PCI for Late presentation

(beyond 12 hrs)

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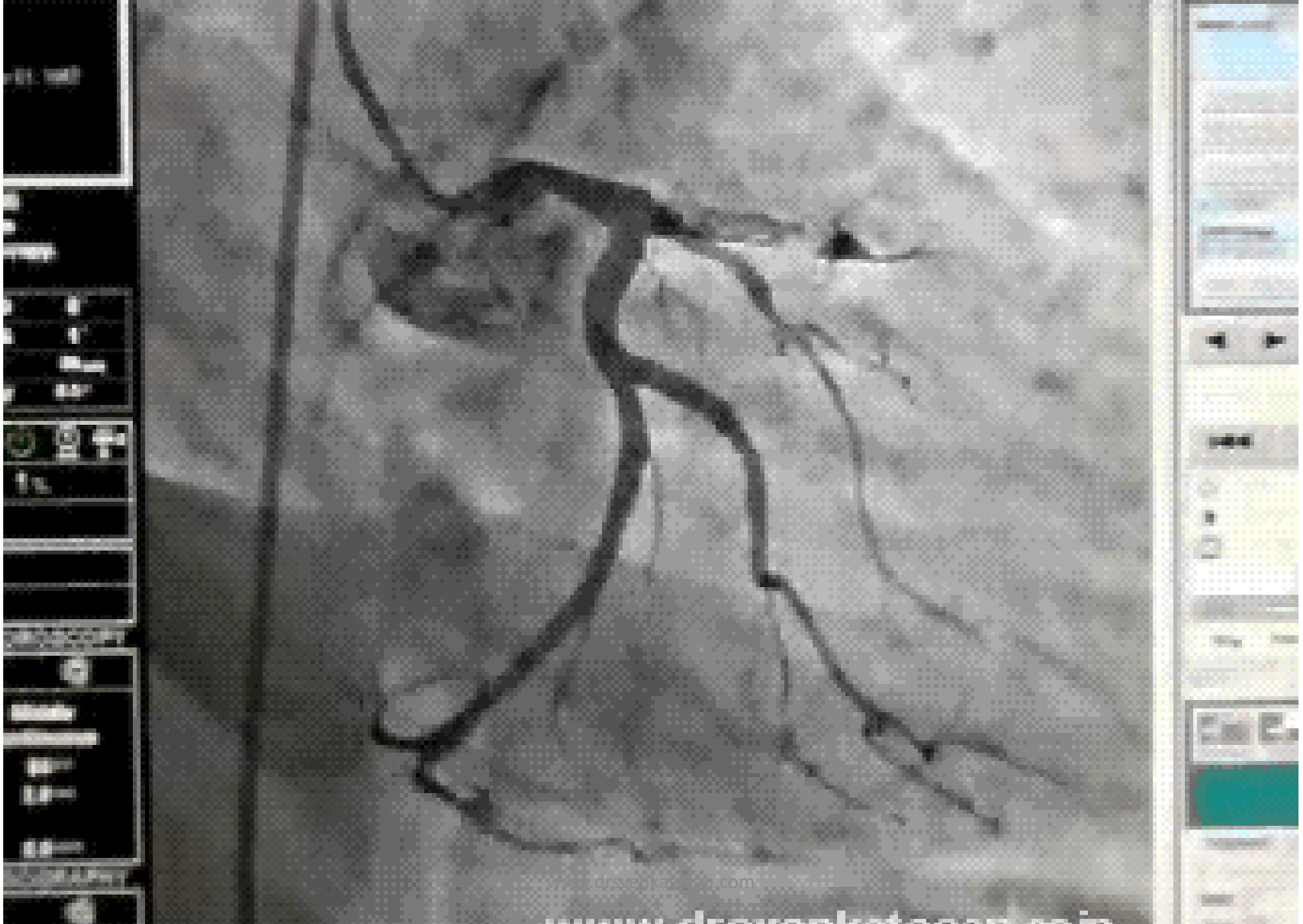
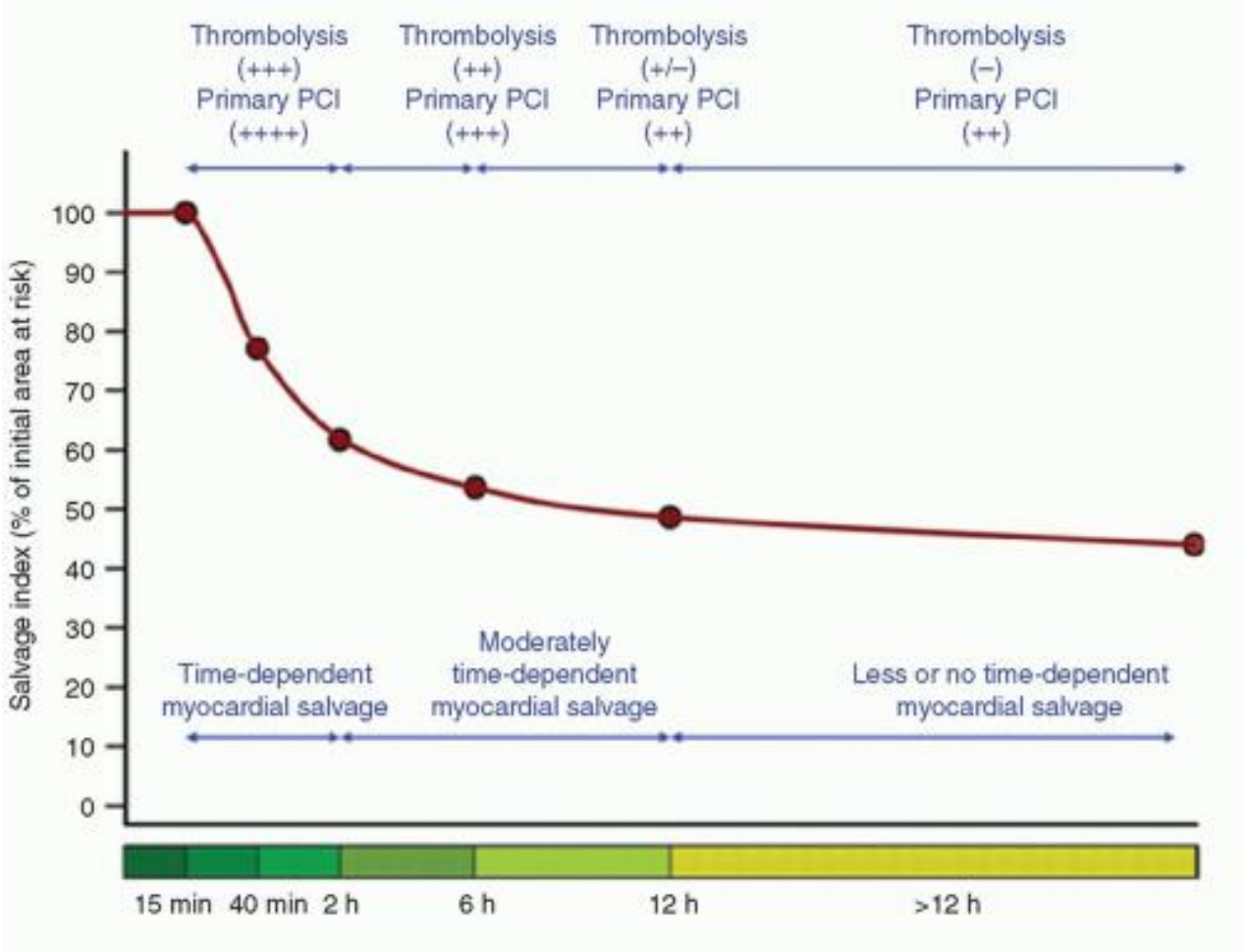


The most vital curve in STEMI



From: Schömig A, et al. *Eur Heart J.* 2006;27:1900-1907,

We are in best of times in science

Primary PCI : Superiority confirmed ?

We think so

Primary PCI is far superior to thrombolytic therapy when delivered in centres of excellence and without delay

Short-term mortality (5.3% versus 7.4%),

Non-fatal re-infarction (2.5% versus 6.8%) and

Stroke (1.0 versus 2.0%).[35,36] TIMI-3 flow may be restored in 95% of patients compared with 54% after thrombolysis.

(Zijlstra et al., 1999; Keeley et al., 2003; Andersen et al., 2003; Steg et al., 2012).

But … *Conditions apply

Pathological cascade

Table 10–3 Evolution of Morphologic Changes in Myocardial Infarction

Time Frame	Gross Features	Light Microscopic Findings	Electron Microscopic Findings
Reversible Injury			
0–½ hours	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½–4 hours	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4–12 hours	Occasionally dark mottling	Beginning coagulation necrosis; edema; hemorrhage	
12–24 hours	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; hypereosinophilic appearance of myocytes; marginal contraction band necrosis; beginning neutrophilic infiltrate	
1–3 days	Mottling with yellow-tan infarct center	Coagulation necrosis with loss of nuclei and striations; interstitial infiltrate of neutrophils	
3–7 days	Hyperemic border; central yellow-tan softening	Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border	
7–10 days	Maximally yellow-tan and soft, with depressed red-tan margins	Well-developed phagocytosis of dead cells; early formation of fibrovascular granulation tissue at margins	
10–14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2–8 weeks	Gray-white scar; progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 months	Scarring complete	Dense collagenous scar	

Is a race against time … not money

Contraction band necrosis

Myocardium won’t wait for traffic jams & Insurance clearance. It would welcome a prehospital ambulance lysis on any day than much glamorous pPCI.

STEMI general principles

Its all time stupid !

Time window errors
FMC vs Symptom to balloon time
Second medical contact always lags
Too many windows to worry about (,door, needel, sheath, balloon, wire etc)

No modality can beat prehospital lysis

What is late PCI ?

Late PCI

Some harsh questions before going for late PCI

How good is primary PCI for late presentation ? ie < 12 hrs

Great ?
Fair ?
Modest ?
Miniscule ?

Even in early , fast and furious primary PCI , the myocardium is not fully salvaged .In > 75% we are left with LV dysfunction.

Why do we think late PCI can defy myocardial patho clock ?

Trying to catch a late opportunity to salvage myocardium ?

At what cost ?

What risk ?

Open artery hypothesis

Closed myocardium ?

Time Independent benefits (Electrical stability , Favourable remodelling , Less progression to CHF Future collateral channel)

Benefits hard to accrue/ Not universal

Hypothetical ? Evidence base strong or shaky

BRAVE 2 study

The only meaningful study in favor of late PCI … still not cleared the confusion

Schömig A, et al. Mechanical Reperfusion in Patients With Acute Myocardial Infarction Presenting More Than 12 Hours From Symptom Onset. JAMA. 2005;293:2865-2872.

KAMIR NIH from Korea

(Tried hard to prove a point in favor of late PCI … but, curiously concluded delayed PCI is as effective as (or as Ineffective as ?) immediate PCI among late comers.

https://doi.org/10.1161/CIRCINTERVENTIONS.120.009863Circulation: Cardiovascular Interventions. 2021;14:e009863

Late PCI : Some personalised thoughts

Never hesitate to condole a late STEMI myocardial demise with a RIP

Fortunately RIP* is not death in myocardionomics(*Even TIMI 1 flow can stun it)

Expecting a miracle with late PCI is ok .But it is not a harmless exercise.

Routine poking of IRAs late after the accident invites more trouble as we are interfering its sleep with reperfusion injury.(Angry bird ?)

Late PCI following thrombolysis

How is this different from late Primary PCI ?

Who can define the mystery term “Ongoing ischemia” post STEMI ?

Late Pharmaco –Invasive approach : Need fresh data

By the way ,What is ongoing Ischemia ?

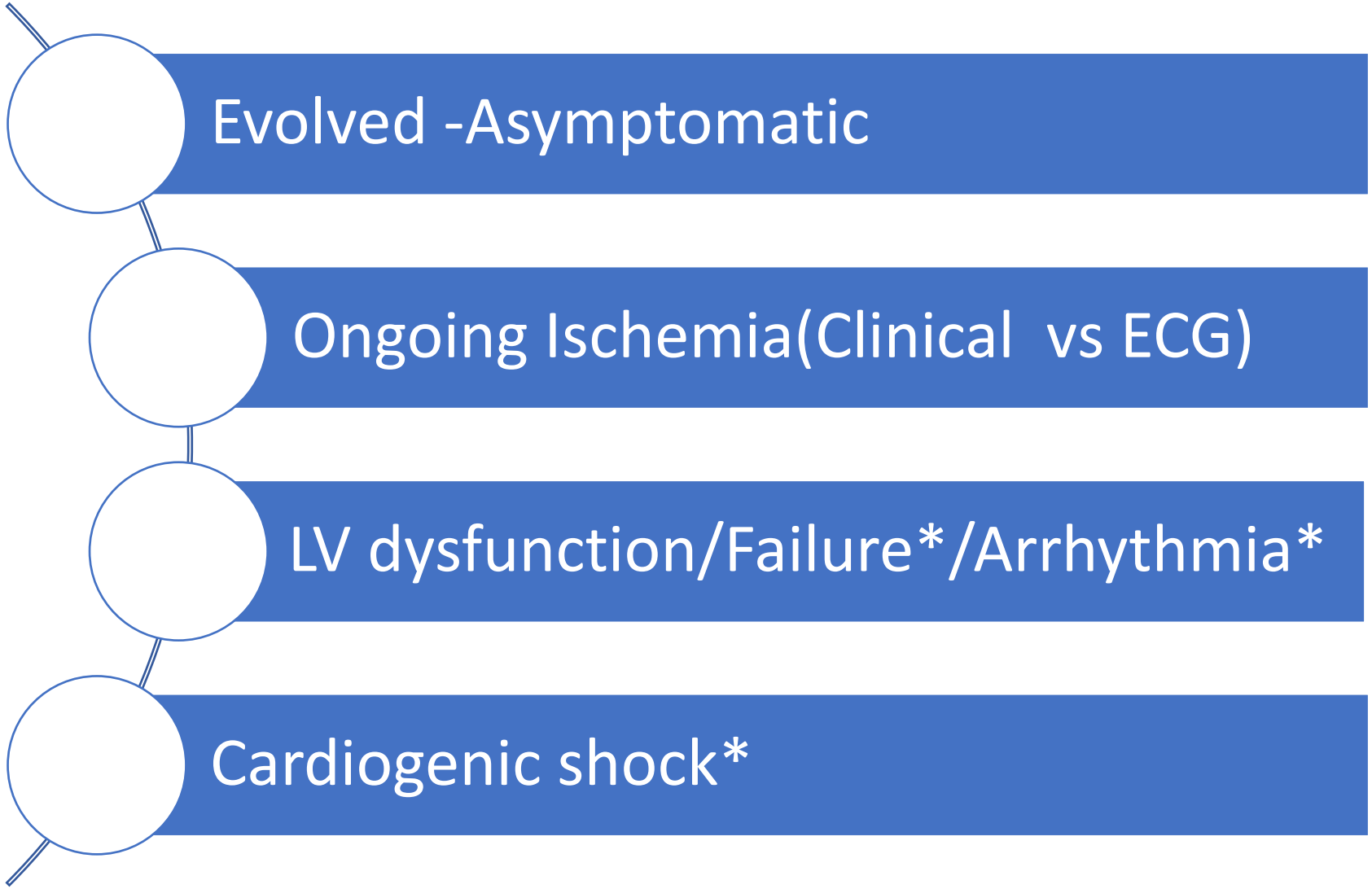
& where is it going !

Is it clinical, biochemical, ECG , WMA ?

Is residual TIMI 1- 2 flow Indicate ongoing Ischemia ?

No ,sub optimal TIMI flow can never be considered ongoing Ischemia if asymptomatic and ST segment resolute . (Many times It is due to transient increase in IMR* which will eventually reduce) Intramyocardial resistance

Latecomer subsets



* PCI not guaranteed benefit

Late PCI : Some more principles

Late IRA vs Non IRA PCI

Some times what is late for IRA is perfect prime time for non IRA critical lesion.

The concept of dual ACS is real (Manifest STEMI /Unmanifest or masked NSTEMI)

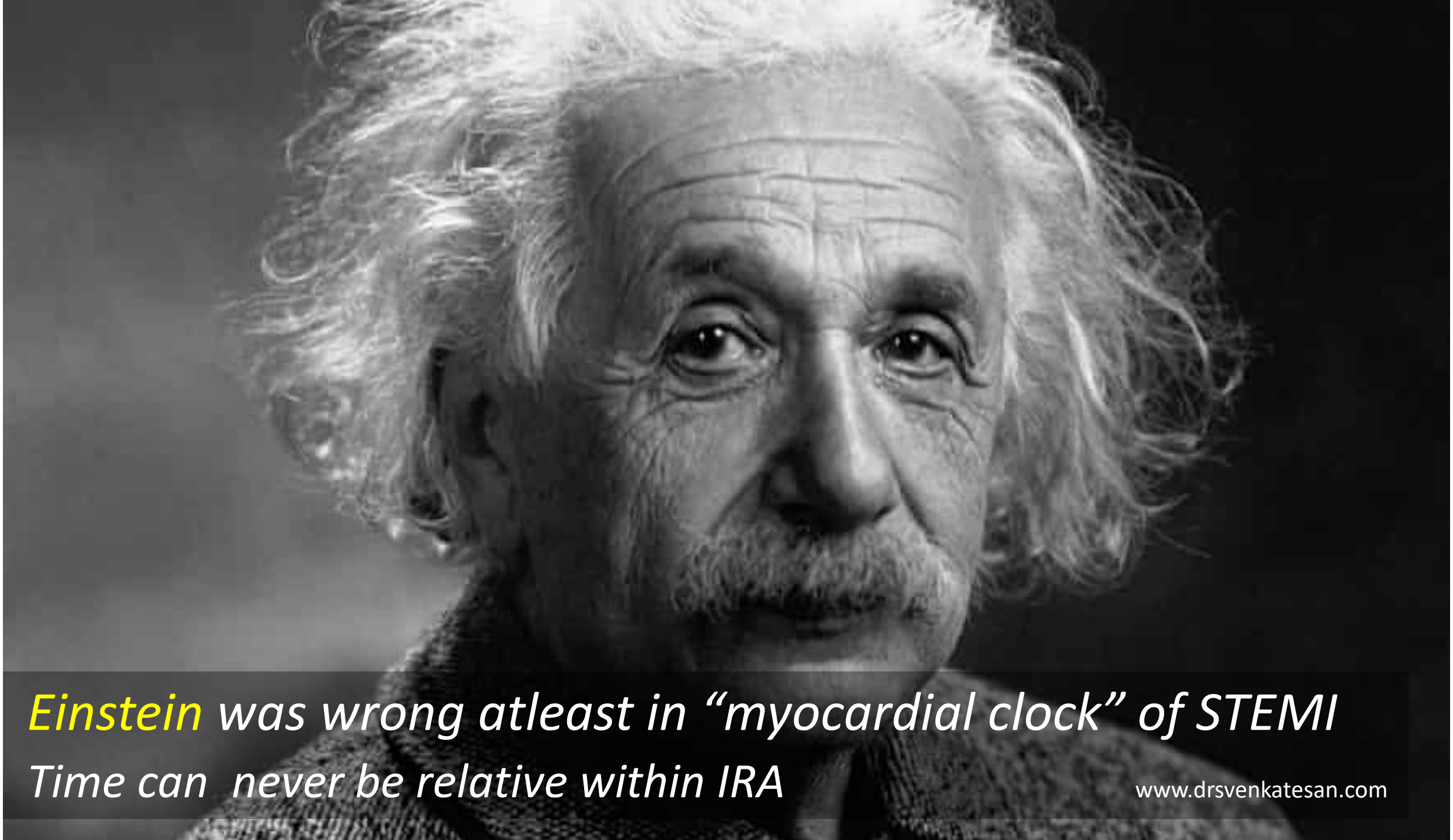
Lets try some conclusions

Time is not-relative within IRA

Late PCI : True "lateness" start from 6-12 hrs and not by 24 after IRA occlusion

PCI is not a magic modality. We can't allow it to enjoy a extended time window, that has been wrongly conferred by few of us.

*However , late PCI do have a life saving value **in a small** minority of STEMI with turbulent & Ischemic IRA*


Einstein was wrong atleast in “myocardial clock” of STEMI
Time can never be relative within IRA
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