Nová antidiabetika (gliflozíny a semaglutid) *- co je důležité pro IM*

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ICU, Landesklinikum Baden bei Wien, Austria

University of applied Sciences

Hochschule Campus Wien, Austria

Akutně 2026

Nová antidiabetika (SGLT2-i a GLP1ra)

- co je důležité pro IM

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no conflict of interest

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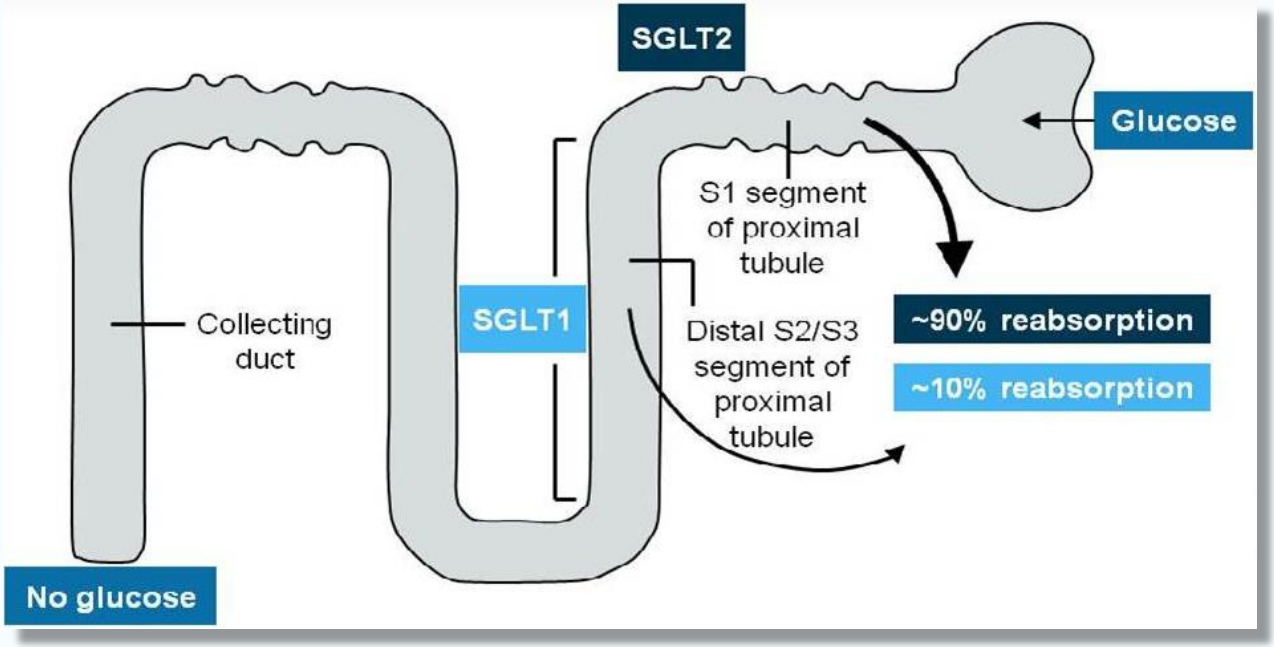
přehled

1. co jsou SGLT2i?
2. terapeutické efekty
3. indikace
4. rizika relevantní pro ICU
5. euglykemická diabetická ketoacidóza
6. perioperační management
7. GLP-1 ra

- **Sodium-Glucose Linked Transporter 2** (SGLT2) inhibitors are a new class of oral drugs to treat type 2 diabetes mellitus (Type 2 DM).
- **Sodium-Glucose Linked Transporters (SGLT)** or **Sodium-dependent glucose cotransporters** are a family of glucose transporter.

- There are two kinds of SGLTs: SGLT1 and SGLT2.
- **SGLT1** is found in intestinal mucosa of the small intestine and proximal straight tubule (PST) of nephron.
- **SGLT2** is found in proximal convoluted tubule (PCT) of nephron.

- Both SGLT1 and SGLT2 are responsible for the renal glucose reabsorption.
- Approximately 90% of glucose is reabsorbed by SGLT2 at PCT and only around 10% of glucose is reabsorbed by SGLT1 at PST.



SGLT2 Inhibitors

- SGLT2 Inhibitors include:
  - Dapagliflozin (**Farxiga**)
  - Canagliflozin (**Invokana**)
  - Empagliflozin (**Jardiance**)
  - Ipragliflozin
  - Tofogliflozin
  - Remogliflozin etabonate
  - Sergliflozin etabonate

- **Dapagliflozin** is the first drug to be developed in this class. But, its approval was rejected in 2012 by Food and Drug Administration (FDA) due to safety concerns. Later it was approved by FDA on 8th Jan 2014. Previously it was approved by EU in 2011.
- **Canagliflozin** is the first drug to be approved by FDA which got the approval by 29th Mar 2013.
- **Empagliflozin** got the FDA approval by 1st Aug 2014.
- **Ipragliflozin** (ASP-1941), is in Phase III clinical trials
- **Tofogliflozin**, is also in Phase III clinical trials
- **Remogliflozin etabonate**, is in phase IIb trials.
- **Sergliflozin etabonate**, discontinued after Phase II trials.

- SGLT2 inhibitors can improve glycemic control in conjunction with exercise and a healthy diet.
- They can be used either alone or in combination with other antidiabetics such as metformin, sulfonylureas, pioglitazone and insulin.

SGLT2 Inhibitors

Generic Name	Brand Name	Company Producing	Date of FDA Approval
Canagliflozin	Invokana	Janssen	March 29, 2013
Dapagliflozin	Farxiga	Astrazeneca	January 8, 2014
Empagliflozin	Jardiance	Boehringer Ingelheim	August 1, 2014
Canagliflozin + metformin	Invokamet	Janssen	August 8, 2014
Dapagliflozin + metformin	Xigduo	Astrazeneca	October 29, 2014
Empagliflozin + linagliptin	Glyxambi	Boehringer Ingelheim	January 30, 2015
Empagliflozin + metformin	Synjardy	Boehringer Ingelheim	August 29, 2015
Canagliflozin + metformin	Invokamet XR	Janssen	September 20, 2016

Abbreviations: SGLT2, sodium-glucose cotransporter 2; FDA, US Food and Drug Administration.

DNES



arena, sál pro filhar
V Brně konč
ODS a její výra

Nové léky na hubnutí míří do Česka

Pro některé pacienty mohou být zdravotním rizikem

Kamila Bendová
spolupracovnice
MF DNES



Ozempic, Mounjaro nebo Wegovy. Tyto léky způsobily revoluci v léčbě obezity po celém světě a v posledních letech se staly hitem i v Česku. Aktuálně je u nás užívá už více než 50 tisíc lidí, kteří si chválí jejich účinky nejen na hubnutí, ale i na srdce. Užívání „hubnoucích" léků může být ale i nebezpečné, zejména u těch, kdo chtějí jen bezpracně zhubnout.

Díky snižující se ceně se léky na obezitu masově šíří. Podle dat Státního ústavu pro kontrolu léčiv (SÚKL) bylo v roce 2025 dodáno do tuzemských lékáren přes 1,37 milionu balení. To znamená více než pětinásobný nárůst za pět let.

Největší je spotřeba léku Ozempic s účinnou látkou semaglutid a Mounjaro s látkou tirzepatid. Loni se k nim přidaly ještě injekce Wegovy se stejnou účinnou látkou jako Ozempic.

Rozdíl je v tom, že zatímco starší lék mohli dostat pouze diabetici 2. typu, ten nový s vyšším obsahem semaglutidu je určen k léčbě obezity dospělých i dětí od 12 let.

Všechny snižují chuť k jídlu

Aktuálně přichází do Česka semaglutid o síle 7,2 mg (dosud byl k dispozici maximálně 2,4mg), který podle studií slibuje pokles až o pětinu výchozí hmotnosti. Následovat bude tabletová forma tohoto léku, která je nyní již k dispozici v USA.

Protože jsou léky na hubnutí součástí obrovského byznysu, farmaceutické firmy neustále vyvíjejí další látky, které by měly mít méně nežádoucích účinků. Schválení další tabletové léčby, orforglipronu, se očekává v roce 2027.

Všechny tyto léky fungují na podobném principu: obsahují látky podobné hormonu, který se po jídle uvolňuje ze střev. Tím, že působí na receptory v mozku a snižují chuť k jídlu.

1,4 mil.
balení léků na hubnutí bylo v roce 2025 dodáno do českých lékáren.

50 tis.
lidí v Česku aktuálně užívá léky na hubnutí.

Ve fázi finálního testování je dále lék MariTide, který se podává jen jednou měsíčně podkožní injekcí (na rozdíl od týdenního podávání). Působí přes dva receptory hormonů z trávicího traktu a pacienti údajně mohou ztratit až 20 procent své hmotnosti za rok.

Vedlejší účinky, jako jsou nevolnost nebo zvracení, byly u MariTide méně časté a většinou se objevovaly pouze při prvních dávkách. Na českém trhu by se mohl objevit v roce 2028.

Pokračování na str. 3

Gliflozin Market

MARKET SIZE · 2024

$6.64B

USD BILLION

FORECAST · 2035

$25.26B

USD BILLION

12.92% CAGR

GROWTH RATE

2024 → 2035

Nearly **3.8× value** over the forecast window

BY TYPE OUTLOOK

- Dapagliflozin
- Empagliflozin
- Canagliflozin
- Ertugliflozin

BY INDICATION OUTLOOK

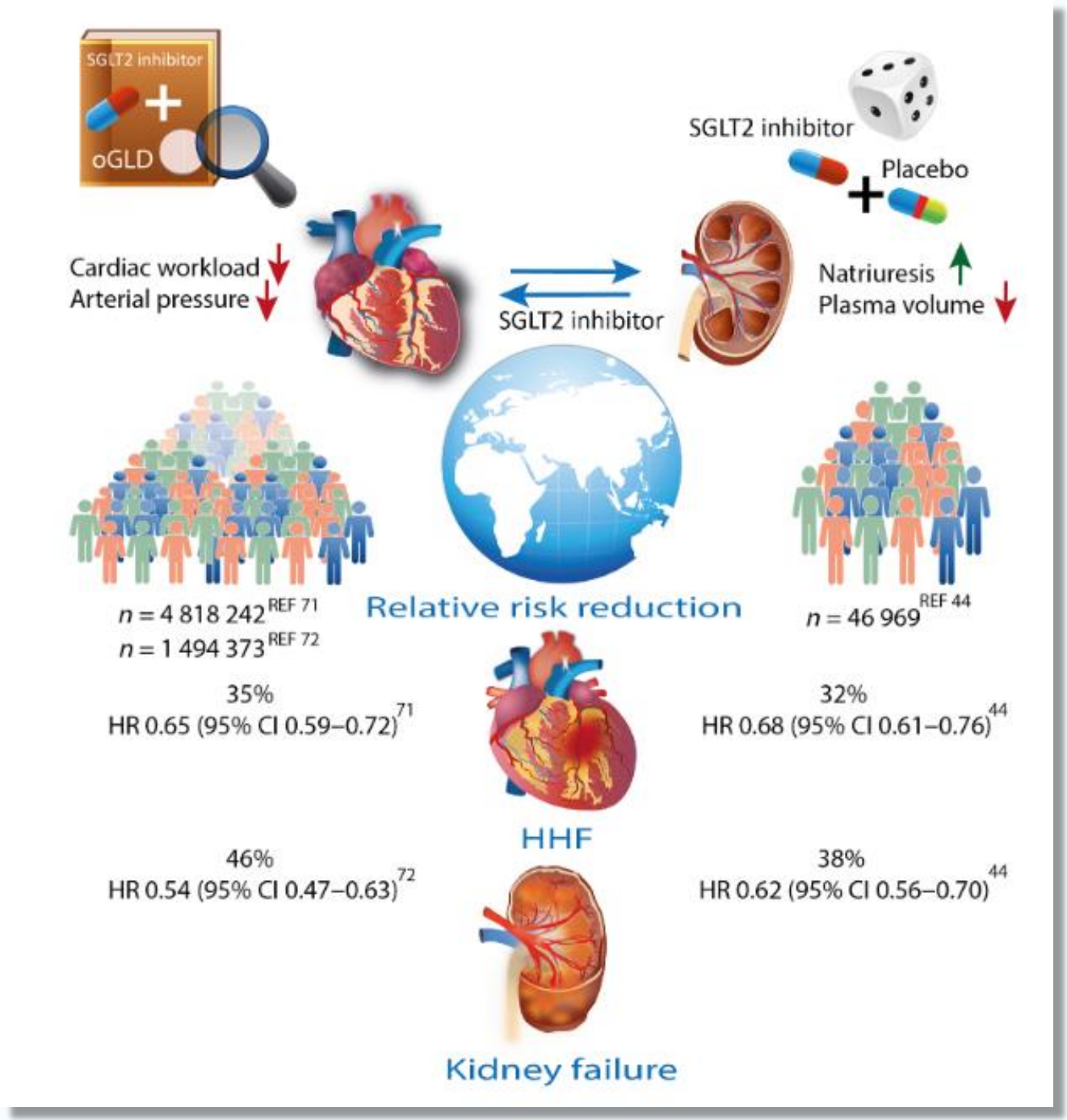
- Type 2 Diabetes
- Heart Failure
- Chronic Kidney Disease

BY ROUTE OUTLOOK

- Oral
- Intravenous

Industry Leaders & Producers

- Boehringer Ingelheim (DE)
- AstraZeneca (GB)
- Merck & Co. (US)
- Bristol-Myers Squibb (US)
- Sanofi (FR)
- Johnson & Johnson (US)
- Eli Lilly and Company (US)
- Novartis (CH)


SGLT2 inhibitor
oGLD
SGLT2 inhibitor
Placebo
Cardiac workload
Arterial pressure
SGLT2 inhibitor
Natriuresis
Plasma volume
n = 4 818 242 REF 71
n = 1 494 373 REF 72
Relative risk reduction
n = 46 969 REF 44
35%
HR 0.65 (95% CI 0.59–0.72) 71
32%
HR 0.68 (95% CI 0.61–0.76) 44
HHF
46%
HR 0.54 (95% CI 0.47–0.63) 72
38%
HR 0.62 (95% CI 0.56–0.70) 44
Kidney failure

therapeutic effects

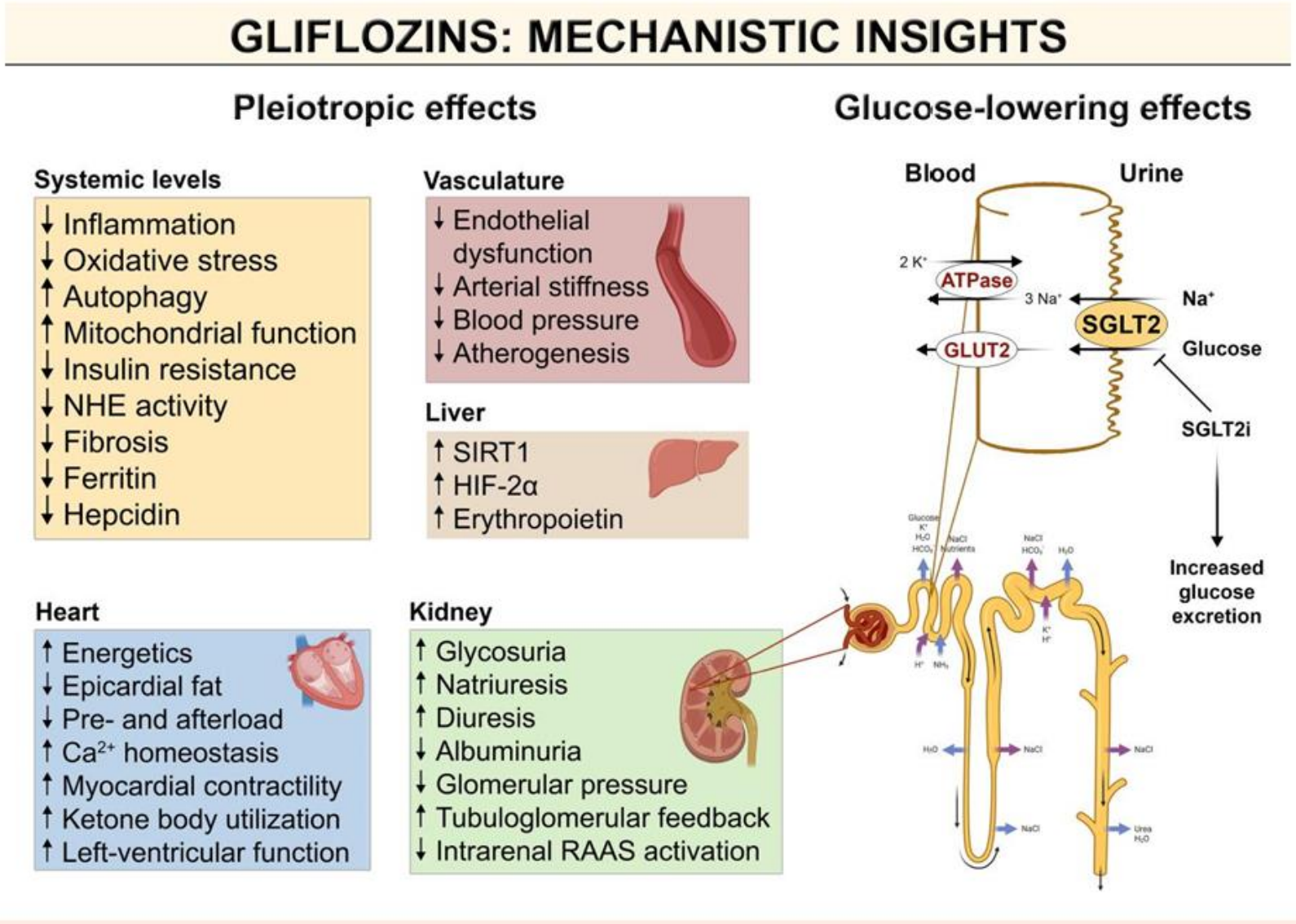


Figure 1 Mechanistic insights into the role of gliflozins at systemic, vascular, cardiac, and renal levels. Preclinical evidence suggests a potential beneficial effect of gliflozins in preclinical models mimicking renal, cardiac, and vascular pathologies. Involving a variety of mechanisms independently of their glucose-lowering effects, SGLT2 inhibition reduces inflammatory signalling, mitigates oxidative stress, improves autophagic flux, and enhances mitochondrial function, thereby contributing to their cardiorenal/-vascular benefits. SGLT2, sodium-glucose transporter 2; NHE, sodium/hydrogen exchanger. Created with content provided by BioRender.com.

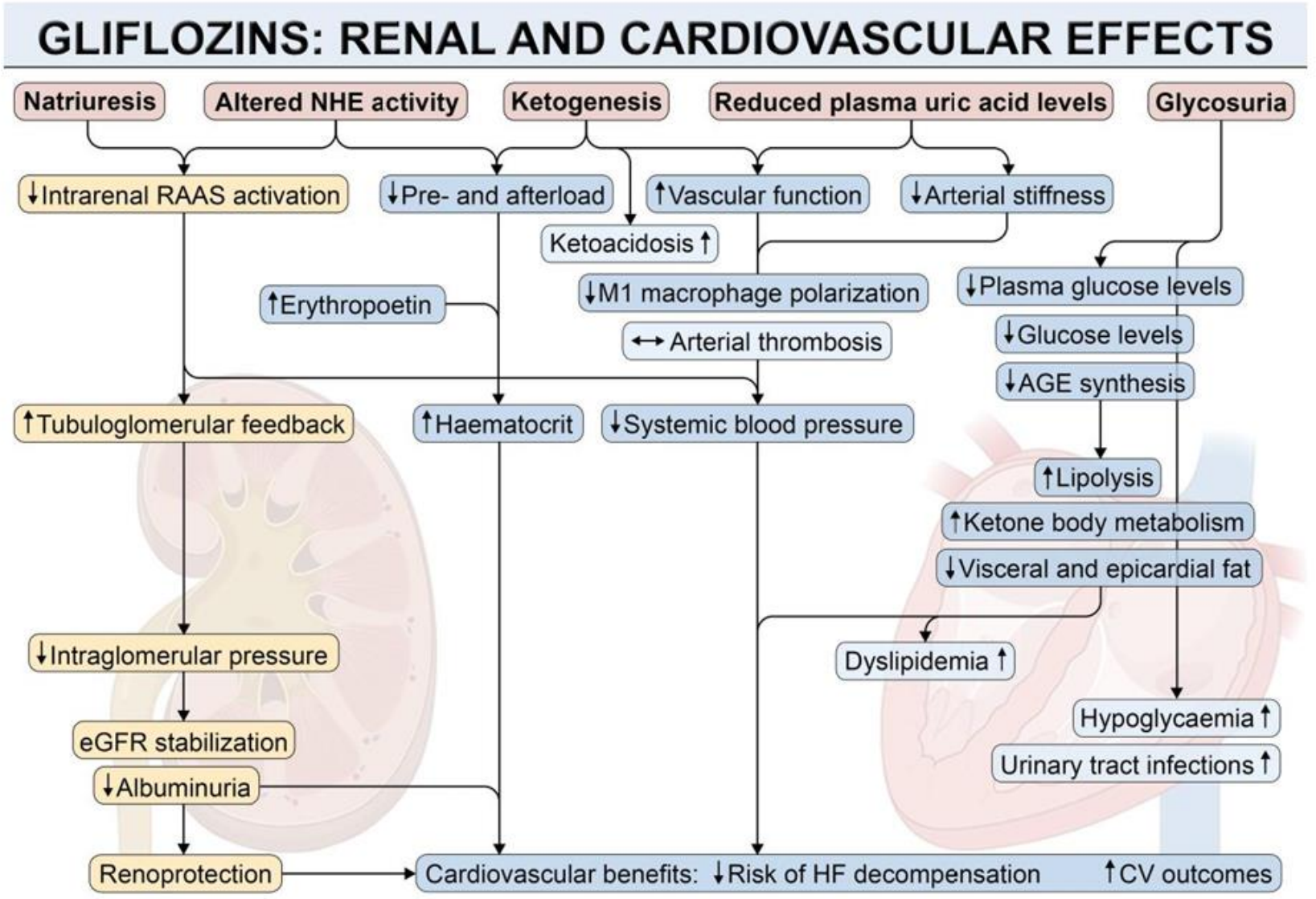


Figure 2 Systemic effects of gliflozins. Gliflozins exert natriuretic effects, impact tissue sodium handling, increase ketogenesis, and reduce both plasma uric acid and glucose levels, leading to a variety of beneficial effects which collectively results in improved renal and CV outcomes. Note that neutral or negative effects are highlighted in bright blue, with lower urinary tract infections ranking among the most frequently observed side-effects. Created with content provided by BioRender.com.

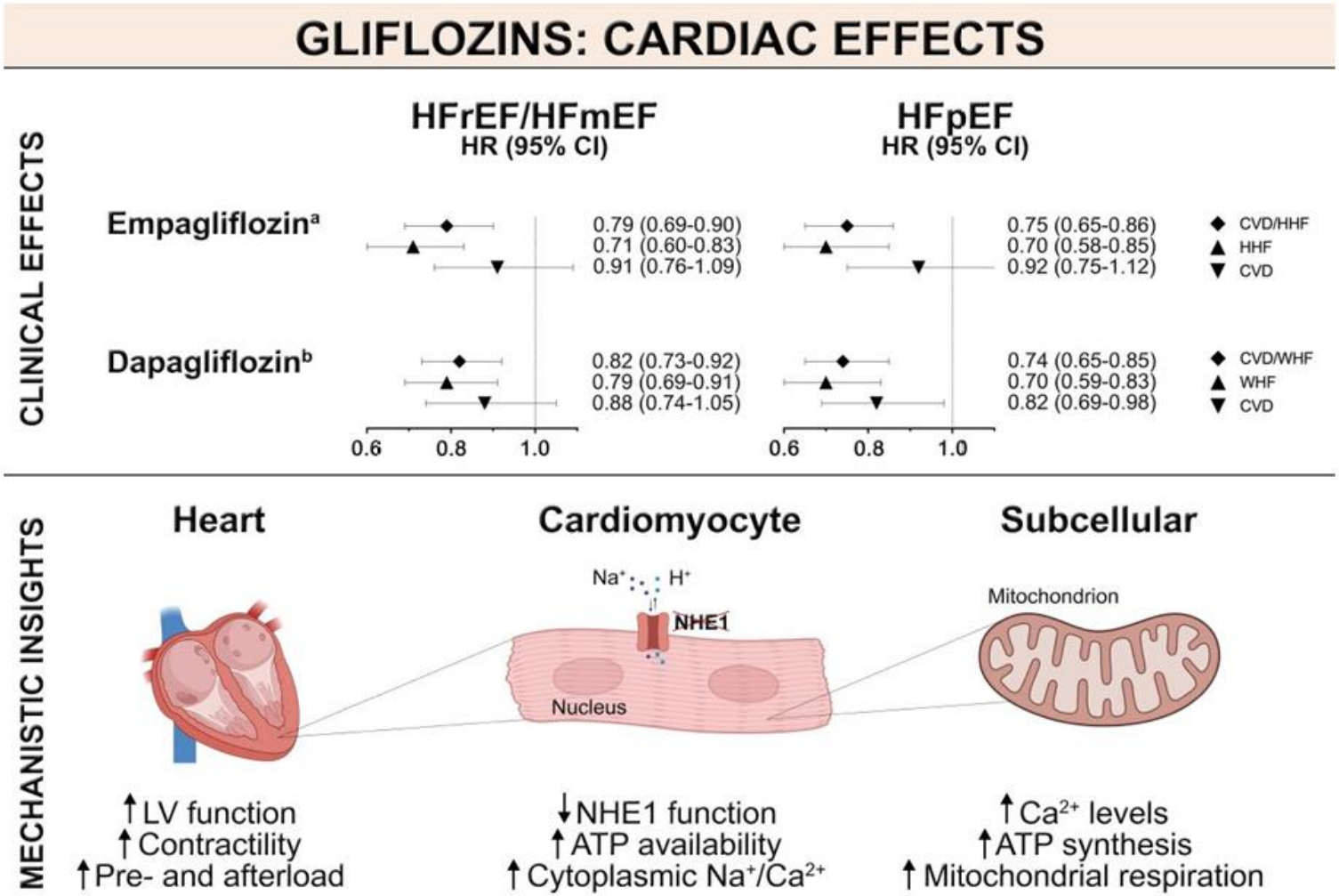


Figure 3 The cardiac effect of gliflozins across the HF spectrum. Empagliflozin and dapagliflozin reduce adverse CV outcomes in patients with HF independently of the ejection fraction. The upper panel shows the clinical benefits of SGLT2 inhibition, with treatment effects shown as hazard ratios (HR) along with their 95% confidence intervals (CI). The lower panel provides insights into potential mechanisms underpinning cardiac benefits. ATP, Adenosine triphosphate; CD, cardiovascular death; CI, confidence interval; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HHF, hospitalization for heart failure; HR, hazard ratio; LV, left-ventricular; NHE, sodium/hydrogen exchanger; WHF, worsening of heart failure. Created with content provided by BioRender.com. [a]Refers to the EMPEROR-Reduced Trial[152] and EMPEROR-Preserved Trial.[153] [b]Refers to the DAPA-HF Trial[154] and DELIVER Trial.[155]

indications

SGLT2 Inhibitors in 2026: Settled Science vs. Clinical Hype

THE SHIFT: FROM GLUCOMETER TO ORGAN PROTECTION

TREATING THE GLUCOMETER

PROTECTING THE HEART & KIDNEYS

FOUNDATIONAL THERAPY FOR HEART FAILURE & CKD (Regardless of Diabetes Status)

SETTLED SCIENCE: PRESCRIBE BY DEFAULT (Class I, Level A)

HEART FAILURE (All Ejection Fractions)
ESC 2026 guidelines: **Class I, Level A** recommendations for HFrEF, HFmrEF, and HFpEF as foundational therapy.

CKD WITH OR WITHOUT DIABETES
Settled for patients with eGFR ≥ 20 ml/min/1.73m². **DAPA-CKD** and **EMPA-KIDNEY** show definitive kidney and cardiovascular protection.

T2D WITH ASCVD OR HIGH RISK
ADA Grade A and **ESC Class I, Level A**. Cardiovascular and kidney risk reduction is a co-primary goal alongside glycemic control.

UNSETTLED / HYPED: EXPERIMENTAL & INVESTIGATIONAL (Neutral or Emerging Data)

POST-MYOCARDIAL INFARCTION (Post-MI)
EMPACT-MI and **DAPA-MI** missed primary endpoints. Not an indication unless the patient has HF, T2D, or CKD.

POST-TAVI (Transcatheter Valve)
Promising results from DapaTAVI trial (HR 0.72 for HF events) but currently rests on a single open-label trial.

MASH AND ATRIAL FIBRILLATION
These remain investigational. MASH resolution looks promising in small trials (DEAN), but larger confirmation is required.

GUIDELINE GRADES (2026)

Indication	ESC 2026 Grade	ADA/KDIGO Grade
HFrEF	Class I, Level A	Class I, Level A
HFpEF	Class I, Level A	IIa (Older AHA)
CKD (eGFR >20 + ACR >200)	N/A	1A (KDIGO)
T2D + ASCVD	Class I, Level A	Grade A
Type 1 Diabetes	Not Recommended	Not Approved (US)

CLINICAL WORKFLOW & PRESCRIBING RULES

EXPECT AND ACCEPT THE eGFR DIP
An early creatinine rise reflects tubuloglomerular feedback, not injury. ADA 2026 advises against stopping the drug for this dip.

OPTIMIZE BACKGROUND THERAPY
Most trials were conducted alongside ACE inhibitors or ARBs; ensure these are optimized rather than replaced.

MANAGE SULFONYLUREA RISK
SGLT2i has low hypoglycemia risk alone, but consider reducing sulfonylurea doses when adding to a regimen to prevent drops.

THE "SICK-DAY" PROTOCOL
Hold the drug during acute illness and 3–4 days before surgery to mitigate the risk of euglycemic DKA.

ALERT: MONITOR FOR EUGLYCEMIC DKA RISK DURING STRESS OR ILLNESS

Gemini Notebook

SGLT2 Inhibitors in 2026: Navigating Indications

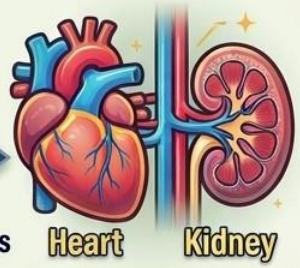
From Glucose Control to Foundation Therapy – How to Tier Your Patients



Tension: Treat glucometer,
Tension: Treat glucometer,
OR **KIDNEY** and **HEART**?

SGLT2i are no longer just glucose drugs.

adds benefits SGLT2i adds benefits



TIER BEFORE YOU PRESCRIBE

SETTLED ARM: prescribe by default

- **HEART FAILURE** (any EF): Foundational Therapy (class I rec.)
- **CKD** (eGFR ≥20, with/without T2D): Kidney/Cardiovascular protection
- **T2D w/ Cardiovascular or Kidney Disease:** Co-primary reduction goals

Back to patient: Sits squarely here. HbA1c not why you prescribe. Kidney is.

UNSETTLED ARM: prescribe with caution/extrapolation

- **In-hospital acute HF:** evidence is pooled, individual trials neutral.
- **Post-MI w/o other indication:** primary results neutral, study design matters.
- **Advanced CKD** (eGFR <20) / **Dialysis / Transpiant:** trials like **RENAL LIFECYCLE** on-going.
- **Post-TAVI:** promising 1-trial data (DapaTAVI), no guideline grade yet.
- **T1D & ertugliflozin:** barriers (DKA, specific data).

KEY CLINICAL PEARLS & WHY

eGFR Dip is Expected: Tubuloglomerular feedback, NOT injury. **DO NOT STOP.**

Mediation by Albuminuria: Most kidney effect linked to ACR reduction.

Review Sulfonylurea: Low SGLT2i hypo risk, but combines with SU.

Counsel on practical risks: Genital infection & volume depletion. **SICK-DAY RULES.** (hold 3-4 days pre-surgery)

DKA

poznámka 1

SGTL2i and kidneys

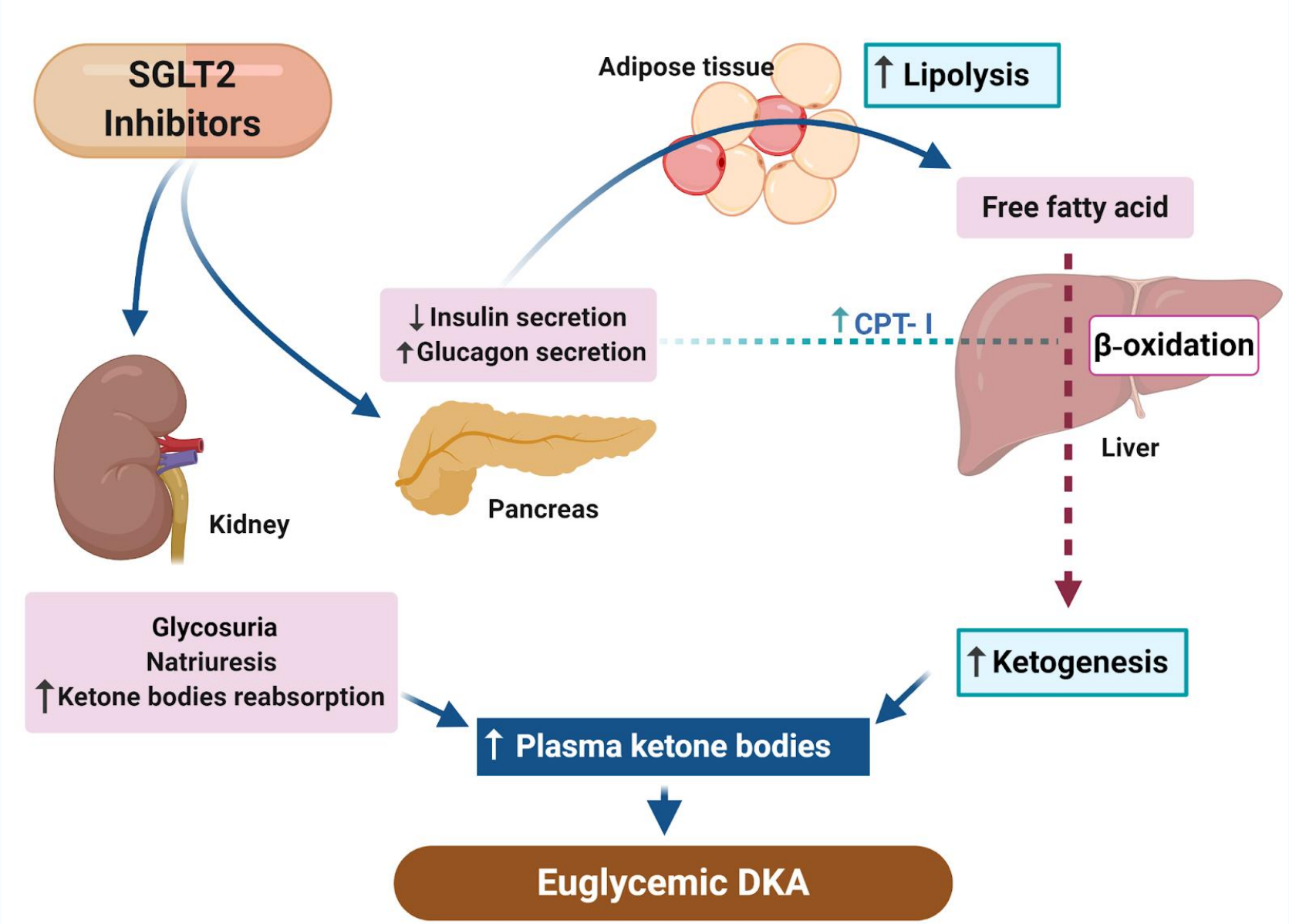
- SGLT2 inhibitors **are not** intrinsically **nephrotoxic**
- they **can cause** an initial, usually **reversible reduction** in **eGFR**
- their **long-term** effect is **renoprotective**
- **AKI** risk in **critically ill** patients: mainly related to **hypovolemia** and **hemodynamic instability**

relevant risks on ICU

ADVERSE EFFECT	CLINICAL RELEVANCE IN ICU
euglycemic DKA (euDKA)	*- most important potentially **life-threatening** complication*
hypovolemia	*- **osmotic** diuresis, **dehydration**, reduced preload*
hypotension	*- particularly relevant in **septic** or **postoperative** patients*
AKI	*- possible secondary to **hypovolemia** and reduced **renal perfusion***
electrolyte disturbances	*- changes in **Na^+** and **K^+**; K^+ depletion during ketoacidosis treatment*
genital mycotic infections	*- increased risk of **Candida** infections*
urinary tract **infections**	*- increased risk; occasionally severe **urosepsis** or **pyelonephritis***
hypoglycemia	*- usually low risk with monotherapy, higher with **insulin** or **sulfonylureas***
Fournier's gangrene	*- very **rare** but **serious** necrotizing infection of the perineum*

euglycemic diabetic ketoacidosis

pathophysiology



EUGLYCEMIC DIABETIC KETOACIDOSIS (EDKA): A CRITICAL ATYPICAL CRISIS

DEFINITION & KEY FEATURES

EUGLYCEMIA



Blood Glucose less than 250 mg/dL

SEVERE METABOLIC ACIDOSIS



pH < 7.3
HCO3 < 18 mEq/L

pH less than 7.3, Bicarbonate less than 18 mEq/L

KETOSIS & KETONEMIA

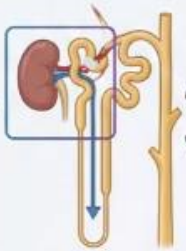


Ketosis with ketonemia

SGLT2 INHIBITORS & EDKA LINK



INHIBIT GLUCOSE REABSORPTION IN PROXIMAL RENAL TUBULES



PROMOTES URINARY GLUCOSE EXCRETION

REDUCES BLOOD GLUCOSE LEVELS

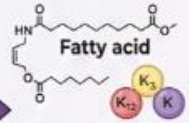
PATHOPHYSIOLOGY OF SGLT2 INHIBITOR-ASSOCIATED EDKA



DECREASED INSULIN PRODUCTION



INCREASED GLUCAGON SECRETION



METABOLIC SHIFT FAVORING LIPOLYSIS & KETOGENESIS

KETOSIS
DESPITE NORMAL BLOOD SUGAR

DKA vs. EDKA: A Critical Management Guide

A concise guide for healthcare professionals comparing classic Diabetic Ketoacidosis (DKA) and Euglycemic DKA (EDKA), highlighting distinct clinical presentations and critical management variations, including the role of SGLT-2 inhibitors.

Classic Diabetic Ketoacidosis (DKA)



The Classic Triad: High Glucose, Acid, Ketones.

Defined by blood glucose well over 250 mg/dL, acidosis, and increased plasma ketones.



Caused by Severe Insulin Deficiency

An absolute or severe relative lack of insulin is the primary trigger.

Euglycemic DKA (EDKA)



The Deceptive Variant: Normal Glucose, but Still Acidotic

Presents with acidosis and ketones, but blood glucose is below 250 mg/dL.



Primarily Driven by SGLT-2 Inhibitors

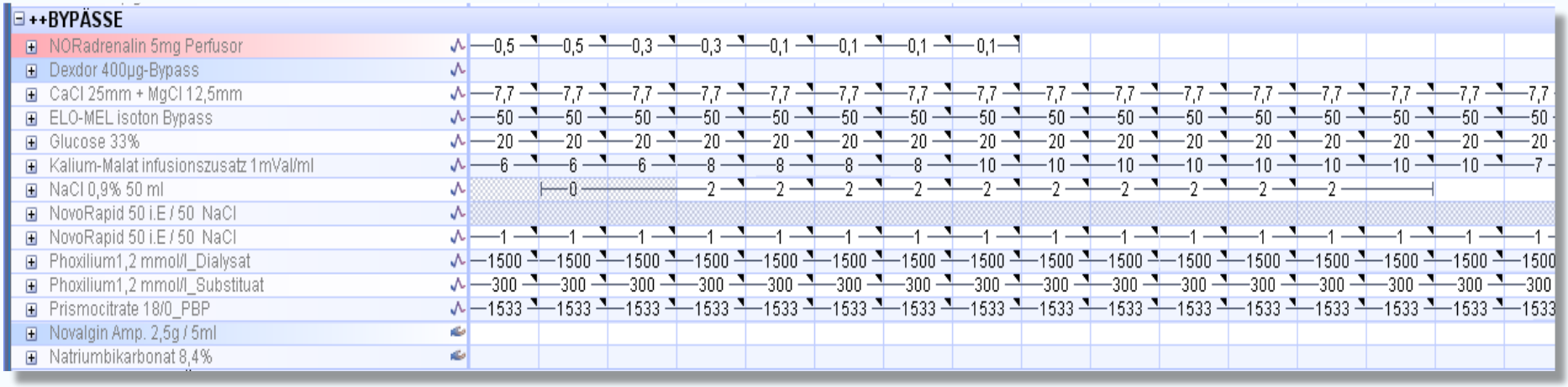
These drugs limit hyperglycemia by expelling glucose in urine, masking the crisis.

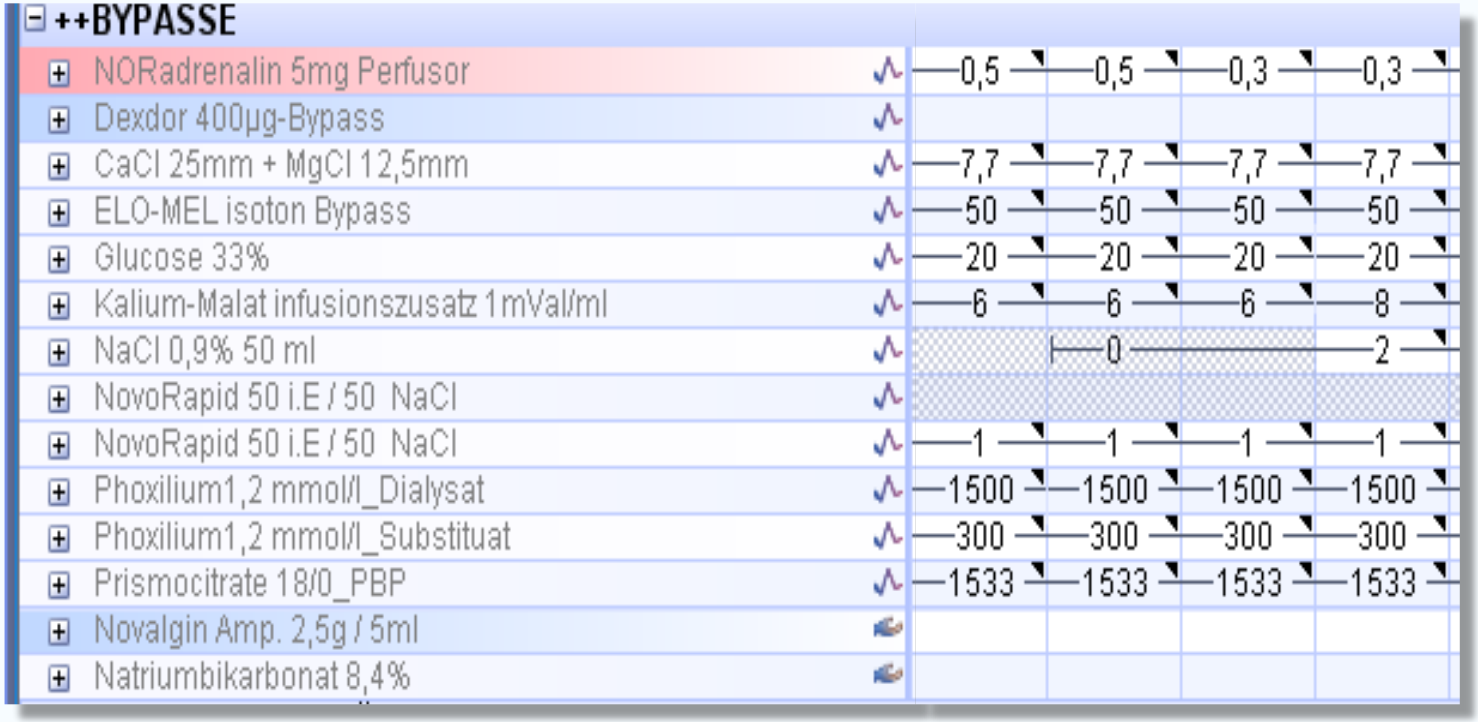
symptoms

- **tachypnea** / Kussmaul respiration
- **nausea** and **vomiting** *(ketone bodies)*
- abdominal **pain**
- unexplained **metabolic acidosis**
- dehydration, **hypovolemia**
- altered **consciousness**

therapy

- **principles** are **similar** to conventional DKA
  *(Glc administration is often required **earlier**)*
- immediately **discontinue** the SGLT2 inhibitor
- **crystalloids** *(guided by hemodynamics and volume status)*
- **continuous insulin** *(usually 0,05–0,1 IU/kg/h, until ketoacidosis resolves, not merely until glucose normalizes!)*
- early **glucose infusion** (5 - 10 - 33% glucose)
  (despite normal blood glucose)
- **K^+ substitution** *(according to serum level and renal function)*
- **frequent monitoring**
  (blood gases, glucose, electrolytes and β-hydroxybutyrate)


++BYPASSE
NORadrenalin 5mg Perfusor
0,5 0,5 0,3 0,3 0,1 0,1 0,1 0,1
Dexdor 400µg-Bypass
CaCl 25mm + MgCl 12,5mm
7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7 7,7
ELO-MEL isoton Bypass
50 50 50 50 50 50 50 50 50 50 50 50 50 50 50 50 50 50
Glucose 33%
20 20 20 20 20 20 20 20 20 20 20 20 20 20 20 20 20 20
Kalium-Malat infusionszusatz 1mVal/ml
6 6 6 8 8 8 8 10 10 10 10 10 10 10 10 7
NaCl 0,9% 50 ml
0 2 2 2 2 2 2 2 2 2 2 2
NovoRapid 50 i.E / 50 NaCl
NovoRapid 50 i.E / 50 NaCl
1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
Phoxilium1,2 mmol/l_Dialysat
1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500 1500
Phoxilium1,2 mmol/l_Substituat
300 300 300 300 300 300 300 300 300 300 300 300 300 300 300 300 300 300
Prismocitrate 18/0_PBP
1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533 1533
Novalgin Amp. 2,5g / 5ml
Natriumbikarbonat 8,4%


++BYPASSE
NORadrenalin 5mg Perfusor
0,5 0,5 0,3 0,3
Dexdor 400µg-Bypass
CaCl 25mm + MgCl 12,5mm
7,7 7,7 7,7 7,7
ELO-MEL isoton Bypass
50 50 50 50
Glucose 33%
20 20 20 20
Kalium-Malat infusionszusatz 1mVal/ml
6 6 6 8
NaCl 0,9% 50 ml
0 2
NovoRapid 50 i.E / 50 NaCl
NovoRapid 50 i.E / 50 NaCl
1 1 1 1
Phoxilium1,2 mmol/l_Dialysat
1500 1500 1500 1500
Phoxilium1,2 mmol/l_Substituat
300 300 300 300
Prismocitrate 18/0_PBP
1533 1533 1533 1533
Novalgin Amp. 2,5g / 5ml
Natriumbikarbonat 8,4%

what's new (different)?

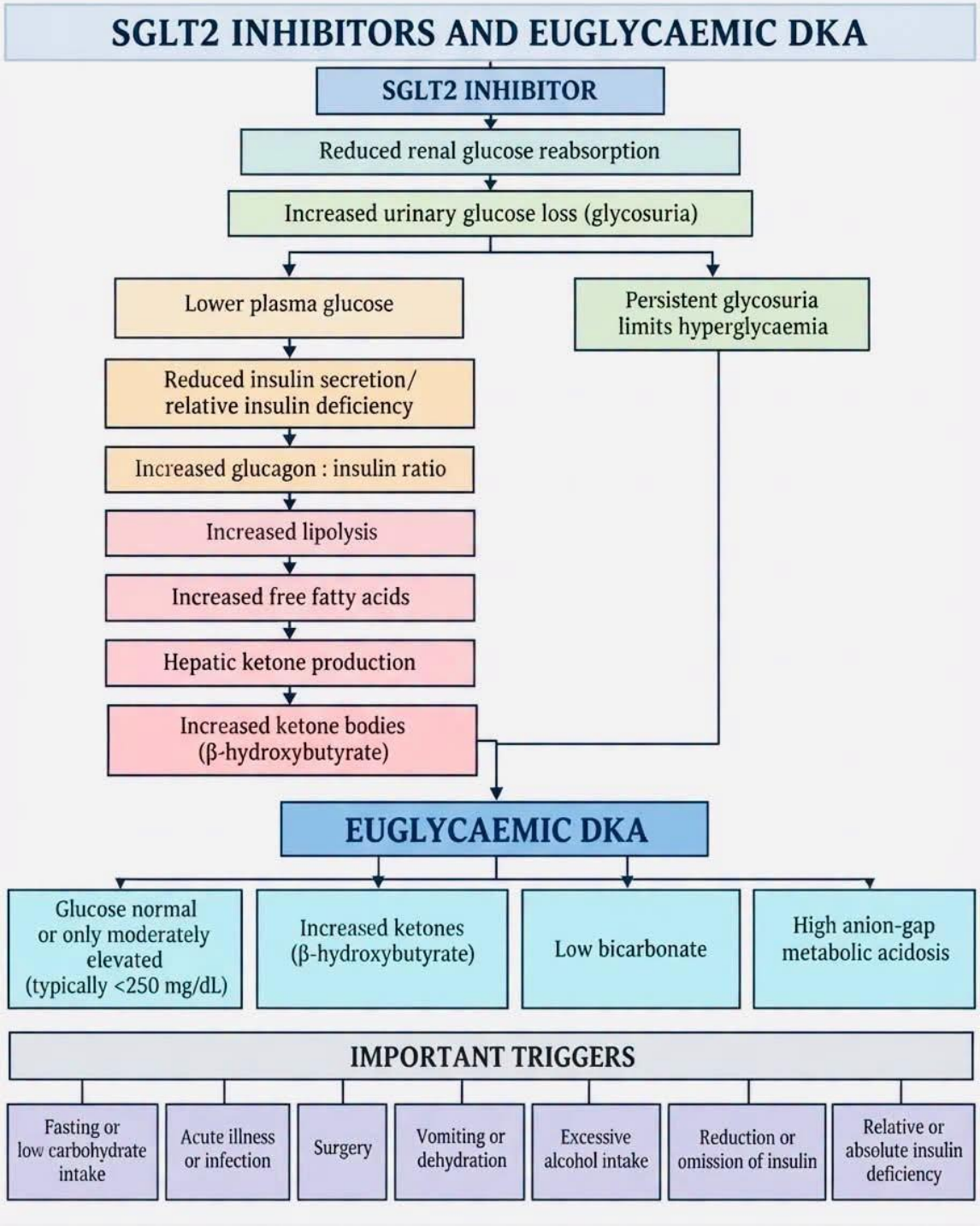
significant differences

- often **normoglycemia** or mild hyperglycemia
- **severe** acidosis *(pH < 7.0 and a **profound anion gap** are possible)*
- **glucosuria** and **ketosis** may **persist** for **days** after the last dose
- the **course** can be **prolonged** *(even when CRRT is initiated)*
- **volume depletion** is **common** because of osmotic diuresis
- normoglycemia leads to **delayed recognition**
- measure **beta-hydroxybutyrate** rather than relying on urine dipsticks *(urine ketone testing is unreliable in SGLT2i-treated patients)*

poznámka 2

SGTL2i and β-hydroxybutyrate)

< 0.6 mmol/l	**normal**
0.6 - 1.5 mmol/l	**“grey zone”** administer fluids (reduce AVP), give Glc (suppress glucagon), and monitor hourly
>1.5 mmol/l	**diagnosis of eDKA probable** initiate full DKA protocol (continous insulin + Glc + aggressive hydration); *do not wait for pH to drop*
>3.0 mmol/l	**diagnosis of eDKA highly probable** full therapy *(incl. CRRT if necessary)*


SGLT2 INHIBITORS AND EUGLYCAEMIC DKA
SGLT2 INHIBITOR
Reduced renal glucose reabsorption
Increased urinary glucose loss (glycosuria)
Lower plasma glucose
Persistent glycosuria limits hyperglycaemia
Reduced insulin secretion/ relative insulin deficiency
Increased glucagon : insulin ratio
Increased lipolysis
Increased free fatty acids
Hepatic ketone production
Increased ketone bodies (β-hydroxybutyrate)
EUGLYCAEMIC DKA
Glucose normal or only moderately elevated (typically <250 mg/dL)
Increased ketones (β-hydroxybutyrate)
Low bicarbonate
High anion-gap metabolic acidosis
IMPORTANT TRIGGERS
Fasting or low carbohydrate intake
Acute illness or infection
Surgery
Vomiting or dehydration
Excessive alcohol intake
Reduction or omission of insulin
Relative or absolute insulin deficiency

Key Differences in Management

DKA vs EDKA

DKA	EDKA (Euglycaemic DKA)
Glucose + 250 mg/dL — High > 250 mg/dL	Normal < 250 mg/dL (Normal Glucose)
Blood Glucose: High > 250 mg/dL	Normal < 250 mg/dL
Trigger Focus: Insulin Omission, Infection	SGLT2 Inhibitors, Fasting, Pregnancy
When Dextrose Started: Later (Glucose < 200-250)	Early / Immediately with Insulin
Hypoglycemia Risk: Later Risk	Early Risk
Insulin & Glucose Strategy: Insulin First, Glucose Added Later	Insulin + Glucose Started Together
Diagnostic Pitfall: High Glucose, Obvious Dx	Normal Glucose, Delayed Dx
Medication Action: No Drug to Stop	Stop SGLT2 Inhibitor

EDKA = Insulin + Dextrose EARLY to Maintain Insulin Despite Normal Glucose

	22	23	00	01	02	03	04	05	06	07	08	09	10	11	12	13	14	15	16	17
Resistance cmH2O/l/s	6	3			7	8		6	6	5	6	6	6	5	5	5	24	15	14	14
.UTGASANALYSE_																				
#PHI-A	7,31			7,22				7,24					7,2		7,2			7,23	7,25	7,26
OxInd	127			193				291							103			70	76	88
#PCO2I-A mmHg	31,7			27,7				26,9					27,9		38,3			46,6	41,2	37,8
#PO2I-A mmHg	57,1			86,9				131					65,7		82,7			55,8	60,5	70,1
#HCO3I-A mmol/l	16,9			13				13,3					12,4		14,7			--- mmol/l	17,3	16,8
#SBEI-A mmol/l	-10,2			-16,3				-15,7					-17,1		-13,2			-8,4	-9,2	-10,2
#SO2I-A %	90,2			96,3				99					91,1		94,9			--- %	89,7	93,7
#LAK-I-A mmol/l	1			1				0,8					1,1		1,4			0,9	0,9	0,9
.....																				
#PHI																				
#PCO2I mmHg																				
#PO2I																				
#HCO3I mmol/l																				
#SBEI mmol/l																				
#SO2I %																				
.																				
#CNAI mmol/l	150			148				148					150		152			155	154	155
#CKI mmol/l	3,5			3,3				3,7					3,4		3,5			4	4	3,9
#CCAI mmol/l	1,39			1,43				1,42					1,45		1,43			1,44	1,44	1,44
#CCLI mmol/l	118			119				120					122		121			120	120	121
..																				
#CTHBI g/dl	9,7			9,7				8,8					9,7		9,9			--- g/dl	9,4	9,7
#HCTI %	29,6			29,9				27					29,6		30,2			--- %	28,8	29,9
...																				
#BZI mg/dl	138			154				170					175		177			182	184	195
#LAK-I mmol/l																				

perioperative management

Perioperative anesthesia care for patients with diabetes mellitus: a review of current literature with focus on oral antidiabetic medication

Sophia Riesemann[a,b], Theresa Tenge[b] and Sebastian Roth[b]

Purpose of review
Every fifth surgical patient presents with diabetes mellitus and the use of modern oral antidiabetic agents is rapidly expanding. Therefore, comprehensive and current knowledge of benefits and risks is essential for anesthesiologists.

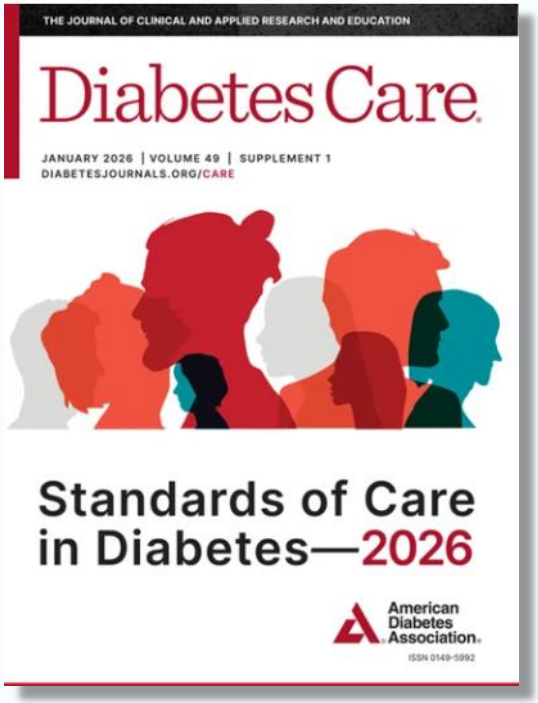
Recent findings
The 2025 international guidelines for perioperative management of patients with type 2 diabetes mellitus recommend continuation of glucagon-like peptide-1 receptor agonists in patients without an elevated risk of aspiration, temporary discontinuation of sodium-glucose-co-transporter-2 inhibitors one to four days before surgery, and do not provide specific recommendations for dipeptidyl peptidase-4 inhibitors. Emerging evidence focuses on two principal areas: the additive metabolic and cardiovascular benefits of combination therapy, and the real-world perioperative safety of these agents. Large cohort studies now demonstrate exceedingly low incidences of pulmonary aspiration and euglycemic ketoacidosis, with adverse events previously documented predominantly in case reports.

Summary
Detailed pathophysiological understanding of the mechanisms, benefits, and potential risks associated with oral antidiabetic agents remains indispensable and should foster individualized, risk-based perioperative management strategies for patients with diabetes mellitus.

Keywords
aspiration, dipeptidyl peptidase-4, glucagon-like peptide-1, ketoacidosis, sodium–glucose-co-transporter-2 inhibitors

Curr Opin Anesthesiol 2026;39:381-9

American Diabetes Association®



SGLT2 Inhibitors & Surgery

Pre-op Reminder:
STOP SGLT2 inhibitors 3–4 days before elective non-cardlac surgery

Medication	Stop ≥ Days Before Surgery
empagliiflozin	3 days
dapagliflozin	3 days
canagliflozin	3 days
ertugliflozin	4 days

! WHY? To reduce risk of euglycemic ketoacidosis, especially in:

- Fasting
- Low-carb intake
- Acute illness
- Volume depletion

Post-op Tip: RESUME only when patient is eating & stable. Monitor for signs of metabolic acidosis.

Diabetes Care. 2026;49(Suppl 1):S1-S377

when can treatment be restarted?

- **acute** illness has **resolved**
- patient is **hemodynamically stable**
- normal **oral nutrition** and **carbohydrate intake** have resumed
- **no ketonemia** or **metabolic acidosis** is present
- **volume status** normalized

in patients with heart failure or CKD,
restarting is important because
of the substantial cardiorenal benefits

SGTL2i and emergency surgery

- **monitor** closely for postoperative **ketoacidosis**
- blood **β-hydroxybutyrate** (not just **urine** ketones)
  (≥ 3.0 mmol/L strongly supports DKA)
- avoid **hypovolemia** and **hypotension**

frontiers | Frontiers in Endocrinology

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The SGLT2 inhibitor checklist: a comprehensive review of perioperative and acute phase safety management

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Stanovisko
k perioperačnímu podávání SGLT2 inhibitorů a GLP-1 receptorových agonistů u dospělých pacientů

Česká společnost anesteziologie, resuscitace a intenzivní medicíny ČLS JEP
Česká společnost intenzivní medicíny ČLS JEP

Černý V., Beneš J., Vymazal T., Bláha J., Tesařík R., Štourač P., Žižka J., Gabrhelík T.

summary I.

- **SGLT2 inhibitors** are **increasingly** used worldwide
- indications: **NIDDM**, **heart failure** and **chronic kidney disease**
- on the **ICU** they **require attention** because of:
  - the risk of **euglycemic diabetic ketoacidosis** (euDKA)
  - the risk of **volume depletion**
  - the risk of **hypotension**
  - the risk of **acute kidney injury** (AKI)

summary II.

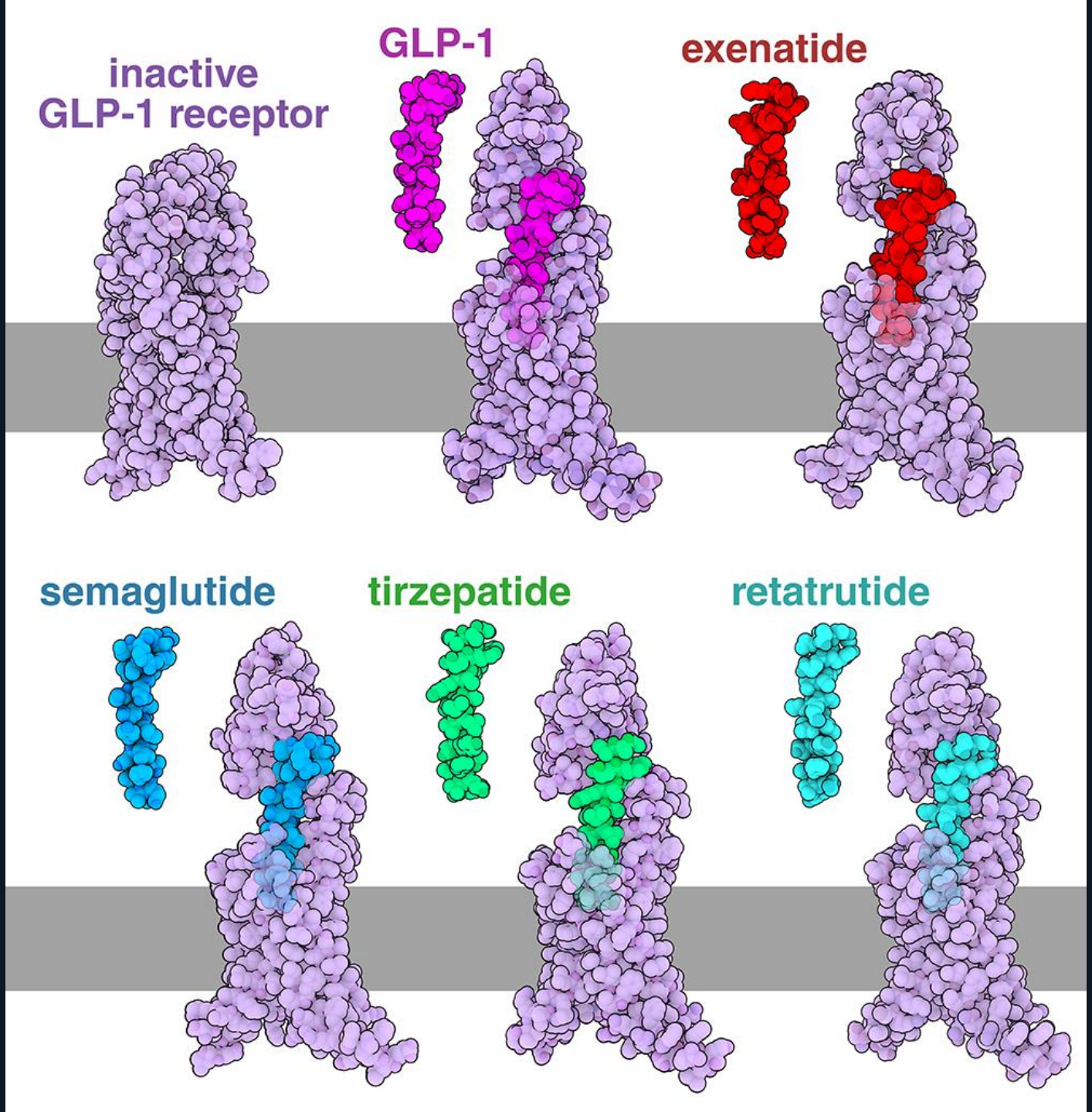
- **don't continue** SGLT2-i routinely in severe **critical illness**
- in **unexplained** metabolic **acidosis**, always **consider euDKA**
- **normal** blood glucose **does not exclude** ketoacidosis
- measure blood **β-hydroxybutyrate**, not just **urine ketones**
- **restart** after clinical recovery and restoration of **normal nutrition**

the most **important** issue for **ICU**:
a patient taking an **SGLT2i** can develop
severe ketoacidosis without significant **hyperglycemia**

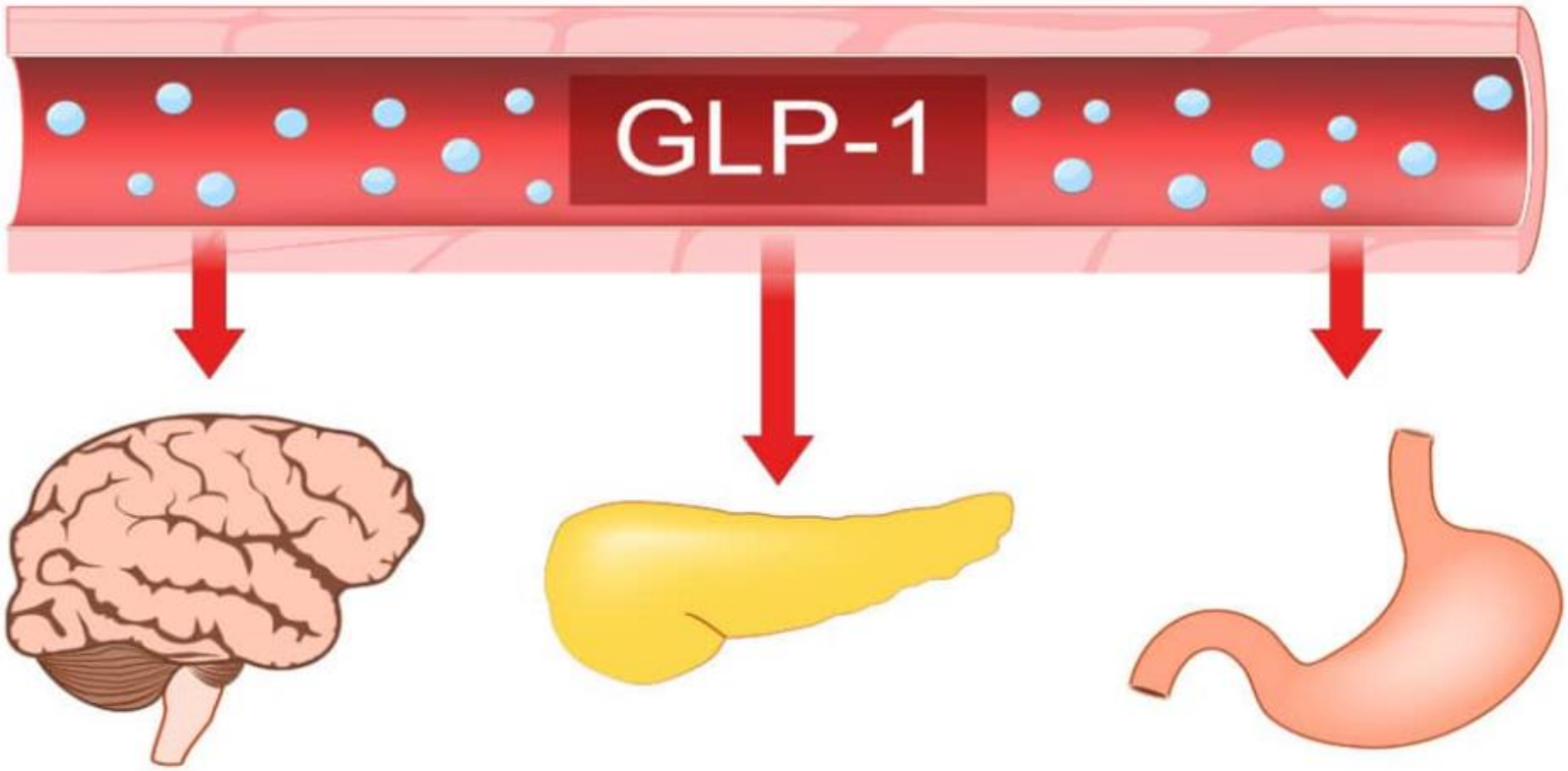
GLP-1
receptor agonist

GLP-1 receptor agonist

- **Glucagon-like peptide-1** (GLP-1) receptor agonists
- class of medications that activate the GLP-1 receptor
- reduced **blood sugar**
- reduced **appetite**
- reduced **energy intake**

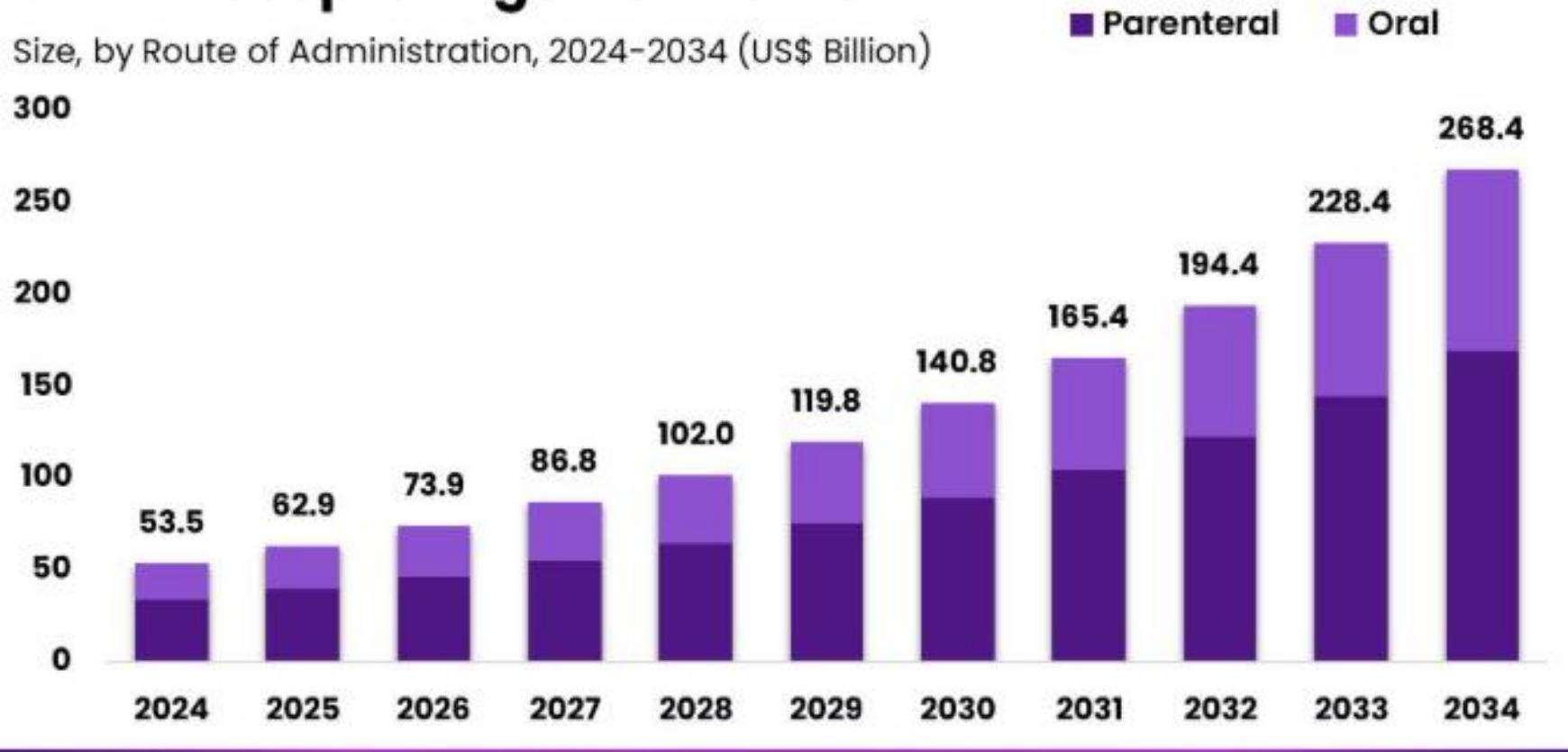

inactive
GLP-1 receptor
GLP-1
exenatide
semaglutide
tirzepatide
retatrutide

Glucagon-like peptide-1



GLP-1 Receptor Agonist Market

Size, by Route of Administration, 2024-2034 (US$ Billion)



The Market will Grow at the CAGR of **17.5%** The Forecasted Market Size for 2034 in US$ **268.4 Bn**

market.us

Přehled agonistů GLP-1 receptoru

Léčivá látka	Obchodní názvy	Podávání	Poznámka
Exenatid	Byetta	s.c., 2× denně	Krátkodobě působící
Exenatid LAR	Bydureon (BCise)	s.c., 1× týdně	Dlouhodobě působící, v řadě zemí již nedostupný
Lixisenatid	Lyxumia	s.c., 1× denně	Krátkodobě působící, mimo trh
Liraglutid	Victoza (diabetes), Saxenda (obezita)	s.c., 1× denně	Dlouhodobě působící, kardiovaskulární benefit (LEADER)
Dulaglutid	Trulicity	s.c., 1× týdně	Kardiovaskulární benefit (REWIND)
Semaglutid	Ozempic (diabetes), Wegovy (obezita)	s.c., 1× týdně	Kardiovaskulární a renální benefit
Semaglutid perorální	Rybelsus	p.o., 1× denně	První perorální GLP-1 RA
Albiglutid	Eperzan / Tanzeum	s.c., 1× týdně	Staženo z trhu

Duální agonisté (GIP/GLP-1)

Léčivá látka	Obchodní názvy	Podávání
Tirzepatid	Mounjaro (diabetes), Zepbound (obezita)	s.c., 1× týdně

Fixní kombinace s bazálním inzulinem

Přípravek	Složení
Xultophy	inzulin degludek + liraglutid
Suliqua	inzulin glargin + lixisenatid

GLP-1a on ICU

- GLP-1 RAs **delay gastric emptying**

- GLP-1 RAs can **worsen nausea**, **vomiting**, **ileus** and feeding intolerance

- ↑ **regurgitation** and **aspiration** have been reported during intubation

- interaction with **SGLT2i**: an additive **metabolic vulnerability** *(patients on both have a **higher risk of** euDKA when oral intake drops)*

- **volume** problems: vomiting, diarrhea and reduced fluid intake can cause dehydration and **prerenal AKI**

GLP-1a on ICU

- consider **rapid sequence induction** *(RSI)*
- place a **gastric tube** before intubation if feasible and sensible
- gastric **ultrasound** (POCUS) *(it is not always practical)*
- **pancreatitis** and **biliary disease** can complicate ICU management *(think of this with epigastric pain and vomiting, check lipase)*
- s**topping** the drug **does not** protect **immediately!** *(with long-acting agents e.g. weekly semaglutide, the effect persists for days to weeks after the last dose)*

Practical checklist on ICU:

1. On admission, ask about GLP-1 RAs (semaglutide, liraglutide, dulaglutide, tirzepatide, exenatide, including weight-loss products such as Wegovy, Zepbound, Saxenda), with dose, interval and last dose.
2. For airway management, assume a full stomach, consider RSI, place a gastric tube and, if possible, do gastric ultrasound.
3. Pause the drug in the ICU. Restart only with stable enteral nutrition and hemodynamics.
4. Nutrition: for intolerance, consider a prokinetic (e.g. erythromycin) and a post-pyloric tube.
5. For abdominal pain and vomiting: check lipase, biliary ultrasound, think of pancreatitis.
6. With an SGLT2 inhibitor or reduced insulin: check blood ketones and anion gap.
7. Keep monitoring glucose closely.


...děkuji Vám za pozornost