**CORONARY ARTERY DISEASE**

**FIVE-YEAR-FOLLOW-UP OF PATIENTS WITH UNSTABLE ANGINA: SURGICAL VERSUS MEDICAL TREATMENT.**

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Among patients (pts) with coronary artery disease those with sympoems of an unstable angina pectoris form a subset particularly jeopardized with regard to threatening myocardial infarction (MI) or cardiac death (CD). We analyzed over 5.4±2.1 years (Y) the clinical course of 123 pts, who between 1977 and 1982 had to be admitted to the intensive care unit for reasons of persisting angina at rest. Within the first 24 hours no patient revealed a significant elevation of serum creatine kinase or typical alterations in the ECG due to acute MI (new Q-waves). During their stay in hospital (19±7 days) 43 pts (37 men, 6 women; age 58±7 Y) were subjected to bypass graft surgery, 60 pts (60 men, 20 women; age 58±10 Y) were medically treated, 13 of whom underwent subsequent bypass graft surgery because of aggravation of symptoms. The table presents a survey of cardiac mortality and incidence of MI in the collectives with medical and surgical treatment during the stay in hospital and 1, 3 and 5 Y after dismissal (calculated according to the life-table method of Kaplan-Meier).

<table>
<thead>
<tr>
<th>Treatment</th>
<th>n = 123</th>
<th>n = 110</th>
</tr>
</thead>
<tbody>
<tr>
<td>in hospital</td>
<td>CD</td>
<td>MI or CD</td>
</tr>
<tr>
<td>1Y</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>3Y</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>5Y</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>* Medically treated pts with subsequent coronary surgery</td>
<td>n = 13</td>
<td></td>
</tr>
</tbody>
</table>

Hence, during the initial hospitalisation infarction and mortality rate in the medically treated group indeed were smaller than in the surgical collective; however, after dismissal this beneficial mortality rate turned into the opposite in the course of the following years. In this group nearly every MI was fatal.

**PLATELET REACTIVITY AND FIBRINOGEN LEVELS IN UNSTABLE CORONARY ARTERY DISEASE (UCAD) .**

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Unstable angina and non-Q-wave myocardial infarction represents the unstable phase of coronary artery disease (CAD). In UCAD a thrombosis in a coronary artery could be the triggering factor of the unstable phase. Increased platelet reactivity and hypercoagulability could be the predisposing factor for developing this condition. Therefore, in patients with UCAD the platelet reactivity was measured as the aggregation response to ADP and collagen, the platelet sensitivity to PGi2 and the release of platelet derived proteins. As an indicator of hypercoagulability the fibrinogen level in plasma was analysed. The control group consisted of patients with chest pain but without CAD. Blood samples for platelet tests and fibrinogen analysis were obtained in the acute phase and after one year.

**Results:**

The only difference in platelet reactivity between cases and controls was noted in their sensitivity to PGi2. The difference remained in the females but not in the males UCAD group after one year.

**Acute phase:**
UCAD Controls
PGi2 1.0ng/ml
Men 43.8±3.0 55.8±2.5
One year:
PGi2 1.0ng/ml
Men 46.6±2.0 50.5±1.7
Women 35.9±2.8 46.2±3.0

In the acute phase a diagnosis of UCAD, elevated weight index and smoking contributed independently to increased fibrinogen levels.

**Conclusion:** The increased fibrinogen level and decreased platelet sensitivity to PGi2 might reflect a hypercoagulable state in patients with UCAD.

**FOUR CASES WITH STABILIZATION OF UNSTABLE ANGINA PECTORIS BY THROMBOLYTIC THERAPY.**

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Acute myocardial infarctions in the vast majority of cases are caused by coronary artery thrombosis at the site of complicated atherosclerotic plaques. By several trials evidence has been given, that myocard can be preserved, when thrombolytic therapy is started within a short period after thrombotic coronary occlusion. Recently, angiographic evidence has been given, that the unstable angina pectoris syndrome frequently is associated with coronary artery thrombosis, too. Thus, thrombolytic therapy should be of comparable benefit for patients suffering from unstable angina pectoris syndrome. Up to now, we have treated four patients suffering from unstable angina pectoris syndrome (two with documented spontaneous reversible ST-segment elevations, two with newly complained recurrent nocturnal episodes of severe angina) with thrombolytic therapy (Pat. 1: 1.500 IE Streptokinase; Pat. 2: 100 mg rt-PA; Pat. 3: 150 mg rt-PA; Pat. 4: 200 000 IE UK). After thrombolytic therapy, all four patients were free of symptoms for at least 60 h. Pat. 3 had recurrence of chest pain with spontaneous reversible ST-segment elevations on the third day after therapy. Pat. 1, 2, and 4 were without clinical symptoms until angiography and secondary intervention (angioplasty (PTCA) /by-pass operation (CABG)). Cardiac catheterization was performed within one week after thrombolytic therapy. In all four patients angiography revealed no patent stenosis at angiography. We conclude, that in unstable angina pectoris syndromes with newly developed nocturnal symptoms and/or spontaneous reversible ST-segment elevations in the ECG can be stabilized by thrombolytic therapy. After thrombolyis, however, recurrence of chest pain may be soon, and PTCA or CABG should be performed as soon as possible.

**HEPARIN AND ACETYL-SALICYLIC ACID (ASA) 75 MG/DAY IN UNSTABLE CORONARY ARTERY DISEASE - EFFECTS ON PLATELET REACTIVITY.**

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In unstable coronary artery disease (UCAD), i.e. unstable angina pectoris (UAP) or non-Q-myocardial infarction (NMI), treatment with heparin or ASA have given encouraging results. The present study attempts to verify the effects of i.v. heparin (5 days) and to evaluate the utility of ASA 75 mg/day (one year). Patients, admitted because of chest pain, who either develops NMI or signs of ischemia in resting or exercise ECGs are included. Within 72 hours patients are randomized to obtain Heparin+ASA, Heparin+Placebo, Placebo+ASA or Placebo+Placebo . Platelet reactivity is studied in vitro in platelet rich plasma (PRP) in a subgroup of patients. The aggregation response is studied after addition of collagen and ADP and after preincubation with prostacyclin before aggregation with ADP. The figures present results from 85 randomised patients tested before, 6 days, one month and one year after start of therapy.

**Conclusion:** In patients with unstable CAD long term treatment with ASA 75 mg/day inhibits collagen induced platelet aggregation and hampers the ADP response. I.v. heparin tends to raise platelet reactivity and reduce the inhibitory effect of prostacyclin. Heparin induced platelet activation is reduced by simultaneous ASA therapy.