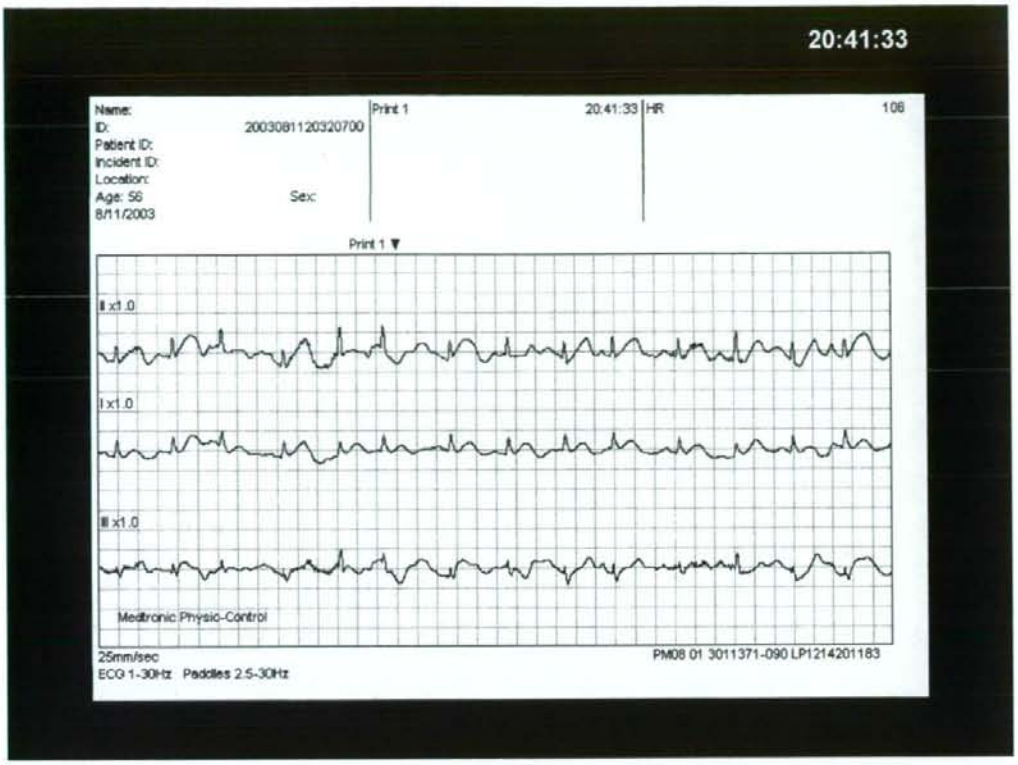
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Age: 56
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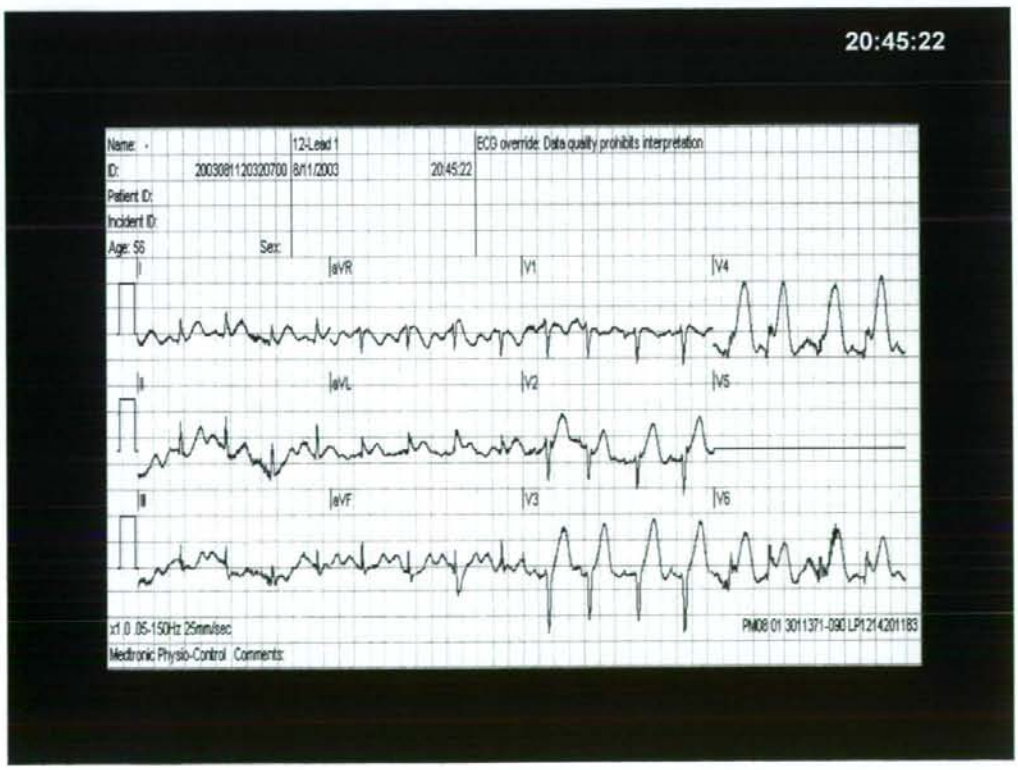
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Incident ID:
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Sex:
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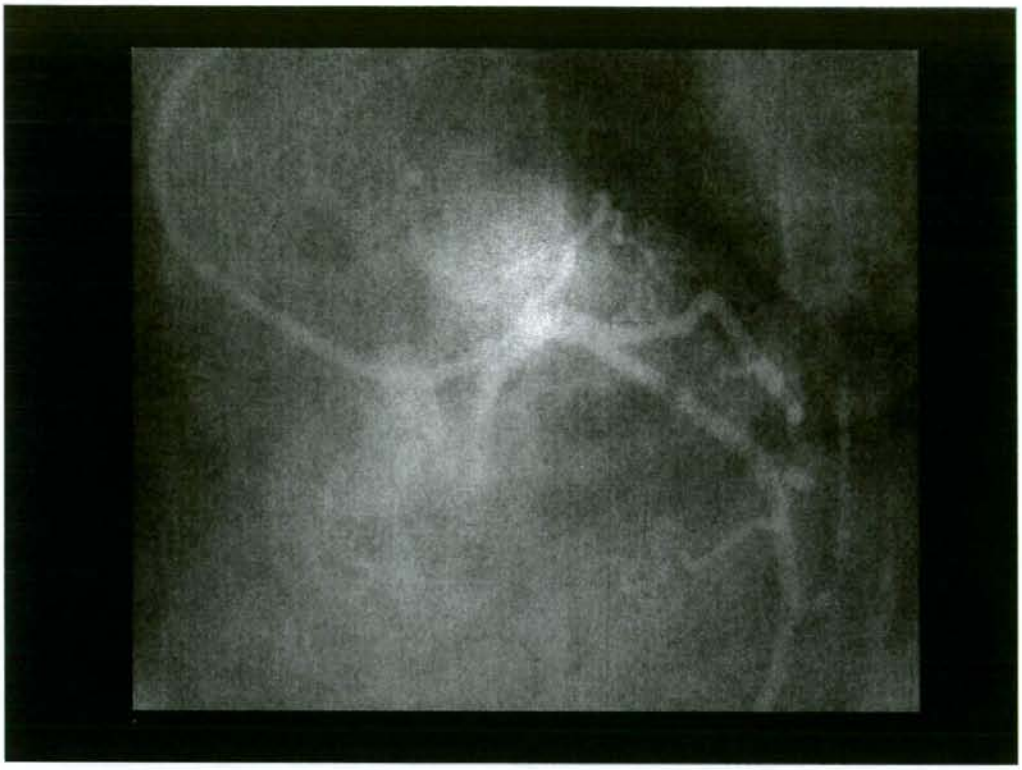
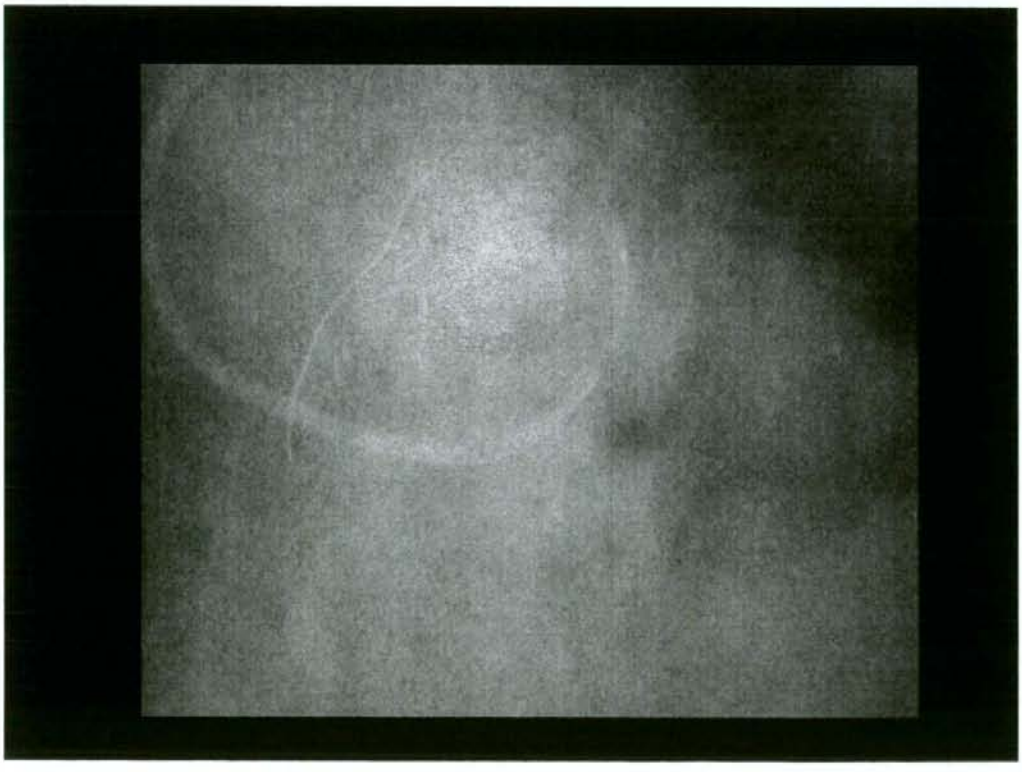
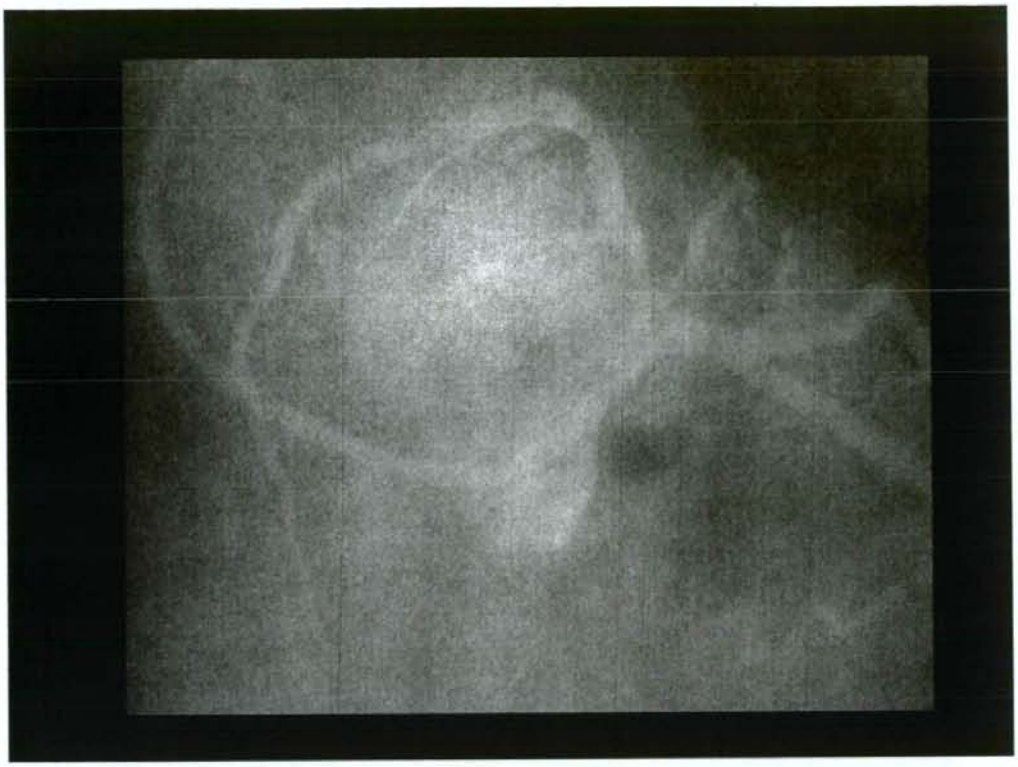
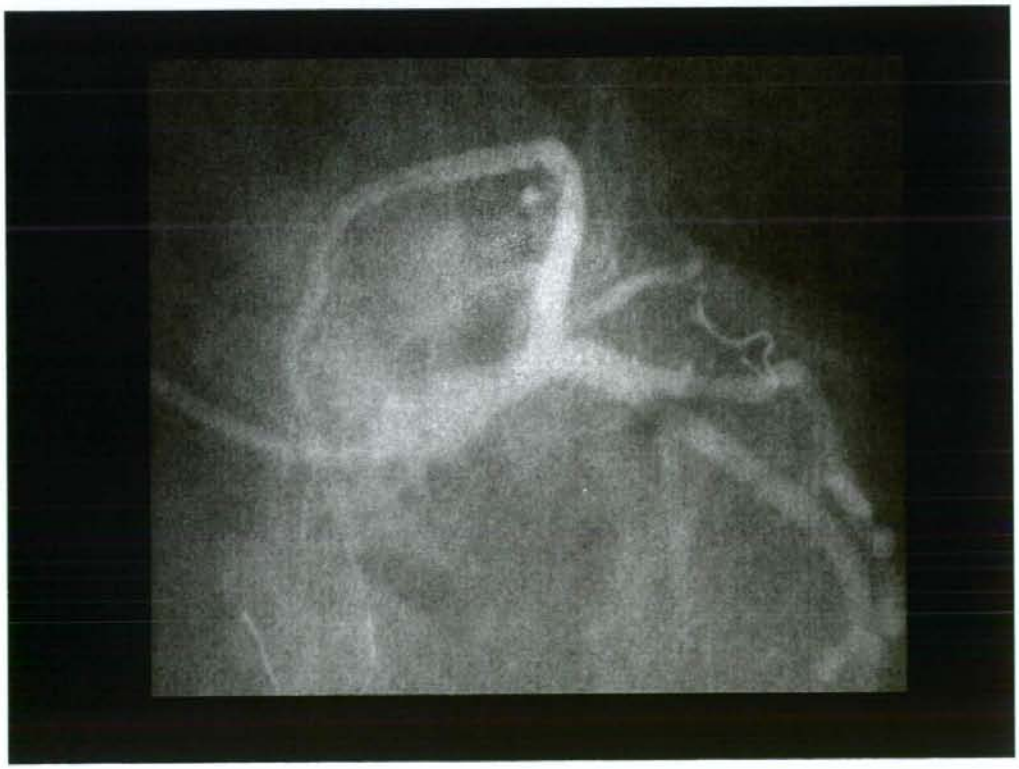








Early Cardiac Catheterization and PCI

- Who should undergo such?
- When should it be done?
- Does it Improve Outcome?

Spaulding et al.

- 1994-1996
- 1762 patients with OOH CA
  - 910 had resuscitation efforts attempted
    - 312 were resuscitated in the field
    - 126/312 died in route to hospital
    - 102/312 were excluded for non-cardiac causes of their CA
    - 84/312 were taken to cath lab after successful resuscitation
- 60/84 (71%) had clinically significant CAD
- 40/84 (48%) had total occlusions
  - 37/84 (44%) had PCI attempted
    - 28/37 (76%) were successful
- In-hospital survivor rate was 38%

NEJM 1997;336:1629

Spaulding et al.

"Clinical and electrocardiographic findings, such as chest pain and or ST elevation on the ECG were poor predictors of acute coronary occlusion."

NEJM 1997;336:1629

Coronary Intervention for STEMI: Post Resuscitation ?

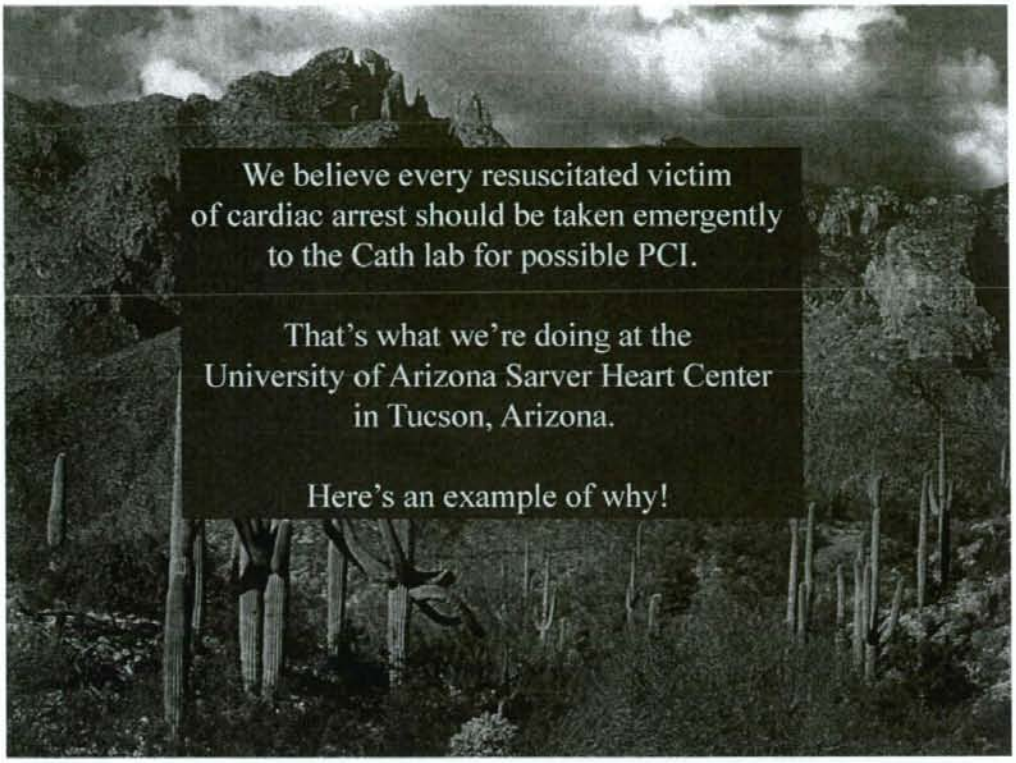
- 13 clinical series have been reported in the recent era
  - N = 744 patients
    - 462/744 (62%) survived to hospital discharge
    - 308/376 (82%) of survivors had good neuro fx

Combined Therapeutic Hypothermia and Coronary Intervention Post Resuscitation

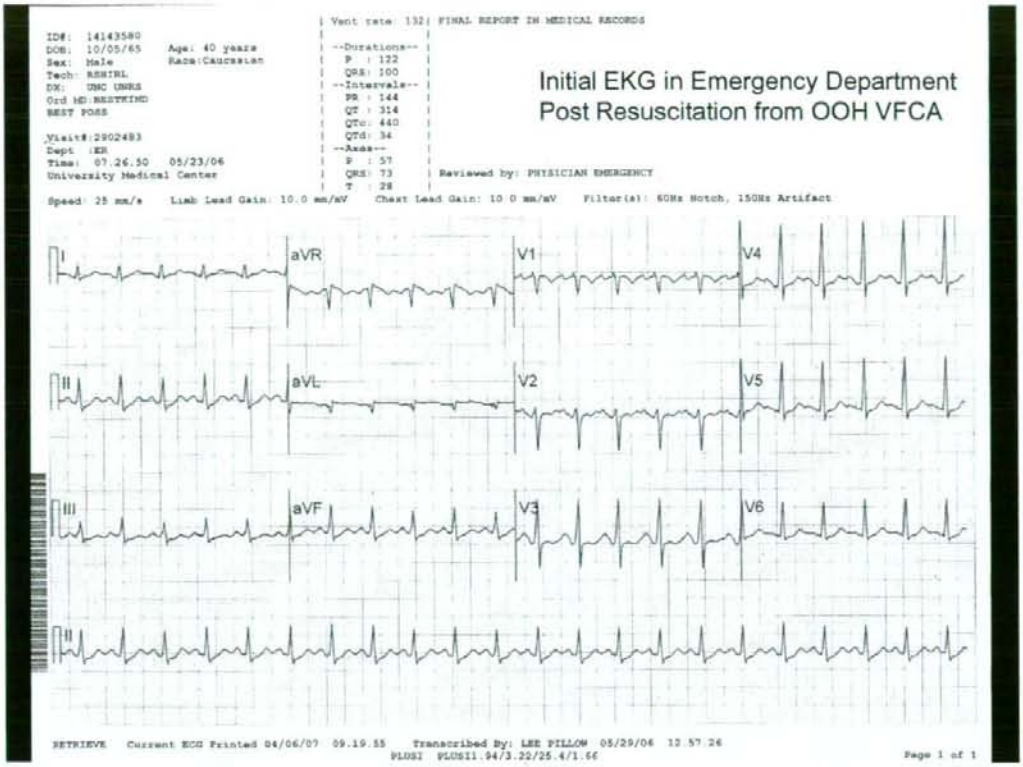
- 3 clinical reports now
  - N = 106 patients
    - 483/106 (78%) survived to hospital discharge
    - 67/83 (81%) of survivors had good neuro fx

Immediate Coronary Angiography Post Resuscitation

- Resuscitated patients with STEMI on ECG
  - Yes !
- Resuscitated patients without STEMI on ECG
  - Yes/No? ⟶ Less certain but Probably
    - Can't always tell by 12 lead ECG post resuscitation who has an occluded coronary vessel!

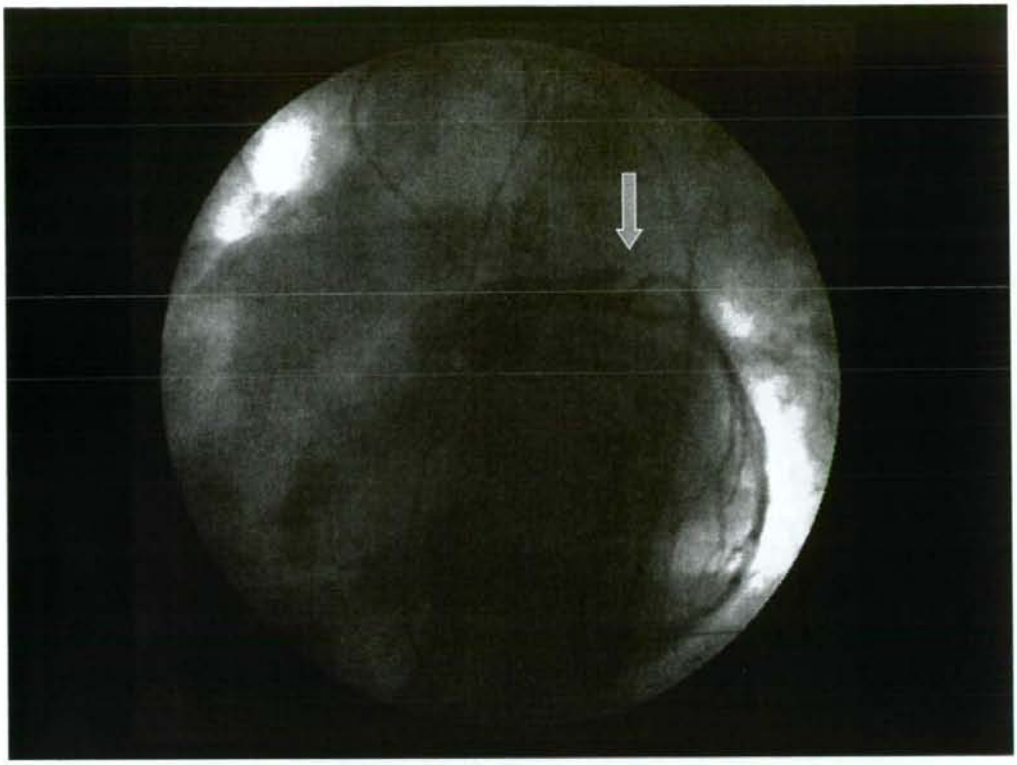


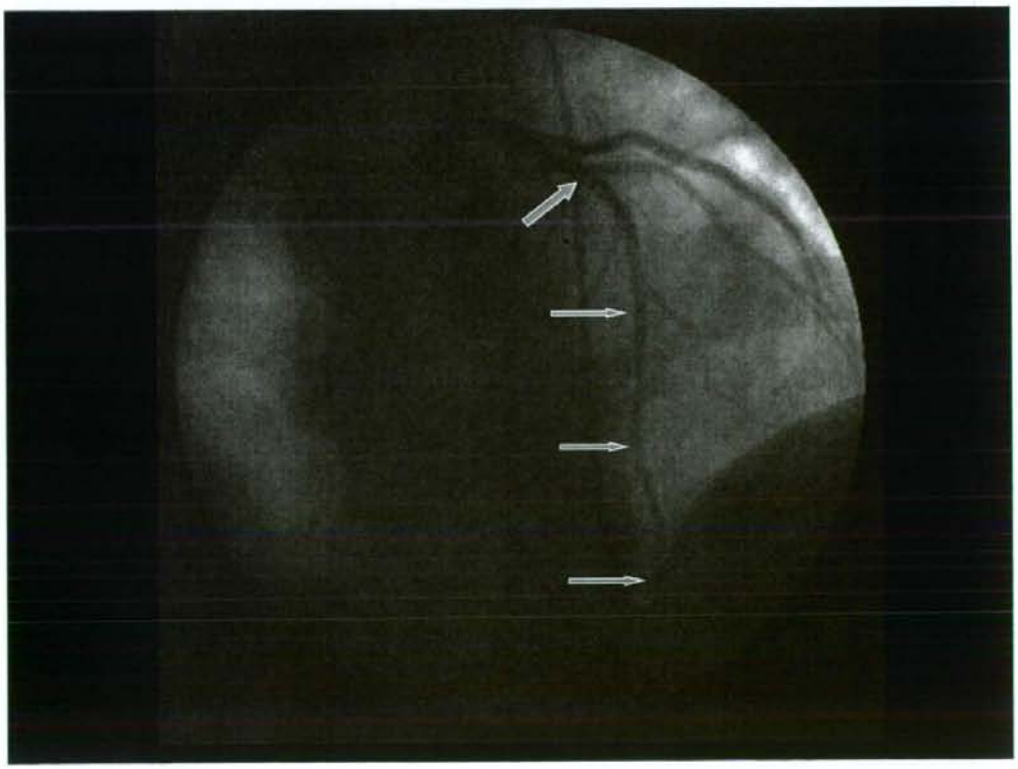
- 40 yr old male
  - Athletic swimmer collapsed in shower post work out
  - Chest compression only BLS immediately
  - AED brought w/i 5 min and 2 shocks given
  - Pulse present on EMS arrival, but comatose



Immediate Angiography/PCI or NOT ?

- Stat ED echocardiographic exam: decreased anterior wall motion
- Decision was then made to take to the CCL
- Mild therapeutic hypothermia started while in the ED





- Echo after PCI: LVEF = 20%

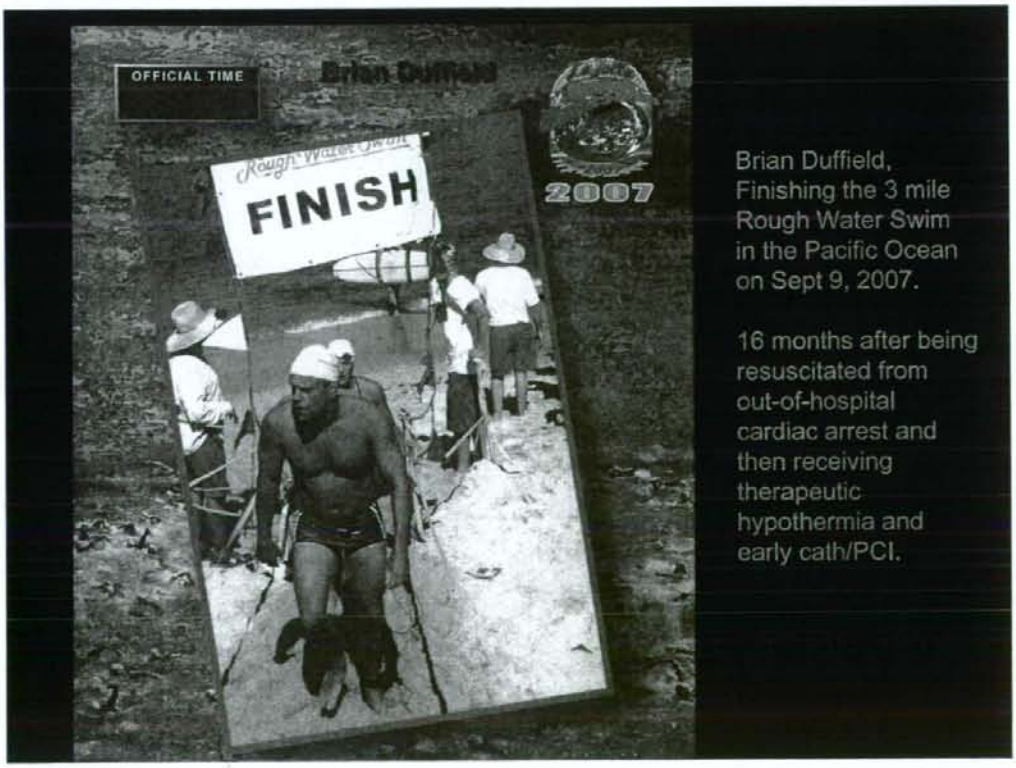
- Warmed up after 24 hours
- COMPLETELY NORMAL CNS Function

- Discharged 5 days later
- Business trip the following week

- Repeat Echo 6 weeks later:

  - LVEF = 50% with minimal septal hypokinesis

Newsweek Cover July 23, 2007



Brian Duffield, Finishing the 3 mile Rough Water Swim in the Pacific Ocean on Sept 9, 2007.

16 months after being resuscitated from out-of-hospital cardiac arrest and then receiving therapeutic hypothermia and early cath/PCI.

Brian and Carolyn Duffield at the AHA Heart Ball, Tucson, Arizona February 2, 2008.

Brian was the Guest of Honor and spoke about his experience with Sudden Cardiac Death.

Aggressive Post Resuscitation Care is the 3rd Pillar of Cardiocerebral Resuscitation

What Can the Survival Rates Be with All Three Pillars of CCR ???

Doubling of the survival rates achieved with the first two pillars:

All rhythms:
7% to ~15% ?

Witnessed Ventricular Fibrillation:
25% to ~50% ?

MORE

CCR Saves Lives !

THE UNIVERSITY OF ARIZONA
SARVER HEART CENTER

Cardiocerebral Resuscitation Really Does Saves More Lives !!


Another Opportunity to
Improve …

Ⅷ. 業績集

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Letter to the Editor

Hyperintensity on T2-weighted magnetic resonance imaging in Takotsubo cardiomyopathy

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Received 17 May 2007; received in revised form 13 July 2007; accepted 3 August 2007
Available online 20 February 2008

Abstract

The mechanism underlying the association between Takotsubo cardiomyopathy (TC) and myocardial injury is unknown. We describe a signal hyperintensity zone on T2-weighted magnetic resonance imaging (T2WI) in 2 cases of atypical and typical TC. In these cases, a transmural hyperintensity zone on T2WI agrees with a region of abnormal left ventricular wall motion. We suggest that edematous viable myocardium is present transmurally in the acute phase of TC.

Keywords: Cardiomyopathy; Magnetic resonance imaging; Pathogenesis

1. Introduction

Takotsubo cardiomyopathy (TC) is usually recognized as a transient left ventricular apical ballooning, which has been widely reported in Japan [1]. The diagnosis of TC consists of chest symptoms, ECG changes mimicking acute myocardial infarction, a reversible left ventricular local asynergy (usually apical ballooning) without significant coronary artery stenosis and a limited release of cardiac markers disproportionate to the extent of asynergy. Recently, several variant abnormal wall motions of TC have been also reported [2–4]. Although reportedly TC is closely related to the activity of catecholamine [5], the underlying mechanism remains unknown. In the present report, we demonstrate hyperintensity on T2-weighted magnetic resonance imaging (T2WI) of 2 cases with TC and suggest one possible aspect of the pathogenesis of TC.

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0167-5273/$ - see front matter
doi:10.1016/j.ijcard.2007.08.046

2. Case report

2.1. Case 1

A 28-year-old woman was admitted to the hospital because of dyspnea after sudden onset of vomiting and headache. Chest radiography showed pulmonary congestion and the ECG showed ST elevation in lead aVL and ST depression in leads II, III, aVF, and V_4 through V_6. Emergency coronary angiography revealed normal coronary arteries. Transthoracic echocardiography showed akinesis of the basal and mid-ventricle and hyperkinesis of the apical wall (Fig. 1a), the so-called "inverted TC" contractile pattern. The patient was treated with dobutamine infusion and stabilized on day 3. Repeated echocardiography on day 7 revealed normal wall motion. A computed tomography scan of the abdomen revealed a left adrenal mass and ^{123}I-metaiodobenzylguanidine scintigraphy showed uptake at the left adrenal region, establishing the diagnosis of pheochromocytoma. Cardiac magnetic resonance (CMR) imaging showed a transmural hyperintensity zone on T2WI (Fig. 1c) without late gadolinium enhancement (Fig. 1b). This hyperintensity zone on T2WI agreed with the region of

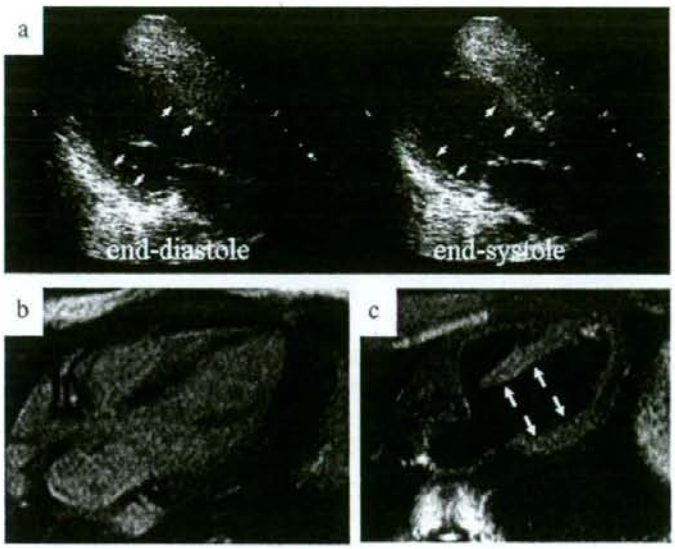

Fig. 1. a. Arrows indicate the endocardiac aspects both in the end-systolic and end-diastolic phases on transthoracic echocardiography, showing akinesis of the basal and mid-ventricle and hyperkinesis of the apical wall, the so-called "inverted Takotsubo cardiomyopathy" contractile pattern. b. No obvious late gadolinium enhancement on cardiac magnetic resonance. c. Demonstration of the transmural hyperintensity zone (arrows) in the T2-weighted image consistent with mid-ventricle asynergy by transthoracic echocardiography.

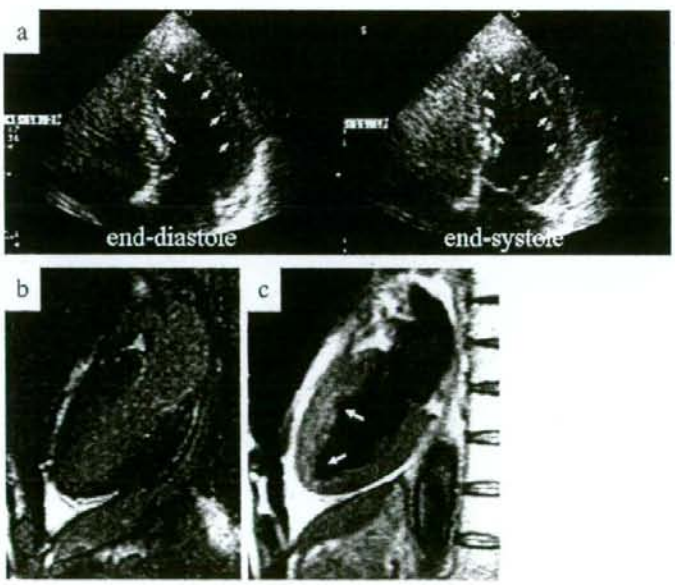

Fig. 2. a. Arrows indicate the endocardiac aspects both in the end-systolic and end-diastolic phases on transthoracic echocardiography, showing akinesis of the apical wall, which is a typical Takotsubo cardiomyopathy contractile pattern. b. No obvious late gadolinium enhancement on cardiac magnetic resonance. c. Demonstration of the transmural hyperintensity zone (arrows) in the T2-weighted image consistent with apical asynergy by transthoracic echocardiography.

abnormal left ventricular wall motion detected by transthoracic echocardiography.

2.2. Case 2

A 68-year-old woman was admitted to the hospital because of chest pain and progressive dyspnea on exertion. Chest radiography showed pneumothorax of the left lung and the ECG showed ST elevation and inverted T waves in leads II, III, aVF and V_2 through V_6. Coronary angiography revealed normal coronary arteries and coronary vasospasm was not provoked by ergonovine maleate. Transthoracic echocardiography showed akinesis of the apical wall, which was a typical TC contractile pattern (Fig. 2a). Pneumothorax was treated using a trocar thoracic catheter. A repeated echocardiography on day 18 revealed normal wall motion without any treatment. CMR on day 9 showed a transmural hyperintensity zone on T2WI (Fig. 2c) without late gadolinium enhancement (Fig. 2b). This hyperintensity zone on T2WI agreed with the region of abnormal left ventricular wall motion detected by transthoracic echocardiography.

3. Discussion

To the best of our knowledge, this is the first report of T2WI in TC. An absence of late gadolinium enhancement on CMR has been recently reported in the heart with TC in spite of severe ventricular dysfunction [6]. The present two cases with TC did not show late gadolinium enhancement on CMR, but demonstrated a transmural hyperintensity zone on the T2WI in the region of the abnormal left ventricular wall motion.

CMR is established as a major technique on pathophysiology of cardiovascular disease and solving clinical problems [7]. It has been reported that late gadolinium enhancement on CMR can be used to assess the infarct size [8] and potential myocardial viability [9] in the patients with myocardial infarction. Several authors also reported that T2WI is a useful technique to distinguish the acute myocardial infarction from old myocardial infarction by the presence or absence of a transmural hyperintensity zone. Although theoretically many factors can affect the myocardial T2WI, tissue T2WI is a method that assesses magnetic image contrast which is directly affected by change in tissue biochemistry, especially tissue water content [10]. Reimer and Jennings demonstrated that edema and inflammation are pathological findings in an animal myocardial infraction model, not only in the infarcted myocardium but also transmurally in the ischemic myocardium with increased total water content [11]. Moreover, T2WI abnormalities were closely correlated with increased myocardial total water content in animal models [12,13]. These findings suggest that abnormality on T2WI can be useful in identifying myocardial edema. On the other hand, late gadolinium enhancement [14,15] for myocardial viability assessment depends on gadolinium-DTPA, which does not traverse cell membranes but remains within the extracellular space [16,17]. Importantly, late gadolinium enhancement indicates the relative changes of the extracellular and intracellular volumes, which is the volume of distribution [18,19]. The acute myocardial infracted zone shows late gadolinium enhancement on CMR as a result of the disproportionately increased apparent extracellular space [20]. For edematous viable myocardium, the ratio of the extracellular and intracellular volumes is not substantially altered [21]. Therefore, the peri-infarct zone, which involves the edematous viable myocardium but not the infracted non-viable myocardium, does not exhibit significant late gadolinium enhancement on CMR.

The ST elevation on ECG appears in almost all patients with TC in the acute phase [1], indicating the transmural myocardial injury. In the present report, we demonstrated that edematous viable myocardium indicated by T2WI is present transmurally in the acute phase of TC. Although the mechanisms underlying the association between TC and myocardial injury are unknown, one possible aspect may be direct myocardial injury by catecholamine [5] but not persistent microvascular obstruction. We conclude that edematous viable myocardium may be present transmurally in the acute phase of TC.

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Original Article

Effects of the Y Chromosome on Cardiovascular Risk Factors in Japanese Men

Yumiko HIURA[1], Yasue FUKUSHIMA[1], Yoshihiro KOKUBO[2], Tomonori OKAMURA[2], Yoichi GOTO[3], Hiroshi NONOGI[3], Rie TAKAHASHI[1], and Naoharu IWAI[1,3]

Excess cardiovascular risk in men compared with women has been suggested to be partly explained by effects of the Y chromosome. However, inconsistent results have been reported on the Y chromosome's genetic influence on blood pressure and lipid levels. The purpose of the present study was to settle the question whether genetic variants of the Y chromosome influence cardiovascular risk factors using a large epidemiological cohort, the Suita study. Possible influences of the Y chromosome polymorphisms (Y chromosome *Alu* insertion polymorphism [YAP], M175 and SRY+465) on cardiovascular risk factors were assessed in 974 Japanese men. The frequency of the YAP(+) allele in our study sample was 0.31. The prevalence of hypertension tended to be higher in YAP(+) than in YAP(–) men, and this tendency was found to be stronger among men aged 65 years or older. Men with the YAP(+) genotype had higher levels of high density lipoprotein (HDL) cholesterol compared with those with the YAP(–) genotype, even after adjustment for age, body mass index, and daily ethanol and cigarette consumption (57.0±14.6 mg/dL *vs.* 54.2±14.2 mg/dL, nominal $p=0.011$, adjusted $p=0.0062$). However, these observed nominal associations disappeared after adjusting for multiple testing (Bonferroni). No association was detected between the YAP genotype and myocardial infarction. Similarly, none of the associations with M175 and SRY+465 attained significance when multiple testing was taken into account. In conclusion, Y chromosome polymorphisms (YAP, M175 and SRY+465) do not appear to be associated with cardiovascular risk factors in Japanese men. Studies using much larger sample sizes and/or additional independent samples will be required for definitive conclusions. (*Hypertens Res* 2008; 31: 1687–1694)

Key Words: Y chromosome, polymorphism, risk factors

Introduction

Higher mortality rates from cardiovascular disease in men than in women are consistent findings among studies of different populations including the Japanese (*1–3*), and male gender has been recognized as an important risk factor. The greater risk associated with male gender may be partly due to sex-related differences in the prevalence of hypertension (*4, 5*) and/or dyslipidemia (*6*). Although the level of significance was not given, in a study of 8,168 Japanese subjects undergoing general health screening, the mean levels of systolic blood pressure (SBP), diastolic blood pressure (DBP), low density lipoprotein (LDL), cholesterol, and triglyceride (TG) were all higher in men ($n=5,244$) than in women ($n=2,924$) (*7*). In that study, mean high density lipoprotein (HDL) cholesterol levels were lower in men than in women. Sex-related differences in HDL cholesterol levels were also evident among eld-

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The present study was supported by a research grant from the Program for the Promotion of Fundamental Studies in Health Sciences of the National Institute of Biomedical Innovation, Japan.

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Received April 2, 2008; Accepted in revised form June 12, 2008.