

924-118 The January 17, 1994 Los Angeles Earthquake as a Trigger for Acute Myocardial Infarction

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Background; The January 17, 1994 Los Angeles earthquake (EQ) provided a unique opportunity to examine the effect of extensive environmental disaster associated with tremendous emotional stress on the incidence of acute cardiac events. **Methods;** We surveyed 75 coronary care units (CCUs) in Southern California comparing the number of admissions for myocardial infarction (MI), unstable angina (UA), total CCU admissions and related in-hospital mortality in the week before (Jan 10–16) and after (Jan 17–23) the EQ. Five CCUs were excluded because of insufficient data. The relative risk for MI following the EQ was assessed in a subgroup of 29 CCUs that were able to provide data on the equivalent period in 1993.

Results; Total number of pts (average number ±SD per CCU):

Cases	Week Before	Week After	p Value
MI	147 (2.1 ± 2.7)	195 (2.9 ± 3.1)	0.01
UA	255 (3.6 ± 5.2)	273 (3.9 ± 5.2)	0.39
Total CCU pts	1096 (15.7 ± 13.2)	1174 (16.8 ± 13.2)	0.18

The % of CCU admissions for MI increased in the week following the EQ [195 of 1174 (16.6%) vs. 147 of 1096 (13.4%); $p = 0.04$]. The % of CCUs reporting increased number of MIs was higher in hospitals located within 15 miles of the EQ epicenter vs. hospitals located >15 miles [11 of 14 (79%) vs. 23 of 56 (41%); $p = 0.02$]. In-hospital mortality (%) was similar for MI and UA pts in the week before and after the EQ (MI: 7% vs. 6%; UA: 1% vs. 0%, respectively). In comparison to 1993 (subgroup of 29 CCUs), there was a significant increase in the risk of developing MI in the week following the EQ vs. the week before (1994: 70 vs. 41 pts; 1993: 49 vs. 68 pts, respectively; odds ratio 2.4, 95% confidence interval 1.4–4.0). **Conclusions;** 1) Our data indicate a significant increase in the incidence of MI but not UA or in-hospital mortality in the week following the EQ. 2) Our observation supports the concept that a stressful event can trigger acute MI.

924-119 Angiographic Findings in Diabetics Treated with Thrombolytic Therapy After Acute Myocardial Infarction

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The GUSTO Angiographic Study has shown that some subgroups with particularly high mortality rates (such as elderly patients) have less recovery of left ventricular (LV) function despite documented infarct related artery (IRA) patency. In order to investigate the correlates of a known higher mortality associated with diabetes mellitus (DM) in the setting of acute myocardial infarction (AMI), we examined IRA patency and LV function in the GUSTO Angiographic Study cohort of 160 patients with DM and 1019 non-diabetics (NDM) randomized to 90 minute and 5–7 day follow-up catheterization after initiation of thrombolytic therapy. The thirty day mortality for DM was 11.9% and 5.8% for NDM ($p = 0.004$). At 90 min angiography, 65 (40.6%) of DM had normal (TIMI 3) IRA flow compared with 385 (37.8%) of NDM ($p = NS$). Total patency rates at 90 min (TIMI 2 and 3 combined) were 70.1% for DM and 66.6% for NDM ($p = NS$). Left ventricular (LV) function indices expressed by ejection fraction (EF), extent of infarct (# of abnormal chords) and regional wall motion (SD/Chord) at 90 min and 5–7 day follow-up ventriculography are shown below (all $p = NS$).

	90 Min Ventriculography		5–7 Day Ventriculography	
	DM	NDM	DM	NDM
EF (%)	60 ± 45	60 ± 0.7	59 ± 1.7	60 ± 0.7
# Chords	20 ± 1.9	22 ± 0.9	15 ± 1.9	17 ± 0.9
SD/Chords	-2.4 ± 0.1	-2.4 ± 0.1	-2.0 ± 0.2	-1.9 ± 0.1

Conclusion; The excess mortality in AMI in diabetic patients cannot be explained by patency rates (equivalent to non-diabetics) nor by initial or follow-up regional or global left ventricular function.

924-120 The Sick-Euthyroid Syndrome in Acute Myocardial Infarction

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The sick-euthyroid syndrome, alterations of thyroid function tests by non-thyroidal illness as manifested by a low free T₃ (FT₃) or both a low FT₃ and a low free T₄ (FT₄), has been well described in non-cardiac patients admitted to medical intensive care units and has been shown to offer prognostic

information. In order to determine whether similar abnormalities are present in patients with myocardial infarction (MI), we studied 658 patients admitted to the Duke Coronary Care Unit (CCU) between 2/92 and 10/93. Only patients with thyroid profiles (FT₃ and FT₄ by Becton Dickinson radioimmunoassay) obtained within 48 hours of a documented MI were included in this analysis. Clinical data were prospectively obtained through the Duke CCU Database. 434 patients (66%) had a low FT₃ (<3 pg/ml) and 131 (20%) had both a low FT₃ and low FT₄ (<0.85 ng/dl). The mortality of the groups vs. those without these abnormalities was: low FT₃ 14.3% versus 4% ($p = 0.0001$); both low FT₃ and low FT₄ 17.6% vs. 9.1% ($p = 0.008$).

To further delineate the relationship of the sick-euthyroid syndrome in MI, we separately analyzed the thyroid profiles of the most critically ill patients, those with cardiogenic shock (CS). In this analysis, there were 533 patients without CS and 125 patients with CS. The mortality of patients without CS was 3.4%, and in those with CS was 42.4% ($p = 0.0001$). We found the sick-euthyroid syndrome to be more common in patients with CS than in those without CS: low FT₃ 86.4% in patients with CS vs. 61.2% in patients without CS ($p = 0.0001$), both low FT₃ and low FT₄ in 29.6% with CS vs. 17.6% in those without CS ($p = 0.004$). The mortality of patients with cardiogenic shock who had a low FT₃ 47.2% was higher than in those without a low FT₃ 11.8% ($p = 0.003$).

We conclude that the sick-euthyroid syndrome, often manifest as a low free T₃, is frequent in patients with MI, is more common as patients develop cardiogenic shock, and is associated with a higher mortality.

925 Hypertrophic Cardiomyopathy

Monday, March 20, 1995, 3:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

925-39 Prenatal Hypertrophic Cardiomyopathy and its Association with Amniotic Fluid Insulin in Fetuses of Diabetic Mothers

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The frequent presence of a disproportionate hypertrophic ventricular septum in infants of diabetic mothers, as well as its benign course, has been widely reported. This phenomenon has also been demonstrated in the fetus. The most accepted hypothesis to explain this disorder, yet to be proved in the prenatal period, is fetal hyperinsulinism due to inadequate control of maternal diabetes. This prospective controlled transversal study was carried out to test the hypothesis that hypertrophic cardiomyopathy during prenatal life in diabetic pregnancies is related to fetal hyperinsulinism as assessed by amniotic fluid insulin levels. Sixty-five fetuses (37 of diabetic mothers and 28 of hypertensive mothers) were studied sequentially and non-intentionally. A comprehensive M-mode and cross-sectional Doppler echocardiogram, with color flow mapping, was performed within one week of the amniocentesis in every case. Fetal insulin levels and interventricular septum thickness were considered abnormal when their values were above 2 standard deviations, according to local nomograms. Statistical analysis utilized Student's "t" test, Fisher's exact test, ANOVA and Kruskal-Wallis test. The mean gestational age at the time of examination was 32.3 ± 3.5 weeks (24–39 weeks) in the gestational diabetes group, 28.5 ± 2.7 weeks (23–33 weeks) in the previous diabetes group and 30.6 ± 3.1 weeks (25–36 weeks) in the control group. Mean septal thickness was 3.7 (2.2 to 6.3 mm) in the group of diabetic mothers and 3.0 (2.0 to 4.3 mm) in the control group ($p < 0.01$). Mean fetal insulin levels was 14.7 ± 16.8 microIU/ml (3.2 to 75.3 microIU/ml) in the group with diabetes and 6.4 ± 3.3 microIU/ml (3.1 to 17 microIU/ml) in the control group ($p < 0.01$). Median insulin levels were 8.7 and 5.5 microIU/ml in the diabetes and in the control group, respectively. Ten out of 37 fetuses of the diabetic group were considered to have hypertrophic cardiomyopathy (27%), while none of the fetuses of the control group showed increase in the septal thickness ($p < 0.01$). A significant association between prenatal hypertrophic cardiomyopathy and fetal hyperinsulinism was demonstrated ($p < 0.007$).

We conclude that there is association between the presence of hypertrophic cardiomyopathy during prenatal life and high levels of fetal insulin in diabetic mothers, thus justifying efforts to improve the glucemic control in pregnancies with this frequent complication.