

# Acute heart failure precipitated by Acute myocardial infarction of the inferior wall accompanied with right ventricular infarction

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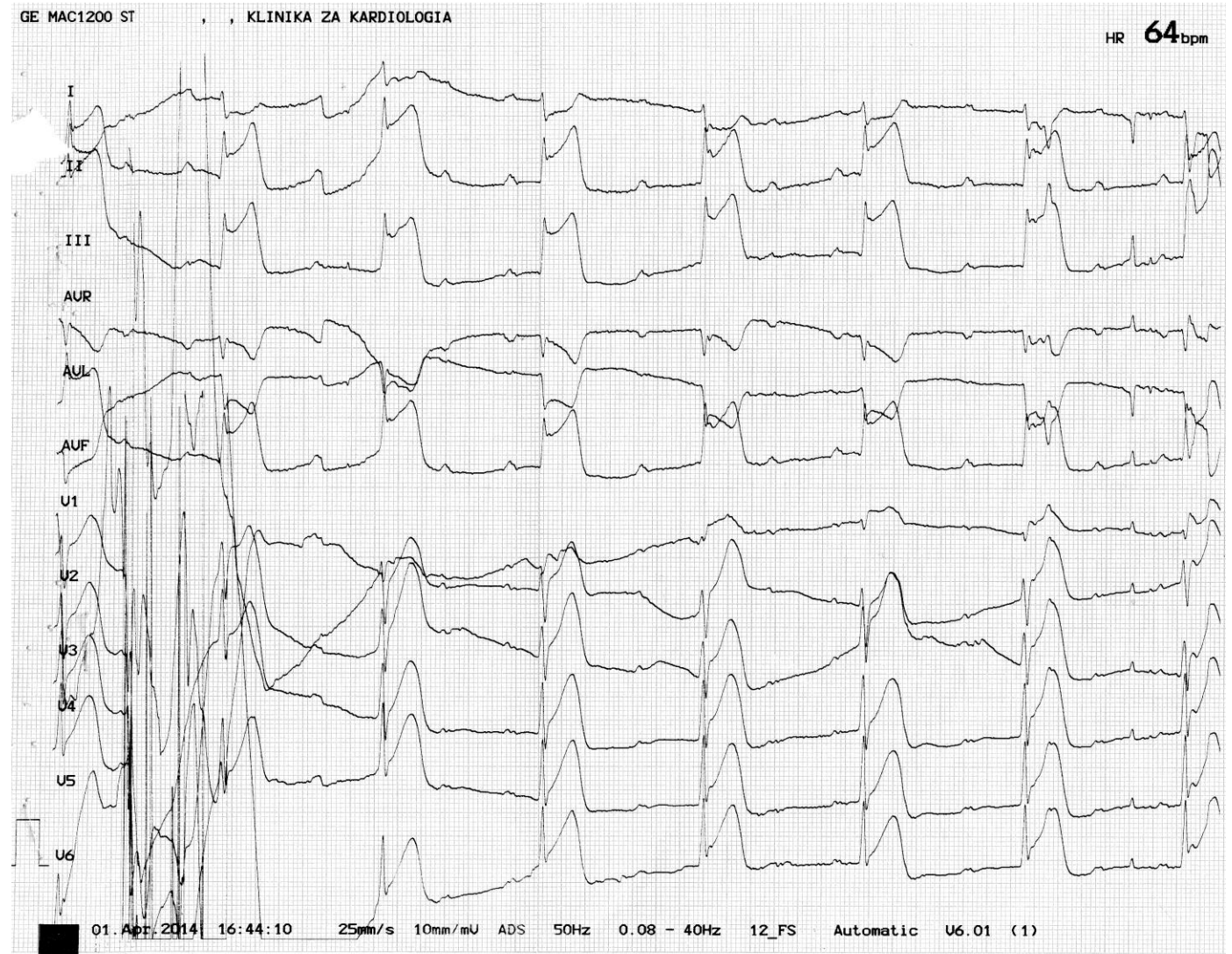
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# CLINICAL CASE

- Male aged 62 years, Smoker
- Newly onset chest pain 1-2 hours prior hospital admission
- BP 60/40mmHg, HR 60/min
- ECG
- TROPONIN T <50ng/L



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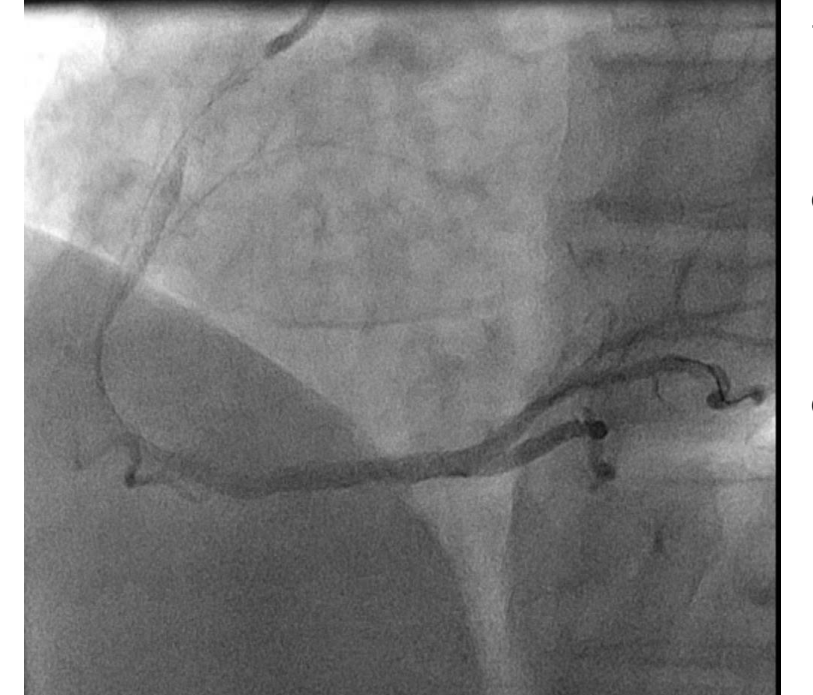
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Recommendations	Class <sup>a</sup>	Level <sup>b</sup>
A 12-lead ECG must be obtained as soon as possible at the point of FMC, with a target delay of ≤10 min.	I	B
ECG monitoring must be initiated as soon as possible in all patients with suspected STEMI.	I	B
Blood sampling for serum markers is recommended routinely in the acute phase but one should not wait for the results before initiating reperfusion treatment.	I	C
The use of additional posterior chest wall leads ( $V_7-V_9 \geq 0.05$ mV) in patients with high suspicion of infero-basal myocardial infarction (circumflex occlusion) should be considered.	IIa	C
Echocardiography may assist in making the diagnosis in uncertain cases but should not delay transfer for angiography.	IIb	C



# PPCI + thromboaspiration + GP IIb/IIIa inhibitor *Tirofiban*

Recommendations	Class <sup>a</sup>	Level <sup>b</sup>
<b>Antiplatelet therapy</b>		
Aspirin oral or i.v. (if unable to swallow) is recommended	I	B
An ADP-receptor blocker is recommended in addition to aspirin. Options are:	I	A
• Prasugrel in clopidogrel-naïve patients, if no history of prior stroke/TIA, age <75 years.	I	B
• Ticagrelor.	I	B
• Clopidogrel, preferably when prasugrel or ticagrelor are either not available or contraindicated.	I	C
GP IIb/IIIa inhibitors should be considered for bailout therapy if there is angiographic evidence of massive thrombus, slow or no-reflow or a thrombotic complication.	IIa	C
Routine use of a GP IIb/IIIa inhibitor as an adjunct to primary PCI performed with unfractionated heparin may be	IIb	B
<b>Procedural aspects of primary PCI</b>		
Stenting is recommended (over balloon angioplasty alone) for primary PCI.	I	A
Primary PCI should be limited to the culprit vessel with the exception of cardiogenic shock and persistent ischaemia after PCI of the supposed culprit lesion.	IIa	B
If performed by an experienced radial operator, radial access should be preferred over femoral access.	IIa	B
If the patient has no contraindications to prolonged DAPT (indication for oral anticoagulation, or estimated high long-term bleeding risk) and is likely to be compliant, DES should be preferred over BMS.	IIa	A
Routine thrombus aspiration should be considered.	IIa	B
Routine use of distal protection devices is not recommended.	III	C
Routine use of IABP (in patients without shock) is not recommended.	III	A



2012 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation



# Atrial fibrillation

## Hypotension/ Shock

BP 60/40mmHg

HR 100-110/min

Diuresis in the first 14 hours 500ml

### ☐ CLINICAL SIGNS AND SYMPTOMS:

hypotension, evidence of low cardiac output (resting tachycardia, altered mental status, oliguria, cool peripheries) and pulmonary congestion.

### ☐ The HAEMODYNAMIC CRITERIA:

cardiac index of  $<2.2$  L/min/m<sup>2</sup> and an increased wedge pressure of  $>18$  mmHg, additionally -diuresis  $<20$  mL/h.

☐ Shock is **also considered present** if **i.v. inotropes** and/or an **IABP** is **needed to maintain a systolic blood pressure  $>90$  mmHg.**





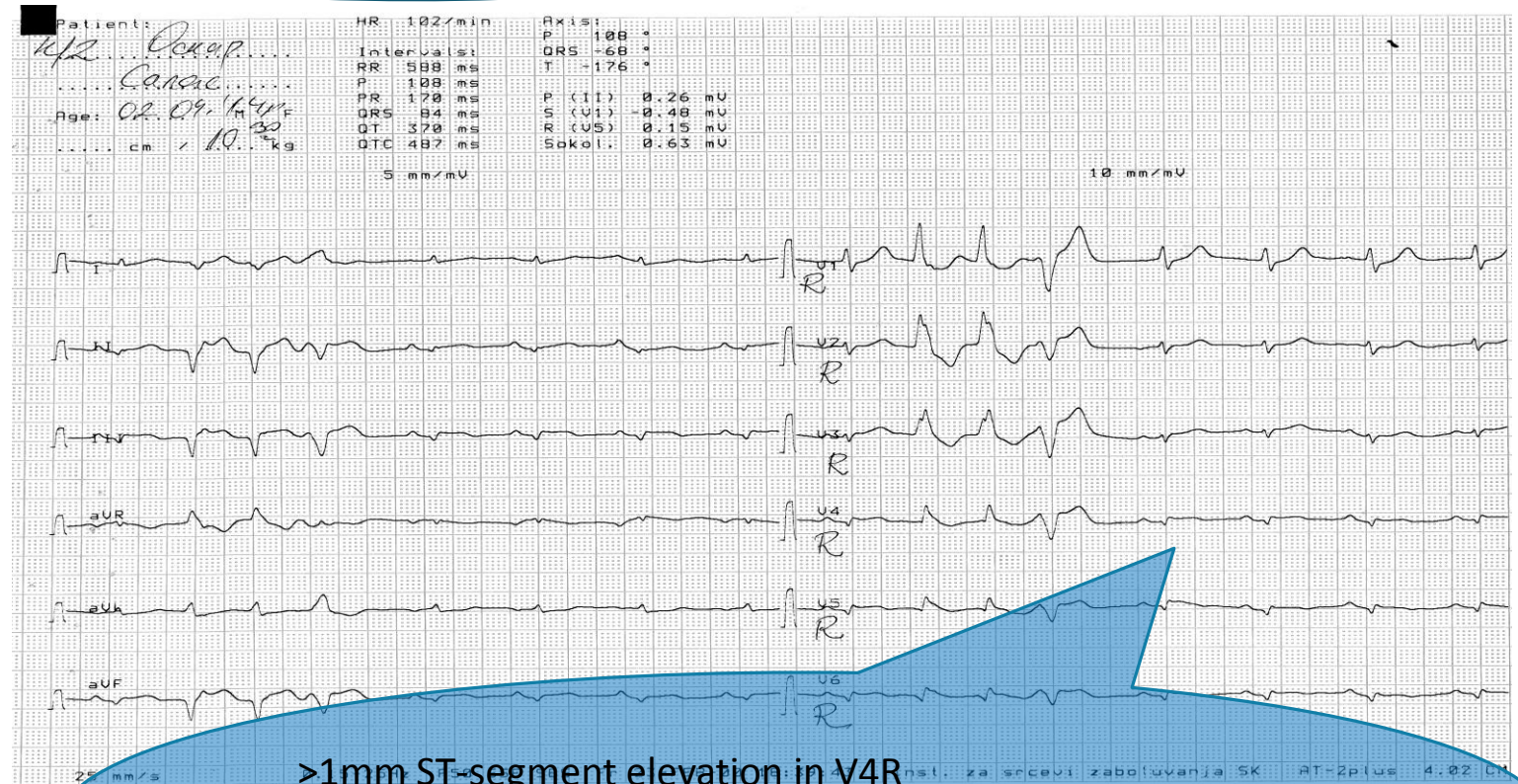
# Infarction of the right ventricle

## RV infarction definition:

- ☐ pathologically
- ☐ haemodynamically
- ☐ electrocardiographically,
- ☐ echocardiographically
- ☐ MRI

**CLINICAL TRIADE:** hypotension, clear lung fields, and elevated jugular venous pressure → markers of RV infarction in patients with IWMl. high specificity (96%) very low sensitivity (25%).

**Kussmaul's venous sign** (distension of the jugular vein on inspiration), may occur with RV infarction.

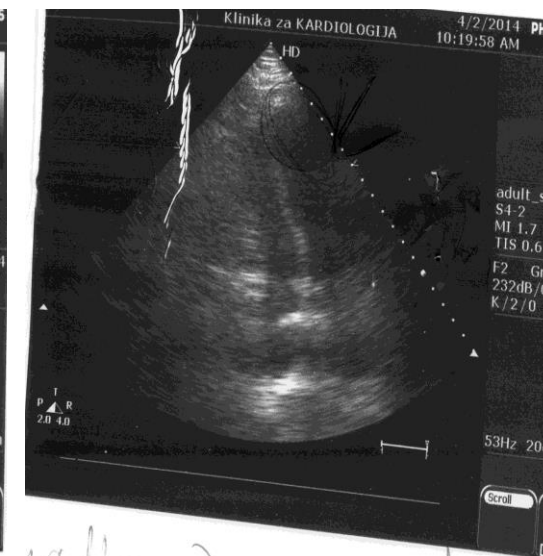
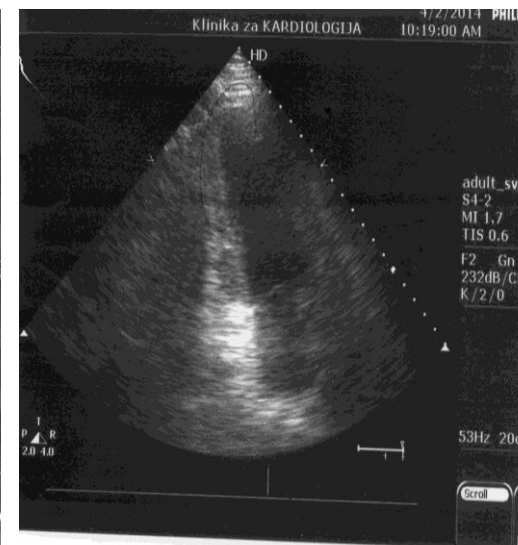
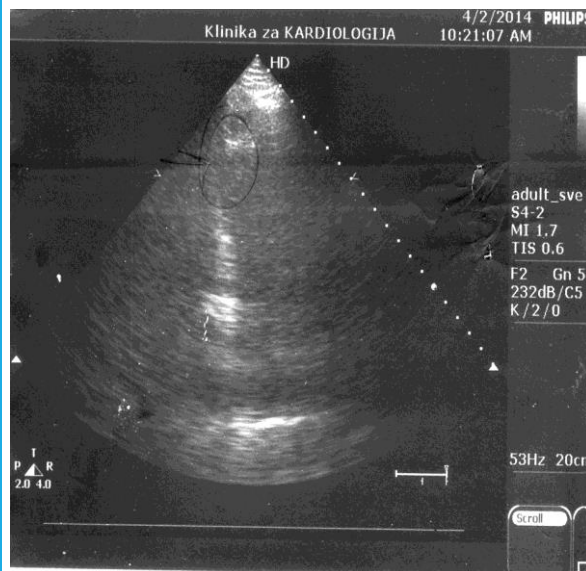


>1mm ST-segment elevation in V4R

A strong independent predictor of major complications and in-hospital mortality in RVI

# ЕШНОСАРДИОГРАФНУ

Перикард	без раздвојување , назначен напред
Кинетика	<div> <div>✓</div> <div>✓</div> <div>✓</div> </div> <div> <div>хипокинезија на апикална третина на ИВС</div> <div>хипокинезија на средна и апикална третина на долен зид</div> <div>Хипокинезија на преден слободен зид на десна комора - кон врвот на срцето</div> </div>
<b>Коментар</b> Прегледот направен во принудна положба , несоодветна за ехокард. преглед . Ехокардиографски видени се уредни димензии на лева комора со уредна глобална ЛК систолна ф-ја .. Концентрична хипертрофија на зидовите на ЛК ... Уредна димензија и на лева предкомора ....Валвуларен апарат уреден Доплер уреден Десна комора зголемена ( поголема од левата ) на базата 56 мм .. среден дел 48 мм .. лонгитудинална дим 79 мм . .... Десна предкомора 59 x 49 мм назначена дим. .. ТВ реглесна TP ..	



Линет згуг - срц. и apex 1CBT - уреден згуг ДК.



## Echocardiography

Information from all available acoustic windows is necessary for the complete assessment of the RV.

Although not validated in acute situations, three-dimensional (3D) echocardiographic RV volumes are comparable to those derived by CMR and are probably more accurate than 2D echocardiographic volumes.

## Cardiac magnetic resonance

CMR using late gadolinium enhancement imaging enables the accurate characterization of ischaemic myocardial injury.

in patients with acute MI indicated that RV involvement was detected significantly more frequently with CMR than with electrocardiography or echocardiography.

Although the recent advances in CMR may contribute to understanding the pathology as well as providing a more accurate diagnosis of RV infarction, further investigations are essential to establish the usefulness of the CMR technique.

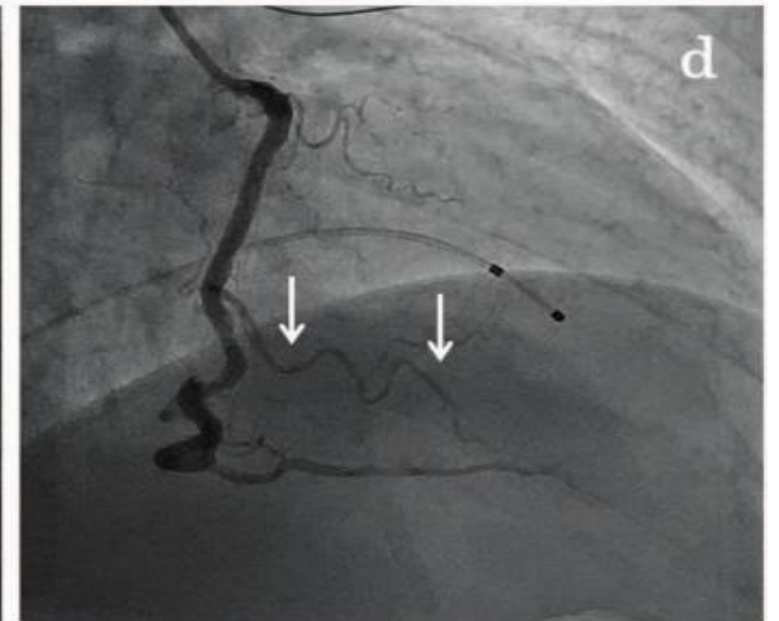
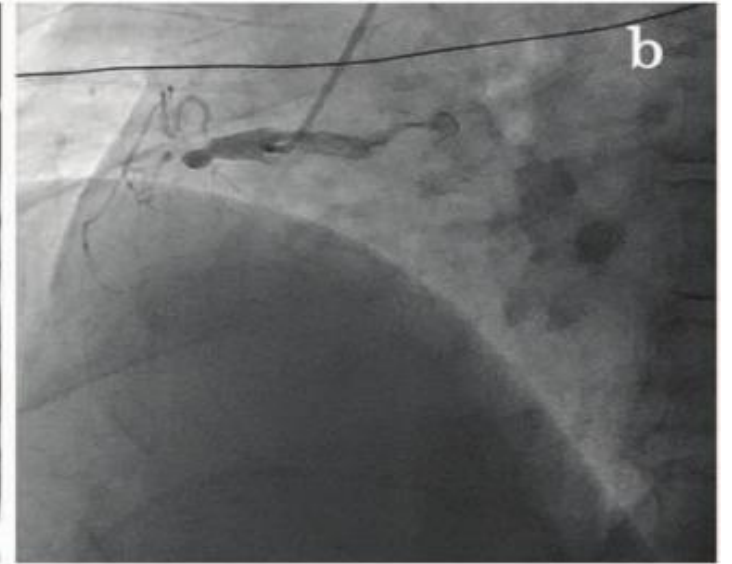
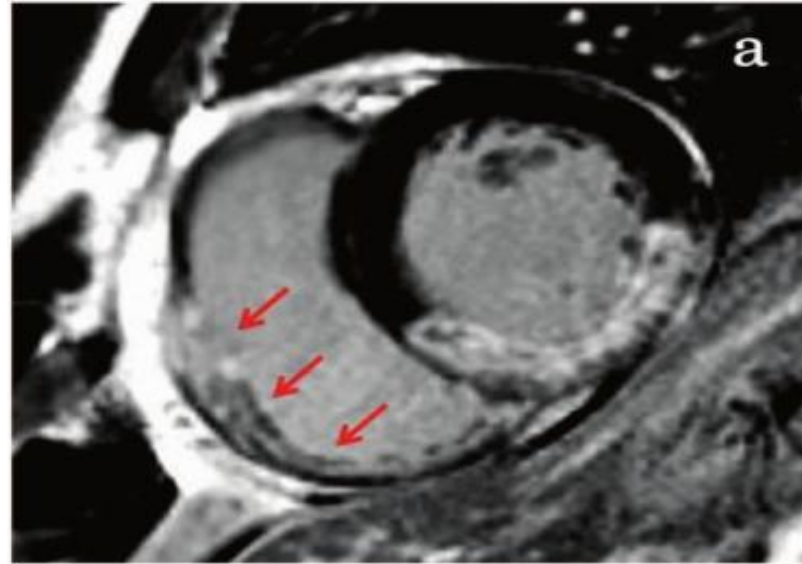
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Contrast-enhanced  
cardiovascular magnetic  
resonance image of right  
ventricular myocardial  
infarction (a)

cine angiogram before (b)  
and after (c, d) PCI



# Treatment of the Shock

Emergent revascularization with PPCI

Hemodynamic stability:

- IABP
- Inotropes (Dopamine)
- Fluids

Management of cardiogenic shock complicating acute myocardial infarction includes:

1. Hemodynamic stability, achieved with medical therapy or mechanical circulatory support, and
2. Emergent revascularization by means of PCI or CABG surgery.

Treatment of cardiogenic shock (Killip class IV)		
Oxygen/mechanical respiratory support is indicated according to blood gasses.	I	C
Urgent echocardiography/Doppler must be performed to detect mechanical complications, assess systolic function and loading conditions.	I	C
High-risk patients must be transferred early to tertiary centres.	I	C
Emergency revascularization with either PCI or CABG in suitable patients must be considered.	I	B
Fibrinolysis should be considered if revascularization is unavailable.	IIa	C
Intra-aortic balloon pumping may be considered.	IIb	B
LV assist devices may be considered for circulatory support in patients in refractory shock.	IIb	C
Haemodynamic assessment with balloon floating catheter may be considered.	IIb	B
Inotropic/vasopressor agents should be considered:	IIa	C
• Dopamine		
• Dobutamine	IIa	C
• Norepinephrine (preferred over dopamine when blood pressure is low).	IIb	B

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# Cardiogenic shock precipitated by ACS-STEMI-RVI

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**RV involvement may be present in as much as 59% (47-57% with MRI), of the patients with IWMI at the initial presentation, but also in some AWTMI (11-65%).**

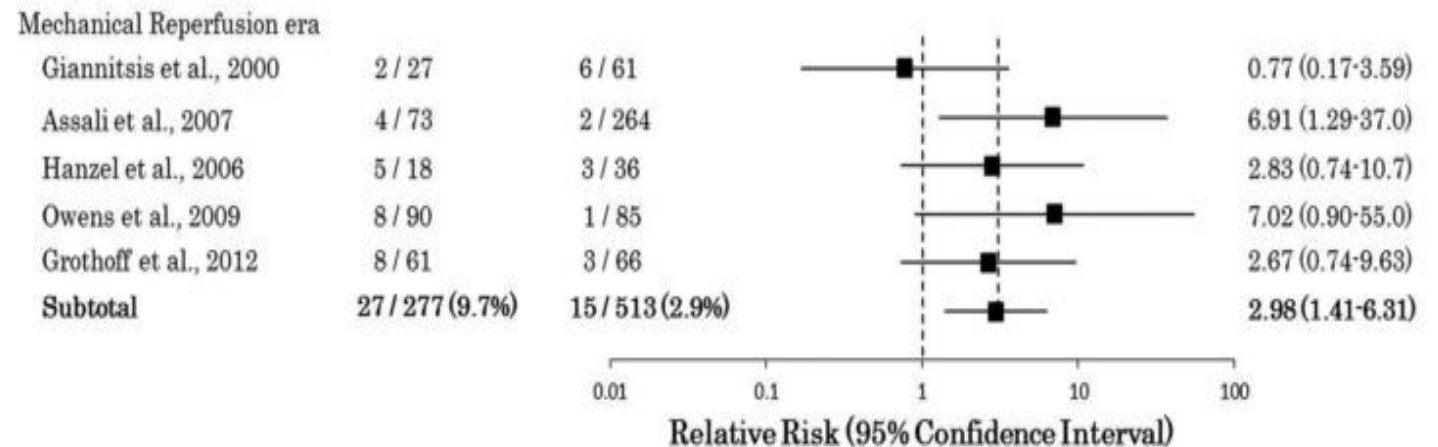
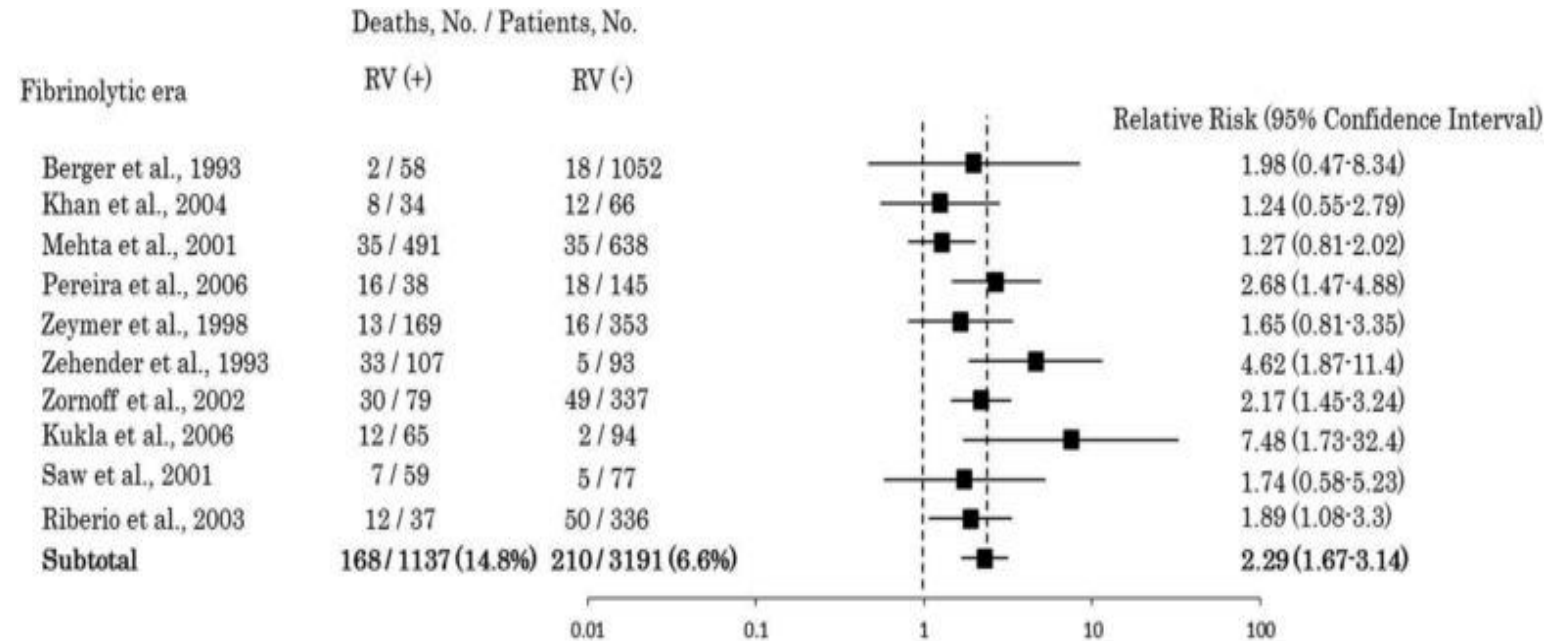
Although the number of patients with **RV infarction** is small, these patients to have a **higher risk of in-hospital mortality (7-9%)** in the mechanical reperfusion era (in comparison with thrombolytic era, without reasonable explanation). There is an increase in the relative risk for in-hospital mortality of RV infarction from 2.29 to 2.98.

Despite worse short term prognosis, the long-term prognosis associated with RV infarction in those patients who survive hospitalization is relatively good.

**Cardiogenic shock complicates 6-10% of all cases of STEMI and remains a leading cause of death, with hospital mortality rates approaching 50%.**

In the SHOCK study, 1 year after revascularization, the survival curves remained relatively stable with an **annual mortality rate of 8-10 deaths per 100 patient-years**. This annual mortality rate is comparable to that reported in a broad cohort of post-percutaneous coronary intervention patients.

# Mortality rate of RVMI (right ventricular myocardial infarction)





**DISCHARGED**  
after 8 days

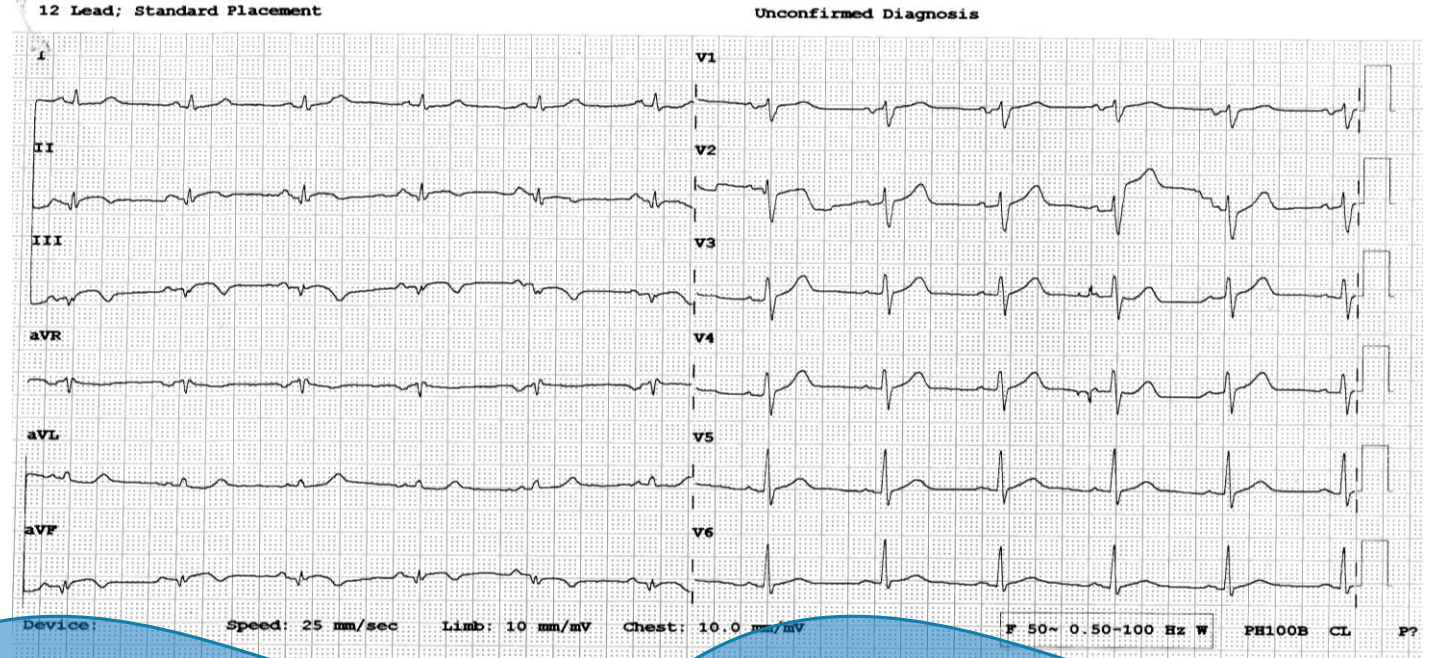
BP 110/70-mmHg

HR 75/min

D 2000ml

Th:

DAPT, high dose statin,  
PPI,



Most of the so-called RVI do not progress to an actual infarction, but the ECG findings usually represent an early, transient phenomenon. (demonstrated recently in a study with CMR).

**Several unique anatomic and physiological characteristics of the RV contribute to recovery from RV infarction:**

1. Pulmonary circulation is approximately one-tenth the length of systemic circulation, and a 5-mmHg perfusion gradient is sufficient to propel blood across the pulmonary circuit.
2. Unlike diastolic flow in the LV, the thin RV free wall allows the biphasic perfusion of coronary blood, with approximately equal contributions during systole and diastole.
3. RV has rich collaterals from the left anterior descending artery. Thus, RV infarcts may be clinically suspected in many patients with a stunned or hibernating RV free wall.

- In patient with inferior wall infarction and persistent hypotension always consider right ventricular infarction
  - Consider performing ECG with right precordial leads and confirmation with echocardiography, if possible with MRI
  - RV involvement is an important contributor to shock, which significantly increases the risk of mortality.
  - **Treatment strategies:**  
EARLY REVASCULARIZATION and  
HEMODYNAMIC STABILIZATION with:  
maintenance of an adequate heart rate, atrioventricular synchrony, volume balance,  
in severe cases: inotropes /vasopressors and IABP or LVAD in the newer era RVAD.
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## TAKE HOME MESSAGES



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