>
<td>Heart rate</td>
<td>86.109 ± 23.2</td>
</tr>
<tr>
<td>Rhythm</td>
<td>N-103 Sinus A-14</td>
</tr>
<tr>
<td>Axis</td>
<td>LAD-16 Normal N-101</td>
</tr>
<tr>
<td>PR. Interval(Sec)</td>
<td>0.136 ± 0.044</td>
</tr>
<tr>
<td>QRS Duration (Sec)</td>
<td>0.070 ± 0.02</td>
</tr>
<tr>
<td>QRS amplitude (mm)</td>
<td>6.204 ± 2.29</td>
</tr>
<tr>
<td>ST Duration(Sec)</td>
<td>0.23 ± 0.047</td>
</tr>
<tr>
<td>T Wave amplitude (mm)</td>
<td>2.48 ± 1.90</td>
</tr>
<tr>
<td>Q Wave amplitude (mm)</td>
<td>2.36 ± 1.98</td>
</tr>
<tr>
<td>ST elevation during infarction (mm)</td>
<td>5 ± 1.8</td>
</tr>
<tr>
<td>ST depression during infarction</td>
<td>4 ± 1.3</td>
</tr>
</tbody>
</table>

Fig 3: Comparison of ECG persistent changes in Pentazocine treated patients of acute myocardial infarction

Fig 4: Comparison of ECG extension of infract changes in Pentazocine and Buprenorphine treated patients of acute myocardial infarction

Fig 5: Comparison of ECG regression changes in Pentazocine treated patients of acute myocardial infarction

Fig 6: Percentage distribution of infarction pattern in Pentazocine treated patients of acute myocardial infarction
Table 3: Serum enzyme studies in Pentazocine and Buprenorphine treated patients of acute myocardial infarction

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pentazocine (n = 117)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDH</td>
<td>638.58 + 307.47</td>
</tr>
<tr>
<td>CPK</td>
<td>472.47 + 856.75</td>
</tr>
<tr>
<td>SGOT</td>
<td>95.445 + 82.617</td>
</tr>
</tbody>
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Discussion

In the present study 66% Pentazocine treated patients were in age group of 45–65 years, while 34% Pentazocine treated patients were in age group 65 – 85 years. Among the total number of patients 68% Pentazocine treated patients were males, while 32% Pentazocine patients were females.

On comparison of ECG changes in Pentazocine treated patients at 6 hours, 12 hours, 24 hours and at discharge, the observations revealed persistent ECG changes, extension of infarct, and regression patterns which were statistically highly significant Pentazocine treated patients.

The study of clinical status of patients at discharge revealed that only 39.31% of Pentazocine treated patients became normal, while the clinical status of 40.17% of Pentazocine treated patients. This study revealed that the clinical status at discharge was statically highly significant. The various parameters which we studied yielded statistically significant or highly significant results in Pentazocine. Pentazocine as the not unlikely cause for the harmful events in the patients of AMI. Furthermore most of the events or the significant changes in the parameter studied were in the first 24 hours after administration of Pentazocine, where as the occurrence of the same. The studies of Keith J et al [17] on opioids receptors and myocardial protection which reported.

The recent studies of Startiz et al[18] which suggested that delta opioid receptors in the intact rat heart mediate cardio protective effects of ischemic preconditioning and opioids. The reduction in binding affinity of kappa receptor was correlated with reduction in vulnerability of ventricles to fibrillate and incidence of ventricular fibrillation. Pentazocine binds to kappa receptors and acts as an agonist at kappa sites. It stimulates K3 receptor and causes sedation and supraspinal analgesia, while K1 stimulation causes spinal analgesia thus the binding to kappa receptor is increased. These data allowed speculation that the cardioprotective effects of both ischemic preconditioning and opioids was in some way related to activity of delta receptors and inversely to activity of kappa receptor.

Additionally Xia and coworkers[19] proposed that activation of kappa receptors may be a contributing factors for arrhythmia induced by myocardial ischemia and reperfusion. In an in vivo rat model of AMI, opioid receptor stimulation has been observed to result in a reduction in infarct size similar to ischemic preconditioning and was due to involvement of myocardial ATP –Sensitive potassium channel (KATP). Further it was suggested that opening 0 20 40 60 80 Normal (Z=6.44) Poor (Z = Death (Z = 7.46) 3.97) Percentage

It is now firmly and finally accepted by one and all that serum enzymology, like SGOT, LDH and CK measurement is one of the specific markers for diagnosing AMI and predicting its outcome. Patients with AMI have elevated levels of SGOT, LDH, and CPK. Our study revealed that Pentazocine had elevated level of SGOT, LDH, and CPK which were highly significant. Anderson and Damsgard have reported that Pentazocine caused rise in CPK (Creatinine Phosphokinase) and LDH (Lactate Dehydrogenase) isoenzymes but in no case did it exceed the upper normal limits[20].

Clinical status of patients at discharge Pentazocine of KATP channel may be an endogenous protective mechanism in
humans also. Opening of KATP channel is differentially involved in antinociceptive effects of some opioids. Morphine as Pentazocine, Fentanyl or Levorphanol. On the basis of analysis of these in vivo data, it was speculated that and Methadone but not in others such Buprenorphine posses previously unrecognized beneficial cardioprotective effects in patients including those undergoing bypass surgery and those experiencing an AMI.

**Conclusion**

The study can be concluded the pentazocine is contraindicated in patients with AMI as an analgesic has it deteriorate the ECG changes by its ability increase myocardial contractility, Heart rate and cardiac work.

**References**

1. Somervillie MA. Opioids for chronic pain of non malignant origin, corecion or consent health care : Analgesic (Anal.): 1998;3: 12


