9:15

IS A CREATINE KINASE LEAK OF PROGNOSTIC IMPORTANCE IN PATIENTS WITH UNSTABLE CORONARY DISEASE ?

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Significant (>2x normal) elevations in serum creatine kinase (CK) are commonly used to distinguish between a small myocardial infarction (MI) and unstable angina (UA) and a further distinction in UA is sometimes made on the basis of a small CK leak (1-2x normal). The aim of this study was to determine whether these relatively small changes in CK are important prognostically or whether all of these pts should be considered together as having unstable coronary disease.

In a prospective study, we examined the prognostic significance of the peak admission CK in 356 consecutive pts, 264 males, 92 females, mean age 58 ± 8 years, with unstable coronary disease. The pts included 231 with UA and a normal CK, 61 with UA and a CK leak (1-2x normal, mean 321 ± 65 u/L) and 64 with a small CK peak (>2x and <5x upper normal, mean 650 ± 194 u/L). Patients were followed for 12 ± 2 months (mean ± sd) after admission (99% complete).

Events	UA (no CK rise) (%)	UA (CK leak) (%)	Small MI (%)	p
MI	12	21	11	ns
Cardiac death	6	5	5	ns
Readmission with UA	22	19	14	ns
Any event	33	35	25	ns

Conclusion: The magnitude of the peak admission CK level is not an important prognostic factor in patients with unstable angina. The clinical distinction between unstable angina and a small myocardial infarction, based on admission CK estimations, does not reflect a difference in prognosis in the year after presentation.

9:30

MULTICENTER EVALUATION OF A NEWLY DEVELOPED ENZYME IMMUNO ASSAY KIT FOR CARDIAC TROPONIN T

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University Hospital, Heidelberg, FR-Germany; Lazarette, Helsingborg, Sweden; University Hospital, Hamburg, FR-Germany; University Hospital, Odense- Aarhus, Denmark; Inselspital, Bern, Swizerland.

Cardiac troponin T is an unique cardiac antigen which is released from disintegrating myofibrils during acute myocardial infarction (AMI). Therefore circulating TnT might be a very sensitive and specific marker for myocardial cell damage in patients.
TnT was measured semiautomatically with a newly developed enzyme immuno assay (EIA) test kit utilising a cardio- and a non-cardiospecific monoclonal antibody. The detection limit of the assay was 0.20 ug/L with an interassay and interday coefficient of variation of 0.02 and 0.03 at 2 ug/L, respectively. The study groups consisted of 87 patients with definite AMI, 17 patients with micro AMI, 148 patients without AMI and 56 patients with skeletal muscle damage.
The sensitivity, specificity, positive and negative predictive values of TnT were 97%, 97%, 97% and 97%, while the values for CKMB-activity were 89%, 97%, 97% and 90%. TnT appeared slightly earlier in serum than CK and was elevated in all patients from 10.5 -140 hours after onset of pain while CKMB-activity was only elevated from 9.5 - 31 hours. Inclusion of the 56 patients with skeletal muscle damage to the control group reduced specificity of TnT measurements by only 4 % but of CKMB by 11 %.
Thus the diagnostic capability of the TnT EIA for the detection of AMI is superior to CKMB measurements. TnT determinations may be particularly useful in patients with suspected AMI and skeletal muscle damage and in patients with AMI admitted after normalisation of CKMB.

9:45

ELECTROCARDIOGRAPHIC DIAGNOSIS OF EVOLVING MYOCARDIAL INFARCTION WITHOUT TELEMETRY FOR PARAMEDIC-INITIATED PRE-HOSPITAL THROMBOLYSIS.

Michael O'Rourke, Alison Cook, David Gallagher, Gerard Carroll, John Hall. St Vincent's Hospital, Sydney, Australia.

Paramedic-initiated thrombolysis without EKG telemetry in evolving myocardial infarction requires extremely high specificity and acceptable sensitivity, so that patients without infarction do not receive therapy while patients with evolving infarction incur no undue delay before hospital treatment. Using a Marquette PC machine with 12 SL algorithm, together with listed ST segment change in 11 leads, we evaluated a series of 505 patients with chest pain syndromes or other suspected or real cardiac disease and 21 patients with acute evolving infarction within 24 hours of symptom onset. The best discriminant for evolving infarction comprised the combination of: (A) the standard TIMI criteria for ST elevation (ST segment elevation of at least 2 mm in 2 or more leads V1-V3, or ar least 1 mm in 2 or more leads V4-V6, aVL or I, or at least 1mm in 2 or more of leads II, III, aVF) together with (B) ST segment depression of at least 0.5 mm in one reciprocal lead and (C) an interpretive comment of "injury" or "infarction". This combination yielded sensitivity of 71% (15/21) and specificity of 98% (496/505). When 9 patients with known previous infarction and persistent ST elevation were excluded, specificity was 100%. These criteria were evaluated prospectively in a series of 179 patients outside hospital by paramedics and fulfilling other criteria for urokinase treatment of evolving infarction. There were 2 apparent false diagnoses (RBBB, and ST elevation without appropriate ST depression); specificity was 98%. Sensitivity remained over 60%. Paramedic-initiated diagnosis of evolving infarction is practical without telemetry provided patients with prior Q wave infarction are excluded.

Thursday, March 7, 1991
8:30AM–10:00AM, Room 360, West Concourse
Doppler Assessment of Left Ventricular Filling Dynamics

8:30

EVALUATION OF THE EFFECT OF PRELOAD ON TRANSMITRAL FLOW VELOCITIES IN SEVERE HEART FAILURE

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It is known that LV systolic dysfunction causes abnormalities of LV filling, but evaluation of LV filling is confounded by changes in preload. To address this problem, 8 dogs were instrumented with micromanometer catheters to measure LA pressure, peak +dP/dt and -dP/dt and tau (time constant of isovolumic pressure decay); and with ultrasonic crystals to measure ejection fraction (EF) and peak rate of LV filling in end-diastolic volumes/s (dV/dt/v). Doppler was used to measure mitral peak E and A velocities, E deceleration time (DT) and deceleration slope (DS). The dogs were then paced 3 weeks at 225-250 bpm. EF fell from 36±6 to 11±6% ($p<0.01$), LA pressure increased from 10±4 to 29±6 mmHg ($p<0.001$) and +dP/dt decreased from 1523±305 to 978±290 mmHg/s ($p<0.01$). LV filling was assessed at high LA pressure (CHF High) and at LA pressure reduced to baseline (CHF Low) by partial inferior vena caval occlusion.
Results: (mean±SD *=p<0.01 vs baseline, ·=p<0.01 vs CHF High).

	Baseline	CHF High	CHF Low
E (cm/s)	60±10	66±11	44±11*·
A (cm/s)	28±5	26±9	33±8
E/A ratio	2.2±0.4	2.7±0.9	1.4±0.3*·
DT (ms)	95±30	58±16*	74±31
ES (cm/s)	680±210	1223±394*	652±220·
Tau (ms)	36±10	42±8	34±11
dV/dt/v (EDV/s)	3.2±0.6	1.5±0.8*	1.0±0.4*
-dP/dt (mmHg/s)	1470±400	992±186*	1021±261*
Heart rate (bpm)	61±22	107±23*	96±19*

Conclusions: 1) High preload causes pseudonormalization of early-diastolic filling velocities. 2) DT and DS primarily reflect changes in preload and not LV filling. 3) dV/dt/v primarily reflects LV filling and is less strongly affected by preload and 4) Tau and -dP/dt appear essentially independent of preload.