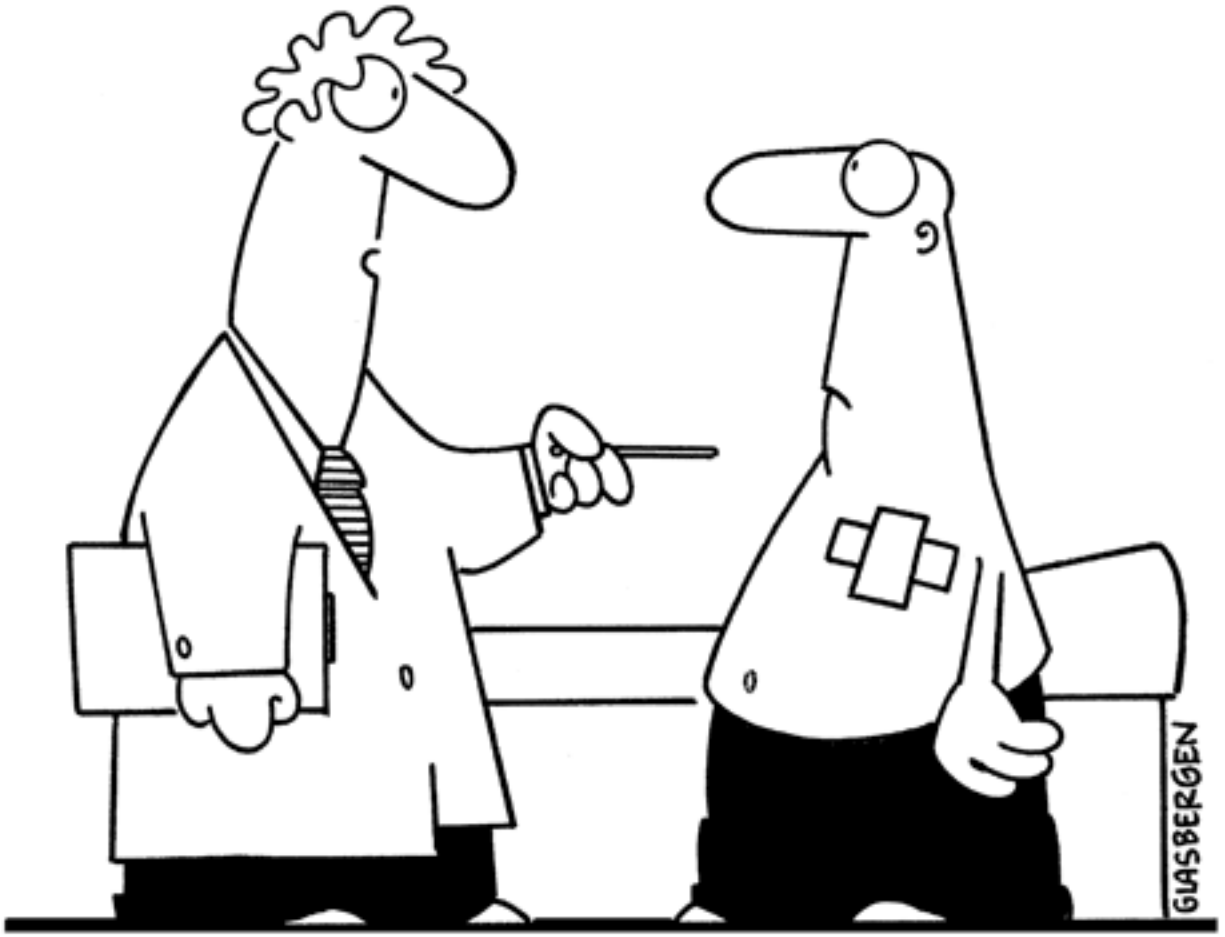
“Takatsubo Cardiomyopathy”
varying clinical presentation

Oct 12 2013

Padma B Hari MD FACC

Summit Cardiology



"It's a pacemaker for your heart, plus you can download apps for your liver, kidneys, lungs, and pancreas!"

Case - 1

CH is a 68 yr old female who presented to the ER with chest pain.

She was a scheduled speaker at roxbury center, for alcohol deaddiction as
She was a recovered alcoholic.
She was very nervous before the speech and noticed some palpitations during the
Speech and subsequently some chest pressure, and shortness of breath.

1-2 days prior was stung by several bees while working in
Her garden with some bushes; She had extensive areas of bee stings on both her
Arms and forearms, on her skin at various stages of healing.
She did not use any epinephrine shots, but was using topical cortisol.

Brief past medical history:
Known CAD- remote RCA stent, normal LV function prior to this episode.
Permanent A fib
Hypertension, hyperlipidemia
History of CHF – diastolic.
Permanent pacemaker for tachy brady syndrome.

Case - 1

Initial labs:
Mildly increased WBC – 16.6
Hbg – 16
Normal chemistry
Troponin- 0.58, normal CK and MB fractions.

Initial EKG

CH

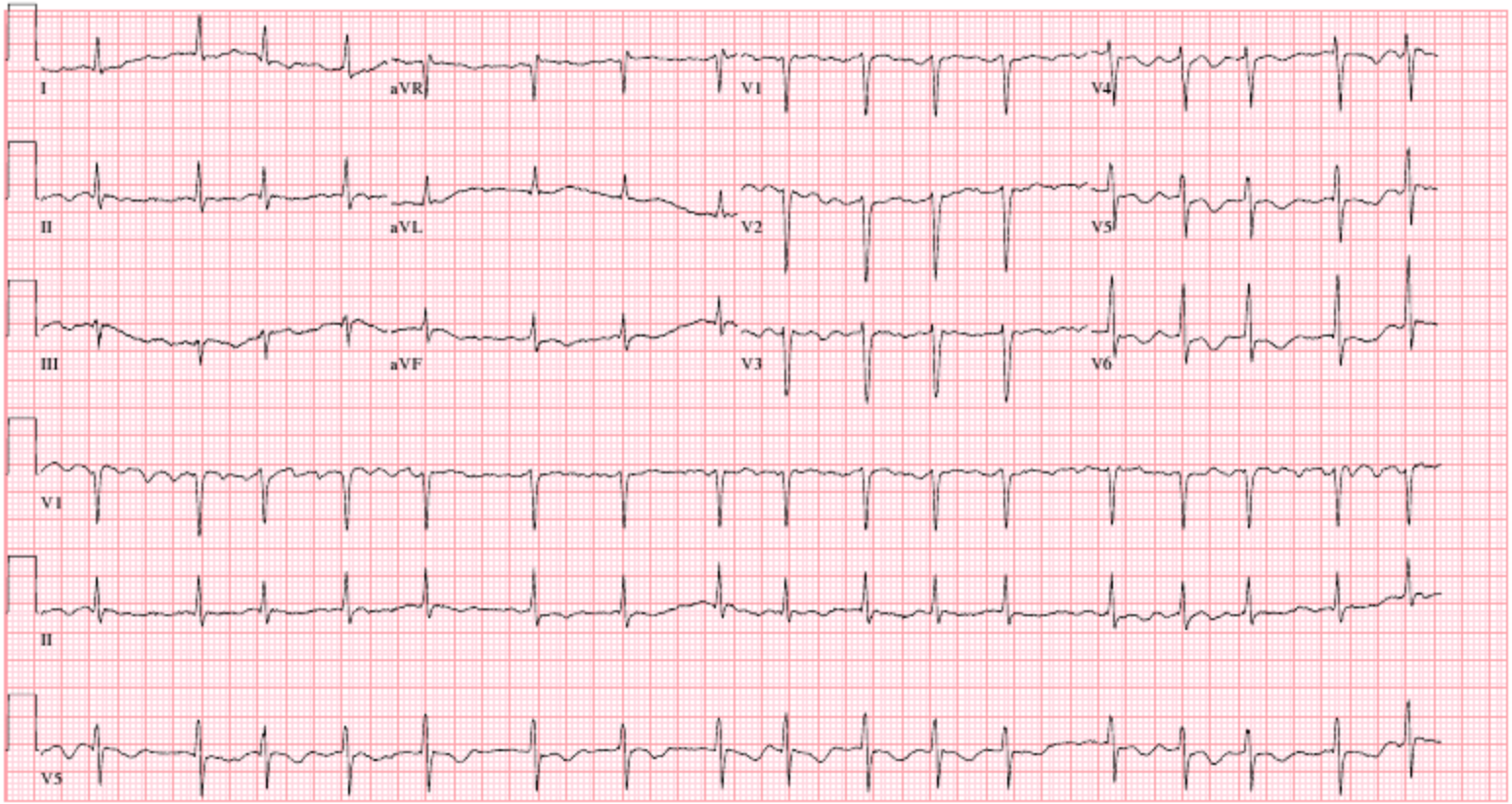
ID:HSM0002337 18-AUG-2009 22:09:02 SUMMIT HEALTH

Vent. rate	102	BPM
PR interval	*	ms
QRS duration	90	ms
QT/QTc	428/557	ms
P-R-T axes	* 10	-52

Technician:JARROD KELLER
Test ind:SHORTNESS OF BREATH

Referred by: Joseph Thornton Electronically signed by: BETSIE FIGUEROA-CRUZ MD

ORDER DR::BOCCHINO COMMENT::



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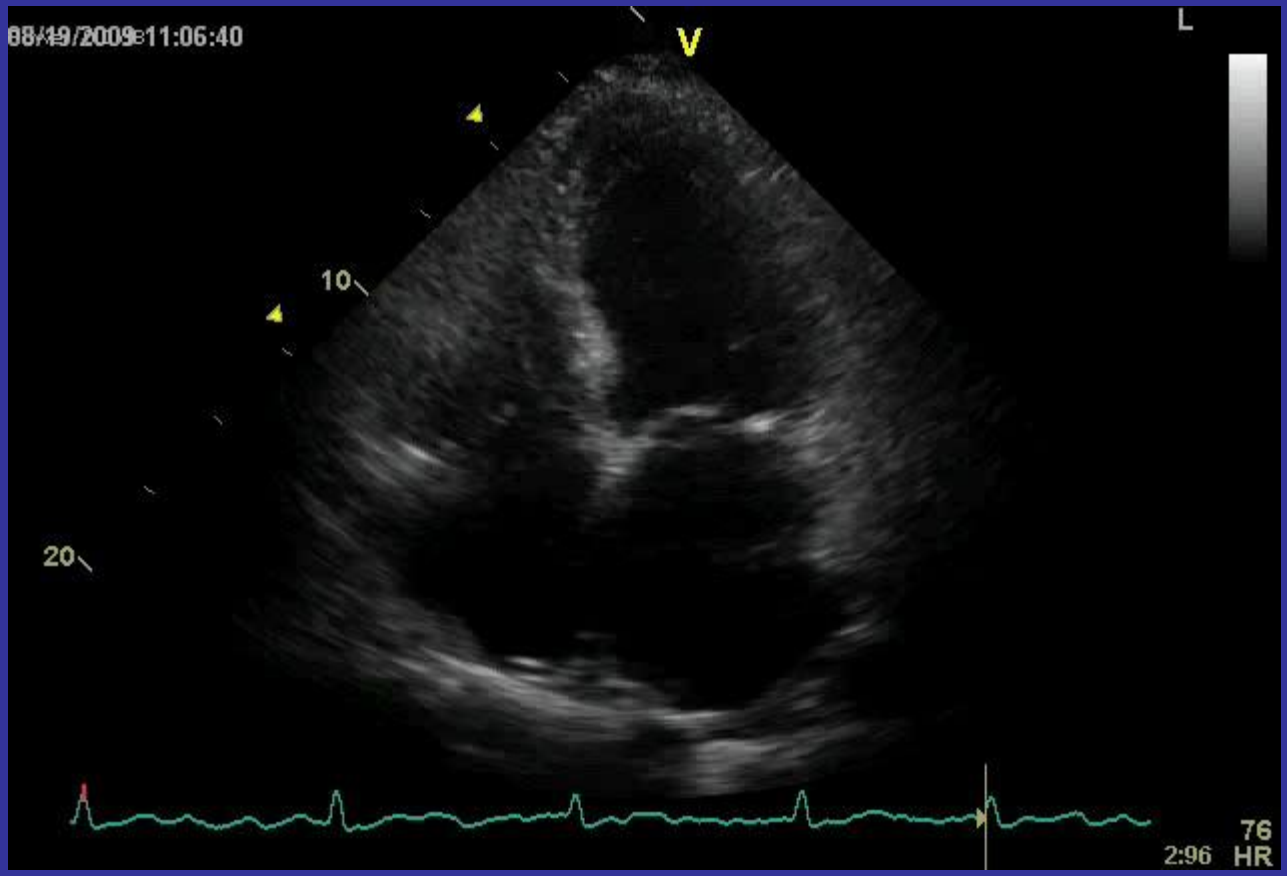
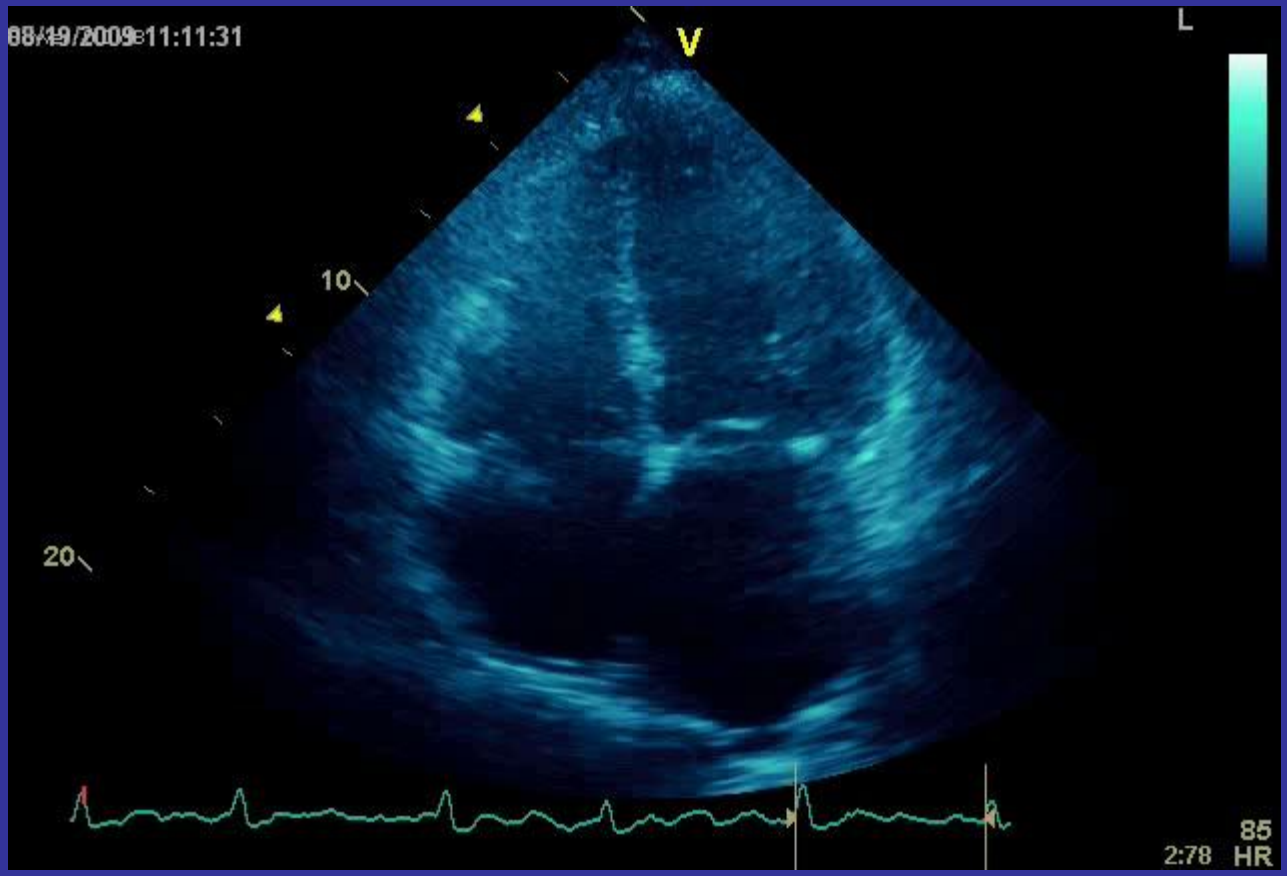
Case - 1

Subsequent hospital course –
Seen by cardiology and stat bedside echo done.
Troponins and CK-MB fraction continued to rise
INR – 2.0

CH episode 1



CH episode 1



Emergent cardiac cath pursued
Had 20% RCA and 30% LCX.
LV gram - 25% ejection fraction, dilated LV and apical dyskinesis
LVEDP moderately increased at 25.

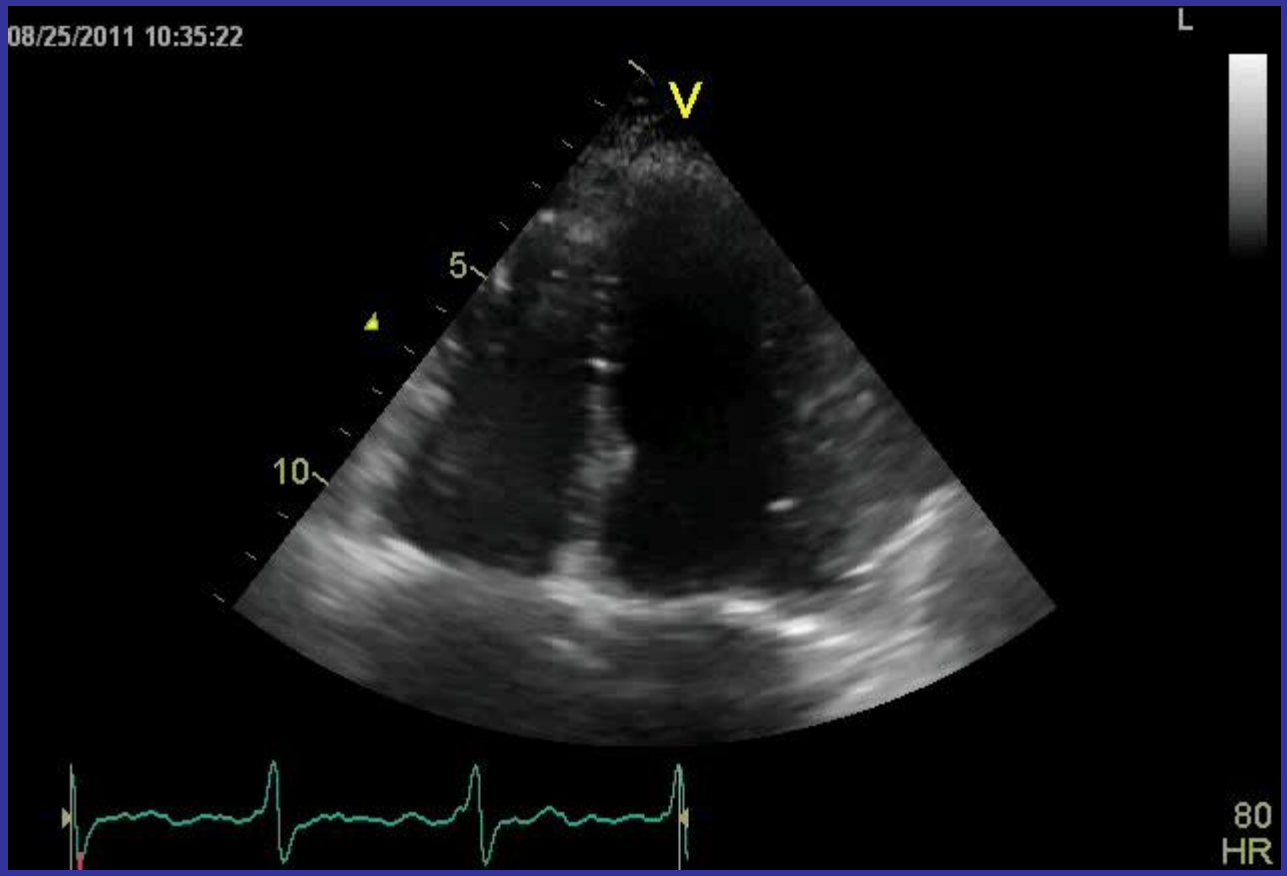
Case -1

Subsequent hospital course
Uneventful; She was started on optimal heart failure meds
Betablockers, ACE inhibitors, aldactone, nitrates and diuretics.

Follow up echo in 11/2009 – Normalized LV function with and EF of
50-55%, improvement of regional wall motion with normal apical wall motion

Had routine office follow ups and another echo 1 yr later 12/2010 revealed a
Sudden drop in her ejection fraction again – 30-35%, with some regional
Wall motion abnormalities.

Clinical suspicion for obstructive CAD was low, given absence of any
Associated clinical symptoms. Reviewing her records, her pacer
Interrogation revealed that she was AV paced – 80-90% of time.
RV pacing induced cardiomyopathy was suspected and she was referred for
An upgrade into a Biventricular device with a defibrillator. EF improved but she
Eventually expired from advanced pancreatic/liver CA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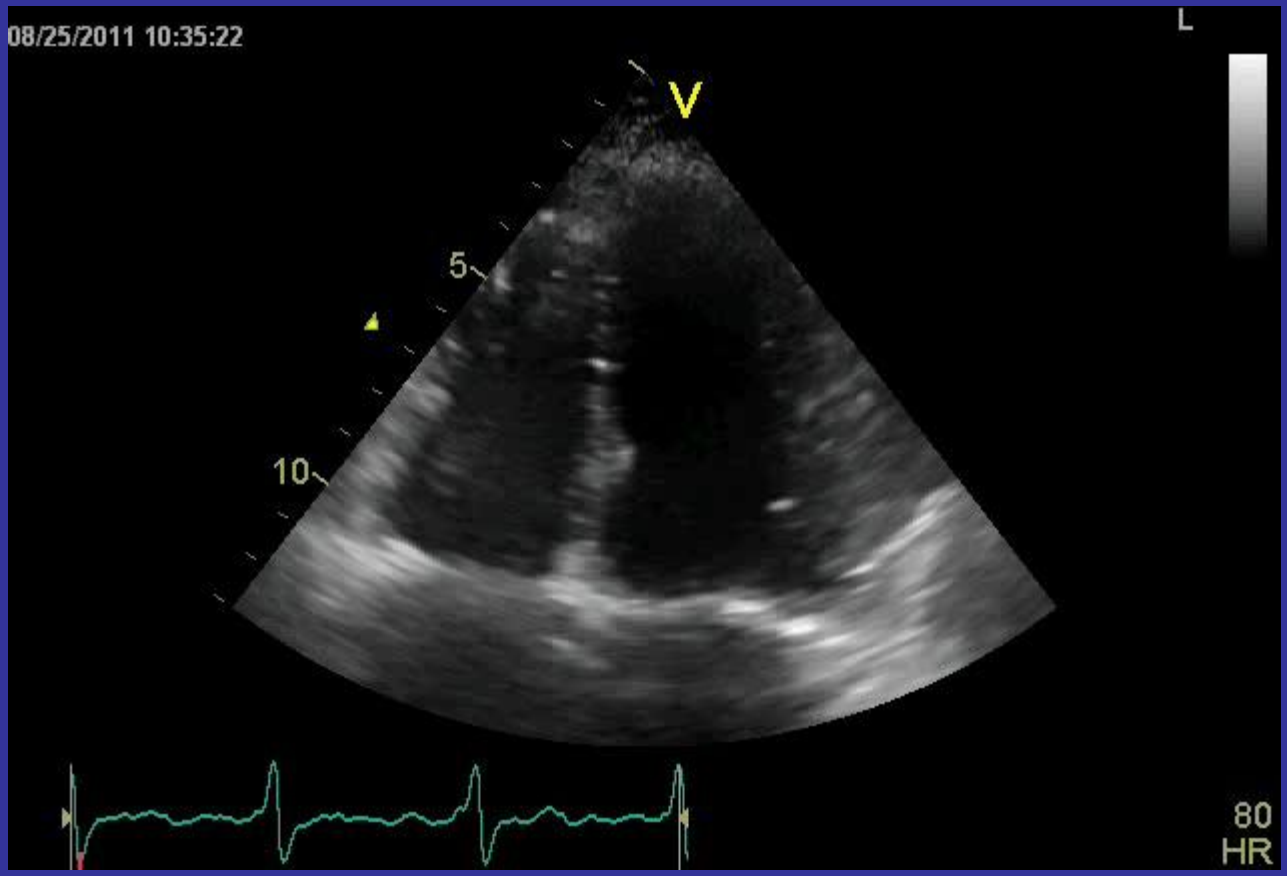
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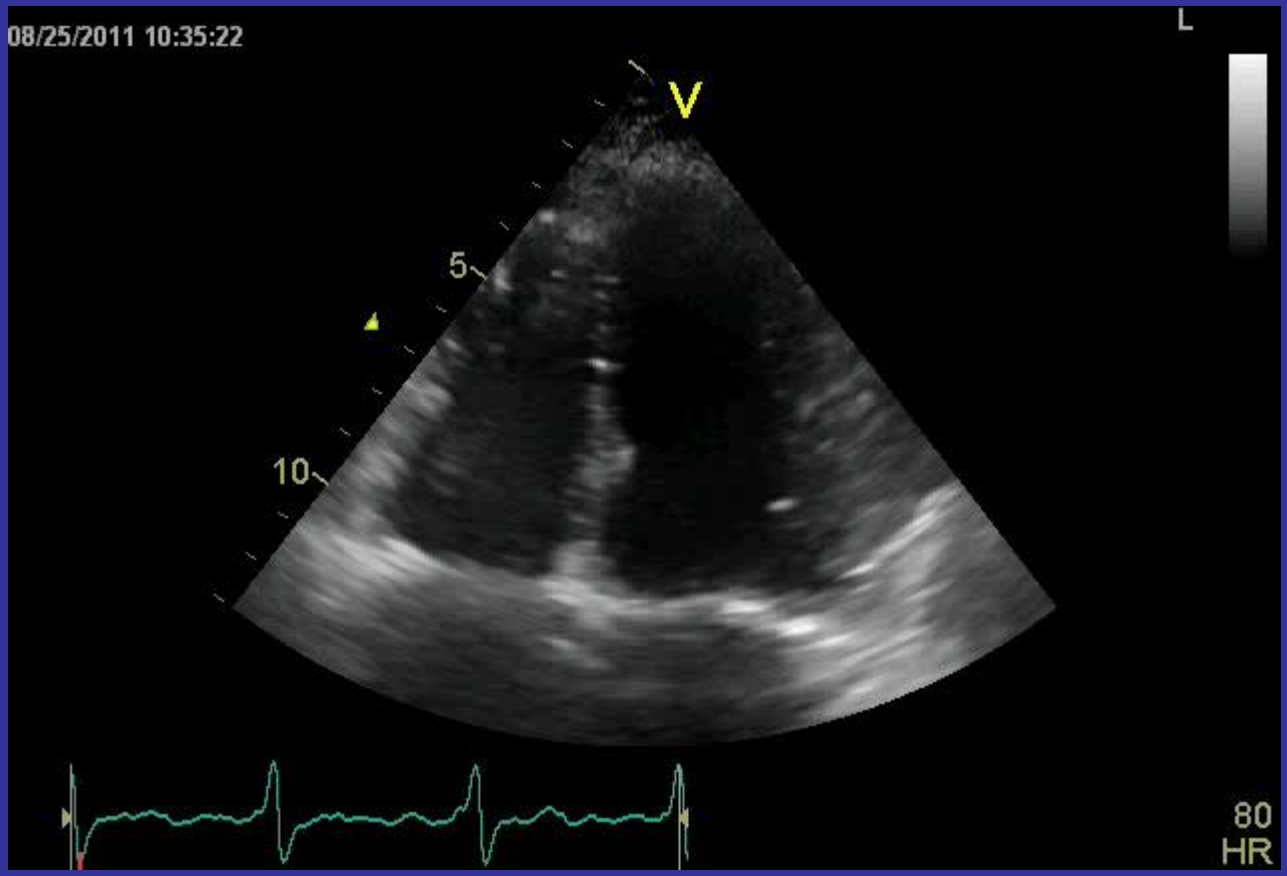
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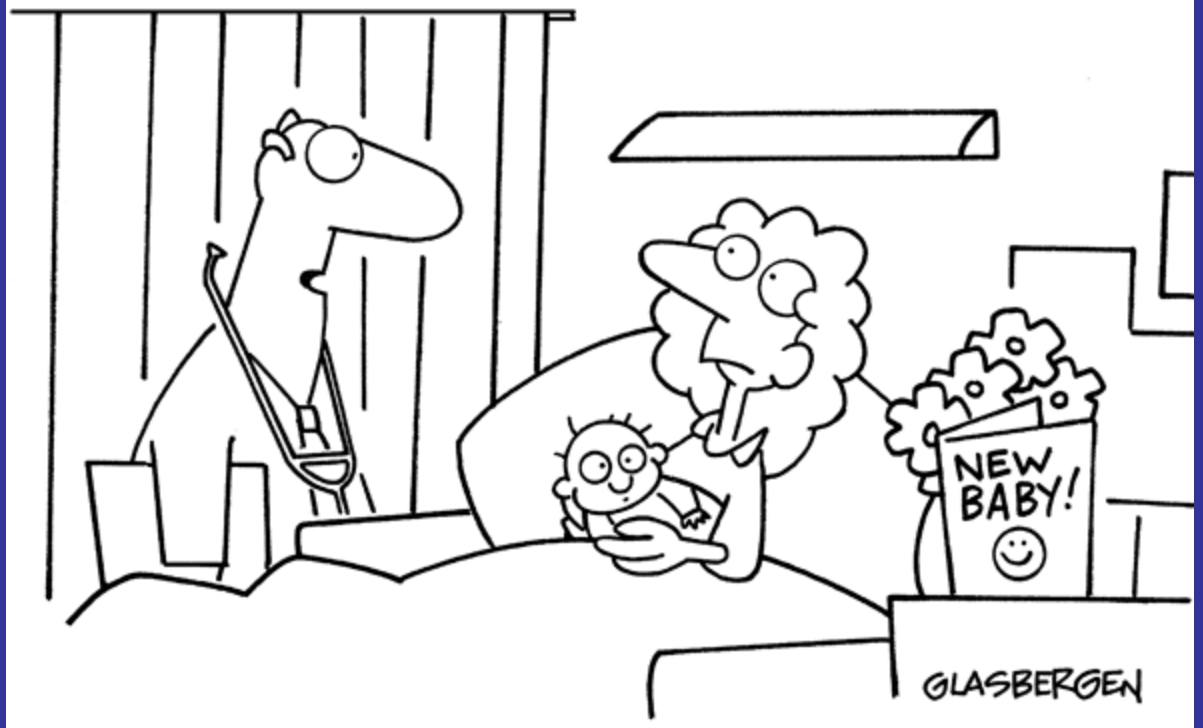


"It's a new medical technology. Instead of crying, we can program your choice of 200 fun ring tones!"

Case -2 - Nov 2007

First hospital course - 11/2007
EO – 79/F
Presents to ER 11/2007 with
CC: severe substernal chest pain radiating to her back
Symptoms started few minutes after hearing the news of her youngest son's death

Past Medical history:
Untreated hyperlipidemia
H/o stroke

Social history:
No smoking, alcohol, lives independently with husband

Physical exam:
CP free after some s/l nitro
Pulse: 80-90/mt, BP initially 170/104mm HG
Cardiac: S1 S2 regular, Lungs : Clear
Abdomen: soft Ext - edema

EO

Case -2 - Nov 2007

SUMMIT HEALTH

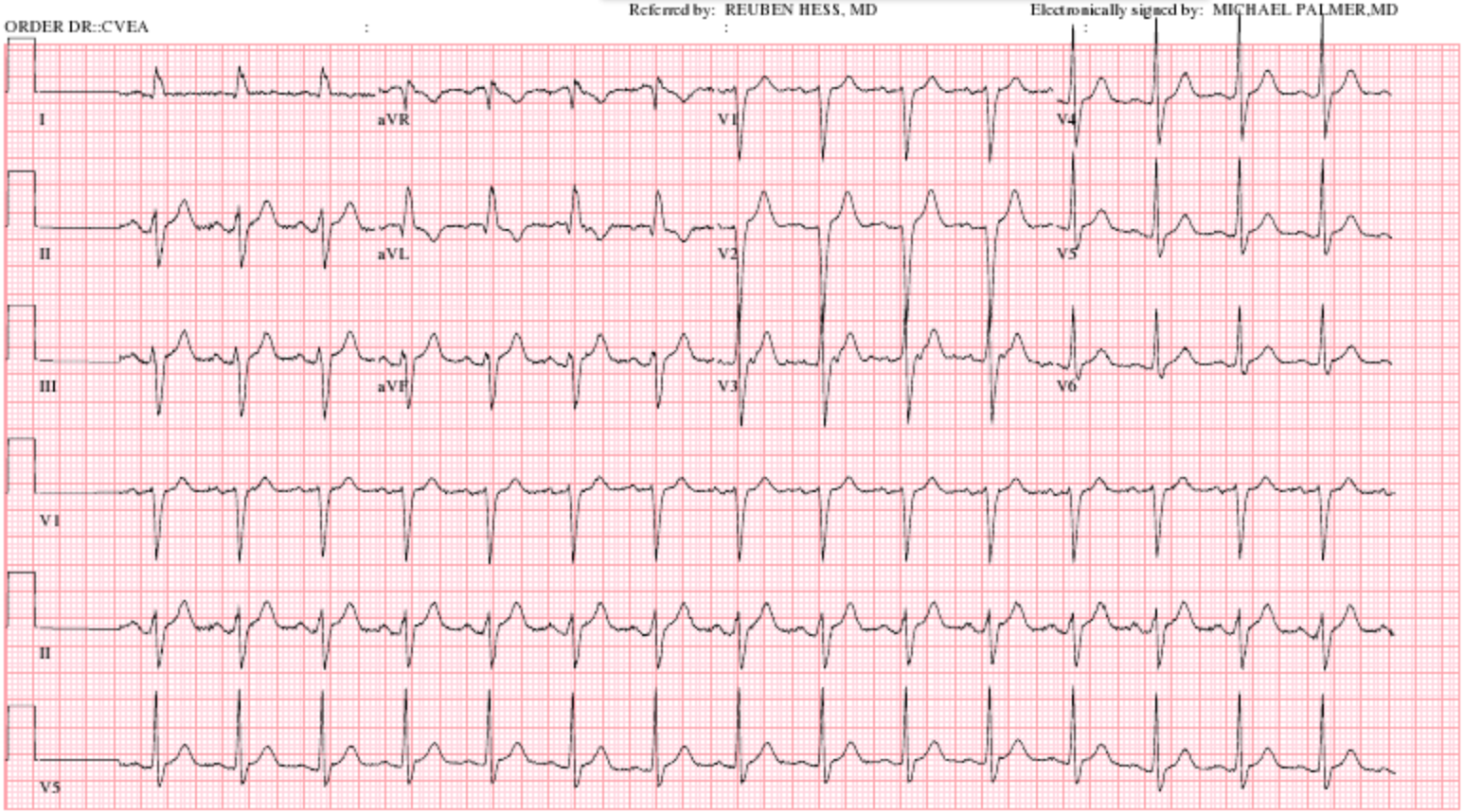
Vent. rate	98	BPM
PR interval	154	ms
QRS duration	112	ms
QT/QTc	356/454	ms
P-R-T axes	60 -52	84

Technician:CANDACE BANFE
Test ind:CHEST PAIN 222

Referred by: REUBEN HESS, MD

Electronically signed by: MICHAEL PALMER,MD

ORDER DR::CVEA



25mm/s 10mm/mV 150Hz 7.1.1 12SL 237 CID: 9

SID: H321496 EID:171 EDT: 11:08 29-NOV-2007 ORDER: 1128-0146 ACCOUNT: H00030500987

Case -2 - Nov 2007

Labs on admission:

Na 131, Bun/Creat – 15/0.8,

Total Ck, CK-MB, troponin- 100/4.3/0.18
Subsequent trend: 142/14.4/4.3; 170/16.1/4.2

BNP on admission- **252 increased to 3017 in 24 hrs.**

Initial plan was to proceed with non invasive ischemia work up once enzymes improved

However in 12 hrs, had recurrence of chest pain and subsequent EKG repeated and echo was done.

EO

Case -2 - Nov 2007

SUMMIT HEALTH

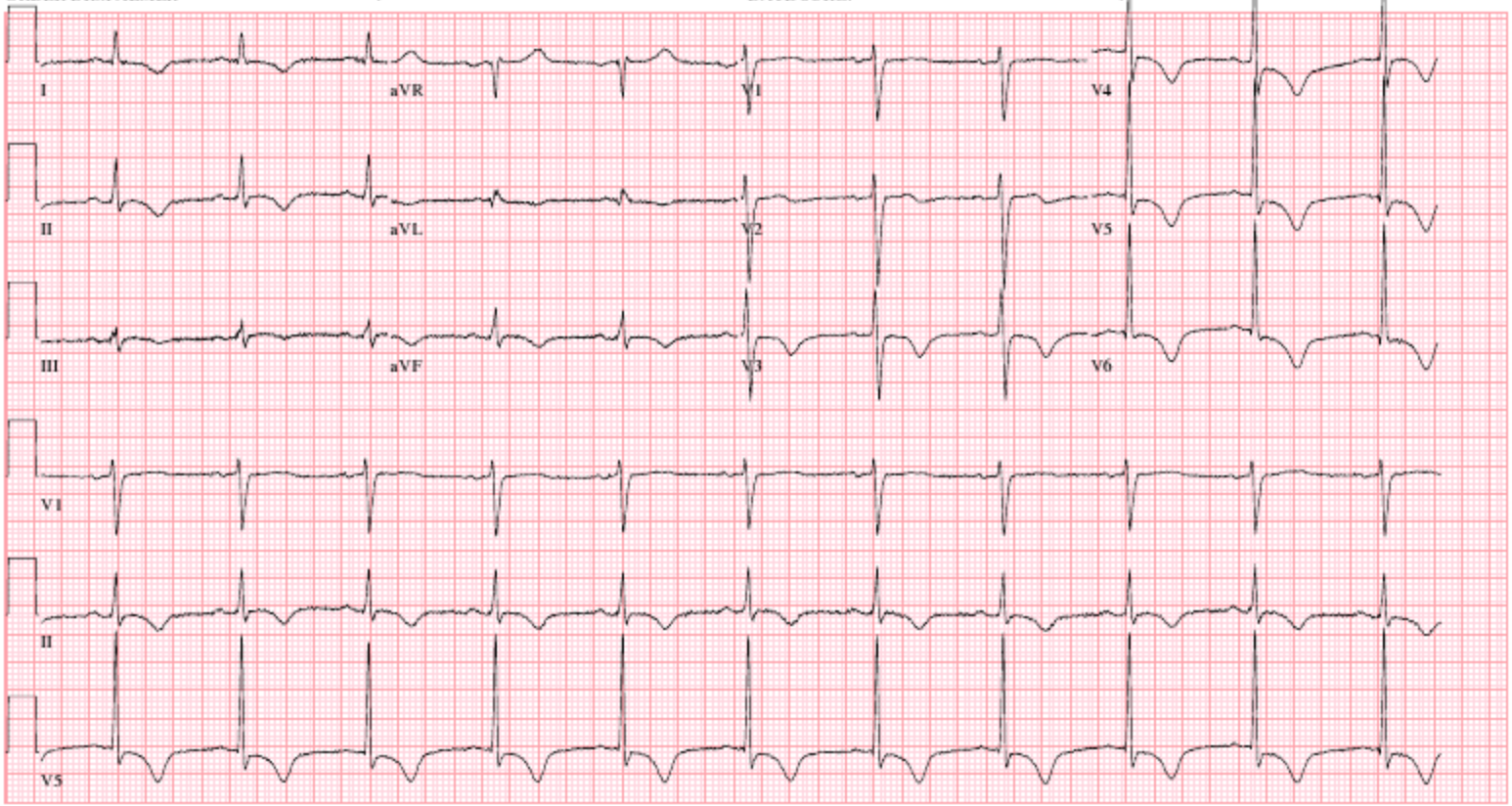
Vent. rate	66	BPM
PR interval	146	ms
QRS duration	100	ms
QT/QTc	458/480	ms
P-R-T axes	5 40 220	

Technician:TERA C. WOOD
Test ind:ACS

Referred by: REUBEN HESS, MD
DATE SURG:

Electronically signed by: AYLMER C. TANG,MD

ORDER DR::PALMER :



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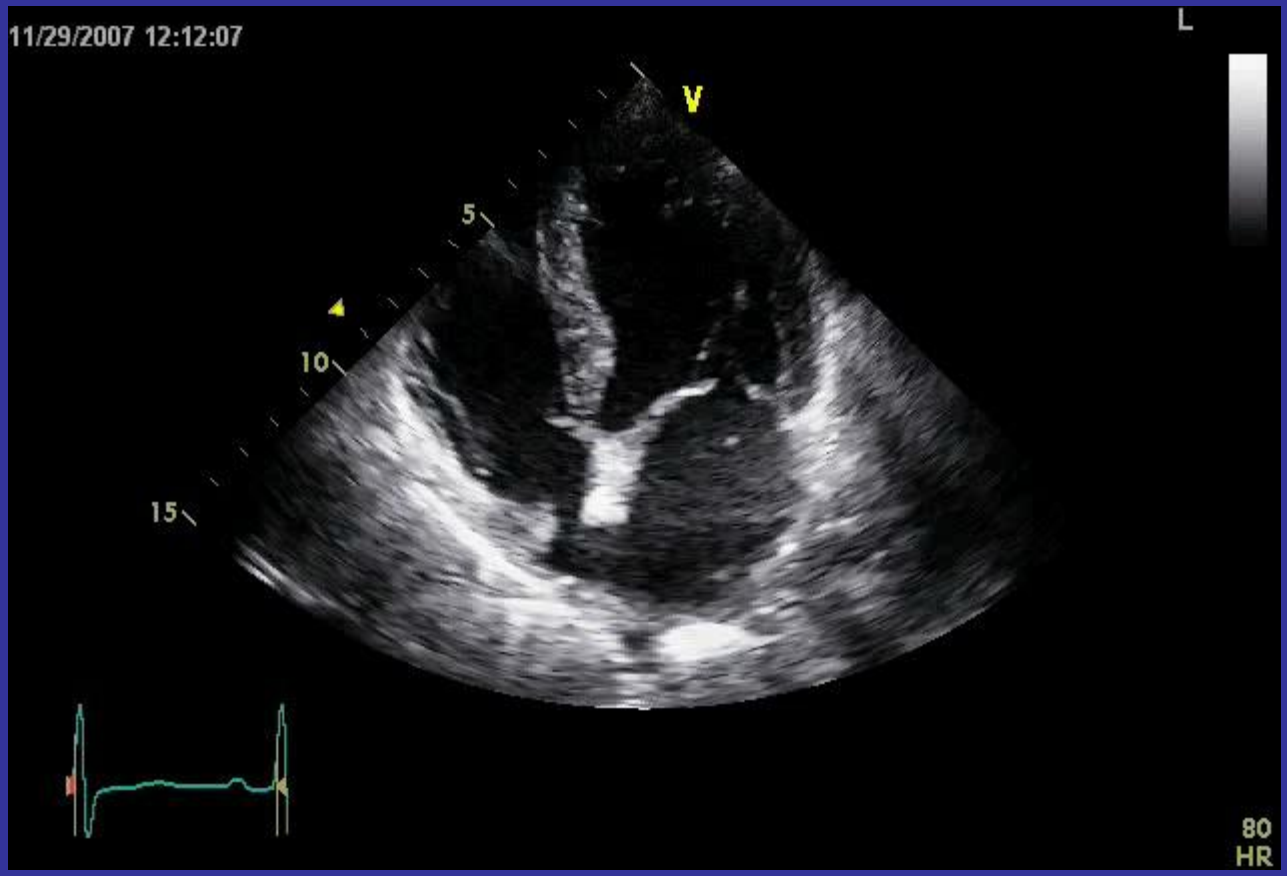
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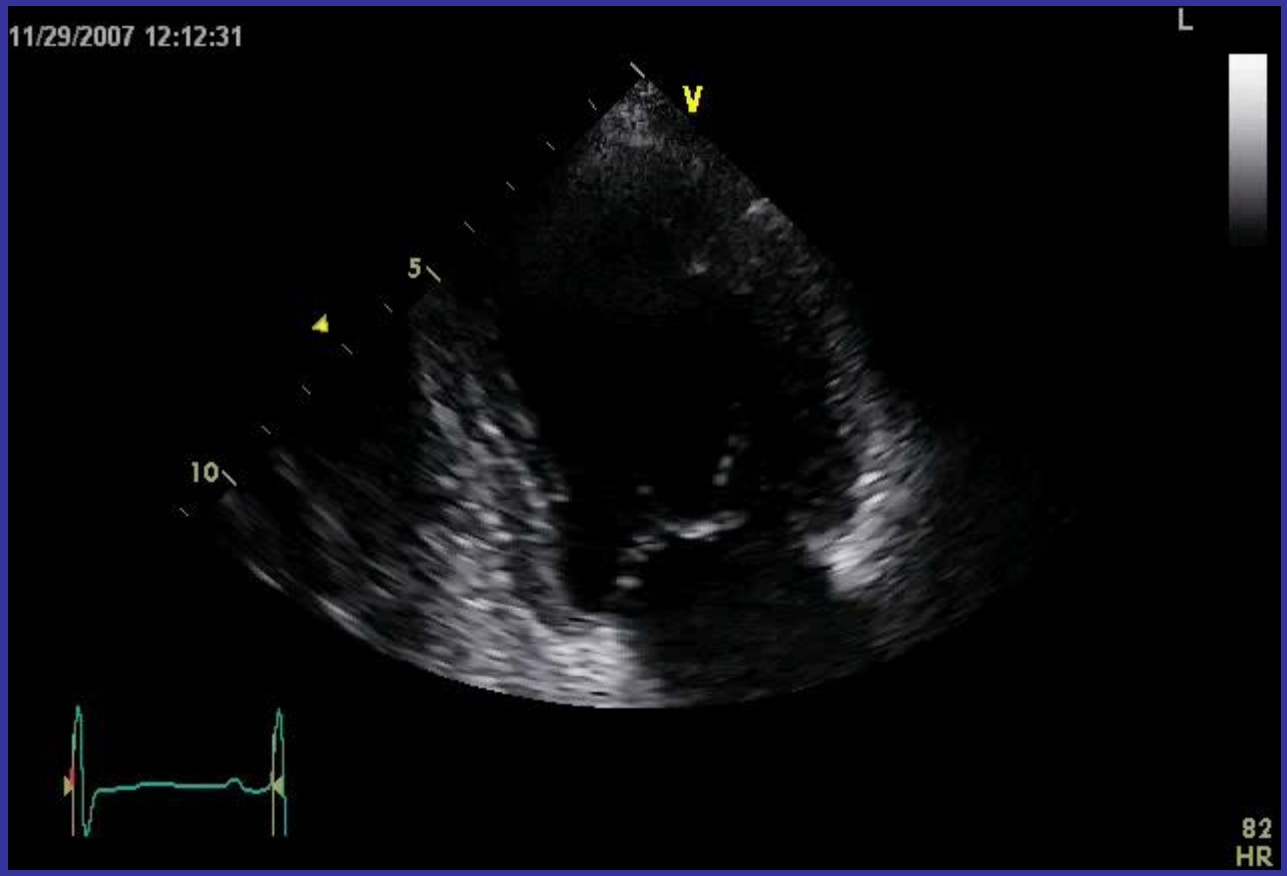
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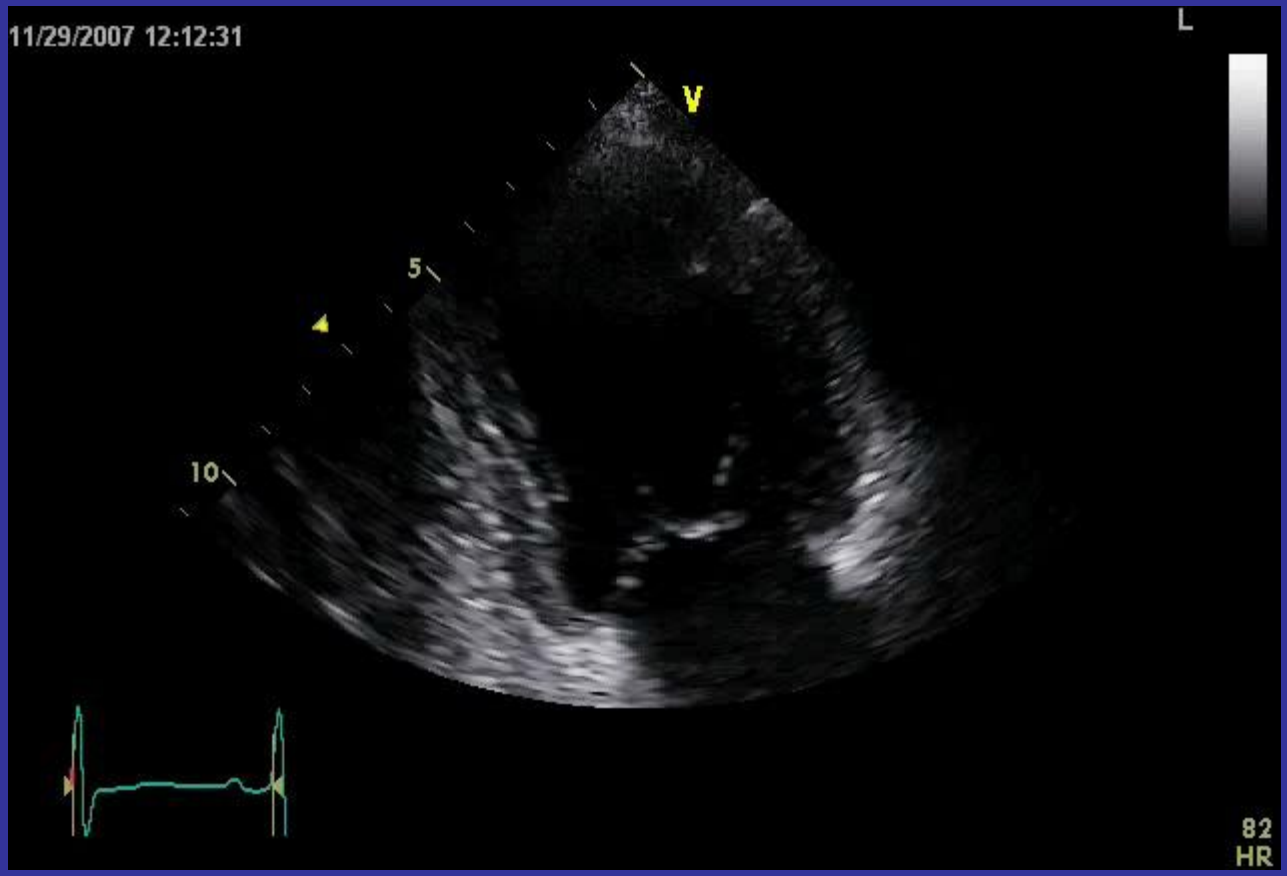
HR


11/29/2007 12:12:31
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HR

Case -2 - Nov 2007

Emergent cardiac cath

Summary:

Hemodynamic assessment demonstrates no systemic hypertension and mildly elevated LVEDP at 19mmHg.

Coronary circulation: - 20% D1

Global left ventricular function was severely depressed. EF estimated by contrast ventriculography was 25 %.

Hospital course Case -2 - Nov 2007

Patient was started on optimum medical therapy
For her LV dysfunction with betablockers, ACEI, Aldactone, diuretics
And referred for cardiac rehab.

Echo repeated in 2/2008 – revealed normal LV function, EF >65%, otherwise
No other major abnormalities.

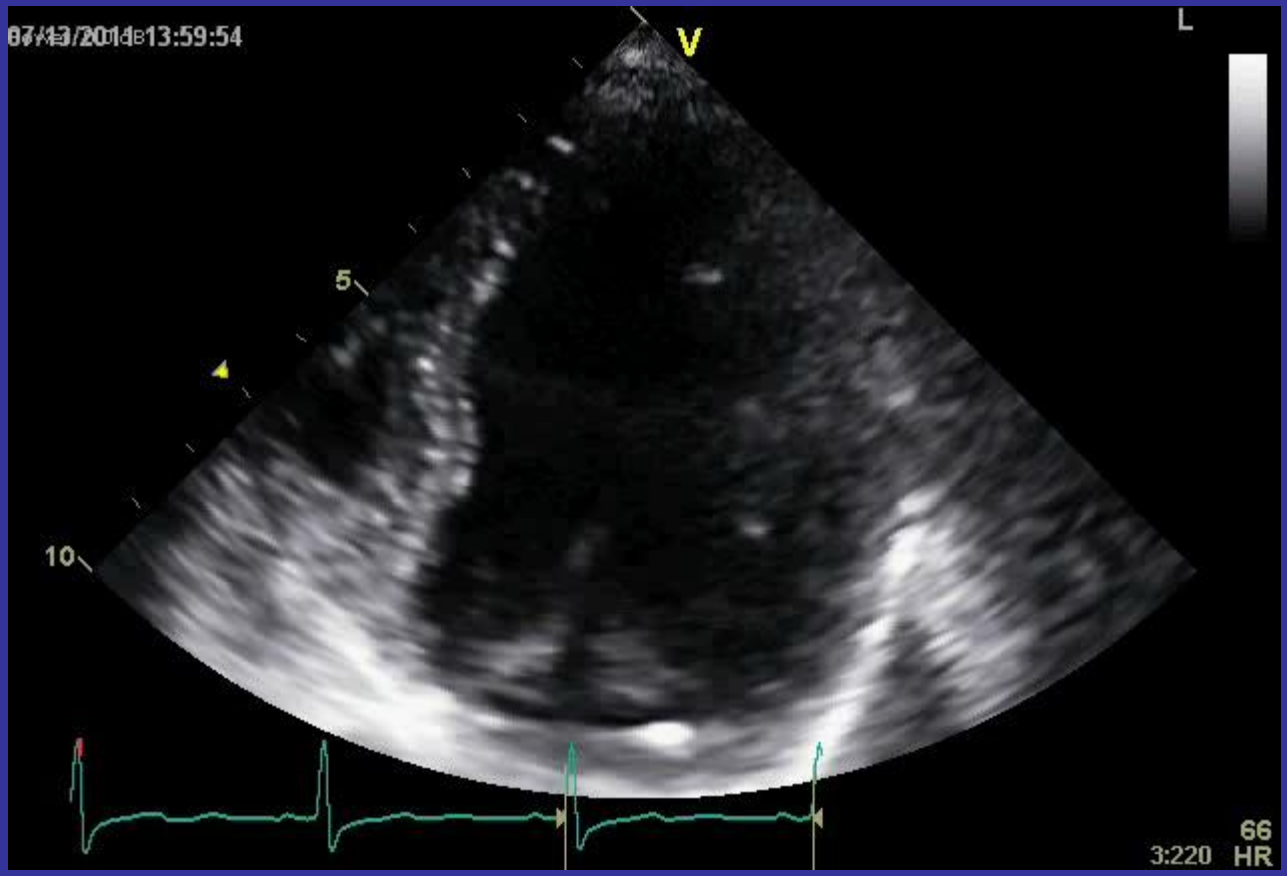
LV function remained normal on a subsequent echo 10/2008

Office visits continued every six months, she was continued on her betablockers
And ACEI, diuretics and aldactone discontinued.

Required a major abdominal pancreatic surgery 10-11/2008, without any
Cardiac problems

Husband passed away 12/2009 – no cardiac problems associated

Patient discharged from cardiology clinic 10/2010 as she had no ongoing active
Cardiac problems, with recommendation to have an echo every 3-5 yrs.


13:59:54
V
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66
3:220 HR

"I'm trying to be more active.
Which one burns more calories,
Twittering, Blogging or Googling?"

Case -2 – Mar /2011

EO – second hospitalization 3/2011

EO presents to the ER with oppressive chest pain for a few hours duration.

Was seen a week prior by her family MD for vague spells of dizziness
And fatigue.

No recent precipitating events like emotional or social stressors, no recent
Illnesses.

In the ER was noted to be hemodynamically stable, with normal BP,
pulse, saturations etc

Labs – mildly anemic (HB -9.8)
Normal chemistry
Initial troponin – 0.82, BNP - 2300

Initial EKG

EO

Case -2 – mar /2011

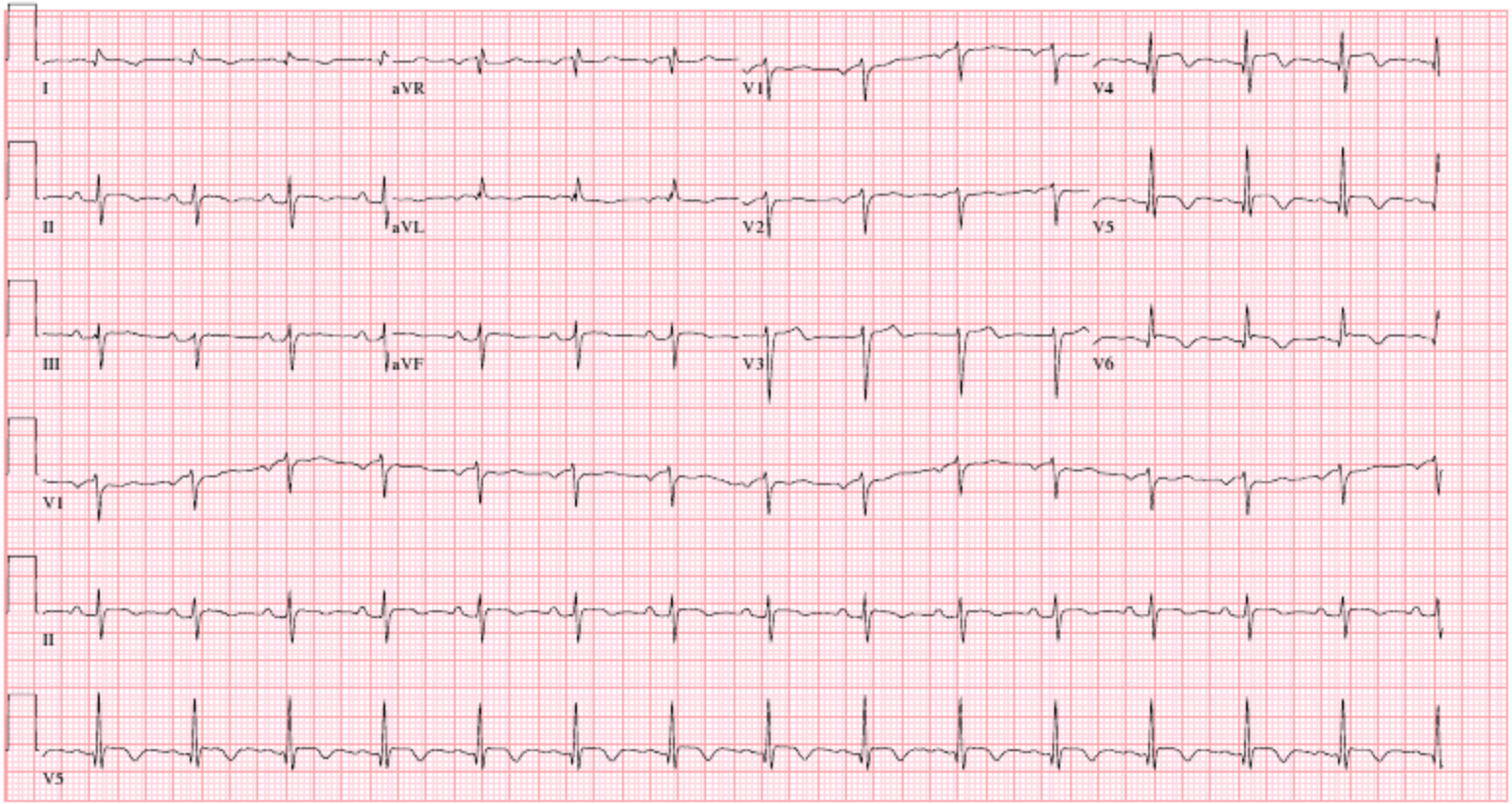
ID:HSM0027255 24-MAR-2011 21:43:36 SUMMIT HEALTH

Vent. rate	88	BPM
PR interval	158	ms
QRS duration	90	ms
QT/QTc	374/452	ms
P-R-T axes	65 -49	98

Technician:RAEGAN MAY
Test ind:SYNCOPE

Referred by: Joe Ferguson Electronically signed by: GARY W LEWIS MD

ORDER DR::TANG COMMENT::



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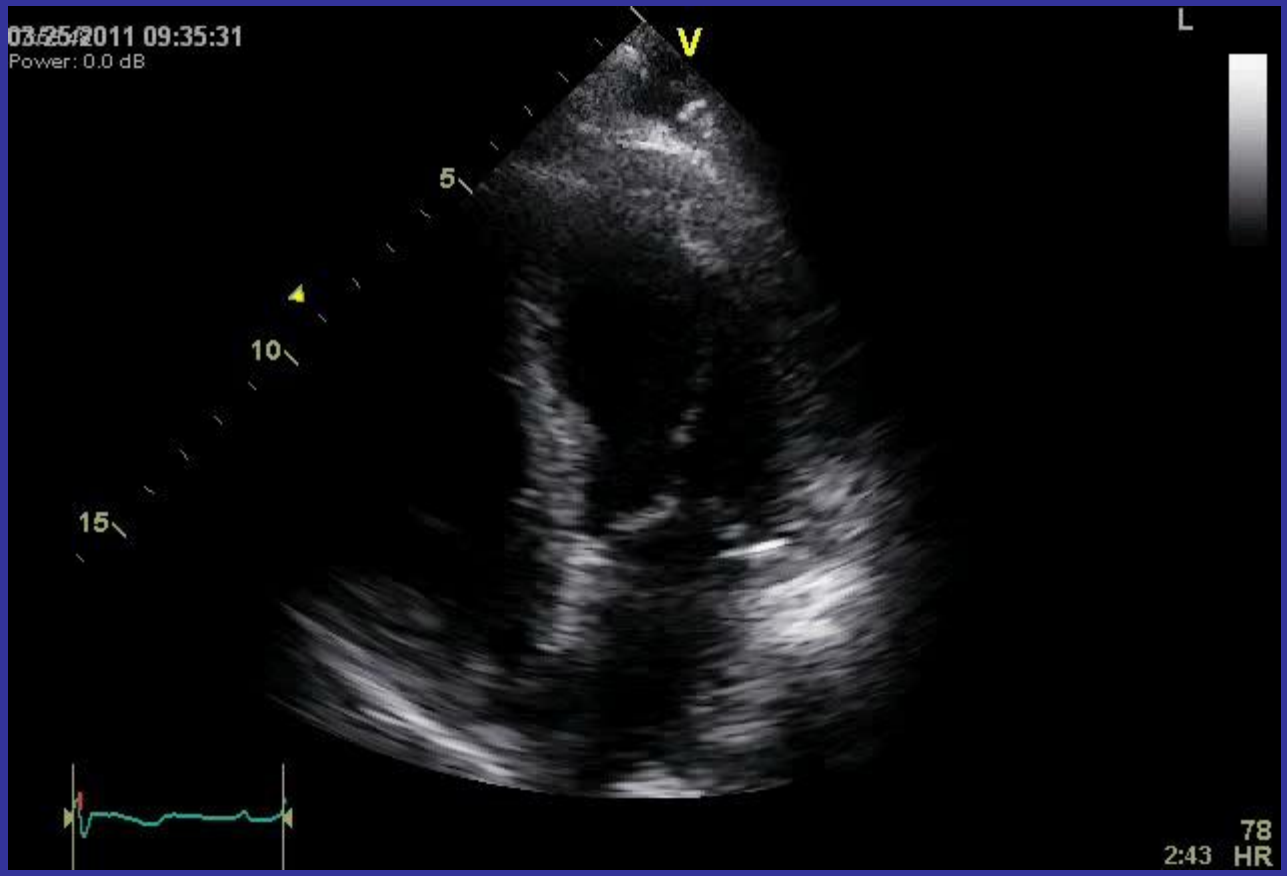
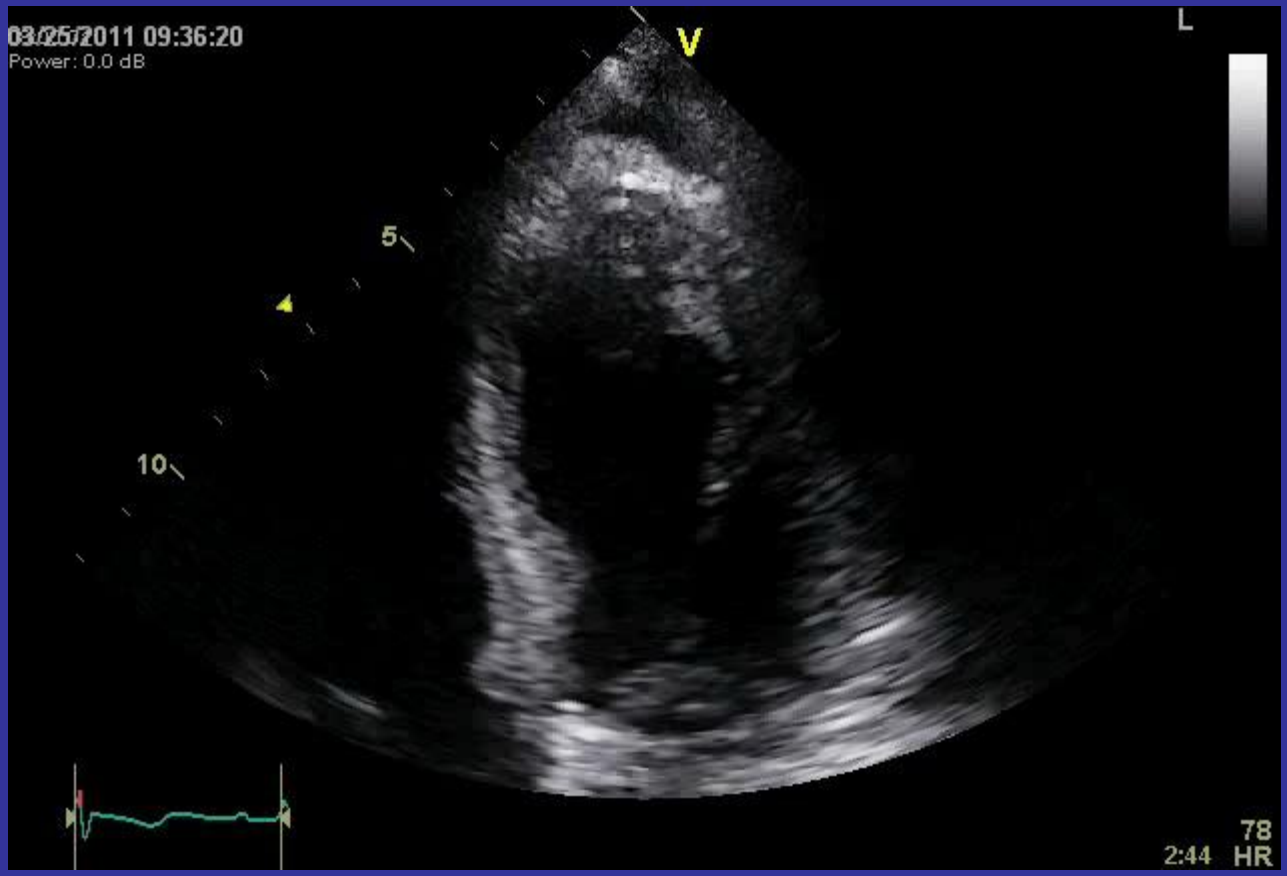
EO second episode

Case -2 – Mar /2011



EO second episode

Case -2 – Mar/2011



Case -2 – mar/2011

EO

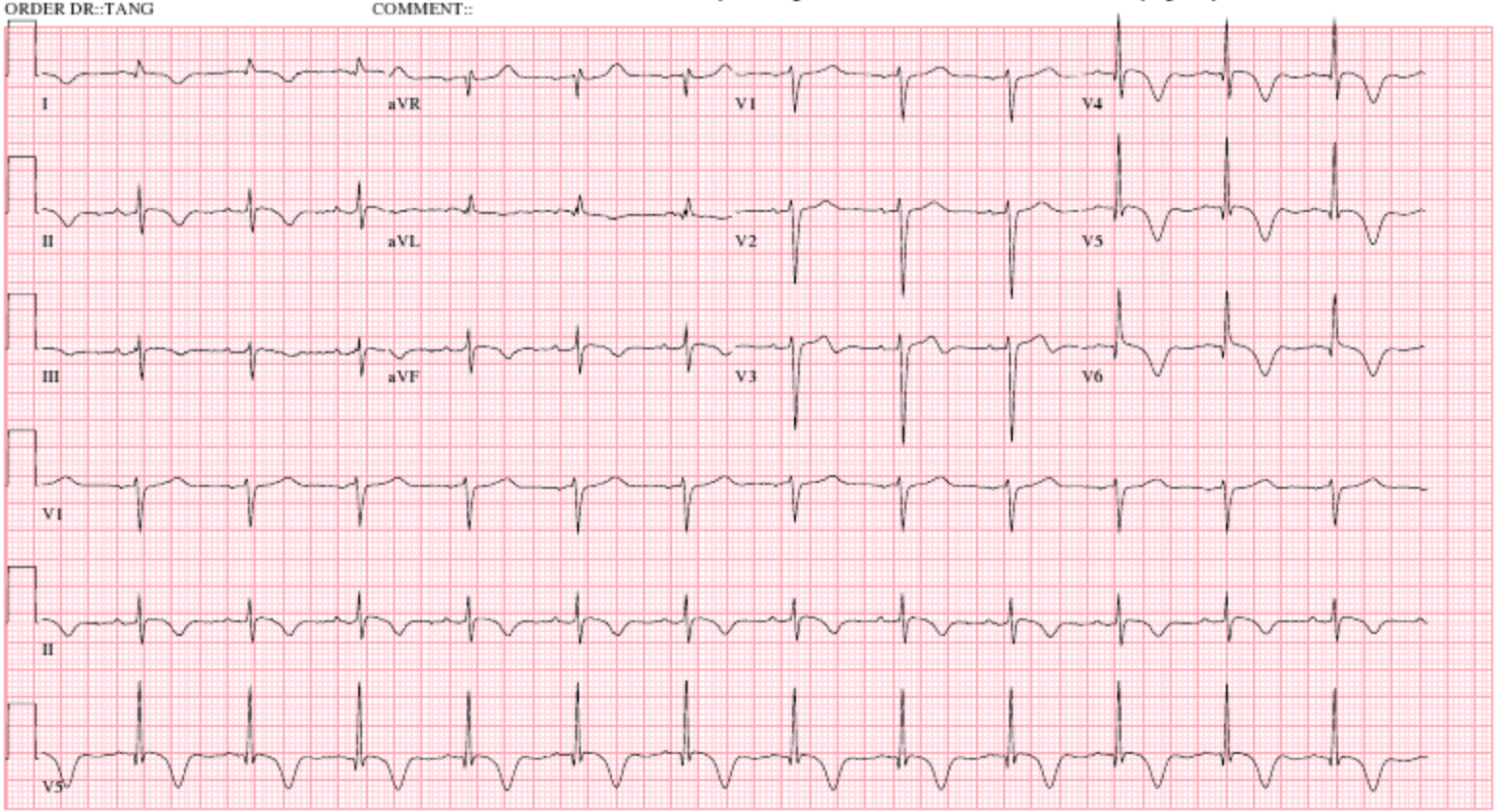
ID:HSM0027255 25-MAR-2011 08:42:49 SUMMIT HEALTH

Vent. rate	77	BPM
PR interval	148	ms
QRS duration	88	ms
QT/QTc	422/477	ms
P-R-T axes	32 -19	214

Technician:PAULA ALLISON
Test ind:POST CATH

Referred by: Joe Ferguson Electronically signed by: HARIGNANASEKERAM MD

ORDER DR::TANG COMMENT::



25mm/s 10mm/mV 40Hz 7.1.1 12SL 237 CID: 6

SID: H321496 EID:6777 EDT: 14:52 25-MAR-2011 ORDER: 0325-0058 ACCOUNT: H00041584988

Case -2 – Mar /2011

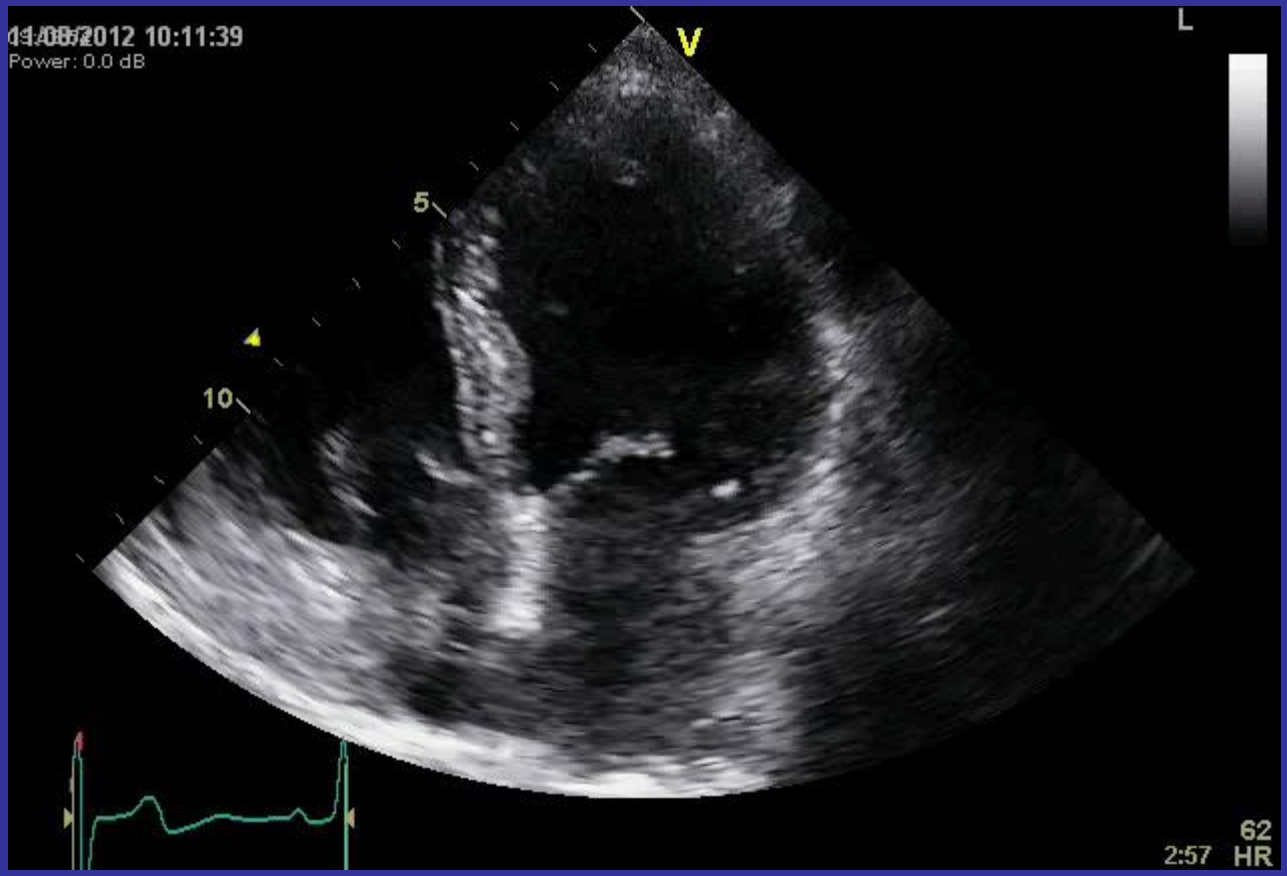
Emergent cardiac cath –

Left main – 20%
LAD – 30% - IVUS confirmed the lesion to be mildly stenotic.
LVEF – 20%
LVEDP -27

Rest of the hospital course was unremarkable and she was discharged
Home again on routine anti-failure meds – beta blockers, calcium channel blockers ACEI, aldactone, diuretics.

Follow up echo – in July 2011 – LVEF 45-50%, mild diffuse hypokinesis.

Remains stable clinically.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"I had to quit drinking coffee. It keeps me awake during presentations."

Takatsubo cardiomyopathy

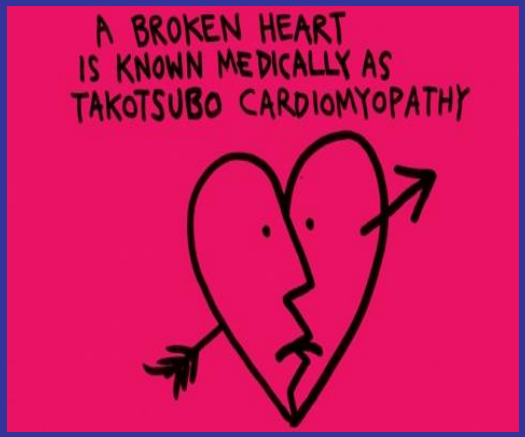
Other names
Apical ballooning syndrome
Broken heart syndrome
Stress induced cardiomyopathy

Definition

Transient systolic dysfunction of the apical and/or mid segments of the left ventricle that mimics myocardial infarction (MI), but in the absence of obstructive coronary artery disease

The name of the disorder is taken from the Japanese name for an octopus trap (takotsubo), which has a shape that is similar to the apical ballooning configuration of the LV in systole in the "typical" form of this disorder.

Takotsubo cardiomyopathy was first described in Japan



In 1990 by Sato et al–
5 patients were reported with a novel, acute cardiac condition characterized by distinctive regional left ventricular (LV) systolic dysfunction, in the absence of significant atherosclerotic coronary artery disease.

Japanese investigators were intrigued by the unusual end-systolic shape of the LV, which resembled the “tako-tsubo,” a fisherman’s pot with a round bottom and narrow neck used for trapping octopuse

Consequently, the term tako-tsubo was introduced to describe a new cardiomyopathic syndrome characterized by reversible LV systolic dysfunction.

Has several clinical hallmarks –
Acute clinical presentation: usually with substernal chest pain, triggered by stressful life circumstance, occurring in elderly women, and requiring differenttial diagnosis from acute coronary syndrome;

LV systolic dysfunction: unique regional contraction abnormalities usually involving the mid-to-distal chamber, not corresponding to the vascular distribution of a single coronary artery;
Reversibility normalization of LV wall motion and global function over several days.


JAPANESE FISHERMEN ABOVE
(octopus trap in japanese)
Tako-tsubo
Calm, happy octopus
leave it in there !
Striped Giraffe Press © December 2005

The LV segments reveal a hypokinesis of the mid-apical segments with Compensatory hyperkinesis of basal segments, producing ballooning of the Apex with systole.

More common in females (post menopausal) versus males

Frequently but not always triggered by an acute medical illness
or by intense emotional or physical stress

Examples of situations are

- eg, death of relatives - particularly if unexpected,
- domestic abuse
- arguments,
- catastrophic medical diagnoses
- devastating financial or gambling losses
- Natural disasters – eg japanese earthquake - 2004

The underlying pathogenesis remains incompletely understood, although some possible mechanisms have been suggested

A number of features of stress-induced cardiomyopathy, including its association with physical or emotional stress suggest that this disorder may be caused by diffuse catecholamine-induced microvascular spasm or dysfunction, resulting in myocardial stunning , or by direct catecholamine-associated myocardial toxicity.

Other postulated mechanisms include, coronary artery spasm, and microvascular dysfunction.

Alternatively, there may be dynamic mid-cavity or left ventricular outflow tract obstruction which may contribute to apical dysfunction

Researchers have implicated gender-related differences in response to sudden bursts of sympethomimetic stimulation as a potential cause of the gender bias seen in this syndrome.

Some have inferred that women may have more potent epinephrine stores available for release, resulting in this gender disparity

,Reduced estrogen levels seen in post-menopausal women may alter endothelial function, making them more vulnerable to sympathetic-mediated myocardial stunning and perhaps stress-related myocardial dysfunction

.

The explanation for apical involvement and basal sparing in takotsubo cardiomyopathy is unknown yet may be related to an increase in apical myocardial sensitivity to sympathetic stimulation or to an increased density of catecholamine-sensitive receptors seen in this region

Measurement of catecholamine levels in patients presenting with takotsubo cardiomyopathy have been conflicting, this may be in part due to the extremely short half-life of epinephrine (3 minutes) and given the fact that most patients arrive to the emergency room at least 30 minutes (>10 half lives) after the inciting event

Prevalence- 1.7-2.2% of all patients presenting with ACS

The following cardiac magnetic resonance (CMR)
imaging features may be helpful in the diagnosis of
stress-induced cardiomyopathy
Late gadolinium enhancement (LGE) on CMR is generally absent in
stress-induced cardiomyopathy in contrast to
myocardial infarction in which intense

Important differential diagnosis includes
-Prinzmetal's angina
-Myocarditis
- cocaine abuse

Proposed Mayo Clinic diagnostic criteria, **all four of which are required for the diagnosis**

-Transient hypokinesis, akinesis or dyskinesis of the left ventricular mid segments with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution. A stressful trigger is often, but not always present.

-Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.

-New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin.

-Absence of pheochromocytoma or myocarditis

It remains unclear why the LV apex is selectively vulnerable and subsequently forms a balloon in typical Takotsubo cardiomyopathy

One of the suggested explanation is

-lack of a 3--layered myocardial structure at the LV apex and the easy loss of elasticity of the LV apex after excessive expansion.

-Histologic findings from patients with takotsubo cardiomyopathy document myofibrillar degeneration, contraction band necrosis and mononuclear leukocyte infiltration, which are forms of myocyte injury witnessed in catecholamine toxicity

RV involvement was reported around 25% in a case series of 34 patients.

Patients with RV dysfunction had lower ejection fractions and In general comprised of a sicker cohort

Complete resolution of RV dysfunction was noted in most patients

Mortality is not high, in- hospital mortality reported around 2-4%

Of patients with in-hospital mortality, 81.4% had underlying critical illnesses.

The presence of underlying critical illness was the main driver of mortality

Men are less susceptible but more likely to die of broken heart syndrome than women.

A case series from Harvard, largest to date, followed for 9 years in 1993, death rate was reported at 49% in men and 30% in women (case series of > 300,000 patients followed.

The mean age of men in the study was 75 years and the mean age of women was 72 years.

Prognosis and Treatment

Supportive therapy and resolution of the physical/emotional stressor results In rapid symptom improvement

Standard meds for LV dysfunction - ACEI, Betablockers, diuretics, digoxin…

Appropriate duration unknown, usually till resolution of LV dysfunction …. However due to chances of recurrence sometimes alpha/betablockade is Continued for lifetime.

Despite this relatively benign prognosis,
more serious complications (cardiogenic shock and ventricular fibrillation) have been reported with in as many as 4.2% and 1.5%, respectively

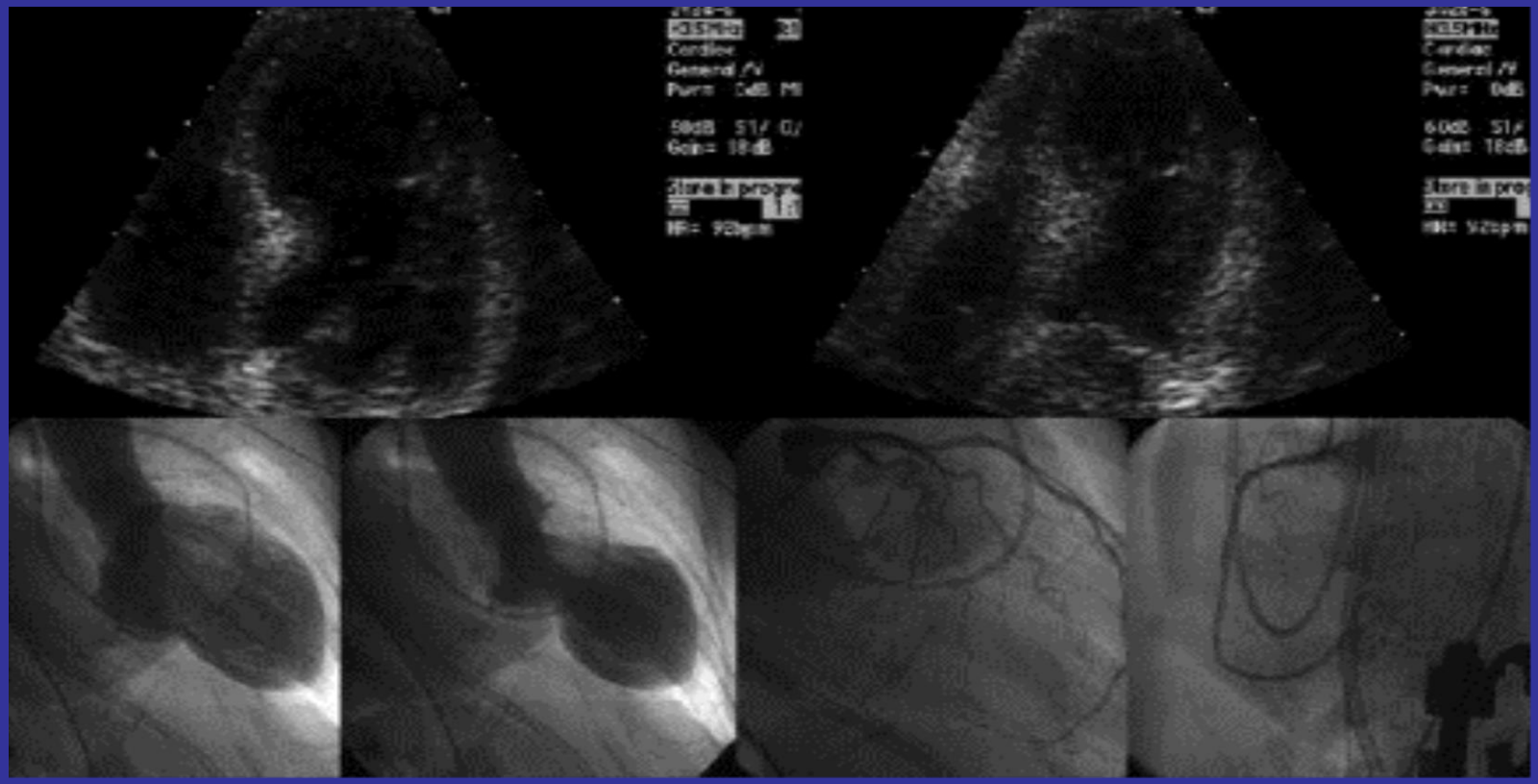
Prognosis and Treatment

Patients may present with cardiogenic shock with or without LV outflow tract obstruction from the hyperdynamic basal segments.

Without LVOT obstruction – If in cardiogenic shock, inotropes may be used safely - Inotropes may cause LVOT obstruction but is usually mild.

With LVOT obstruction – more challenging to treat cardiogenic shock in this setting –
*IABP is a choice with a small risk of worsening obstruction;
*Phenylephrine – a pure alpha agonist may reduce gradient by increasing the Afterload and overall improve hemodynamics –
*Betablockers, to if not hypotensive, betablockers are the main stay of treatment

Prognosis usually good, with full recovery of LV function in 4-6 weeks, small Chance of recurrence 10-25%- based on the data from a series of 100 patients In 2007, followed for 4 yrs


Cardiac
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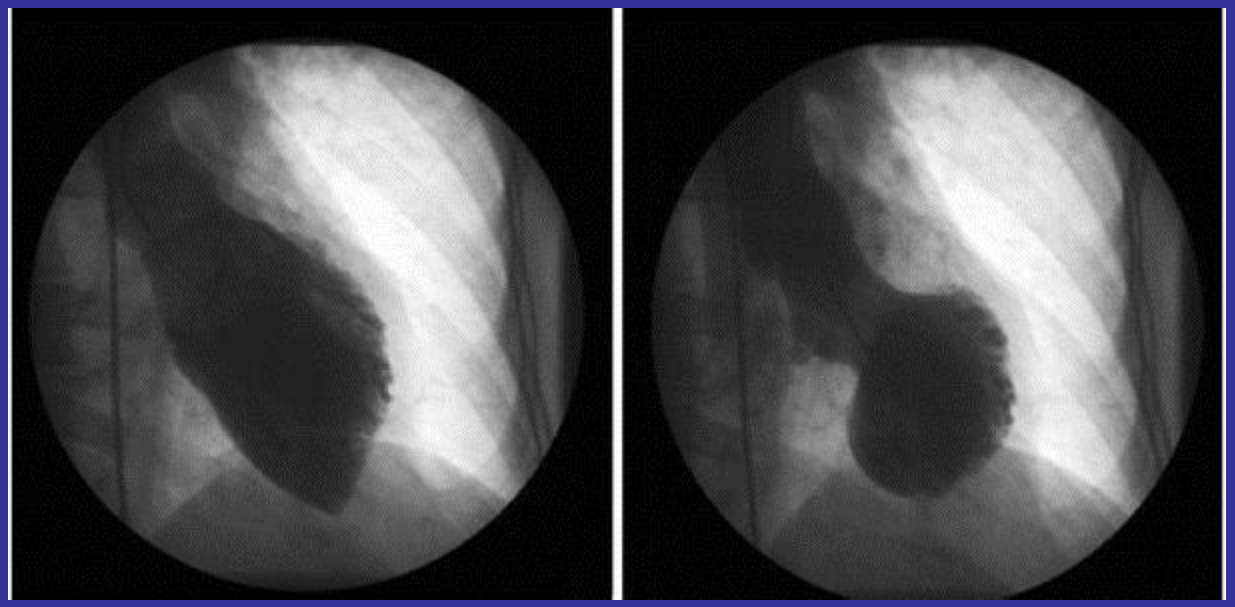
HR= 92bpm



Left ventricular angiogram in diastole and systole.
The findings illustrate normal basal function with severe hypokinesis of the anterolateral, apical, and inferoposterior segments

First described in 2010;

Inverted takotsubo cardiomyopathy

Atypical stress-induced cardiomyopathies without involvement of the LV apex

Most of the cases were instances of transient midventricular ballooning syndrome with midventricular akinesia and normal wall motion of the LV base and apex, although some cases displayed the “inverted Takotsubo” pattern of cardiomyopathy, which is characterized by a hyperdynamic LV apex and akinesia of the LV base and mid-portion

Frequently noted etiology

Acute cerebrovascular accident,

Pheochromocytoma,

Paraglioma,

acute pancreatitis,

amphetamine use

shoulder surgery

Inverted Takotsubo cardiomyopathy triggered by a septic condition or parturition has not been reported.

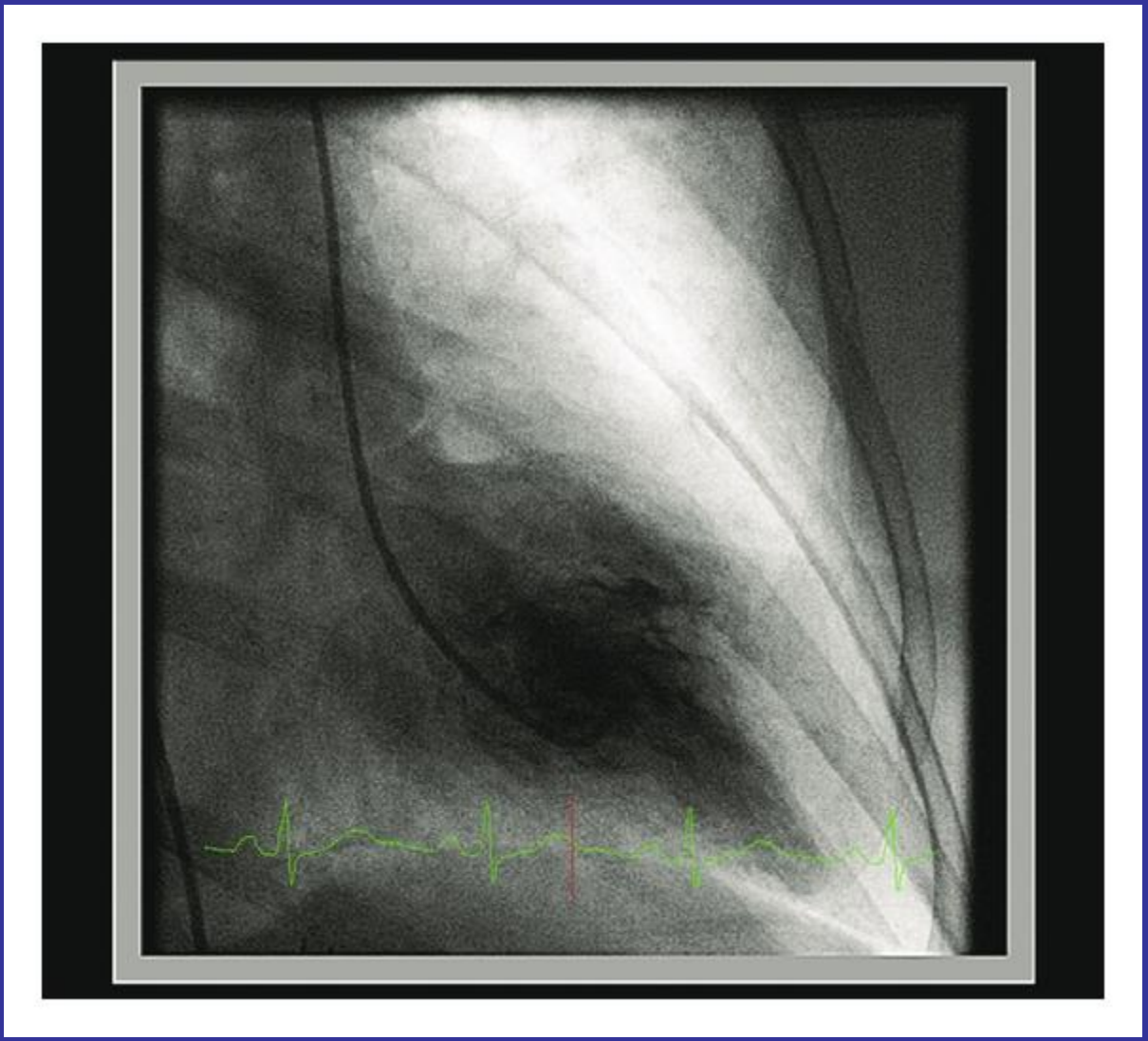
Pathophysiology is similar to Takatsubo's syndrome

Rarer variant of takotsubo cardiomyopathy is important to identify as it tends not to be recognized as readily as the traditional presentation

Patients with reverse takotsubo cardiomyopathy present at a younger age, with a mean age of 36, and often they have an emotional or physical stress trigger

Present with less pulmonary edema, dyspnea and cardiogenic shock than patients with classic takotsubo cardiomyopathy

Biomarker elevations are noted to be higher than the traditional variant likely due to the fact that the extent of myocardium involved in this form, with more myocardial tissue being affected in reverse rather than classic takotsubo cardiomyopathy.



Left ventricular angiogram showing the “ace of spades” appearance of left ventricular contractile function, with basal hypokinesis and normal apex, consistent with reverse Takotsubo cardiomyopathy.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FAST FOOD
FRANCHISE DIVISION
GLASBERGEN
"Our challenge is to convince the public
that heart attacks are sexy."

Questions?????