

3d Libyan Society of Dialysis Annual Conference

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# Coronary Artery Disease in Dialysis Patients

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Tripoli, 2023, July 8<sup>th</sup>

1836, Dr Richard Bright:

*... It is observable, that the hypertrophy of the heart seems, in some degree, to have kept pace with the advance of disease in the kidneys; for in by far the majority of cases, where the muscular power of the heart was increased, the hardness and contraction of the kidney bespoke the probability of a long continuance of the disease.*



Bright R: Tabular view of the morbid appearances in 100 cases connected with albuminous urine, with observations, in Osman AA (ed) Original Papers of Richard Bright on Renal Disease, London, Humphrey Milford, Oxford University Press, 1937, pp 132-148



**Carl Ludwig (1816–1895),**

un pionnier de la physiologie, a présenté un nouveau concept de fonction rénale et d'interaction cardiorénale.

Legend: Picture downloaded from <https://research.uni-leipzig.de/agintern/UNIGESCH/ug175.htm> (accessed on 17 May 2022).



**Frederik Akbar Mahomed (1849-1884)**  
a décrit « *fibrose artério-capillaire du rein* »  
conséquence de l'élévation de PA

Mahomed  
FA. Trans Path Soc 1877;28:394-401



**Arthur C. Guyton 1919-2003**

décrit le SCR comme un dysfonctionnement cardiaque et rénal combiné aggravant progressivement la défaillance des deux organes

# MCV chez les patients atteints de MRC

## The New England Journal of Medicine

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Volume 290

MARCH 28, 1974

ARMANDO LINDNER, M.D., BERNARD GIARRA, M.D., DONALD J. SIERRA,  
BEIDING H. SCRIBNER, M.D.

**Armando Lindner**  
**Veterans Administration Medical Center**  
**4435 Beacon Avenue South**  
**Seattle, WA 98108**

**August 17, 1984**

## NKF 1997,

this observation motivated the members of the NKF to appoint a commission of experts in order to establish a preventive and therapeutic strategy for CVD in uremic patients.

Meyer KB, Levey AS. Controlling the epidemic of cardiovascular disease in chronic renal disease : **Report from the National Kidney Foundation Task Force on cardiovascular disease**. J Am Soc Nephrol 1998 ; 9 (Suppl. 12) : S31-S42.

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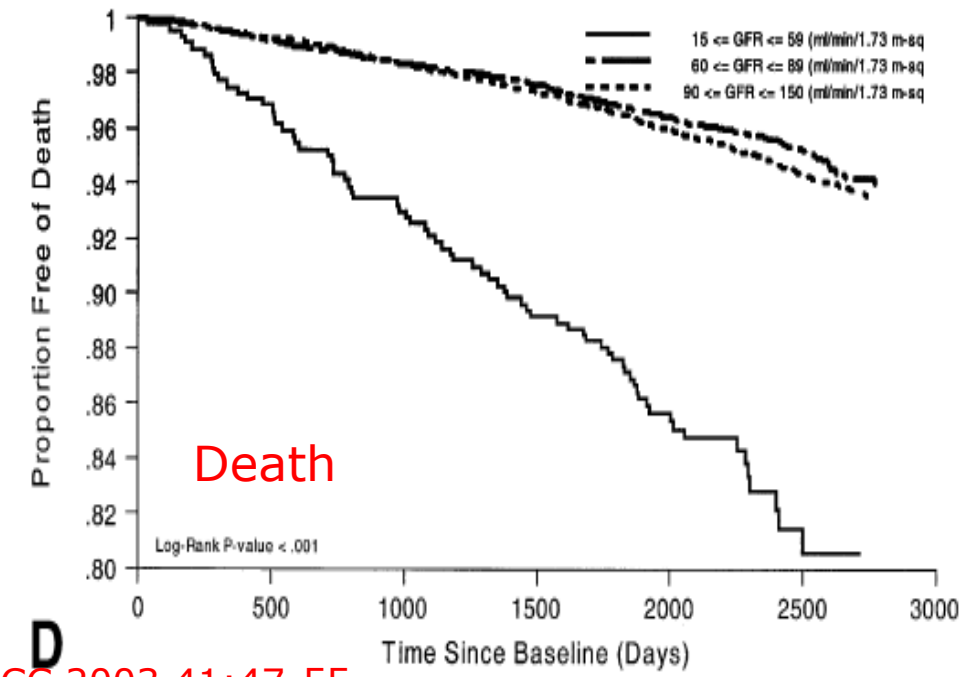
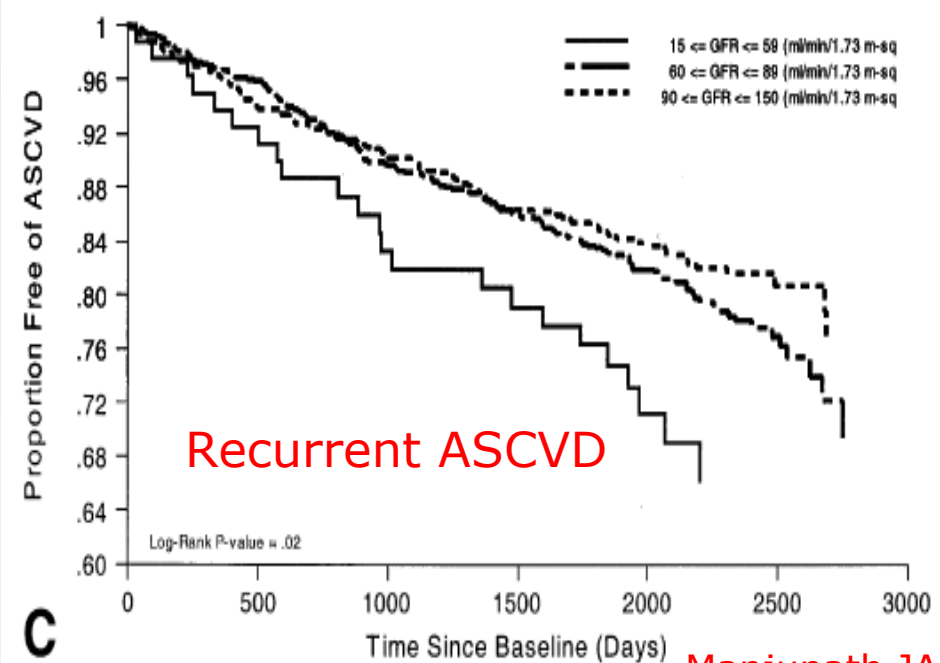
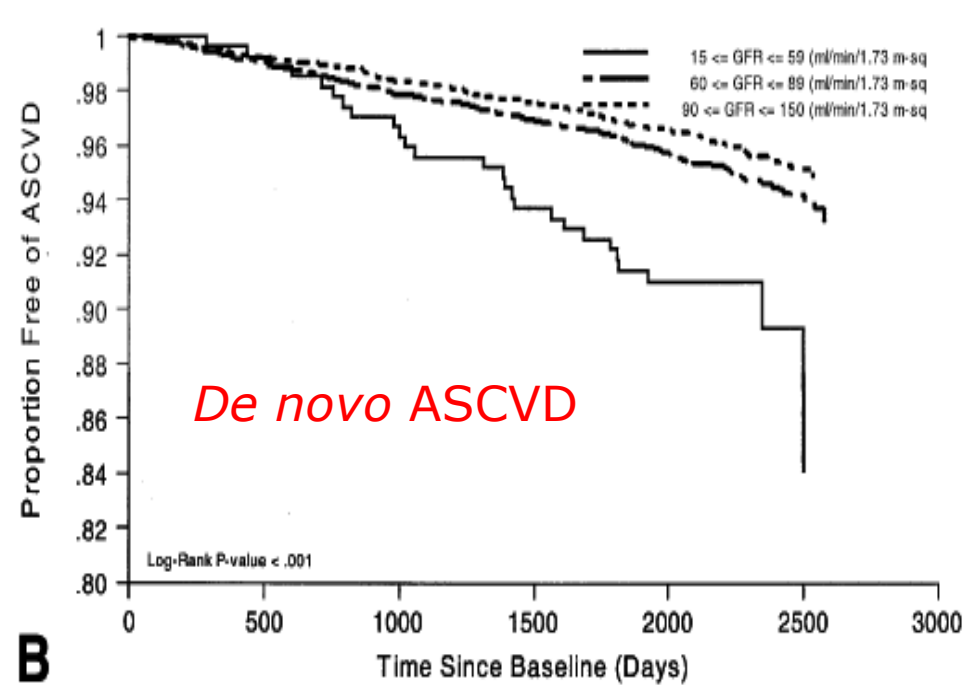
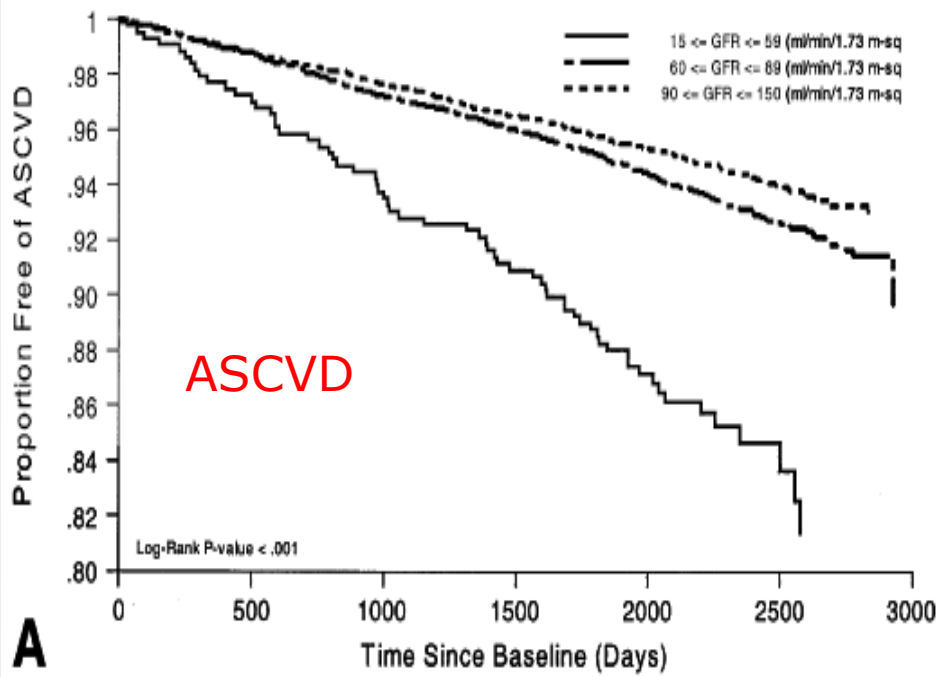
## Level of Kidney Function as a Risk Factor for Atherosclerotic Cardiovascular Outcomes in the Community

Guruprasad Manjunath, MD,\* Hocine Tighiouart, MS,† Hassan Ibrahim, MD,‡ Bonnie MacLeod, BS,†  
Deeb N. Salem, MD,§ John L. Griffith, PHD,† Josef Coresh, MD, PHD,|| Andrew S. Levey, MD,\*  
Mark J. Sarnak, MD\*

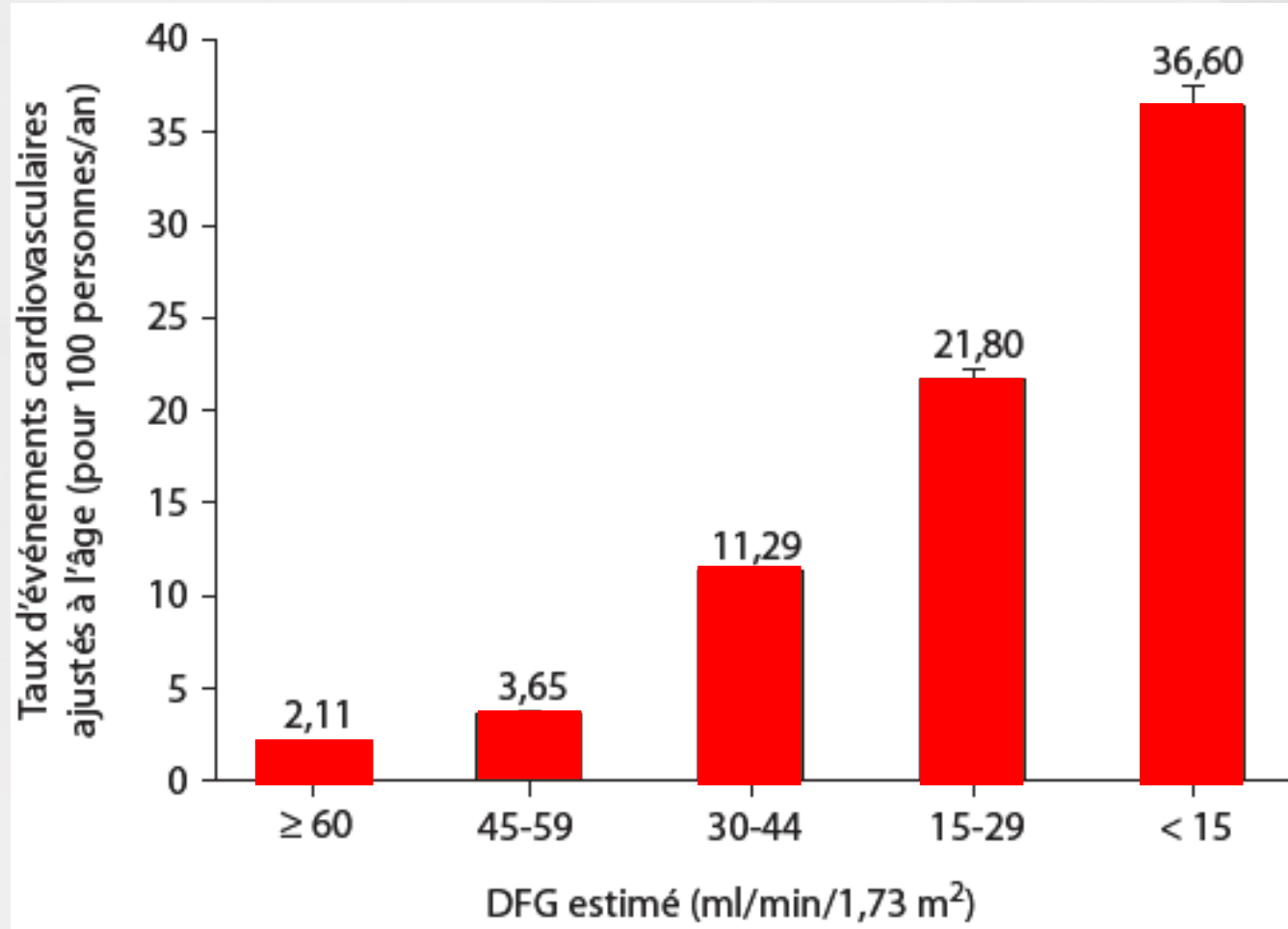
*Boston, Massachusetts; Minneapolis, Minnesota; and Baltimore, Maryland*

From the population based study of ARIC Study  
(Atherosclerosis Risk In Communities)

**Conclusions.** The level of kidney function is an independent risk factor for ASCVD in middle-aged subjects who are not selected for being at higher risk for CVD.



# MACE risk related to GFR rate



Even after adjustment for known CAD risk factors, including diabetes and hypertension, mortality risk progressively increases with worsening CKD

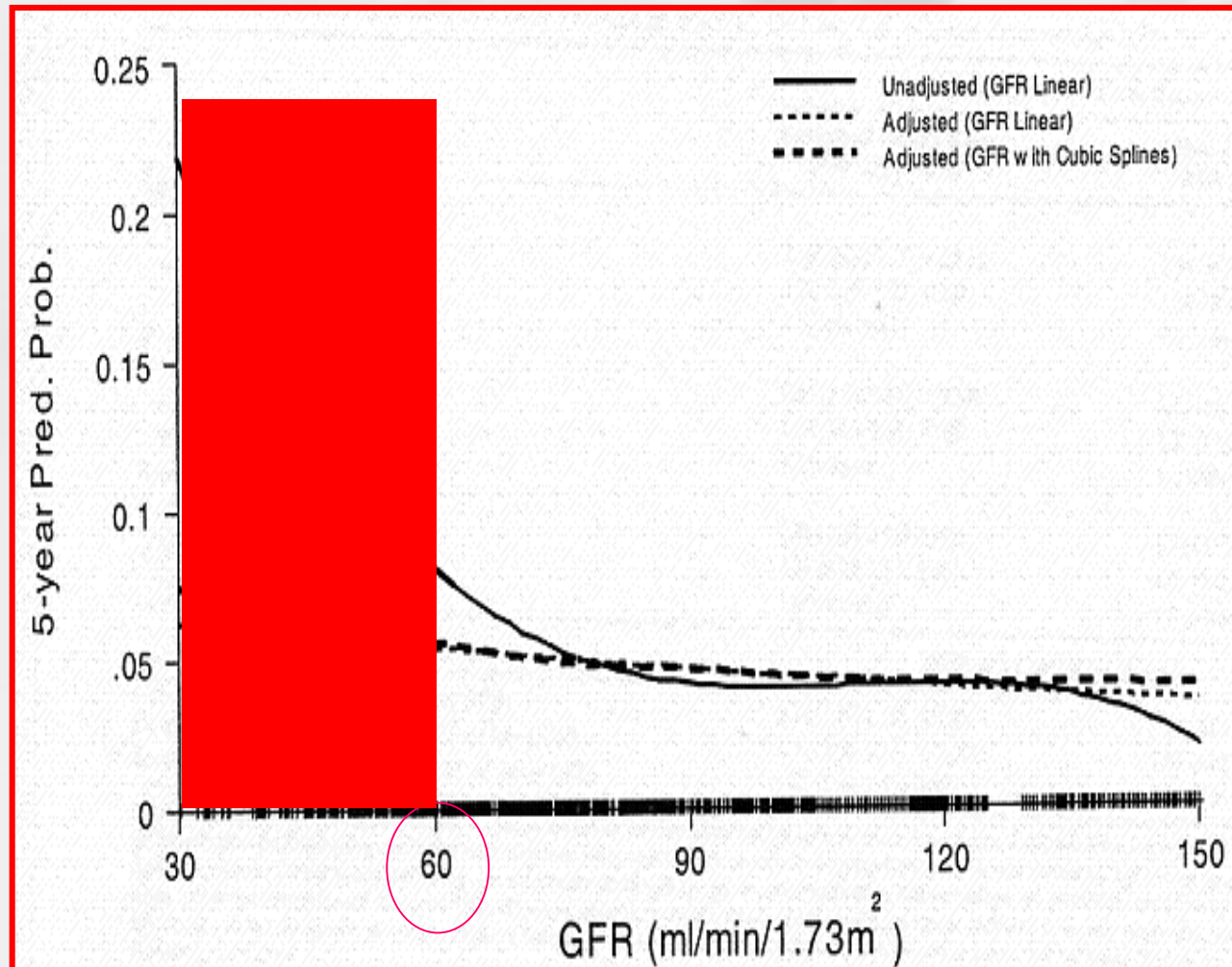
*CKD Prognosis Consortium, Matsushita K, et al. Lancet 2010;375:2073–81.  
Go A S et al. N Engl J Med 2004;351:1296–305.*

Go A S et al. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med* 2004;**351**:1296–305.

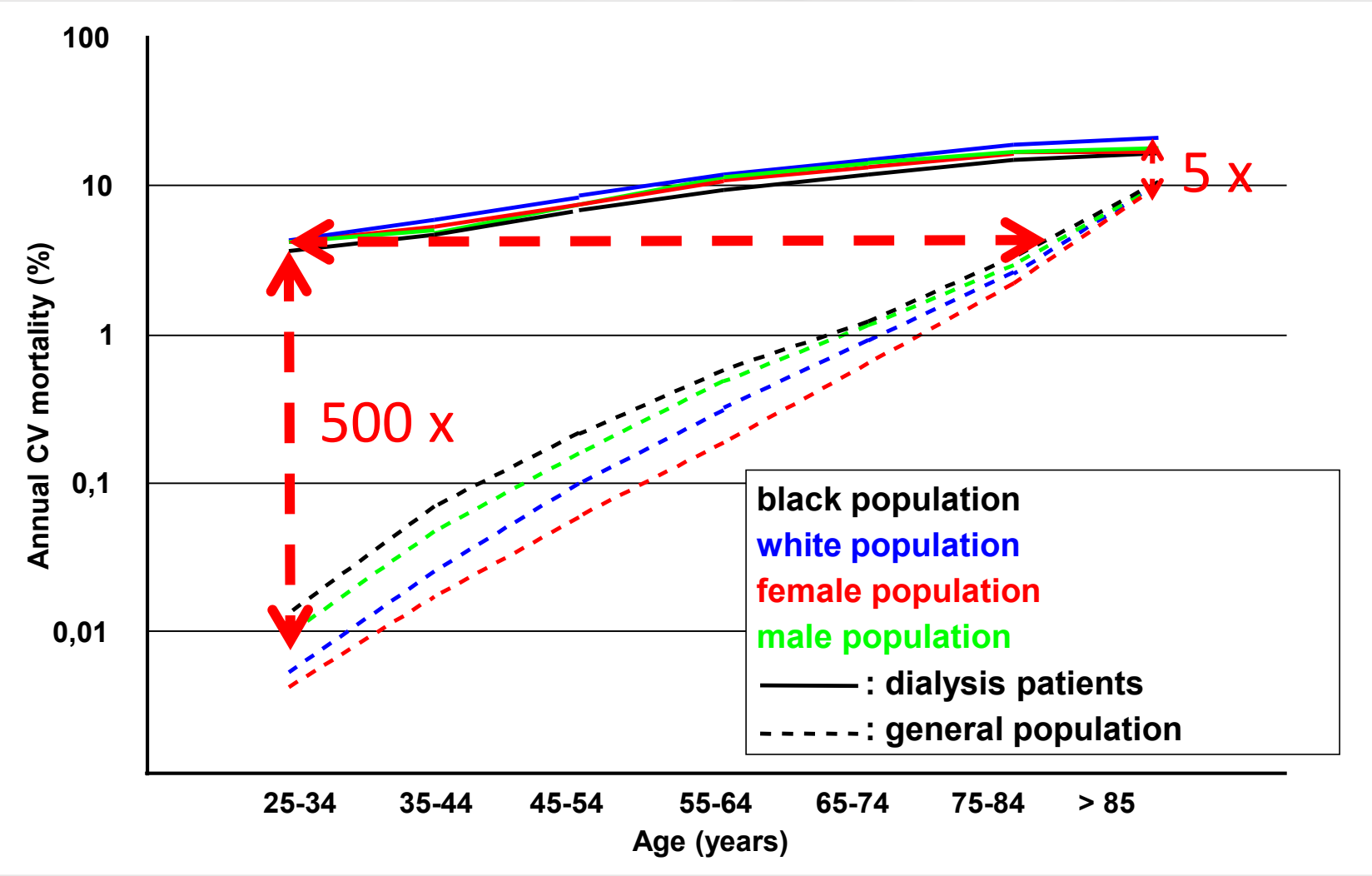


As GFR declines below 60 ml/min/1.73 m<sup>2</sup>,  
the probability of developing CAD increases linearly

*Manjunath JACC 2003,41:47-55*



# Cardiovascular Mortality in dialysis patients is dramatically higher than in the general population



Foley RN et al. AJKD 1998.



ORIGINAL INVESTIGATION

## Longitudinal Follow-up and Outcomes Among a Population With Chronic Kidney Disease in a Large Managed Care Organization

Douglas S. Keith, MD; Gregory A. Nichols, MBA, PhD; Christina M. Gullion, PhD; Jonathan Betz Brown, MPP, PhD; David H. Smith, RPh, PhD

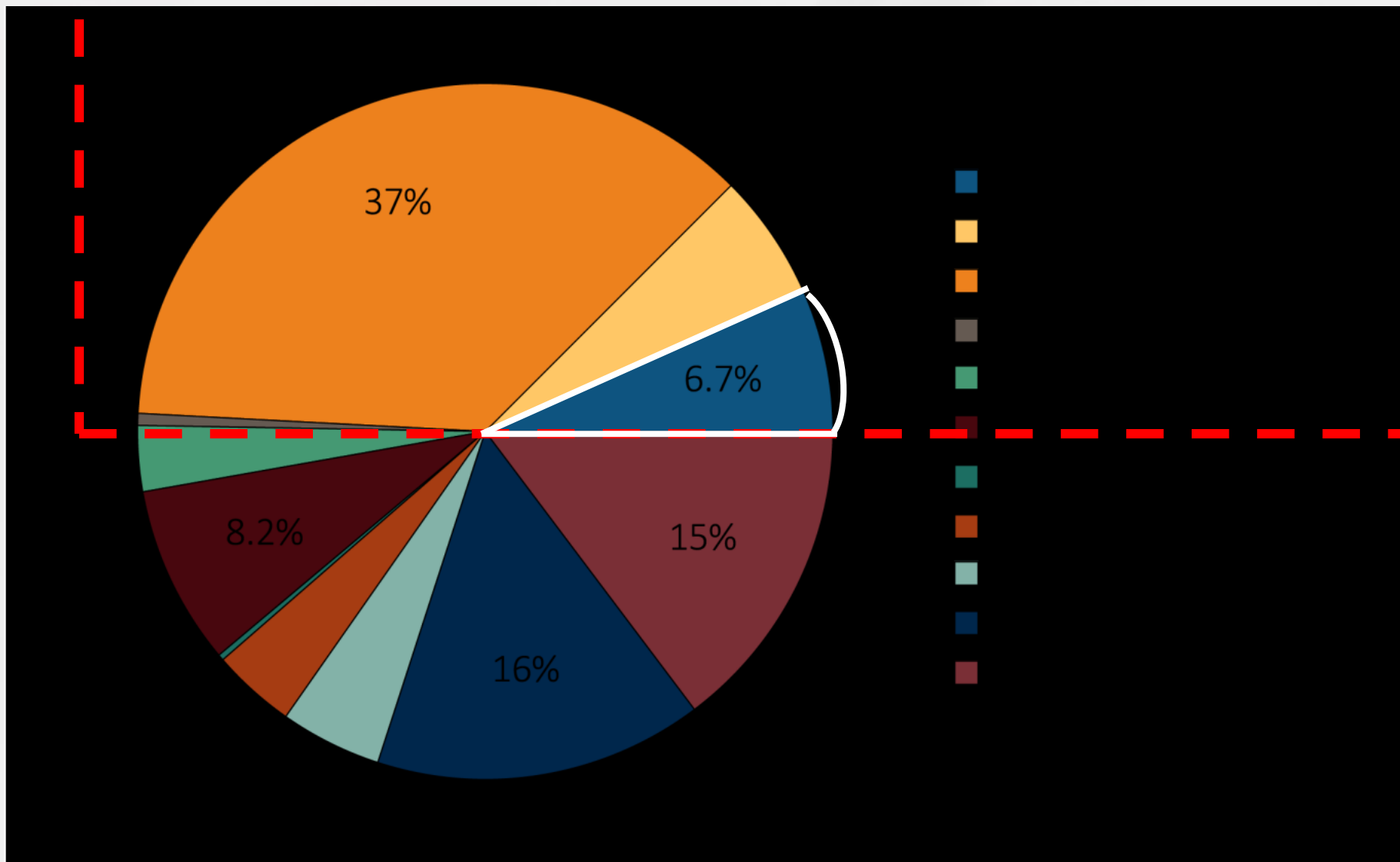
CKD: Survival at 5 years

Critère	G2: 60-89ml/mn	G3: 30-59ml/mn	G4: 15-29ml/mn
Progression to G5 (ESRD)	1.1%	1,3%	19,9%
Death	19,5%	24,3%	45,7%

**Nephrology job in 2023:**  
**Prevent accelerated deterioration of kidney function & prevent CVD**

Keith DS, et al. Arch Intern Med 2004; 104: 659-663

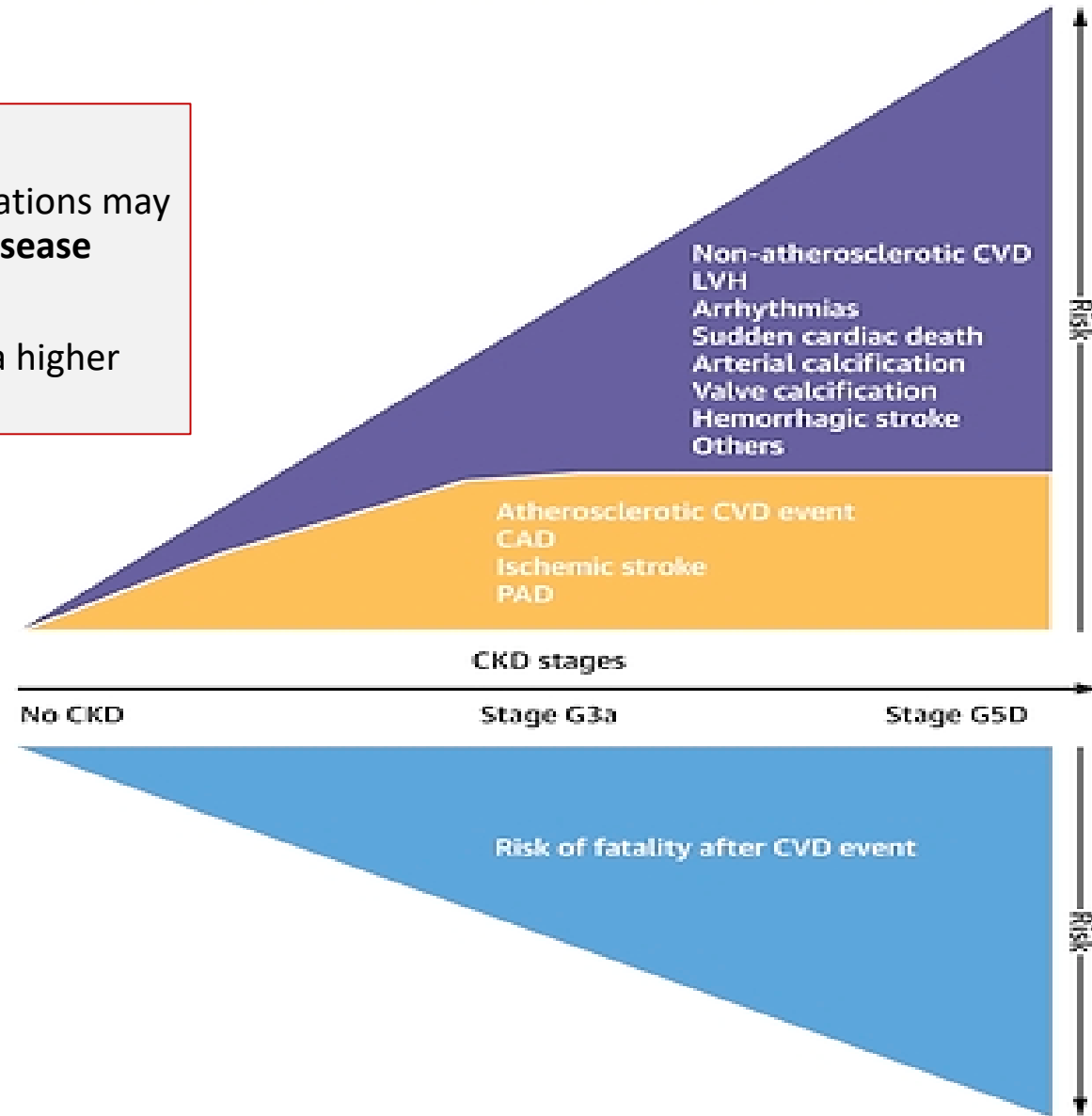
# Causes of death in ESRD patients, 2013



## CENTRAL ILLUSTRATION: Changes in Cardiovascular Disease Risk During Chronic Kidney Disease Progression

Sudden death is particularly common in ESKD, the shifts in volume, electrolytes, and drug concentrations may **trigger arrhythmias in patients with a myocardial disease** (LVH and heart failure).

As GFR declines, nonatherosclerotic events assume a higher proportion of the CVD events



# CKD and ESKD modify the clinical presentation and cardinal symptoms of CAD.

CKD patients: more likely to have an AMI, rather than stable exertional angina = as initial clinical manifestation of CAD

*Go AS et al. J Am Coll Cardiol 2011;58:1600–7.*

**“oligo-symptomatic” presentation is common:**

Patients with CKD  $<60\text{ml}/\text{mn}/1.73\text{m}^2$  who present with AMI report:

- chest, arm, shoulder, or neck pain in only **44%** (vs **72%** of patients with preserved kidney function),
- More likely be **dyspneic**

*Sosnov J, et al. Am J Kidney Dis 2006;47:378–84*

**Dialysis** patients with AMI presentations report

- chest pain in **44%** vs **68%** of nondialysis patients

*Herzog Caet al. Circulation 2007;116:1465–72.*

Atypical presentation of coronary syndromes in CKD patients  
=> Anginal equivalents/ **shortness of breath or fatigue.+++**

A low functional capacity, (common among ESKD patients), *Burton JO et al. Clin J Am Soc Nephrol 2009;4:1925–31. 7.*

Intradialytic hypotension and myocardial stunning =HD-specific syndromes associated with mortality

*Stefansson BV et al. . Clin J Am Soc Nephrol 2014;9: 2124–32.*

# It is more likely to be a non-NSTEMI than an ST-segment elevation myocardial infarction (STEMI) among patients on dialysis

*Shroff GR et al. J Am Soc Nephrol 2017;28: 1379–83.*

**Table 2.** Trends in prevalence of AMI, demographic characteristics, and claims for primary versus secondary AMI among patients on dialysis

Prevalence, Characteristics, and Claims	Year			
	1993	1998	2003	2008
Total period prevalent patients on dialysis, n	178,846	254,255	320,038	383,354
Prevalence of AMI, n (% total patients on dialysis)				
Total	4494 (2.5)	8081 (3.2)	14,232 (4.4)	16,361 (4.3)
NSTEMI	1898 (1.1)	4502 (1.8)	9701 (3.0)	13,210 (3.4)
STEMI	1658 (0.9)	2245 (0.9)	2331 (0.7)	1571 (0.4)
Unspecified	938 (0.5)	1334 (0.5)	2200 (0.7)	1580 (0.4)
Primary AMI (coded as principal diagnosis), n (% total AMI)	2904 (64.6)	5094 (63.0)	8363 (58.8)	8505 (52.0)
Type of AMI, n (% of primary AMI)				
NSTEMI	1329 (45.8)	2959 (58.1)	5831 (69.7)	6914 (81.3)
STEMI	1158 (39.9)	1589 (31.2)	1583 (18.9)	1020 (12.0)
Unspecified	417 (14.4)	546 (10.7)	949 (11.3)	571 (6.7)
Secondary AMI (not coded as principal diagnosis), n (% total AMI)	1590 (35.4)	2987 (37.0)	5869 (41.2)	7856 (48.0)
Type of AMI, n (% of secondary AMI)				
NSTEMI	576 (36.2)	1553 (52.0)	3877 (66.1)	6305 (80.3)
STEMI	488 (30.7)	642 (21.5)	725 (12.4)	505 (6.4)
Unspecified	526 (33.1)	792 (26.5)	1267 (21.6)	1046 (13.3)

Denominator estimated from period prevalent patients on hemodialysis and on peritoneal dialysis reported in figure 1 of volume 2 of the 2012 US Renal Data System Annual Data Report (<https://www.usrds.org/atlas12.aspx>).

These Non-STEMI presentations may reflect

- a supply-demand mismatch,
- ischemic pre-conditioning,
- collateralization of blood vessels,
- perhaps a higher prevalence of LVH altering the ECG findings

there is less plaque rupture with superimposed occlusive thrombus.

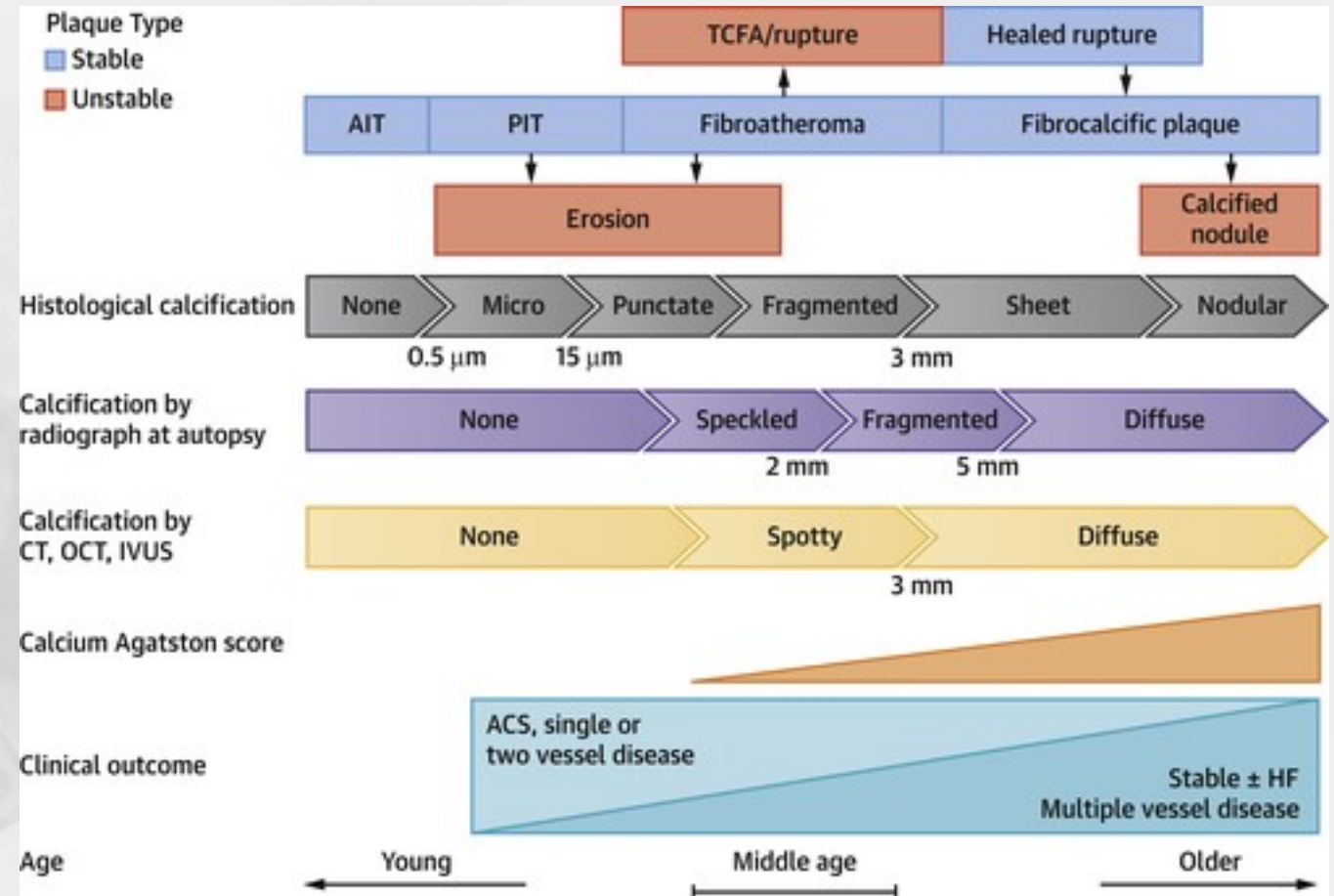
**Microcalcification** occurs primarily in younger patients and is particularly associated with inflammation and plaque instability leading to acute coronary syndromes (ACS).

**Macrocalcifications** tend to occur in older patients with more stable CAD and multivessel CAD

Atherosclerosis in early CKD is driven by traditional CAD risk factors,

Nontraditional risk factors play a predominant role as GFR declines, leading to fibrocalcific lesions

It is not exactly clear how CKD modifies this paradigm although as mentioned calcified and more advanced plaques are highly prevalent in CKD



**Modification of lipoproteins** (e.g., LDL carbamylation, HDL dysfunction) in CKD likely **contributes to accelerated progression of CAD**, and risk factors for calcification include inflammation, senescence, mechanical factors (e.g., shear stress, elastin fatigue), and potentially accumulation of microbiome-dependent metabolites such as trimethylamine N-oxide



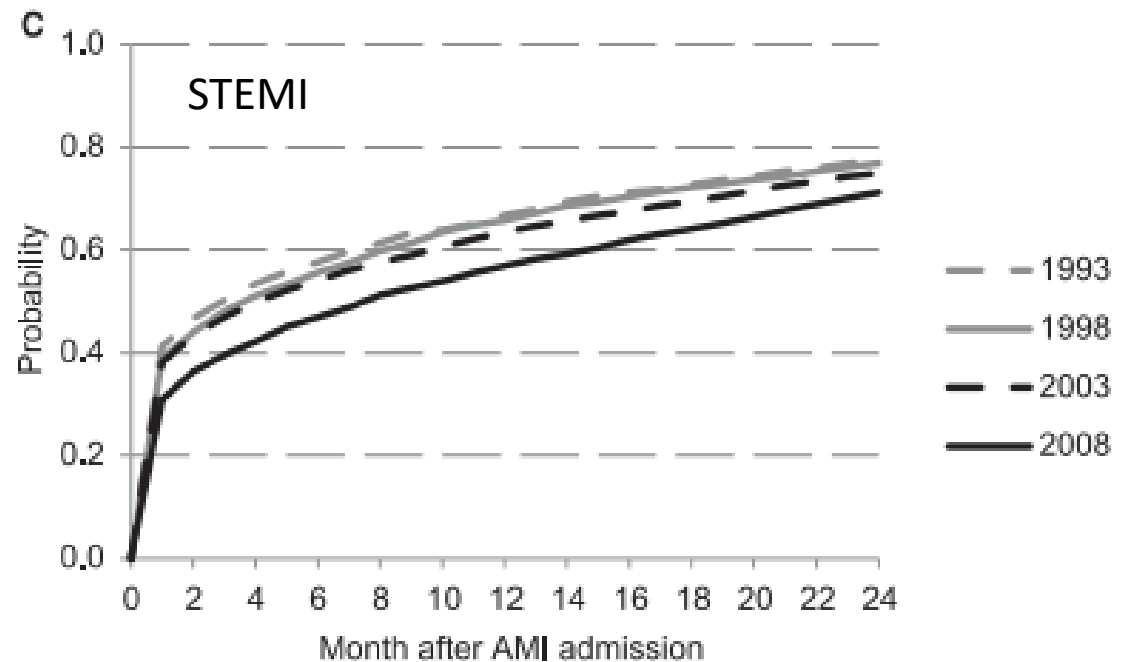
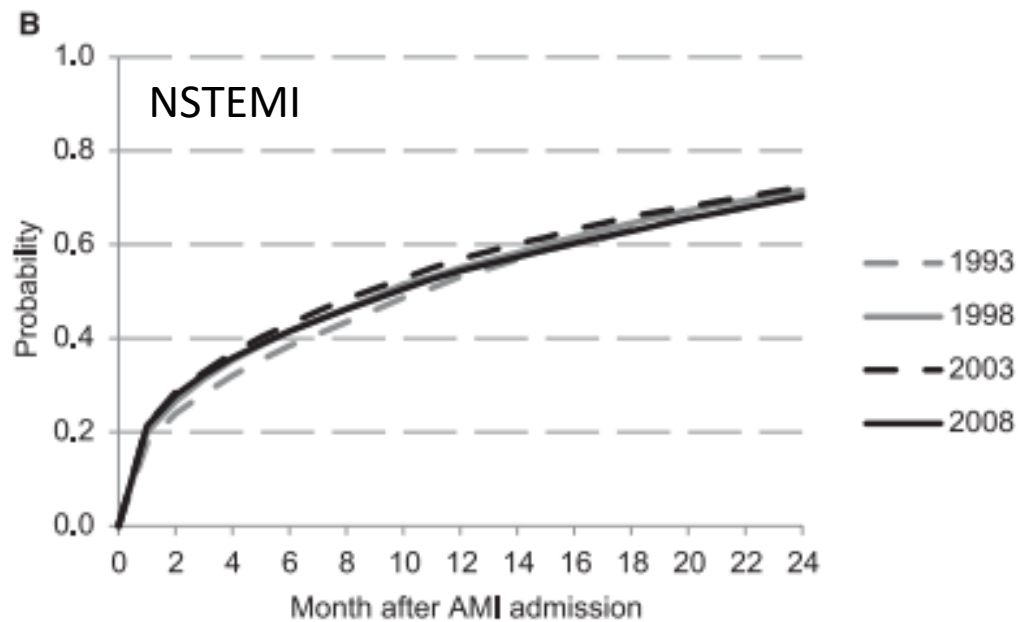
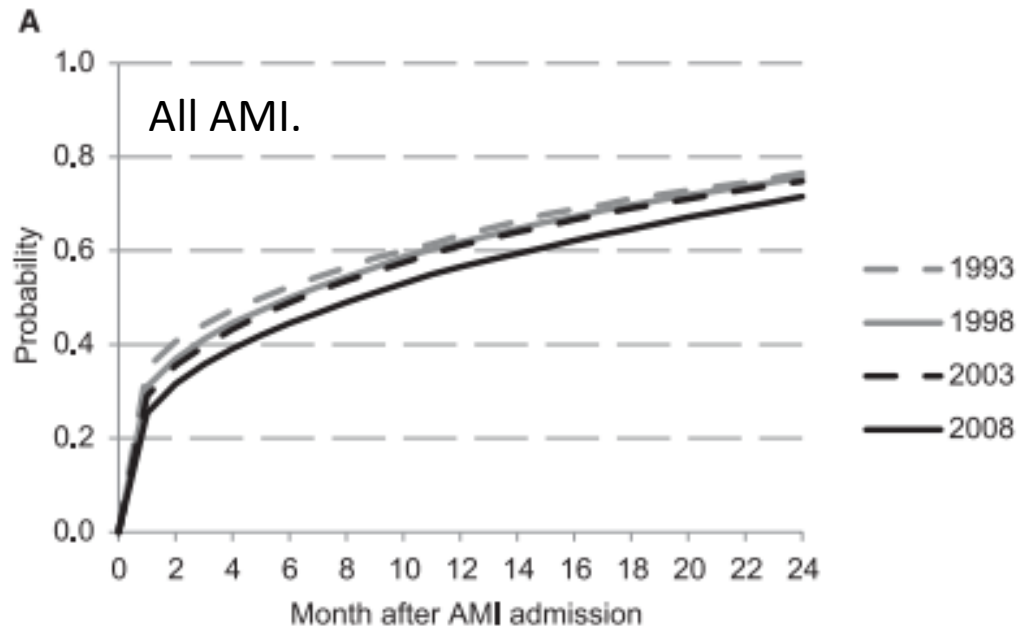


## Trends in Discharge Claims for Acute Myocardial Infarction among Patients on Dialysis

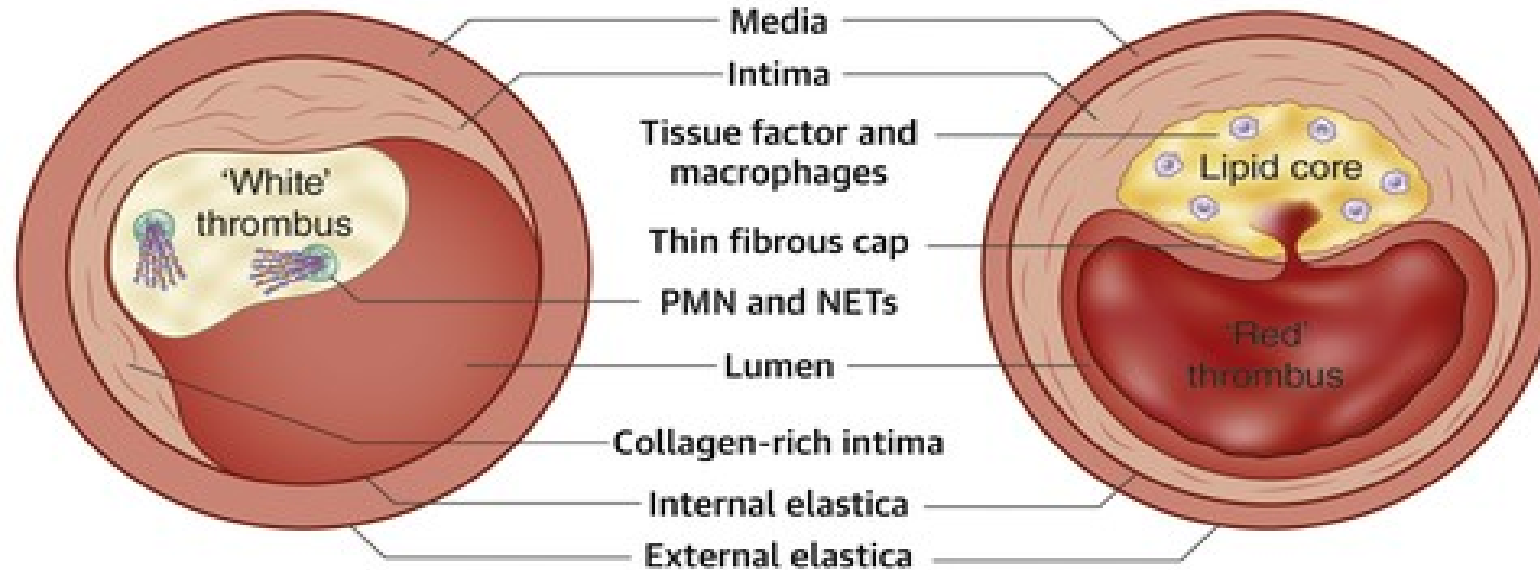
Gautam R. Shroff,\* Shuling Li,† and Charles A. Herzog\*†

### Trends in mortality following AMI among dialysis patients.

Unadjusted cumulative probability of mortality after index hospitalization for AMI in period prevalent patients on dialysis in 1993, 1998, 2003, and 2008.



## Coronary Artery Cross-Sections



### Thrombosis due to erosion

- Fibrous cap thick and intact
- 'White' platelet-rich thrombus
- Collagen trigger
- Smooth muscle cells prominent
- Often sessile, non-occlusive thrombus
- Usually less remodeled outward
- NETs involved
- More frequent in non-STEMI?

### Thrombosis due to rupture

- Thin fibrous cap with fissure
- 'Red' fibrin-rich thrombus
- Tissue factor trigger
- Macrophages prominent
- Often occlusive thrombus
- Usually expansively remodeled
- Less NET involvement?
- More frequently cause STEMI?

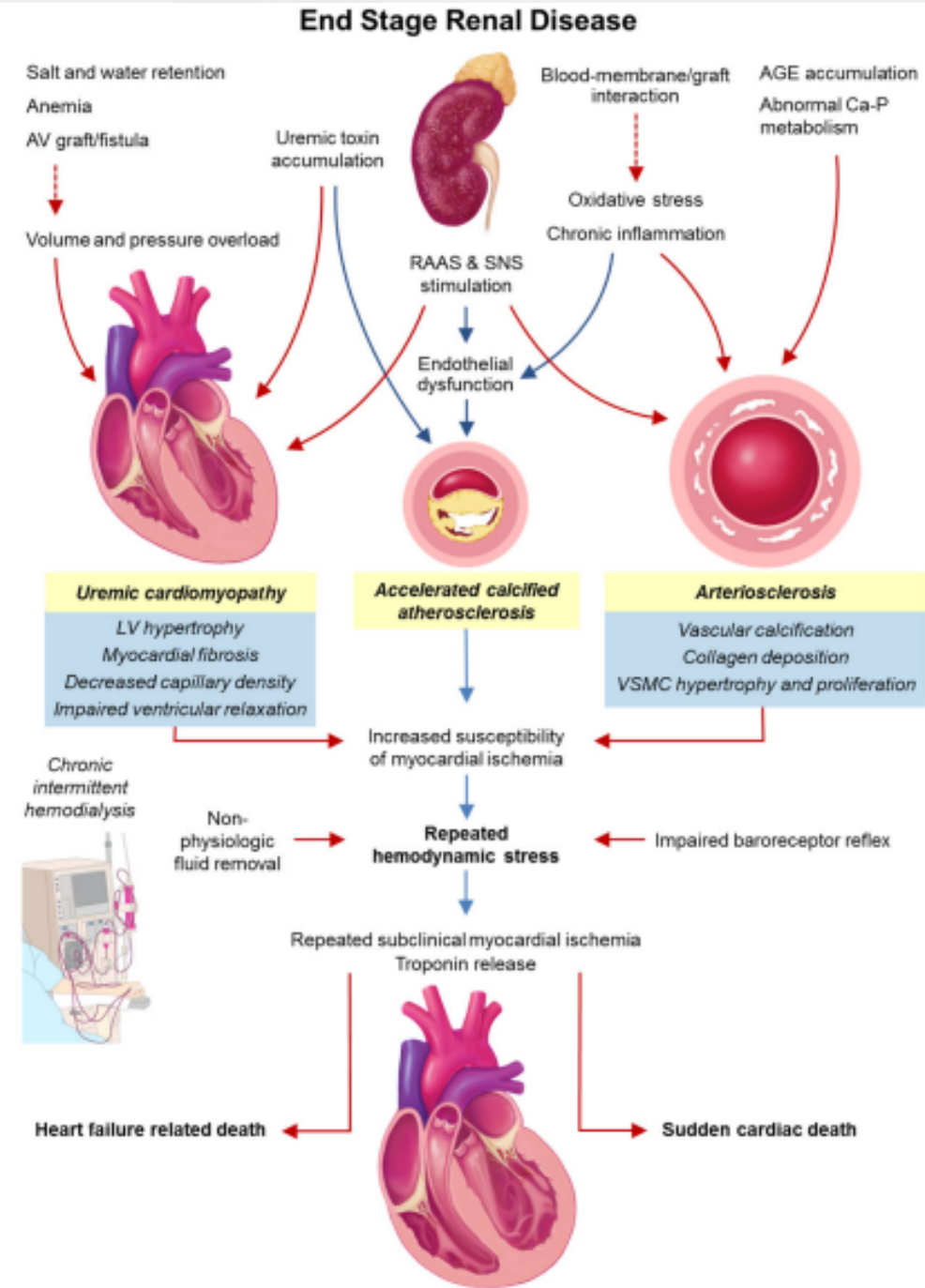
The coronary artery cross section showing thrombosis due to erosion (left) and rupture (right).

ACS :acute coronary syndrome; NET: neutrophil extracellular trap; PMN: polymorphonuclear leukocytes; STEMI: ST-segment elevation myocardial infarction.

# Pathophysiology of hemodialysis-induced myocardial injury

Cardiovascular Impact in Patients Undergoing Maintenance Hemodialysis: Clinical Management Considerations

Int J Cardiol. 2017 April 01; 232: 12–23



Abbreviations: AV, arterio-venous; AGE, advanced glycation end product; RAAS, renin angiotensin aldosterone system; SNS, sympathetic nervous system

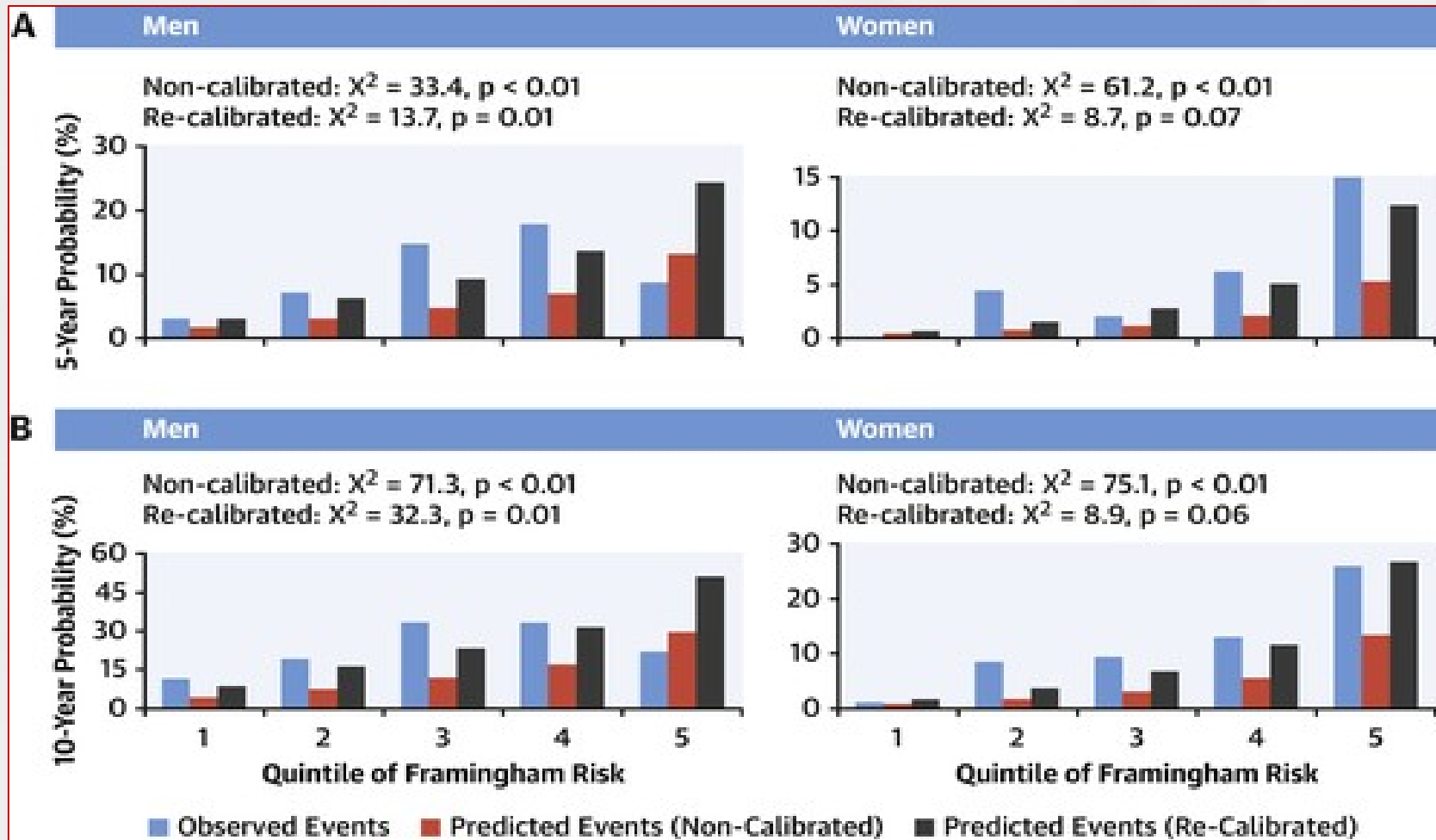
## Regular assessment for atherosclerotic CVD risk should be distinguished from screening for asymptomatic CAD.

- absence of evidence that pre-emptive coronary revascularization is effective in reducing death or MI risk in asymptomatic patients
- screening for underlying anatomic CAD lacks either a rationale or evidence—even in at-risk asymptomatic patients
- there is a rationale for screening in transplant candidates

*Young LH, et al., for the DIAD Investigators. JAMA 2009; 301:1547–55.*

# Risk assessments (e.g., from the pooled cohorts equation) that inform decisions about CAD prevention rest on population studies

Goff DC Jr, et al. *J Am Coll Cardiol* 2014;63:2935–59



Graphical presentation of actual **5-year (A)** and **10-year (B)** risk of **cardiac outcomes** in CKD patients along with predicted risk, with and without recalibration for higher event rates in CKD **stratified by quintile of predicted Framingham risk**

Patients with CKD exemplify the shortcomings of risk assessment from population data, as their predicted risks are well below their observed risk, and model discrimination is poor. Unfortunately, this underestimation is nonuniform, so recalibration of the pooled cohort equations is not sufficient to resolve inaccuracies in risk stratification in CKD

Weiner DE et al. *J Am Coll Cardiol* 2007;50:217–24.

# Diagnosis:

**Exercise testing** : reduced accuracy for detecting CAD in CKD : (higher rate of both false-negative and false-positive tests)

- limited by an inability of CKD patients to reach diagnostic workloads
- baseline ECG abnormalities (e.g., LVH) = limit ability to detect ST-segment changes during exercise
- most existing data were derived from studies of transplant candidates

*Winther S et al. J Am Coll Cardiol Img 2015;8:553–62*

*Patel RK et al. Am J Transplant 2008;8:1673–83*

*Wang LW et al. Cochrane Database Syst Rev 2011;12:CD008691*

## Functional stress testing & noninvasive coronary imaging

- Symptomatic patient
- Asymptomatic potential transplant recipient,

These tests are more widely used in individuals with advanced CKD than in those with preserved kidney function

*Herzog CA et al. J Am Coll Cardiol Img 2019;12:1420–6.*

kidney transplant candidates: 

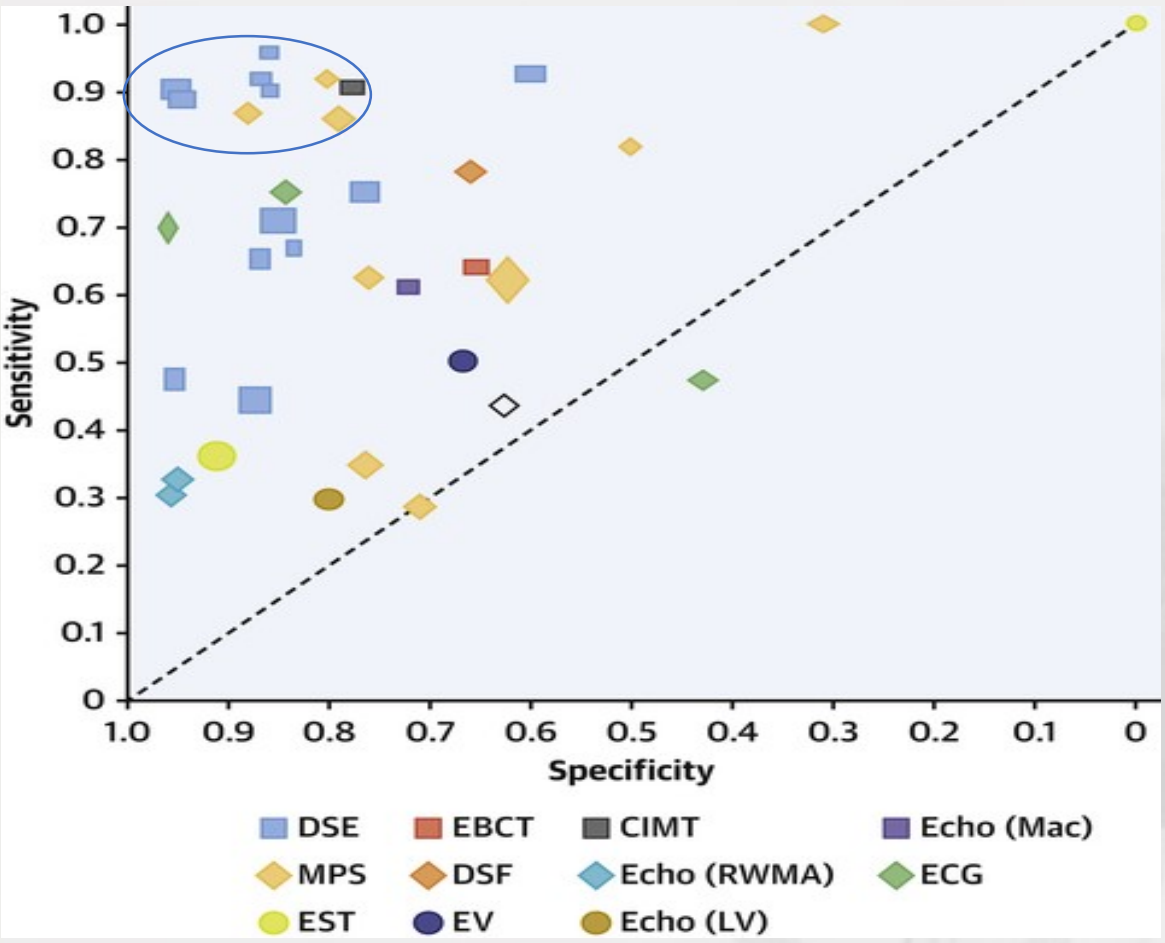
- Myocardial perfusion scintigraphy
- Dobutamine stress echocardiography

moderate accuracy for detecting obstructive atherosclerosis

*the extent to which these data are generalizable to dialysis patients or nontransplant candidates who are likely to have more comorbidities, a lower functional capacity, and a higher burden of atherosclerosis is uncertain*

*Coronarography: if any doubt on a possible CAD*

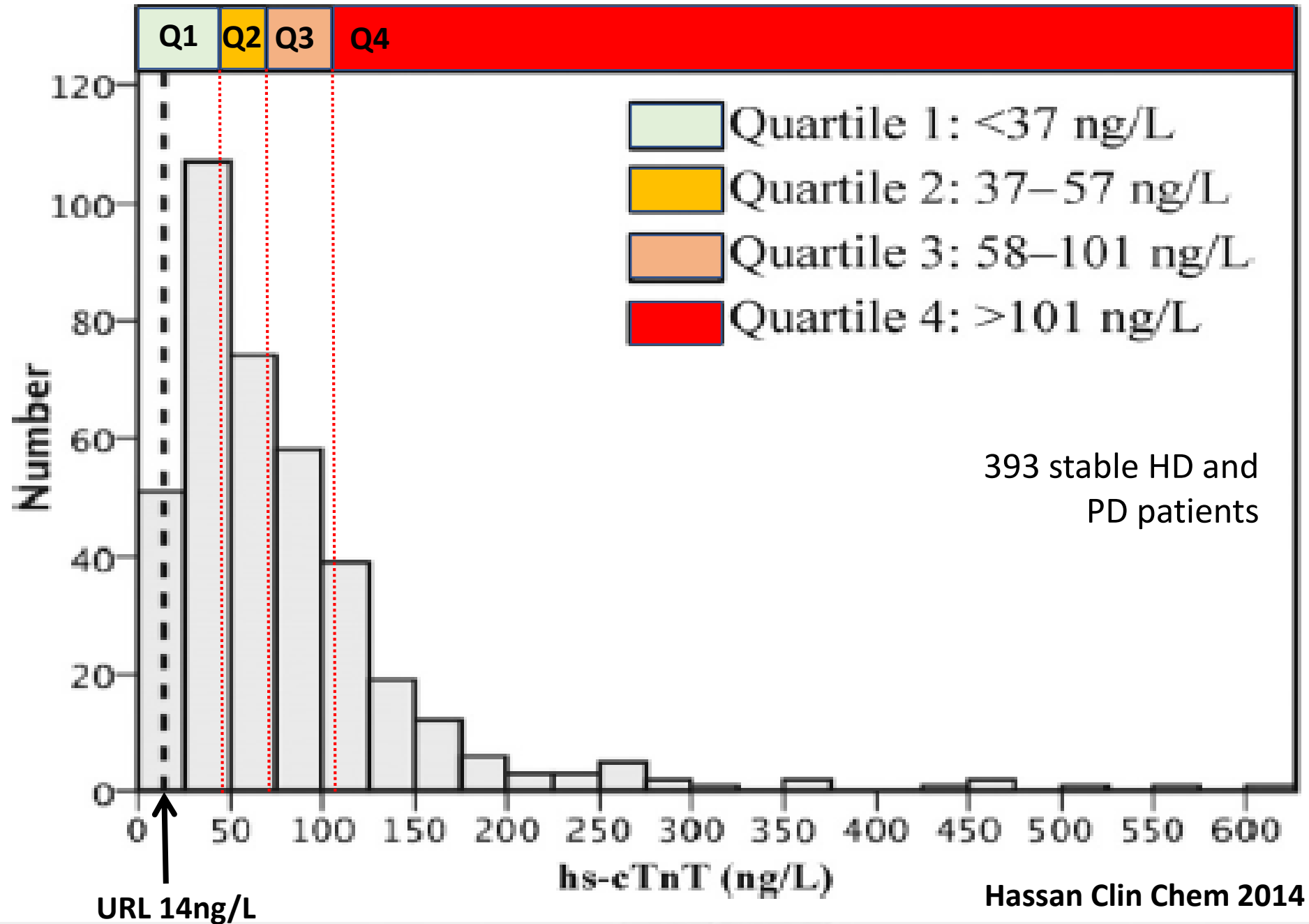




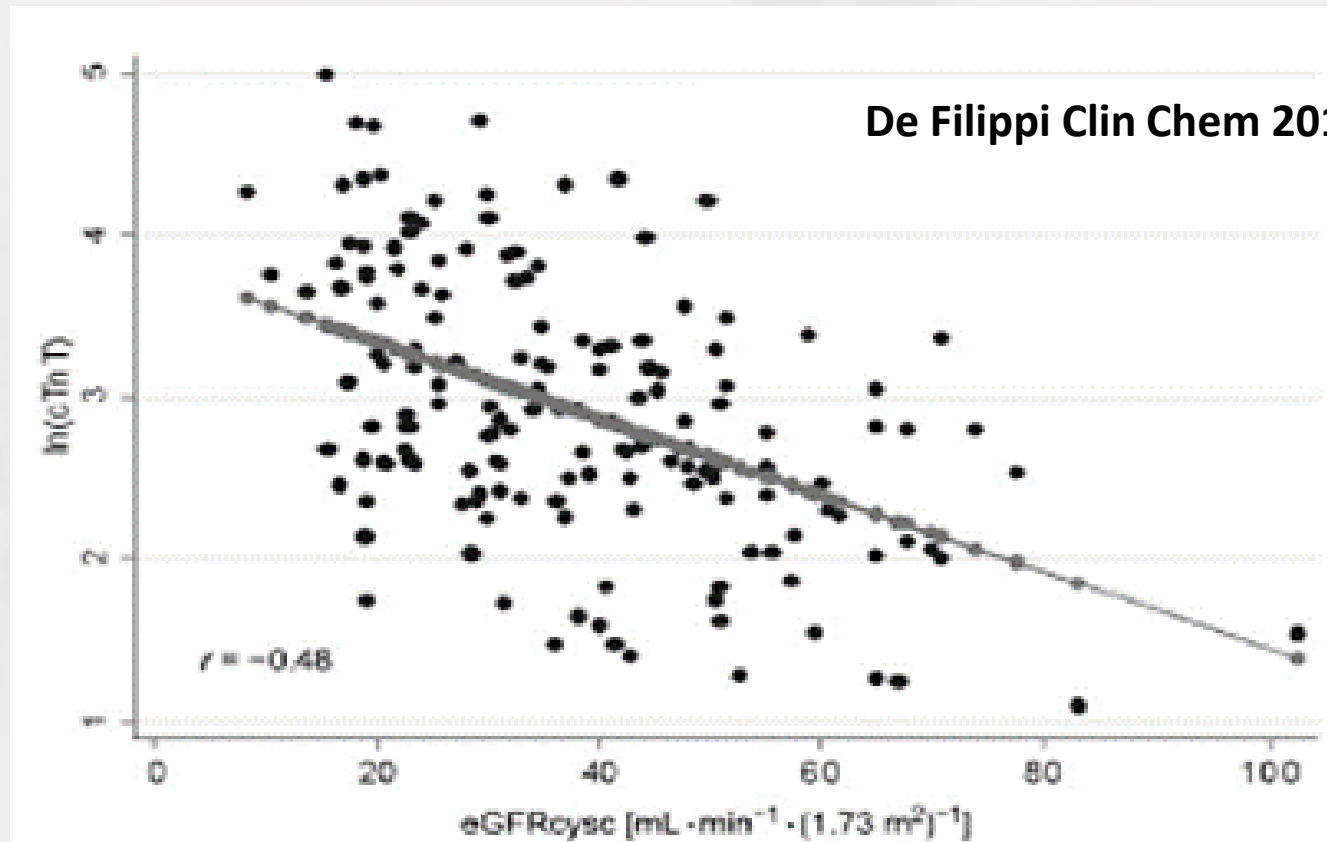
**Plot of sensitivity versus specificity for different stress tests compared with a gold standard of coronary angiography in potential kidney transplant recipients.**

Height and width of symbols are proportional to the inverse standard error of the sensitivity and specificity, respectively. Each symbol represents results of a single report.

- CAD : coronary artery disease;
- CIMT : carotid intimal medial thickness;
- DSE : dobutamine stress echocardiography;**
- DSF : digital subtraction fluorography;
- EBCT: electron beam computed tomography;
- ECG : resting electrocardiography;
- Echo (LV) : echocardiography (left ventricular dysfunction or cardiomegaly);
- Echo (MAC): echocardiography (mitral annular calcification);
- Echo (RWMA): echocardiography (resting wall motion abnormality);
- EST : exercise stress electrocardiography;
- EV : exercise ventriculography;
- MPS : myocardial perfusion scintigraphy**



## eGFR & cTnT



“... the diagnostic accuracy for presence of AMI of a baseline measurement of high-sensitive troponin in patients with renal insufficiency was poor and resembles tossing a coin.”

Pfortmueller Am J Cardiol 2013

## cTNT

Most patients who have had a heart attack have increased troponin levels within 6 h. After 12 h, almost everyone who has had a heart attack will have raised levels. Troponin levels may remain high for 1 to 2 weeks after a heart attack.

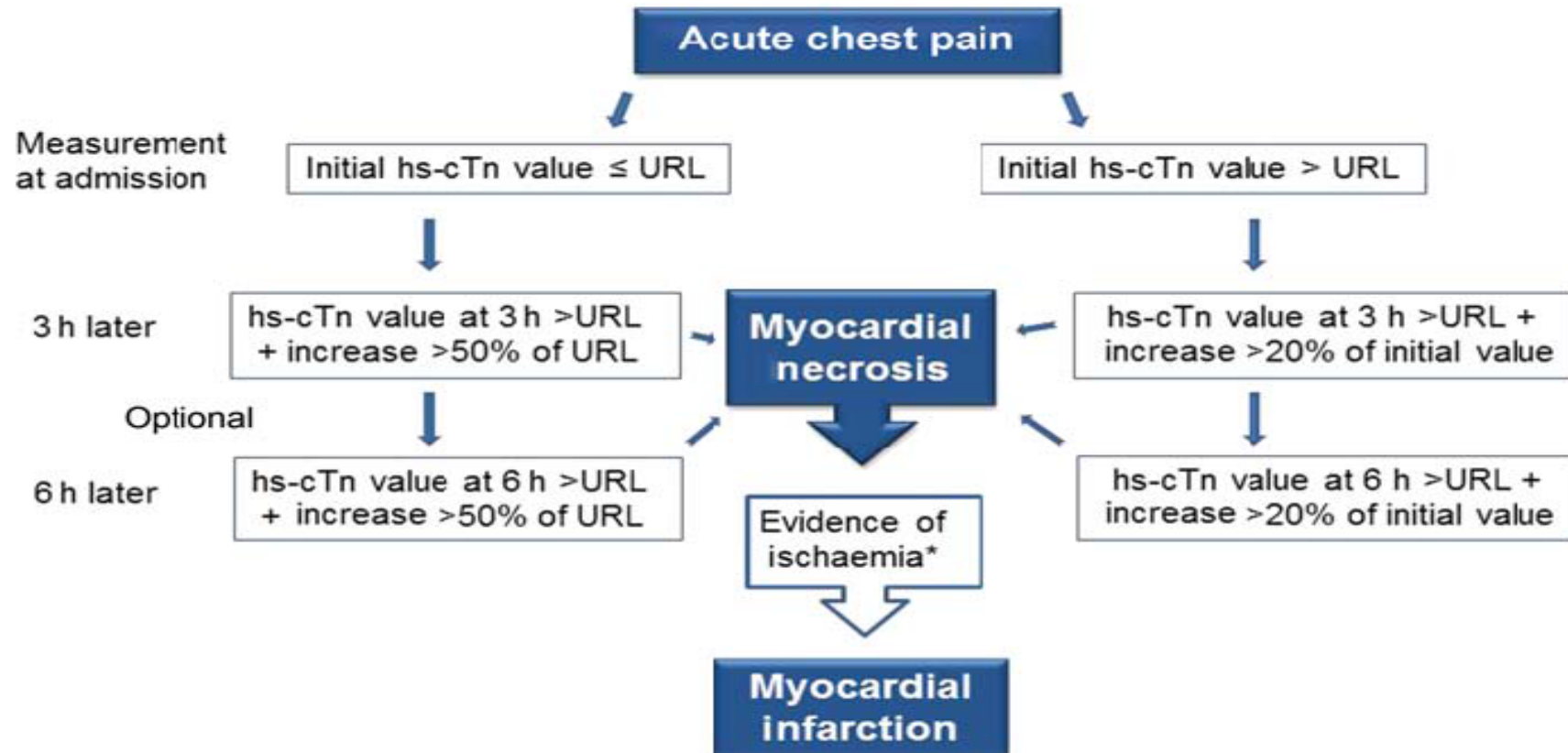
Increased troponin levels may also be due to

- Tachyarrhythmias
  - Heart failure
  - Hypertensive emergencies
  - Critical illness (e.g. shock/sepsis/burns)
  - Myocarditis
  - Takotsubo syndrome
  - Valvular heart disease (e.g. aortic stenosis)
  - Aortic dissection
  - Pulmonary embolism, pulmonary hypertension
  - Renal dysfunction and associated cardiac disease
  - Acute neurological event (e.g. stroke or subarachnoid haemorrhage)
  - Cardiac contusion or cardiac procedures (CABG, PCI, ablation, pacing, cardioversion, or endomyocardial biopsy)
  - Hypo- and hyperthyroidism
  - Infiltrative diseases (e.g. amyloidosis, haemochromatosis, sarcoidosis, scleroderma)
  - Myocardial drug toxicity or poisoning (e.g. doxorubicin, 5-fluorouracil, herceptin, snake venoms)
  - Extreme endurance efforts Rhabdomyolysis
- Troponin increases with CKD severity: normal Tn assay may be sufficient to rule out infarction, but elevated values are less definitive.
  - Upper Limit of Normal (ULN) for the general population (99th percentile) is not applicable in dialysis patients
  - Troponin predicts AMI & CV Mortality
  - Repeating measurements allows setting an upper limit value for each patient (baseline value)

There is minimal variability in  $_{\text{hsTnT}}$  of stable dialysis patients, so routine outpatient testing to establish a baseline “healthy” TnT value in stable CKD could improve diagnosis of ACS.

**When Acute Coronary Syndrome suspected (in NSTEMI),  
how to make decision on toponin check-up?**

**Rapid early rule-in of AMI with high-sensitivity cardiac troponin**



## 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

### MINOCA:

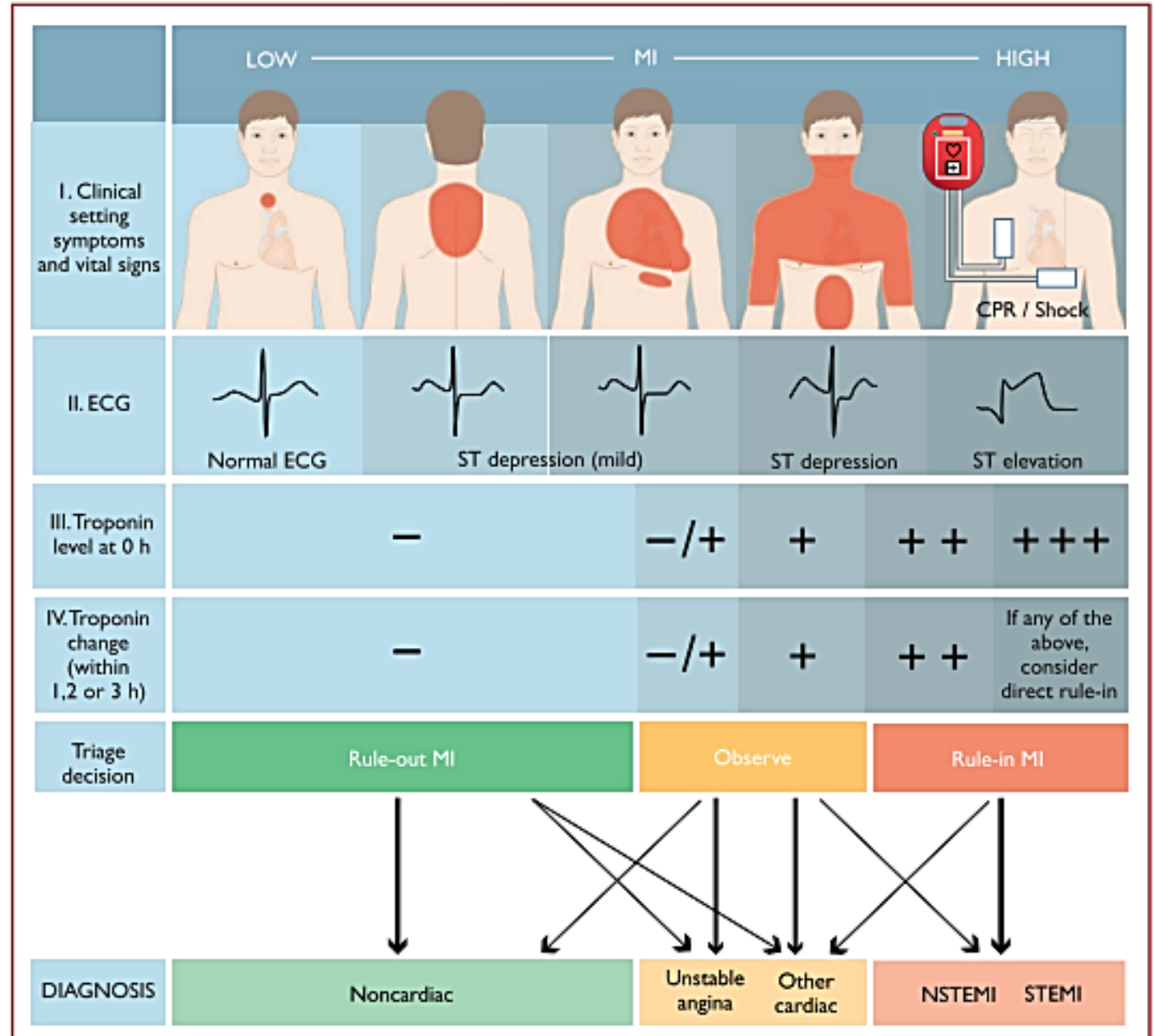
Myocardial infarction with non-obstructive coronary arteries

## 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation

## 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes

The Task Force for the diagnosis and management of chronic coronary syndromes of the European Society of Cardiology (ESC)

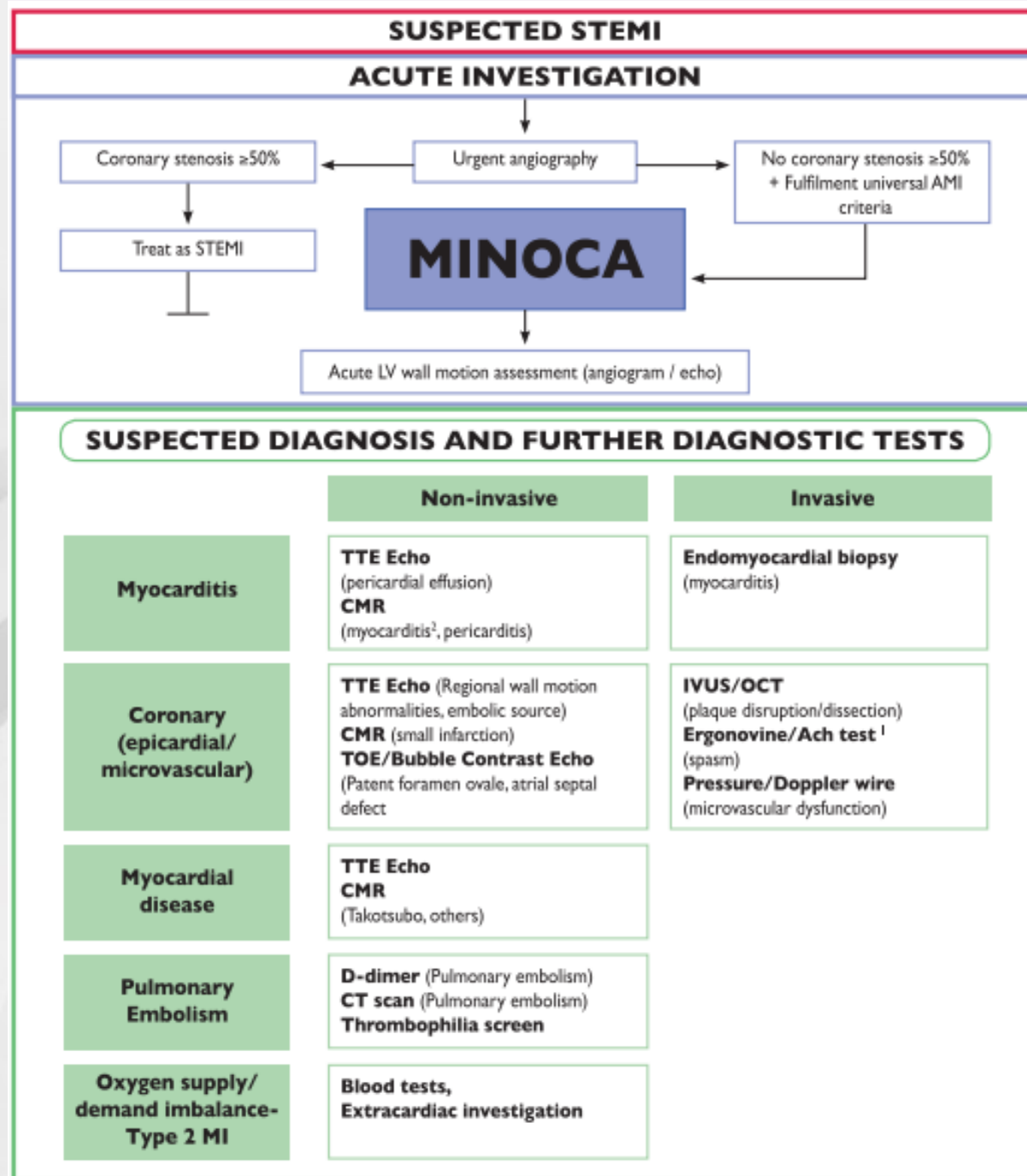
## Diagnostic algorithm and triage in acute coronary syndrome





## 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation

# MINOCA: Myocardial infarction with non-obstructive coronary arteries



## CAD in CKD patients:

- **Coronary lesions:** more numerous, longer, more calcified, pathological downstream bed
- **More difficult access** with stent
- **Associated co-morbidities:** diabetes, hypertension....

Classically:

- Less good initial results
- More hospital complications
- More restenosis

**= unfavorable prognosis**



## 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes

*“Data on patients on haemodialysis are very limited, making generalizable treatment recommendations difficult”.*

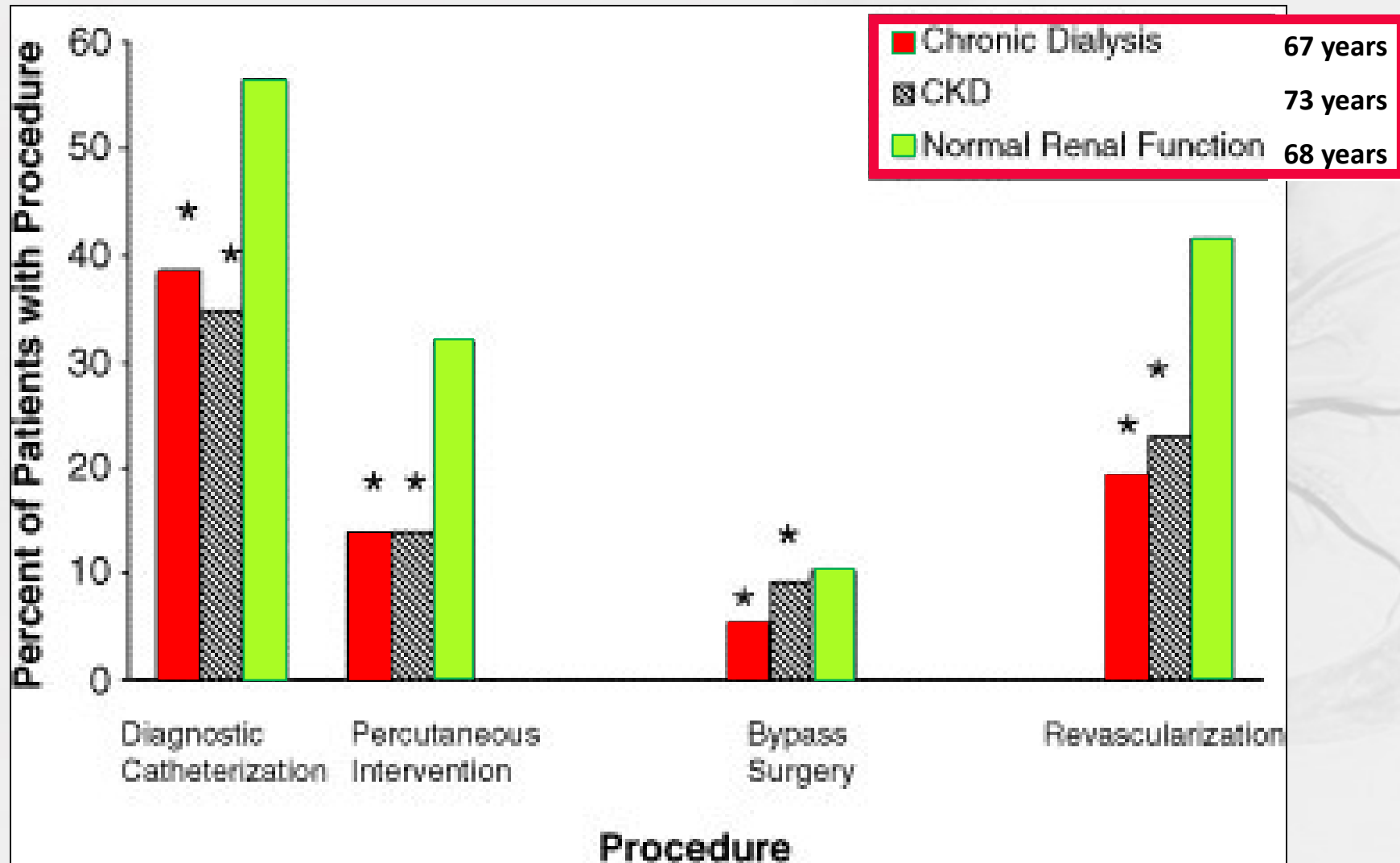
### Seminars in Dialysis

[Explore this journal >](#)

## Cardiovascular Disease and Dialysis Patients: Is Therapeutic Nihilism Justified?

Charles A. Herzog

# 154 692 patients admitted because of AMI in 2001



**CKD: Therapeutic Nihilism**

## 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation

**Table 7** Major and minor criteria for high bleeding risk according to the Academic Research Consortium for High Bleeding Risk at the time of percutaneous coronary intervention (bleeding risk is high if at least one major or two minor criteria are met)

Major	Minor
<ul style="list-style-type: none"><li>● Anticipated use of long-term OAC<sup>a</sup></li></ul>	<ul style="list-style-type: none"><li>● Age <math>\geq</math> 75 years</li></ul>
<ul style="list-style-type: none"><li>● Severe or end-stage CKD (eGFR <math>&lt;</math>30 mL/min)</li></ul>	<ul style="list-style-type: none"><li>● Moderate CKD (eGFR 30–59 mL/min)</li></ul>
<ul style="list-style-type: none"><li>● Haemoglobin <math>&lt;</math>11 g/dL</li></ul>	<ul style="list-style-type: none"><li>● Haemoglobin 11–12.9 g/dL for men or 11–11.9 g/dL for women</li></ul>
<ul style="list-style-type: none"><li>● Spontaneous bleeding requiring hospitalization and/or transfusion in the past 6 months or at any time, if recurrent</li></ul>	<ul style="list-style-type: none"><li>● Spontaneous bleeding requiring hospitalization and/or transfusion within the past 12 months not meeting the major criterion</li></ul>
<ul style="list-style-type: none"><li>● Moderate or severe baseline thrombocytopenia<sup>b</sup> (platelet count <math>&lt;</math>100 <math>\times</math> 10<sup>9</sup>/L)</li></ul>	<ul style="list-style-type: none"><li>● Chronic use of oral non-steroidal anti-inflammatory drugs or steroids</li></ul>
<ul style="list-style-type: none"><li>● Chronic bleeding diathesis</li></ul>	<ul style="list-style-type: none"><li>● Any ischaemic stroke at any time not meeting the major criterion</li></ul>
<ul style="list-style-type: none"><li>● Liver cirrhosis with portal hypertension</li></ul>	
<ul style="list-style-type: none"><li>● Active malignancy<sup>c</sup> (excluding non-melanoma skin cancer) within the past 12 months</li></ul>	
<ul style="list-style-type: none"><li>● Previous spontaneous intracranial haemorrhage (at any time)</li><li>● Previous traumatic intracranial haemorrhage within the past 12 months</li><li>● Presence of a brain arteriovenous malformation</li><li>● Moderate or severe ischaemic stroke<sup>d</sup> within the past 6 months</li></ul>	
<ul style="list-style-type: none"><li>● Recent major surgery or major trauma within 30 days prior to PCI</li><li>● Non-deferrable major surgery on DAPT</li></ul>	

CKD = chronic kidney disease; DAPT = dual antiplatelet therapy; eGFR = estimated glomerular filtration rate; OAC = oral anticoagulation/anticoagulant; PCI = percutaneous coronary intervention.

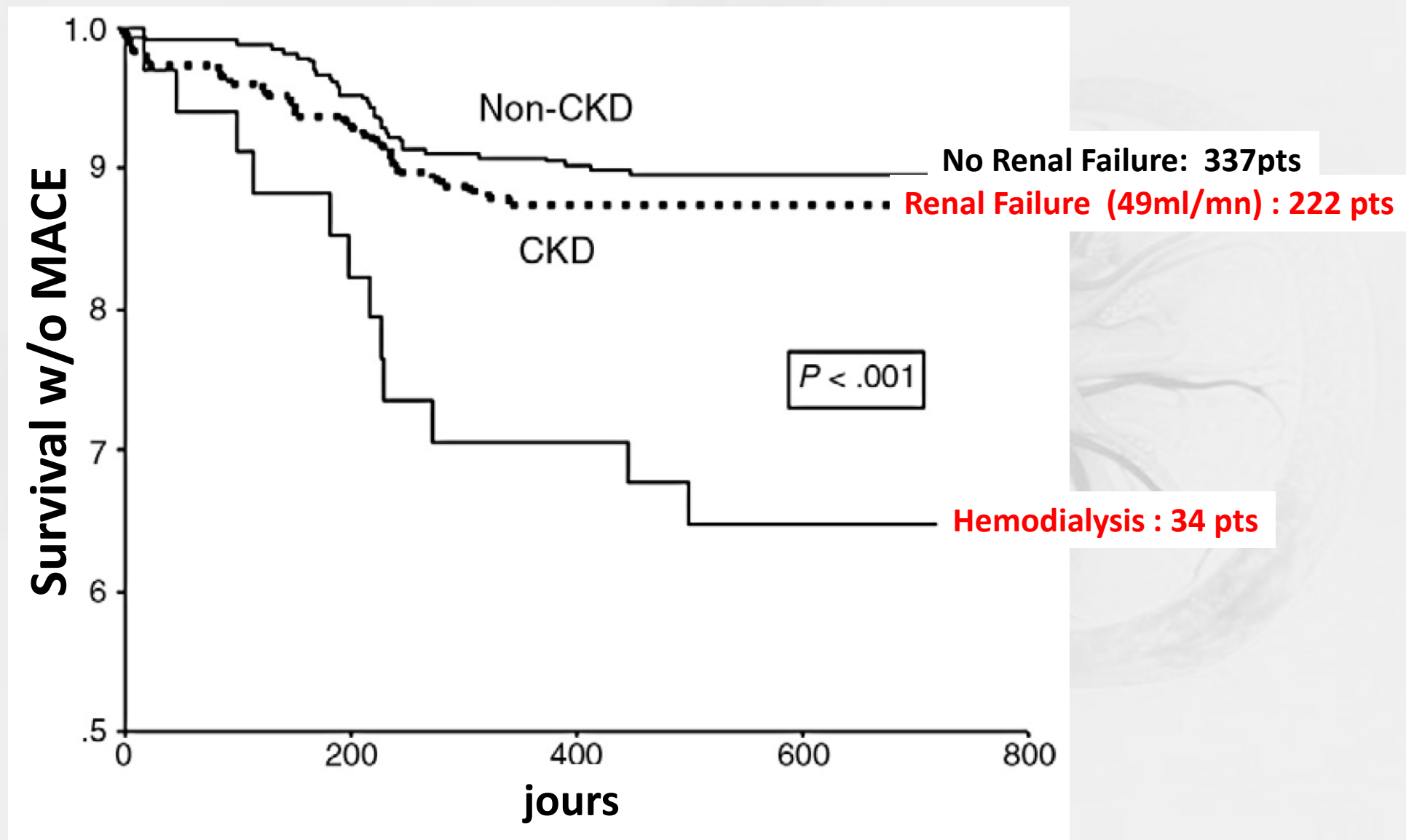
<sup>a</sup>This excludes vascular protection doses.<sup>162</sup>

<sup>b</sup>Baseline thrombocytopenia is defined as thrombocytopenia before PCI.

<sup>c</sup>Active malignancy is defined as diagnosis within 12 months and/or ongoing requirement for treatment (including surgery, chemotherapy, or radiotherapy).

<sup>d</sup>National Institutes of Health Stroke Scale score  $>$ 5.

# Registry: SES à 2 ans





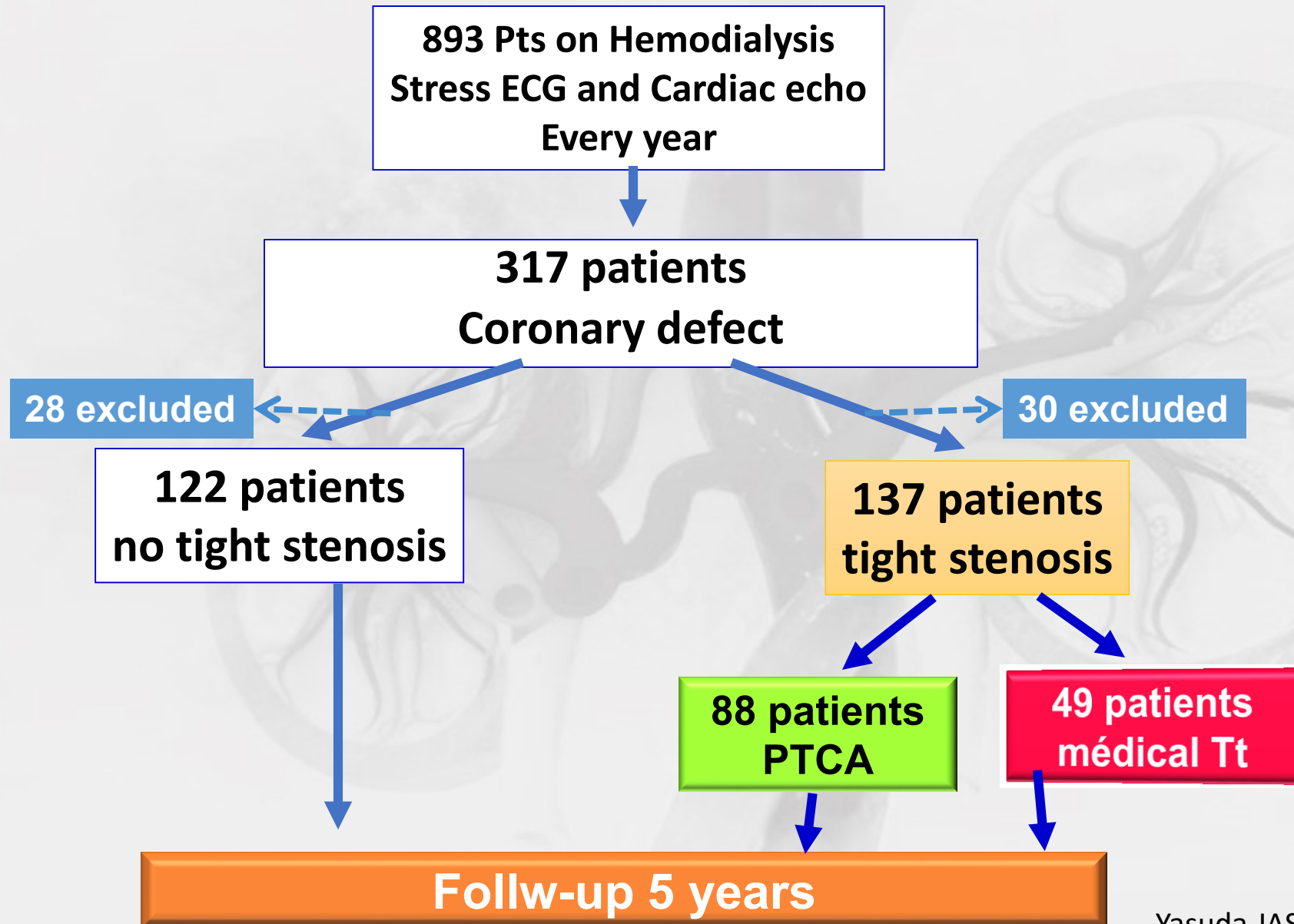


**Revascularization + Medical treatment  
Vs  
Medical treatment alone**

Revascularization options in patients with CKD include CABG and PCI.

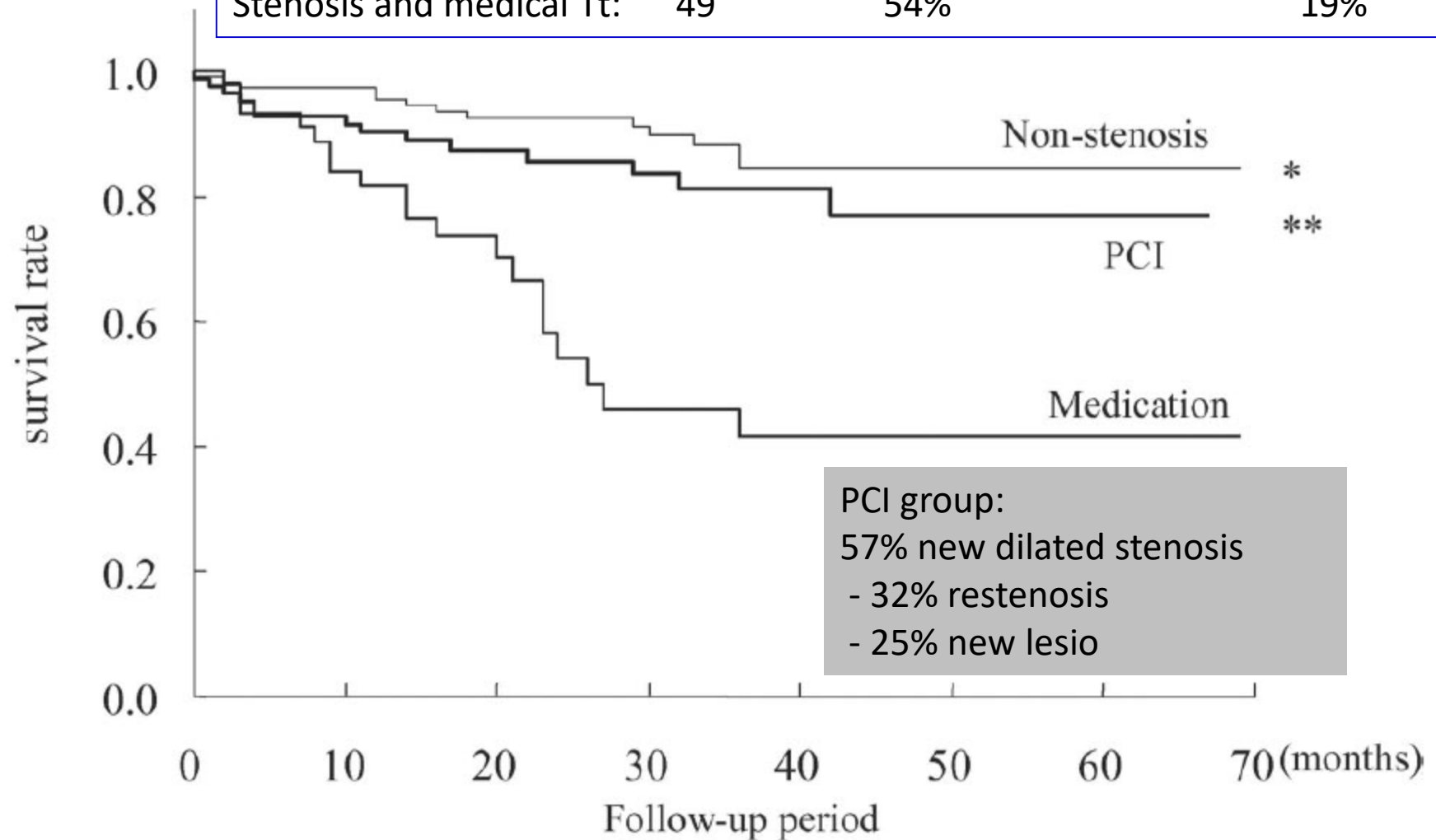
Meta-analyses suggest that CABG is associated with higher short-term risk of death, stroke, and repeat revascularization, whereas PCI with a new-generation DES is associated with a higher long-term risk of repeat revascularization.

# Patients on dialysis: PCI vs medical treatment: Prospective study



# Dialysis patients: survival rate w/o CV event

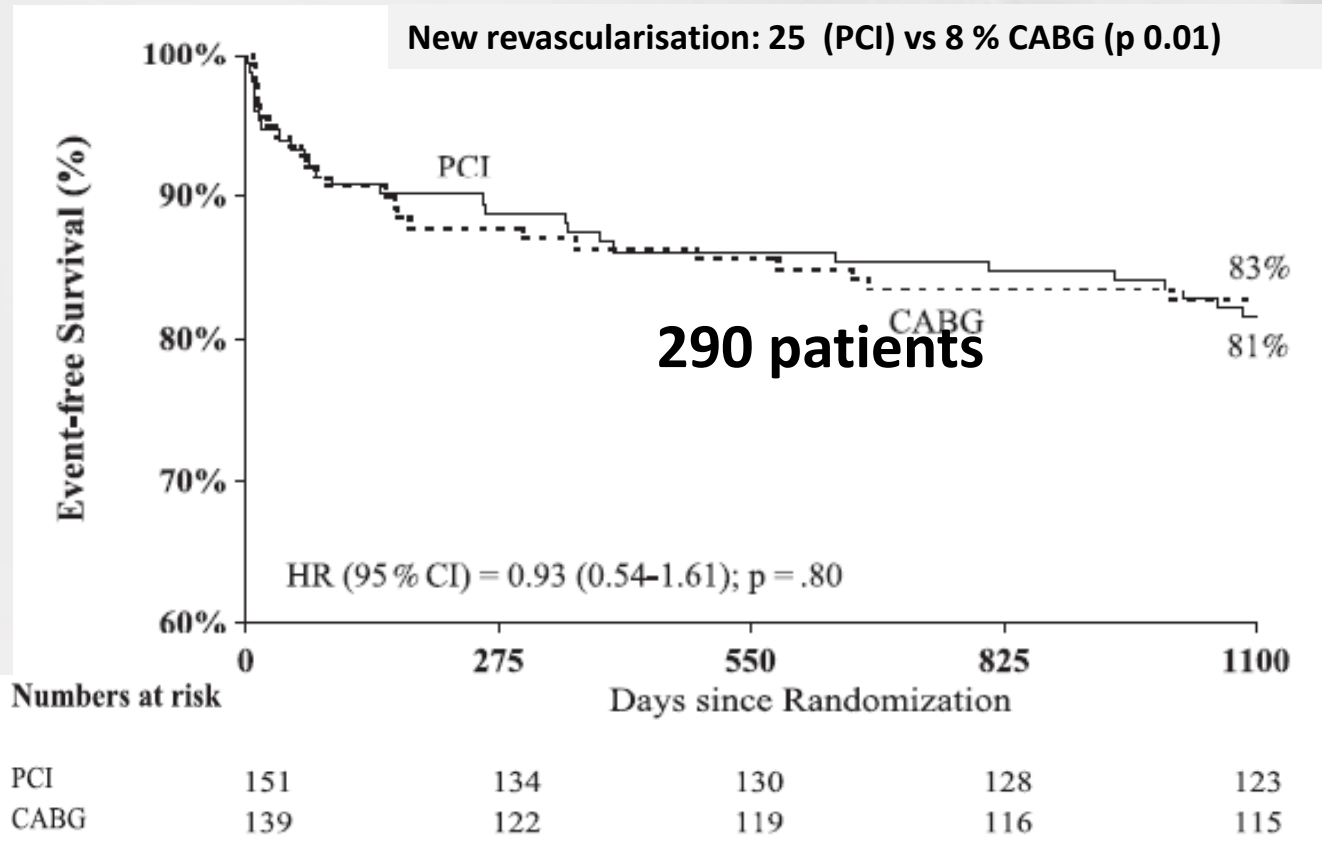
Groups	Nb Pts	PluriTronculars	Instable Angina
Non stenosis:	122	-	13%
Stenosis and PCI:	88	73%	42%
Stenosis and medical Tt:	49	54%	19%



PCI group:  
 57% new dilated stenosis  
 - 32% restenosis  
 - 25% new lesio

**ARTS trial = PRCT**  
**Angioplasty (PCI + non covered stent) vs Bypass graft (CABG)**

**1205 patients with multitruncular lesions**  
**Renal failure(mean 49 ml/mn)**

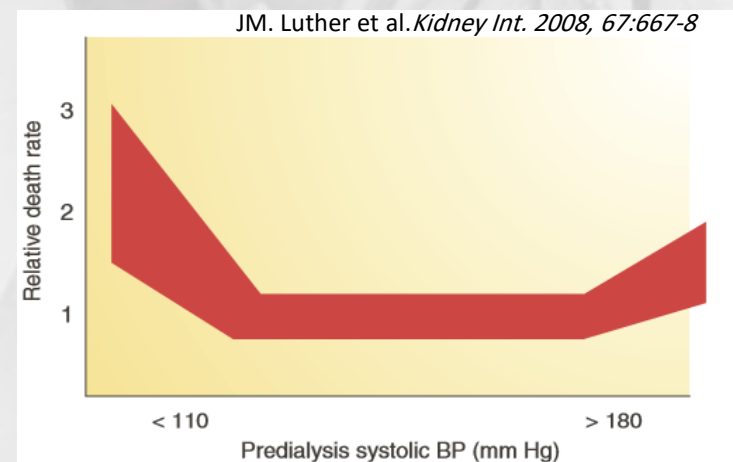
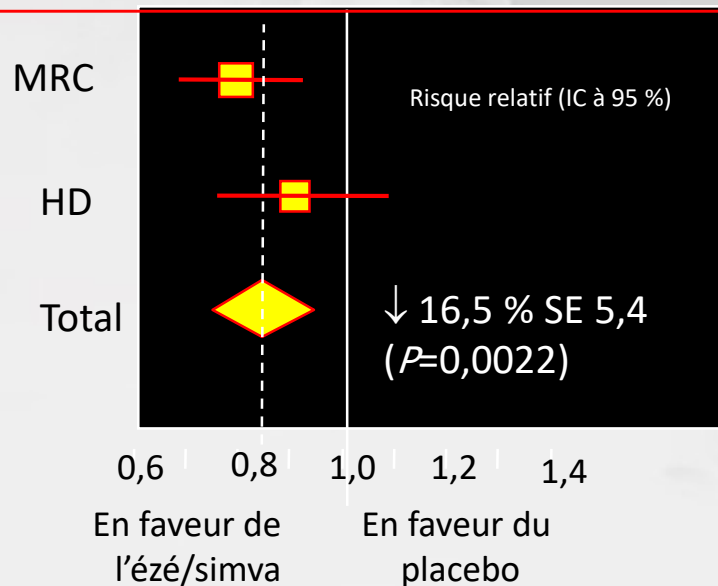
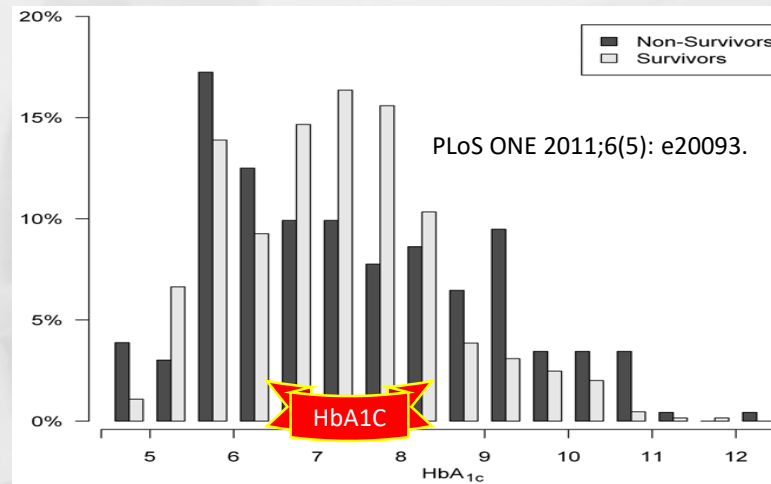


**Comparison of coronary artery bypass grafting and drug-eluting stents in patients with chronic kidney disease and multivessel disease: A meta-analysis**

11 studies  
 29,246 patients  
 17,928 DES patients + 11,318 CABG

DES had higher long-term all-cause mortality. OR, 1.22; p < 0.00001, cardiac mortality (OR, 1.29; p < 0.00001), myocardial infarction (OR, 1.89; p = 0.02), repeat revascularization (OR, 3.47; p < 0.00001) major adverse C/CV events (MACCE) (OR, 2.00; p = 0.002), but lower short-term all-cause mortality (OR, 0.33; p < 0.00001) cerebrovascular accident (OR, 0.64; p = 0.0001).

## CV Prevention in CKD patients: Early detection and aggressive treatment of all CV Risk Factors



# Major randomized controlled trials of cardiovascular medications in hemodialysis patients

First author, year (ref #)	Inclusion criteria	Intervention	Duration, years	Composite fatal and non-fatal cardiovascular events			All-cause mortality (%)			Cardiovascular mortality (%)		
				Intervention	Placebo	HR (95%CI)	Intervention	Placebo	HR (95%CI)	Intervention	Placebo	HR (95%CI)
FOSIDIAL, 2006 <sup>(95)</sup> .	HD, LVH (n=397)	Fosinopril titrated up to 20 mg/day	2	NA	NA	0.80 (0.59–1.1)	-	-	-	-	-	-
Takahashi et al., 2006 <sup>(96)</sup> .	HD (n=80)	Candesartan 4–8 mg/day	19.4 mo	16.3	45.9	0.29 (0.12–0.70)	-	18.9	NA	-	-	-
Suzuki et al., 2008 <sup>(97)</sup> .	HD, SBP >160 mmHg or >150 mmHg if receiving anti-HT drugs (n=366)	Losartan up to 100 mg, or candesartan up to 12 mg/day or valsartan up to 160 mg/day	3	19	33	0.51 (0.33–0.79)	14	21	0.64 (0.39–1.06)	-	-	-
OCTOPUS, 2013 <sup>(98)</sup> .	HD, BP≥140/90 mmHg (n=469)	Olmesartan 10–40 mg/day until achieved target BP of <140/90 mmHg	3.5	35.3	34	1.00 (0.71–1.40)	24	22.2	0.97 (0.62–1.52)	-	-	-
DOHAS, 2014 <sup>(101)</sup> .	HD, serum K <6.5 mEq/L (n=309)	Spirololactone 25 mg/day	3	5.7	15.1	0.40 (0.20–0.81)	6.4	19.7	0.36 (0.19–0.66)	2.5	4.6	0.57 (.18–1.87)
4D, 2005 <sup>(6)</sup> .	HD, type 2 DM, LDL 90–180 mg/dL (n=1255)	Atorvastatin 20 mg/day	4	37	38	0.92 (0.77–1.10)	48	50	0.93 (0.79–1.08)	20	23	0.81 (0.64–1.03)
AURORA, 2009 <sup>(26)</sup> .	HD (n=2776)	Rosuvastatin 10 mg/day	3.8	28.5	29.5	0.96 (0.84–1.11)	45.8	47.7	0.96 (0.86–1.07)	23.3	23.4	1.00 (0.85–1.16)
SHARP, 2011 <sup>(8)</sup> .	CKD Cr≥1.7 mg/dL in men or 1.5 mg/dL in women (n = 9270)	Simvastatin 20 mg/day plus ezetimibe 10 mg/day	4.9	11.3	13.4	0.83 (0.74–0.94)	24.6	24.1	1.02 (0.94–1.1)	5.4	5.9	0.93 (0.78–1.10)
	Dialysis subgroup (n=3023; HD=2527, PD=496)	Simvastatin 20 mg/day plus ezetimibe 10 mg/day	4.9	15	16.5	0.90 (0.75–1.08)	-	-	-	-	-	-

First author, year (ref #)	Inclusion criteria	Intervention	Duration, years	Concomitant drugs (%)			All-cause mortality (%)			Cardiovascular mortality (%)			HF hospitalization (%)		
				ACEI	ARB	BB	Intervention	Placebo	HR (95% CI)	Intervention	Placebo	HR (95% CI)	Intervention	Placebo	HR (95% CI)
Cice et al., 2003 <sup>(105)</sup> .	HD, NYHA II-III HF, LVEF<3 5% (n=114)	Carvedilol titrated up to 25 mg twice a day	2	98	2	-	51.7	73.2	0.51 (0.32–0.82)	29.3	67.9	0.32 (0.18–0.57)	13.8	57.1	0.19 (0.09–0.41)
Cice et al., 2010 <sup>(99)</sup> .	HD, NYHA II-III HF, LVEF≤4 0%, on ACEI (n=332)	Telmisartan titrated up to 80 mg/day	3	100	-	60	31.5	54.4	0.51 (0.32–0.82)	30.3	43.7	0.32 (0.18–0.57)	33.9	55.1	0.38 (0.19–0.51)



# Coronary Artery Disease in Dialysis Patients

## Take Home Messages

- There is a linear increase in the risk of cardiovascular mortality with decreasing GFR
- RR 36.6 increase in dialysis patients for total CV Events.
- CAD is highly prevalent in patients with Dialysis, but not the major CVD
- Medical treatment for risk-factor control during renal function failing (lipids, BP, and glucose) can improve outcomes.
- Special attention during the workup for dialysis patients with suspected obstructive CAD should be paid to the fact that angina is less common and silent ischemia more common (Dyspnea, arrhythmia, unusual fatigue)
- Level of hs-TroponinT is increased in dialysis patient: Basal value should be known, then controlled if any CAS suspicion
- Additionally, non-invasive stress testing shows reduced accuracy in patients with CKD.
- NSTEMI is more frequent than STEMI, diagnosis is mostly made at AMI
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- Dialysis patients should have vascular doppler control and a cardiologist check up (echo) almost yearly
- Functional stress testing & noninvasive coronary imaging
  - Symptomatic patient
  - Asymptomatic potential transplant recipient,
- Interestingly, patients with CKD are less likely to receive invasive management for treatment of CAD compared with those without, although benefits of invasive management have been reported.
- Revascularization options in patients with CKD include CABG and PCI.
- Meta-analyses suggest that CABG is associated with higher short-term risk of death, stroke, and repeat revascularization, whereas PCI with a new-generation DES is associated with a higher long-term risk of repeat revascularization



Thank you  
Merci  
شكرا لكم

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