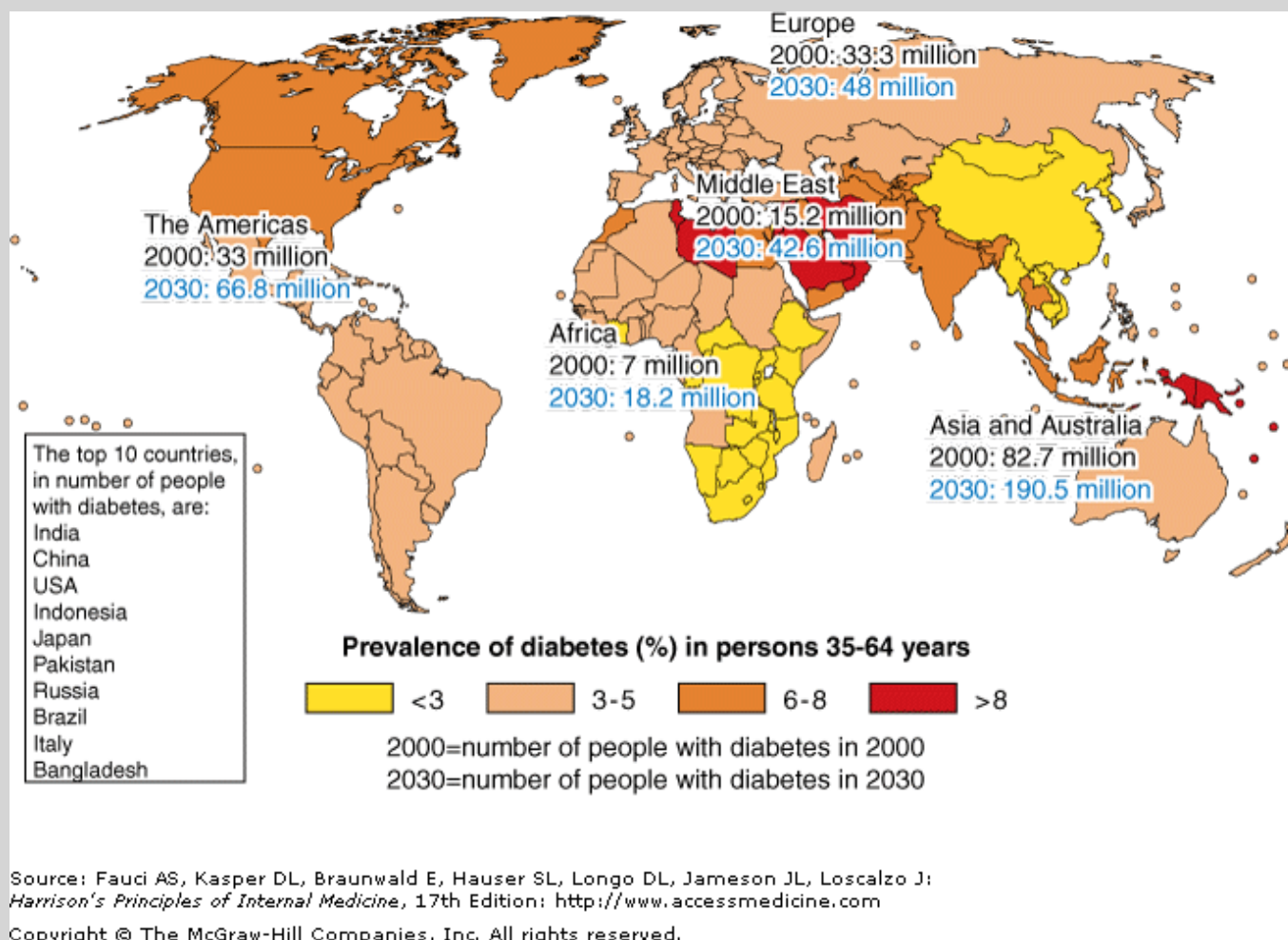


Đái tháo đường và bệnh tim mạch: Biện pháp giảm biến cố tim mạch (2018)

PGS. TS. BS Phạm Nguyễn Vinh
BV Tim Tâm Đức
Viện Tim TP. HCM
ĐH Y khoa Phạm Ngọc Thạch

Tần suất bệnh đái tháo đường/ thế giới năm 2000 và 2030



Tiêu chuẩn chẩn đoán bệnh đái tháo đường

1. HbA1c $\geq 6,5\%$ - Phòng xét nghiệm chuẩn (NGSP certified/chuẩn hoá theo DCCT assay)
2. Đường máu đói ≥ 126 mg/dL (7.0 mmol/L)- Nhịn đói 8 giờ
3. Trắc nghiệm dung nạp đường : ≥ 200 mg/dL (11.1 mmol/L) 2 giờ sau uống đường (75g glucose) quy trình WHO
4. Đường máu bất kỳ ≥ 200 mg/dL kèm triệu chứng ĐTĐ

Đái tháo đường týp 2: giết người thầm lặng

- Khoảng 30% bệnh nhân ĐTĐ không được chẩn đoán
- Khoảng 50% bệnh nhân được chẩn đoán ĐTĐ đã có biến chứng

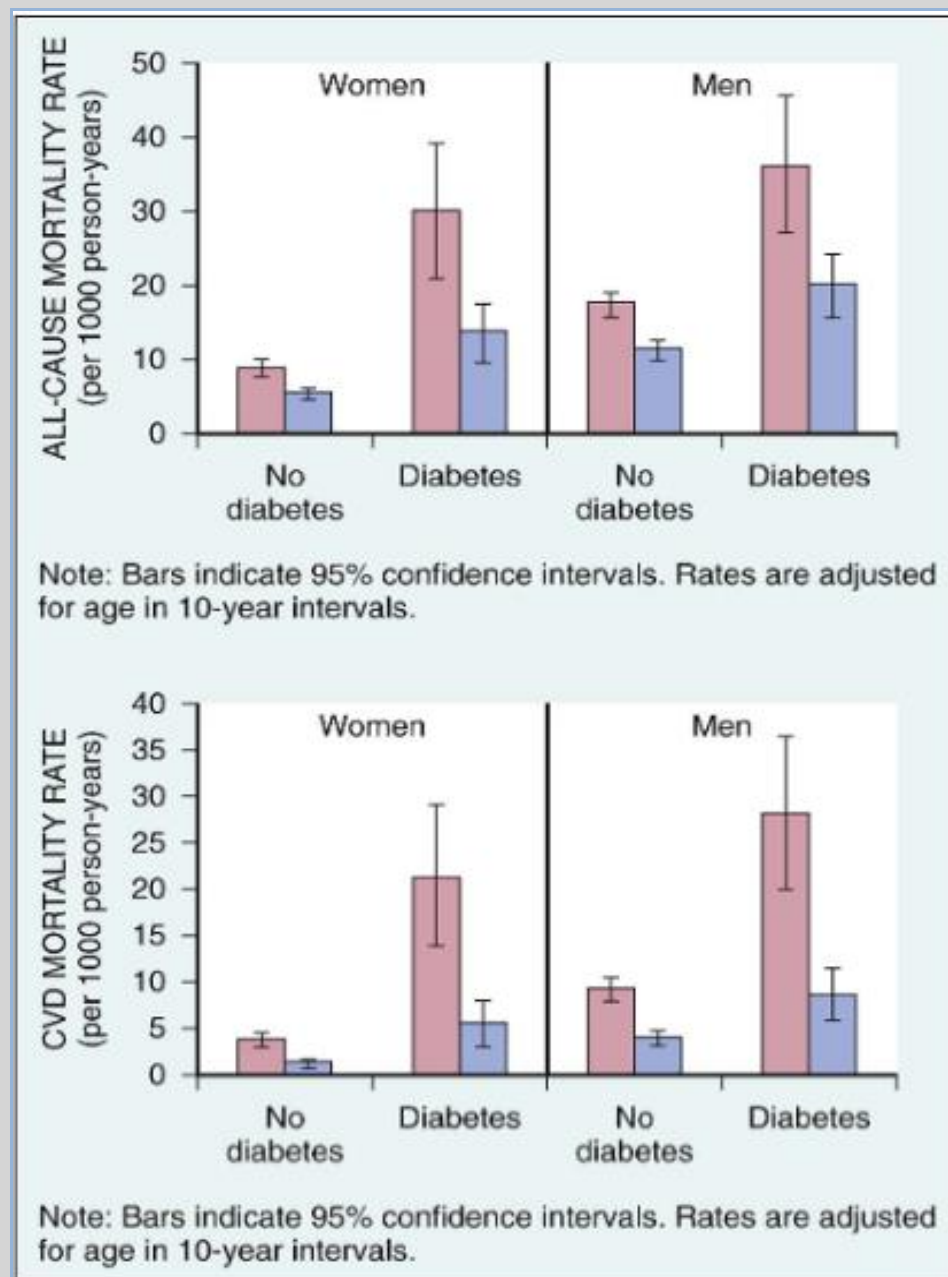
Tầm quan trọng của bệnh tim mạch trên B/n ĐTĐ

- ❑ Hầu hết b/n ĐTĐ tử vong vì bệnh tim mạch; 80% do xơ vữa động mạch
- ❑ 75% tử vong tim mạch/ ĐTĐ do bệnh ĐMV, 25% do bệnh mạch máu não hay mạch ngoại vi

Tử vong mọi nguyên nhân và tử vong tim mạch/ b/n ĐTĐ

- Cột hồng (bên trái): thời gian từ 1950-1975
- cột xanh (bên phải): thời gian từ 1976-2001

TL: Preis SR et al. Circulation 119: 1728, 2009



Các biến chứng mạch máu của Đái tháo đường (ĐTĐ)

- Biến chứng vi mạch:
 - Bệnh võng mạc
 - Bệnh thận
 - Bệnh thần kinh (neuropathy)
- Biến chứng mạch máu lớn:
 - Bệnh ĐMV
 - Bệnh mạch máu não
 - Bệnh mạch máu ngoại vi

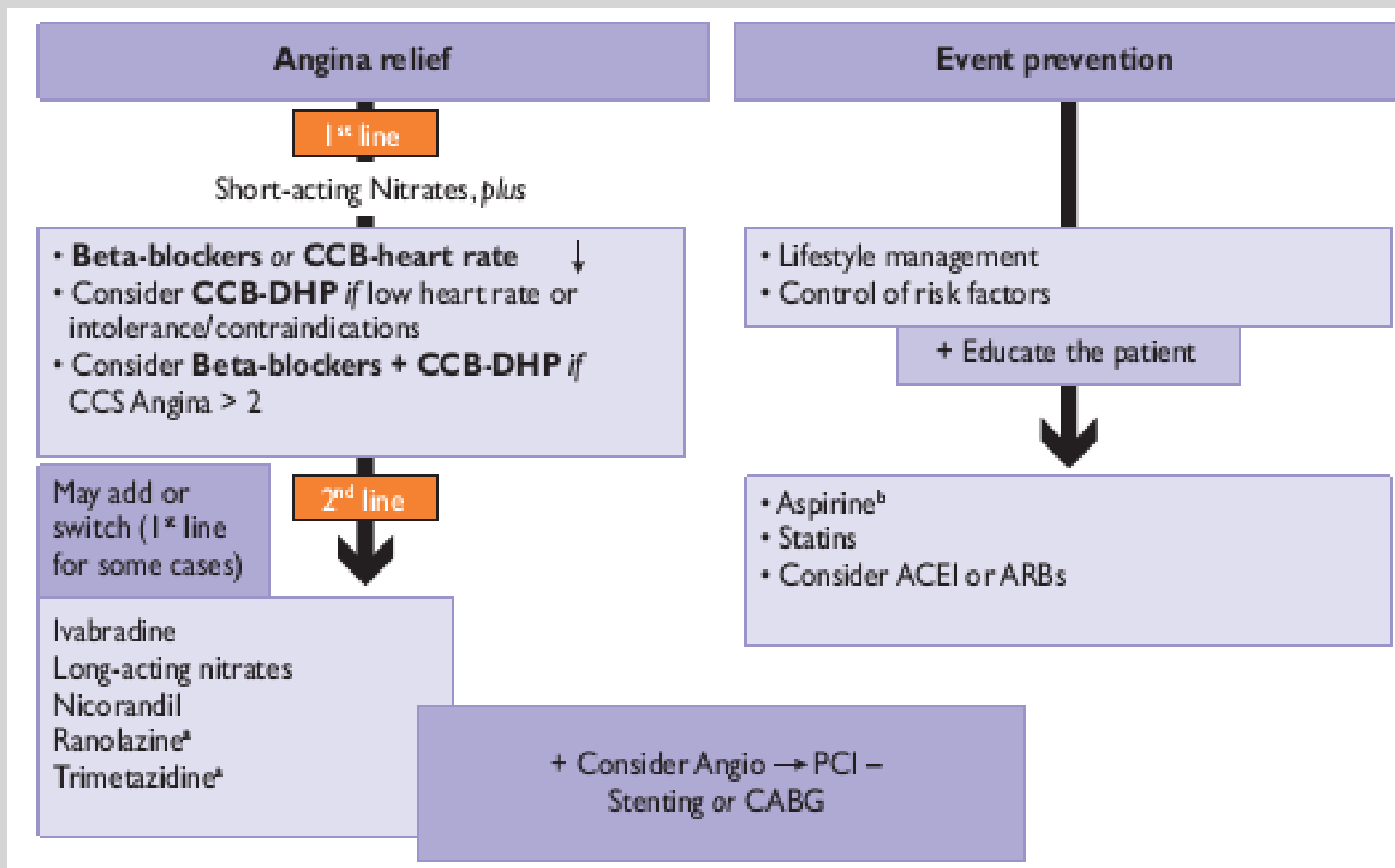
Hai vấn đề lớn về tim mạch/ ĐTĐ

- Bệnh động mạch vành/ bệnh nhân ĐTĐ
- Suy tim trên b/n ĐTĐ

Các mục tiêu điều trị BĐMV/b/n ĐTĐ

- Xử trí các yếu tố nguy cơ BĐMV: THA, rối loạn lipid máu, ngưng thuốc lá, thay đổi lối sống
- Điều trị các biểu hiện BĐMV: tái lưu thông ĐMV/ hội chứng ĐMV cấp hoặc bệnh động mạch vành ổn định; suy tim do BĐMV

Điều trị nội bệnh nhân BĐMV ổn định



Đái tháo đường: gia tăng nguy cơ suy tim

- ĐTĐ: yếu tố nguy cơ độc lập tăng suy tim 2-5 lần*
- ĐTĐ: tăng tử vong 60%/ suy tim so với không ĐTĐ*

*TL: Mc Guire D. K. Braunwald's Heart Disease, 10th ed. 2015, Elsevier Saunders, p 1392-1407

Sinh lý bệnh về rối loạn chức năng tim, suy tim và tiên lượng xấu/ ĐTĐ

Sympathetic nervous system activation

Renin-angiotensin system activation

Increased sodium and free water retention

Decreased vascular compliance

Elevated endothelin levels (in diabetes)

Loss of “dipping” nocturnal blood pressure pattern

Increased free fatty acid levels

Dysregulated myocardial glucose and fatty acid metabolism

Increased left ventricular hypertrophy or mass via myocyte hypertrophy

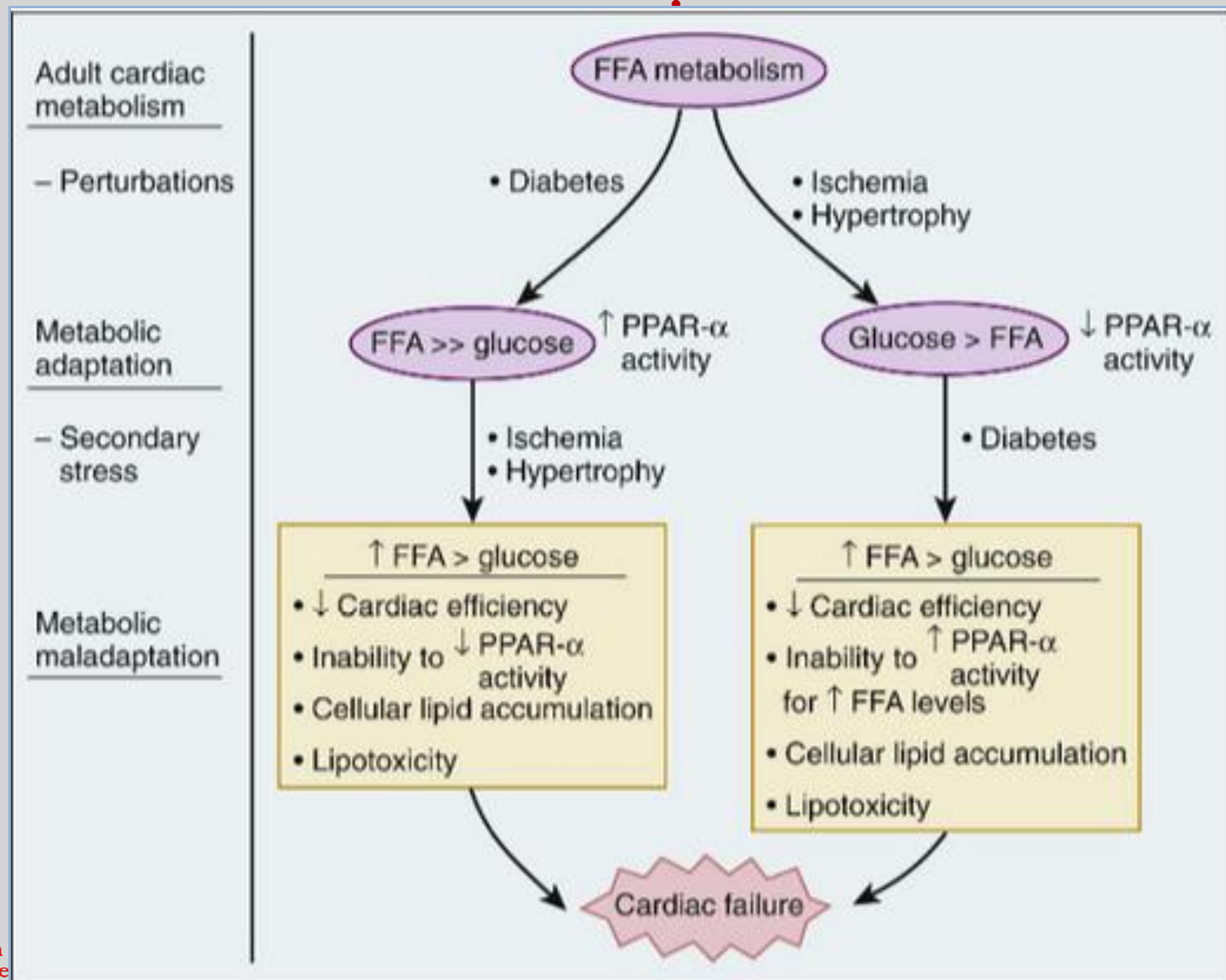
Deposition of advanced glycation end products in extracellular matrix

Increased cardiac fibrosis

Increased cardiac steatosis



Sơ đồ tóm tắt cơ chế bệnh cơ tim do ĐTĐ



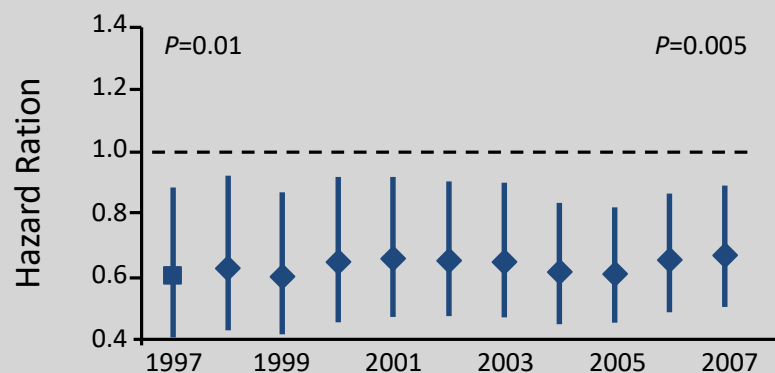
Phòng ngừa suy tim trên bệnh nhân ĐTĐ

- Điều trị hiệu quả tăng huyết áp ($< 130/80\text{mmHg}$ hoặc $< 140/90\text{ mmHg}$): n/c UKPDS
- Kiểm soát chặt đường huyết: n/c UKPDS, n/c Iribarren & c/s ($\text{HbA}_{1\text{C}} < 7\%$)
- Các thuốc có hiệu quả phòng ngừa hay điều trị suy tim do ĐTĐ:
 - UCMC, chẹn thụ thể AGII, eplerenone, spironolactone
 - Chẹn beta

- Thuốc điều trị ĐTĐ có thể tăng nguy cơ tim mạch?
- Kiểm soát chặt đường máu có giảm nguy cơ tim mạch?

Lợi điểm của kiểm soát chặt và sớm đường máu: kết quả theo dõi thêm 10 năm của nghiên cứu UKPDS

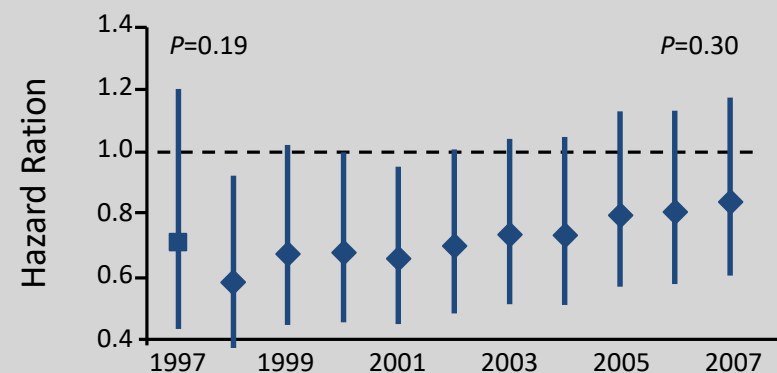
Myocardial Infarction



No. of Events

Conventional therapy	73	83	92	106	118	126
Metformin	39	45	55	64	68	81

Microvascular Disease



UKPDS, UK Prospective Diabetes Study



Pham
Nguyen
Vinh

Holman R, et al. *NEJM*. 2008;359:1577-1589.

Hiệu quả kiểm soát chặt đường huyết bằng Metformin trên bệnh nhân ĐTĐ dư cân: n/c UKPDS 34

Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes

	Metformin (n=342)	Insulin/Sulfonylurea (n=951)	Conventional (n=411)
Absolute risk (events per 1,000 patient-years)			
Myocardial infarction	11.0*	14.4	18.0
Stroke	3.3	6.2[†]	5.5
All-cause mortality	13.5*[†]	18.9	20.6

*p<0.05 vs conventional; *[†] p<0.05 vs insulin/SU

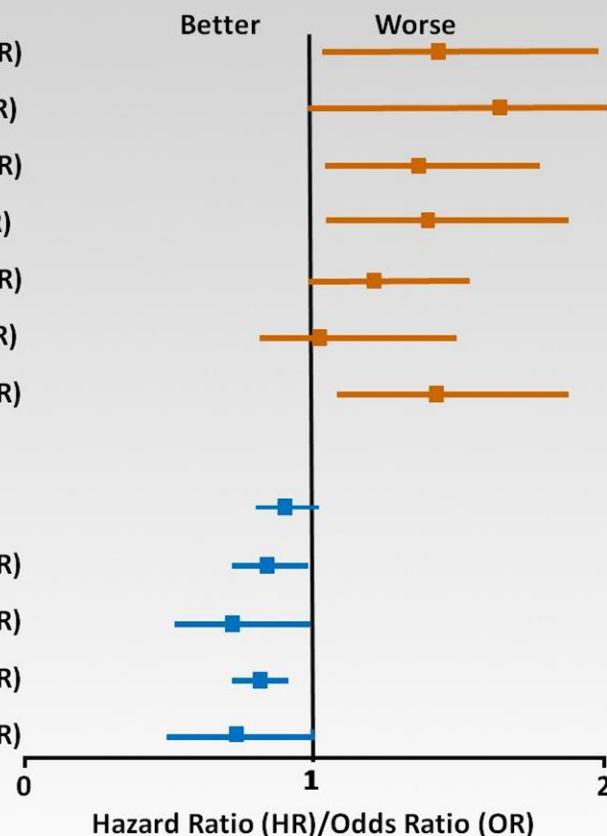
Thiazolinedione và các biến cố tim mạch: Rosiglitazone so với Pioglitazone

Rosiglitazone

Meta-analysis, Nissen et al ^a	Myocardial infarction (OR)
Meta-analysis, Nissen et al ^a	Cardiovascular death (OR)
Meta-analysis, Krall ^b	Myocardial infarction (OR)
Meta-analysis of 42 trials, FDA ^c	Myocardial ischemia (OR)
Data from Nissen and RECORD ^d	Myocardial infarction (OR)
Data from Nissen and RECORD ^d	Cardiovascular death (OR)
Meta-analysis, Singh ^e	Myocardial infarction (HR)

Pioglitazone

PROactive ^f	Primary end point (HR)
PROactive ^g	MI, stroke, and death (HR)
PROactive MI subgroup ^h	Myocardial infarction (HR)
Meta-analysis, Lincoff et al ⁱ	MI, stroke, and death (HR)
Meta-analysis, FDA 2007 ^c	MI, stroke, and death (HR)



1. Nissen SE, et al. NEJM 2007;356:2457-2471. 2. Krall RL, et al. Lancet 2007;369:1995-1996. 3. Callaghan F. FDA <http://www.fda.gov/downloads/AdvisoryCommittees/CommitteesMeetingMaterials/Drugs/EndocrinologicandMetabolicDrugsAdvisoryCommittee/UCM218495.pdf>. Accessed March 29, 2013. 4. Bracken MB, et al. NeJM 2007;357:937-938. 5. Singh S, et al. JAMA 2007;298:1189-1195. 6. Diamond GA, et al. Ann Intern Med 2007;147:578-581. 7. Dormandy JA, et al. Lancet 2005;366:1279-1289. 8. Erdmann E, et al. J Am Coll Cardiol 2007;49:1772-1780. 9. Lincoff AM, et al. JAMA 2007;298:1180-1188

FDA cần chứng cứ tim mạch/thuốc điều trị ĐTĐ

- Owing to the potential for CV risk with drugs for T2DM, in December 2008, the FDA issued new guidance for all diabetes drugs in development: **Manufacturers of diabetes drugs and biologics need to provide evidence that therapy will not increase the risk of CV events**

More robust and adequate design and data collection are required for Phase 2/3 clinical trials:

- ✓ New diabetes therapies should not increase CV risk compared with current therapies, especially when used by older patients and in those with advanced diabetes or renal impairment
- ✓ Trials should include patients at higher risk of CV events

CV events occurring during clinical trials should be analyzed by independent committees

- ✓ This includes major events (CV mortality, MI, and stroke) and can also include hospitalization for ACS, urgent revascularization procedures, and other end points

FDA. December 2008. [http://www.fda.gov/downloads/Drugs/Guidance Compliance Regulatory Information/Guidances/ucm071627.p](http://www.fda.gov/downloads/Drugs/Guidance%20Compliance%20Regulatory%20Information/Guidances/ucm071627.p). Accessed Aug 12, 2013.

Nghiên cứu EMPAREG OUTCOME

(Empaglifozin, Cardiovascular outcomes and mortality in type 2 Diabete)

TL: Zinman B et al. N Engl J Med, 2015, Sept 17, doi: 10: 1056/NEJ Med oal 504720

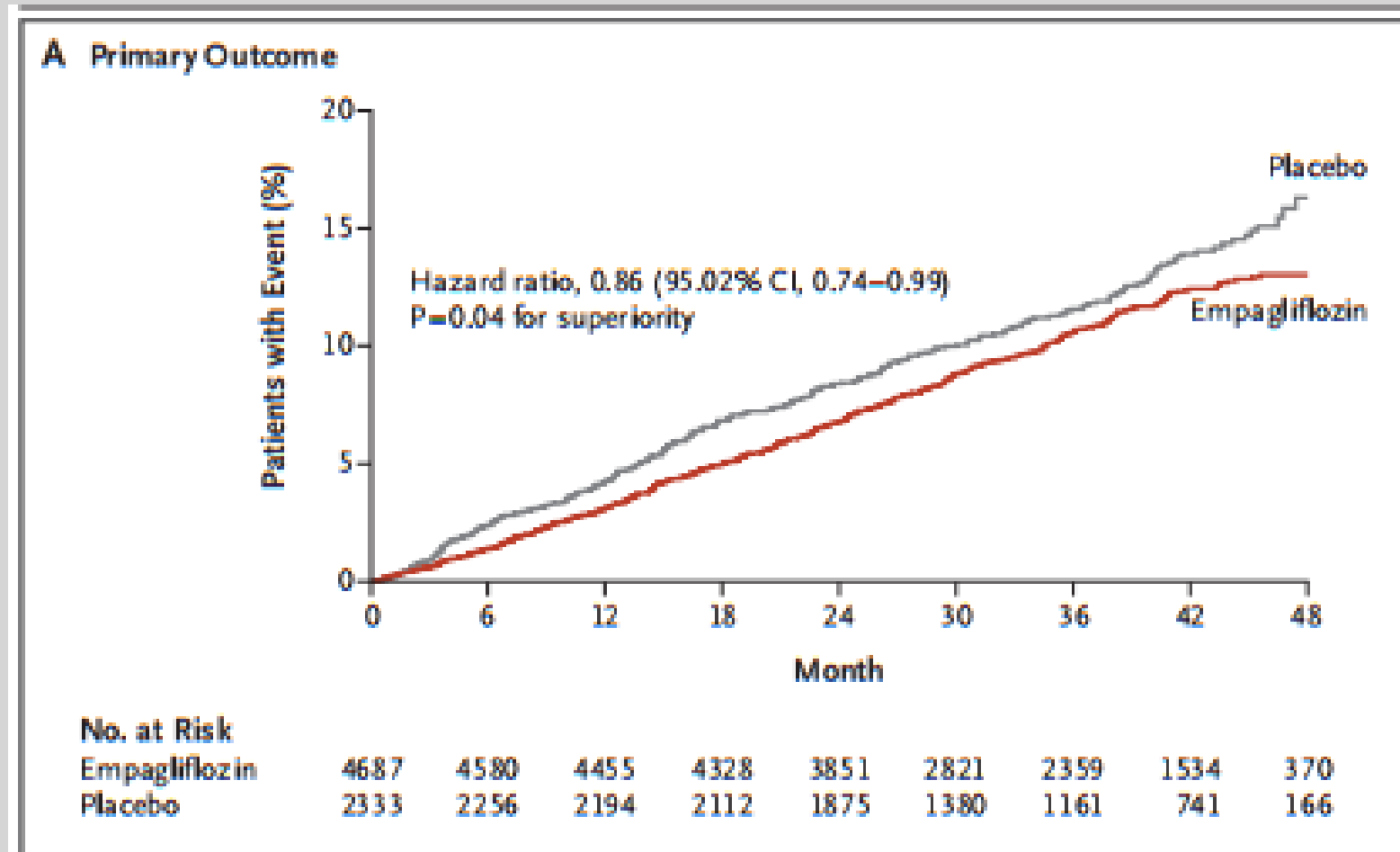
Thiết kế nghiên cứu

- Ngẫu nhiên, mù đôi, đa trung tâm, quốc tế
- 7020 bệnh nhân ĐTĐ2 có nguy cơ tim mạch cao ≥ 18 tuổi
- Nhóm empaglifozin (10 hoặc 25 mg/qđ)
- Nhóm placebo
- Theo dõi trung bình 3,1 năm
- Tiêu chí chính: tử vong tim mạch, NMCT không tử vong, đột quy không tử vong
- Tiêu chí phụ: tiêu chí chính + nhập viện vì đau thắt ngực không ổn định

TL: Zinman B et al. N Engl J Med, 2015, Sept 17, doi: 10: 1056/NEJ Med oal 504720

Kết quả nghiên cứu EMPAREG (1)

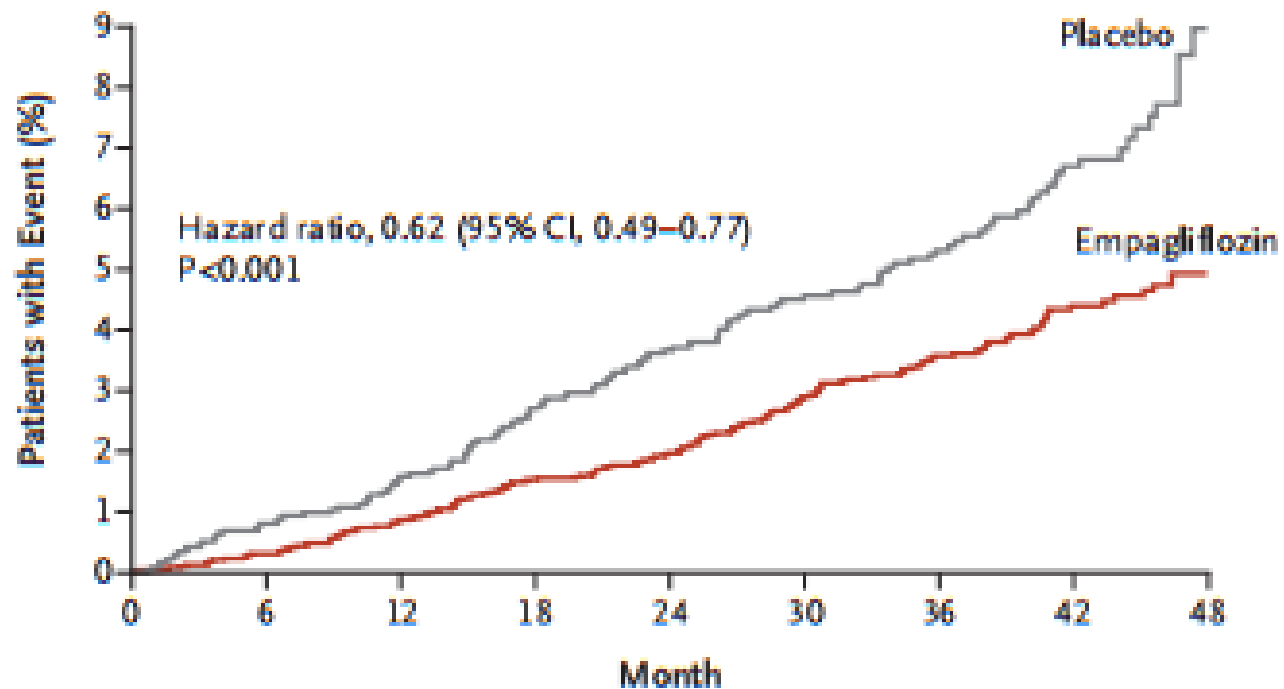
Tiêu chí chính



Kết quả nghiên cứu EMPAREG (2)

Tử vong do tim mạch

B Death from Cardiovascular Causes

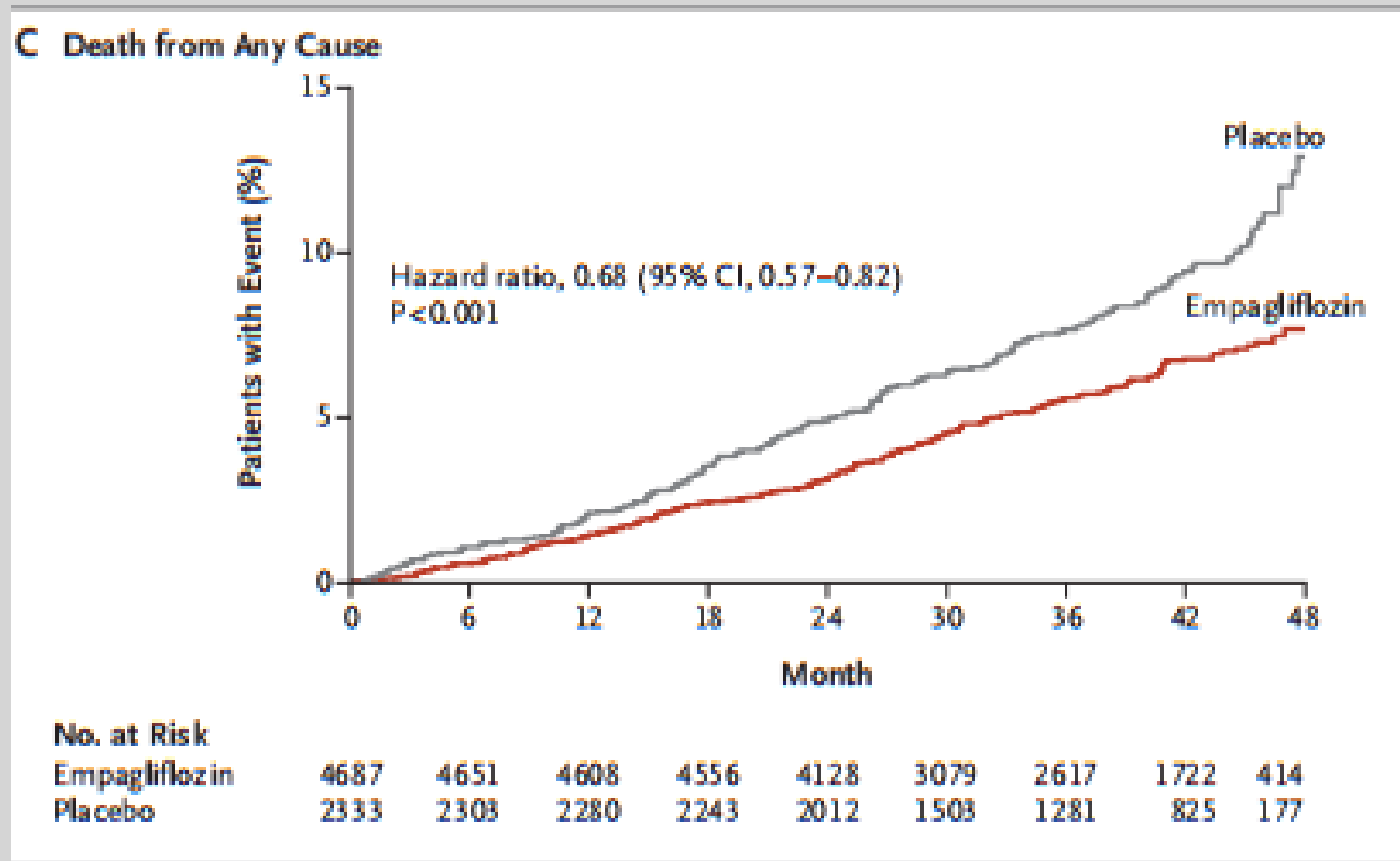


No. at Risk

Empagliflozin	4687	4651	4608	4556	4128	3079	2617	1722	414
Placebo	2333	2303	2280	2243	2012	1503	1281	825	177

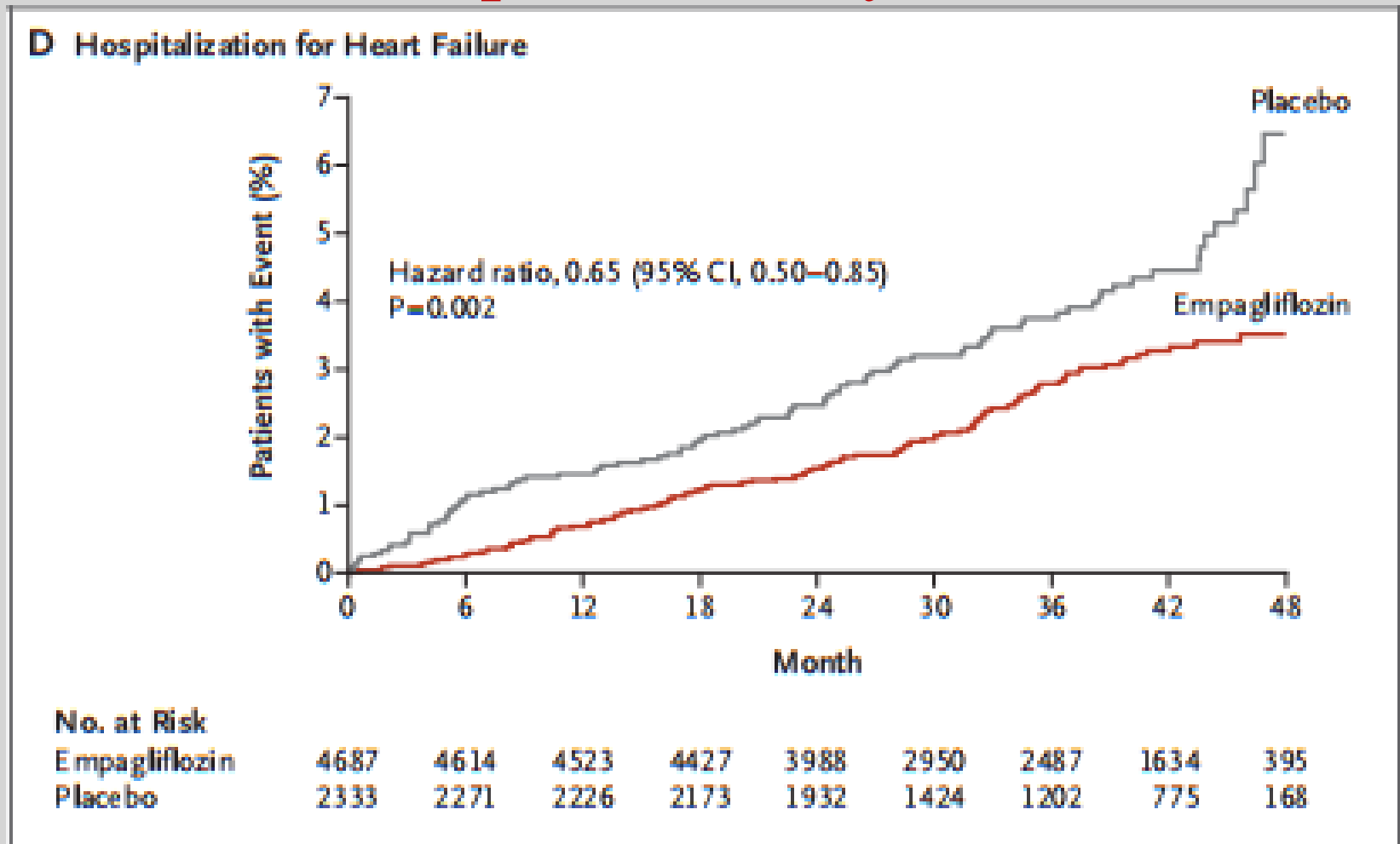


Kết quả nghiên cứu EMPAREG (3) Tử vong mọi nguyên nhân

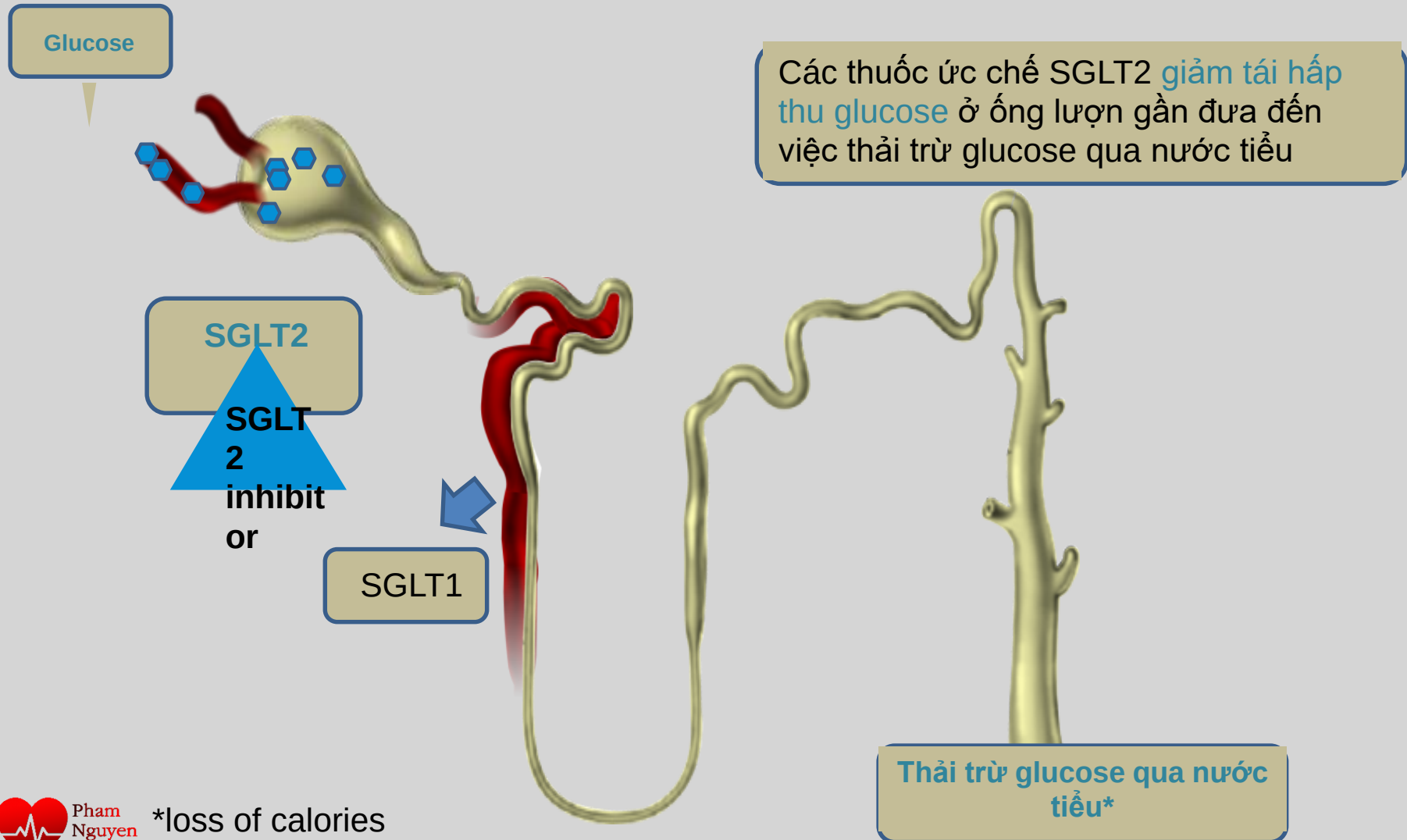


Kết quả nghiên cứu EMPAREG (4)

Nhập viện vì suy tim



Cơ chế hoạt động của các thuốc ức chế SGLT2: Đào thải glucose qua nước tiểu thông qua ức chế SGLT2 ¹



Nghiên cứu đầu tiên chứng minh lợi ích giảm biến cố tim mạch của thuốc ĐTD trên bệnh nhân ĐTD type 2

ORIGINAL ARTICLE

Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes

Bernard Zinman, M.D., Christoph Wanner, M.D., John M. Lachin, Sc.D., David Fitchett, M.D., Erich Bluhmki, Ph.D., Stefan Hantel, Ph.D., Michaela Mattheus, Dipl. Biomath., Theresa Devins, Dr.P.H., Odd Erik Johansen, M.D., Ph.D., Hans J. Woerle, M.D., Uli C. Broedl, M.D., and Silvio E. Inzucchi, M.D., for the EMPA-REG OUTCOME Investigators

ABSTRACT

BACKGROUND

The effects of empagliflozin, an inhibitor of sodium-glucose cotransporter 2, in addition to standard care, on cardiovascular morbidity and mortality in patients with type 2 diabetes at high cardiovascular risk are not known.

METHODS

We randomly assigned patients to receive 10 mg or 25 mg of empagliflozin or placebo once daily. The primary composite outcome was death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke, as analyzed in the pooled empagliflozin group versus the placebo group. The key secondary composite outcome was the primary outcome plus hospitalization for unstable angina.

RESULTS

A total of 7020 patients were treated (median observation time, 3.1 years). The primary outcome occurred in 490 of 4687 patients (10.5%) in the pooled empagliflozin group and in 282 of 2333 patients (12.1%) in the placebo group (hazard ratio in the empagliflozin group, 0.86; 95.02% confidence interval, 0.74 to 0.99; $P=0.04$ for superiority). There were no significant between-group differences in the rates of myocardial infarction or stroke, but in the empagliflozin group there were significantly lower rates of death from cardiovascular causes (3.7%, vs. 5.9% in the placebo group; 38% relative risk reduction), hospitalization for heart failure (2.7% and 4.1%, respectively; 35% relative risk reduction), and death from any cause (5.7% and 8.3%, respectively; 32% relative risk reduction). There was no significant between-group difference in the key secondary outcome ($P=0.08$ for superiority). Among patients receiving empagliflozin, there was an increased rate of genital infection but no increase in other adverse events.

CONCLUSIONS

Patients with type 2 diabetes at high risk for cardiovascular events who received empagliflozin, as compared with placebo, had a lower rate of the primary composite cardiovascular outcome and of death from any cause when the study drug was added to standard care. (Funded by Boehringer Ingelheim and Eli Lilly; EMPA-REG OUTCOME ClinicalTrials.gov number, NCT01131676.)

MAIN PAPER

Sep 2015 (EASD)



VIII

ORIGINAL ARTICLE

Empagliflozin and Progression of Kidney Disease in Type 2 Diabetes

Christoph Wanner, M.D., Silvio E. Inzucchi, M.D., John M. Lachin, Sc.D., David Fitchett, M.D., Maximilian von Eynatten, M.D., Michaela Mattheus, Dipl. Biomath., Odd Erik Johansen, M.D., Ph.D., Hans J. Woerle, M.D., Uli C. Broedl, M.D., and Bernard Zinman, M.D., for the EMPA-REG OUTCOME Investigators*

ABSTRACT

BACKGROUND

Diabetes confers an increased risk of adverse cardiovascular and renal events. In the EMPA-REG OUTCOME trial, empagliflozin, a sodium-glucose cotransporter 2 inhibitor, reduced the risk of major adverse cardiovascular events in patients with type 2 diabetes at high risk for cardiovascular events. We wanted to determine the long-term renal effects of empagliflozin, an analysis that was a prespecified component of the secondary microvascular outcome of that trial.

METHODS

We randomly assigned patients with type 2 diabetes and an estimated glomerular filtration rate of at least 30 ml per minute per 1.73 m² of body-surface area to receive either empagliflozin (at a dose of 10 mg or 25 mg) or placebo once daily. Prespecified renal outcomes included incident or worsening nephropathy (progression to macroalbuminuria, doubling of the serum creatinine level, initiation of renal-replacement therapy, or death from renal disease) and incident albuminuria.

RESULTS

Incident or worsening nephropathy occurred in 525 of 4124 patients (12.7%) in the empagliflozin group and in 388 of 2061 (18.8%) in the placebo group (hazard ratio in the empagliflozin group, 0.61; 95% confidence interval, 0.53 to 0.70; $P<0.001$). Doubling of the serum creatinine level occurred in 70 of 4645 patients (1.5%) in the empagliflozin group and in 60 of 2323 (2.6%) in the placebo group, a significant relative risk reduction of 44%. Renal-replacement therapy was initiated in 13 of 4687 patients (0.3%) in the empagliflozin group and in 14 of 2333 patients (0.6%) in the placebo group, representing a 59% lower relative risk in the empagliflozin group. There was no significant between-group difference in the rate of incident albuminuria. The adverse-event profile of empagliflozin in patients with impaired kidney function at baseline was similar to that reported in the overall trial population.

CONCLUSIONS

In patients with type 2 diabetes at high cardiovascular risk, empagliflozin was associated with slower progression of kidney disease and lower rates of clinically relevant renal events than was placebo when added to standard care. (Funded by the Boehringer Ingelheim and Eli Lilly and Company Diabetes Alliance; EMPA-REG OUTCOME ClinicalTrials.gov number, NCT01131676.)

RENAL PAPER

June 2016 (ADA)



European Heart Journal
doi:10.1093/eurheartj/ehw208

AHA

Heart failure/cardiomyopathy

Heart failure outcomes with empagliflozin in patients with type 2 diabetes at high cardiovascular risk: results of the EMPA-REG OUTCOME® trial

David Fitchett^{1*}, Bernard Zinman^{2,3}, Christoph Wanner⁴, John M. Lachin⁵, Stefan Hantel⁶, Ashin Salsali⁷, Odd Erik Johansen⁸, Hans J. Woerle⁹, Uli C. Broedl⁹, and Silvio E. Inzucchi¹⁰, on behalf of the EMPA-REG OUTCOME® trial investigators

¹Michael J. Hospital, Division of Cardiology, University of Toronto, Toronto, Canada; ²University of Toronto, Toronto, Canada; ³University of Toronto, Toronto, Canada; ⁴Boehringer Ingelheim, Biberach, Germany; ⁵Boehringer Ingelheim, Biberach, Germany; ⁶Boehringer Ingelheim, Biberach, Germany; ⁷Boehringer Ingelheim, Biberach, Germany; ⁸Boehringer Ingelheim, Biberach, Germany; ⁹Boehringer Ingelheim, Biberach, Germany; ¹⁰Boehringer Ingelheim, Biberach, Germany

Received 10 October 2015; revised 23 November 2015; accepted 10 December 2015

Aims

We previously reported that in the EMPA-REG OUTCOME® trial, empagliflozin added to standard of care reduced the risk of 3-point major adverse cardiovascular events, cardiovascular and all-cause death, and hospitalization for heart failure in patients with type 2 diabetes and high cardiovascular risk. We now further investigated heart failure outcomes in all patients and in subgroups, including patients with or without baseline heart failure.

Methods and results

Patients were randomized to receive empagliflozin 10 mg, empagliflozin 25 mg or placebo. Seven thousand and twenty patients were treated; 706 (10.1%) had heart failure at baseline. Heart failure hospitalization or cardiovascular death occurred in a significantly lower percentage of patients treated with empagliflozin [245/1687 patients (5.7%) than with placebo [198/2333 patients (8.5%)] [hazard ratio, HR: 0.66 (95% confidence interval: 0.55–0.79); $P<0.001$], corresponding to a number needed to treat to prevent one heart failure hospitalization or cardiovascular death of 35 over 3 years. Consistent effects of empagliflozin were observed across subgroups defined by baseline characteristics, including patients with vs. without heart failure, and across categories of medications to treat diabetes and/or heart failure. Empagliflozin improved other heart failure outcomes, including hospitalization for or death from heart failure (2.8 vs. 4.3%; HR: 0.61 (0.47–0.79); $P<0.001$) and was associated with a reduction in all-cause hospitalization (36.8 vs. 39.6%; HR: 0.89 (0.82–0.96); $P=0.003$). Serious adverse events and adverse events leading to discontinuation were reported by a higher proportion of patients with vs. without heart failure at baseline in both treatment groups, but were no more common with empagliflozin than with placebo.

Conclusion

In patients with type 2 diabetes and high cardiovascular risk, empagliflozin reduced heart failure hospitalization and cardiovascular death, with a consistent benefit in patients with and without baseline heart failure.

Keywords

Cardiovascular disease • Hospitalization • Mortality

HHF PAPER

Jan 2016

Lợi ích tim mạch của Empagliflozin trong nghiên cứu EMPA-REG OUTCOME® Thay đổi các khuyến cáo quốc tế trong điều trị BN ĐTĐ týp 2



Khuyến cáo của Canada về quản lý thuốc trong điều trị đái tháo đường týp 2¹



EUROPEAN
SOCIETY OF
CARDIOLOGY®

Hướng dẫn ESC về chẩn đoán và điều trị suy tim cấp và mãn trong chẩn đoán và điều trị suy tim và dự phòng bệnh tim mạch trong thực hành lâm sàng^{2,3}



Phác đồ quản lý toàn diện của AACE/ACE 2017 ở bệnh nhân đái tháo đường týp 2⁴



Khuyến cáo của Hội đái tháo đường Mỹ ADA 2017 về các chuẩn chăm sóc y tế ở bệnh nhân đái tháo đường⁵

Does the risk for heart failure modulate the effectiveness of empagliflozin on HF hospitalization or CV death in patients with type 2 diabetes without HF?

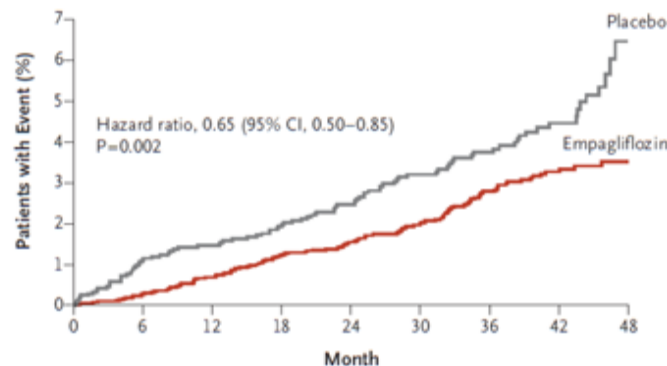
Insights from EMPA-REG OUTCOME Trial

Javed Butler, MD MPH MBA
Professor of Medicine
Director, Division of Cardiovascular Medicine
Stony Brook University, Stony Brook, New York

Background

- In EMPA-REG OUTCOME, in 7020 patients with T2D and CV disease, empagliflozin reduced CV mortality by 38%, HF hospitalization by 35%, and the composite of CV mortality or HF hospitalization by 34%

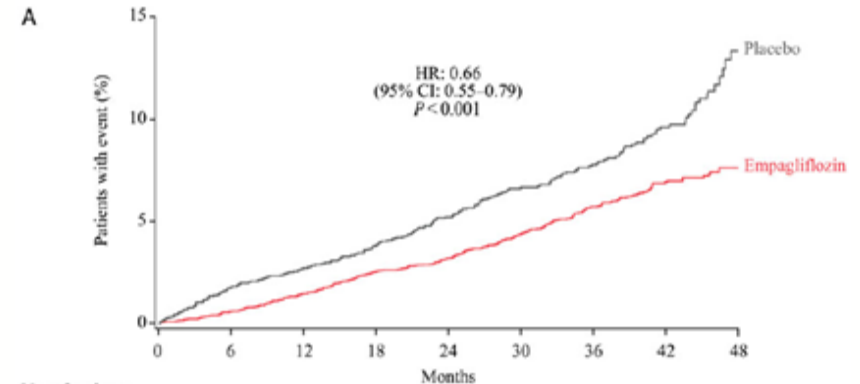
Time to first hospitalization for HF



No. at Risk									
Empagliflozin	4687	4614	4523	4427	3988	2950	2487	1634	395
Placebo	2333	2271	2226	2173	1932	1424	1202	775	168

Zinman et al. *N Engl J Med.* 2015;373:2117-2128

Time to first hospitalization for HF or CV death



No. of patients									
Empagliflozin	4687	4614	4523	4427	3988	2950	2487	1634	395
Placebo	2333	2271	2226	2173	1932	1424	1202	775	168

Fitchett et al. *Eur Heart J* 2016;37:1526-1534

Conclusion

- In patients with T2D and established CV disease, a sizeable proportion without HF are at high or very high risk for HF outcomes
- Empagliflozin reduced HF hospitalisation and CV mortality in type 2 diabetes patients with or without HF at baseline
- In patients without HF at baseline, empagliflozin reduced HF hospitalisation and CV mortality across a spectrum of HF risk

Type 2 diabetes mellitus and heart failure: a position statement from the Heart Failure Association of the European Society of Cardiology

The coexistence of type 2 diabetes mellitus (T2DM) and heart failure (HF), either with reduced (HFrEF) or preserved ejection fraction (HFpEF), is frequent (30–40% of patients) and associated with a higher risk of HF hospitalization, all-cause and cardiovascular (CV) mortality. The most important causes of HF in T2DM are coronary artery disease, arterial hypertension and a direct detrimental effect of T2DM on the myocardium. T2DM is often unrecognized in HF patients, and vice versa, which emphasizes the importance of an active search for both

disorders in the clinical practice. There are no specific limitations to HF treatment in T2DM. Subanalyses of trials addressing HF treatment in the general population have shown that all HF therapies are similarly effective regardless of T2DM. Concerning T2DM treatment in HF patients, most guidelines currently recommend metformin as the first-line choice. Sulphonylureas and insulin have been the traditional second- and third-line therapies although their safety in HF is equivocal. Neither glucagon-like peptide-1 (GLP-1) receptor agonists, nor dipeptidyl peptidase-4 (DPP4) inhibitors reduce the risk for HF hospitalization. Indeed, a DPP4 inhibitor, saxagliptin, has been associated with a higher risk of HF hospitalization. Thiazolidinediones (pioglitazone and rosiglitazone) are contraindicated in patients with (or at risk of) HF. In recent trials, sodium–glucose co-transporter-2 (SGLT2) inhibitors, empagliflozin and canagliflozin, have both shown a significant reduction in HF hospitalization in patients with established CV disease or at risk of CV disease. Several ongoing trials should provide an insight into the effectiveness of SGLT2 inhibitors in patients with HFrEF and HFpEF in the absence of T2DM.



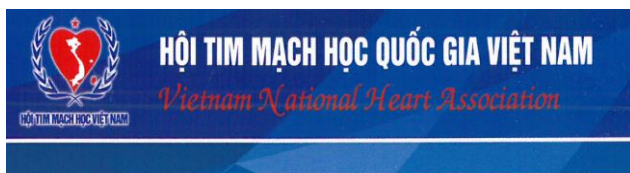
ESC 2016 guidelines for diagnosis and treatment of acute and chronic heart failure



*“Recently, **empagliflozin** has been **shown to improve outcomes** (including the reduction of mortality and HF hospitalizations) in patients with T2D.”*

Recommendations	Class	Level
Treatment of hypertension is recommended to prevent or delay the onset of HF and prolong life	I	A
Treatment with Statins is recommended in patients with or at high-risk of CAD whether or not they have LV systolic dysfunction, in order to prevent or delay the onset of HF and prolong life.	I	A
Counselling and treatment for smoking cessation and alcohol intake reduction is recommended for people who smoke or who consume excess alcohol in order to prevent or delay the onset of HF.	I	C
Treating other risk factors of HF (e.g. obesity, dysglycaemia) should be considered in order to prevent or delay the onset of HF.	IIa	C
Empagliflozin should be considered in patients with type 2 diabetes in order to prevent or delay the onset of HF and prolong life	IIa	B

Khuyến cáo của Hội Tim mạch học Việt Nam



KHUYẾN CÁO VỀ CHẨN ĐOÁN VÀ ĐIỀU TRỊ SUY TIM

(Cập nhật 2017)

www.vnha.org.vn

Chỉ định nhóm IIa:

- UCMC có thể hữu ích phòng ngừa suy tim trên BN có tiền sử bệnh do xơ vữa động mạch hoặc ĐTD hoặc THA có kèm yếu tố nguy cơ tim mạch.
- CTTA có thể hiệu quả tương tự UCMC dù mức bằng chứng kém hơn.
- Cần nhắc sử dụng **Empaglifozin** điều trị ĐTD týp 2 giúp chậm phát triển suy tim và tăng khả năng sống còn.

Kết luận

- Đái tháo đường là bệnh tim mạch:
 - Biến chứng mạch máu lớn
 - Biến chứng mạch máu nhỏ
- Thuốc điều trị ĐTĐ:
 - Không tăng biến cố tim mạch
 - Giúp giảm biến cố tim mạch
- Empaglifozin: thuốc đầu tiên
 - Giảm tử vong tim mạch và tử vong mọi nguyên nhân
 - Giảm suy tim