

CONGRESSO
REGIONALE
SID-AMD
ABRUZZO/MOLISE



**La gestione dell'iperglicemia nel paziente
ospedalizzato (terapia con steroidi,
insufficienza renale, epatica)**

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ASL PESCARA

CITTÀ SANT'ANGELO (PE)
16 NOVEMBRE 2024

Il sottoscritto Fabrizio Febo

ai sensi dell'art. 76 comma 4 dell'Accordo Stato-Regioni del 2 febbraio 2017

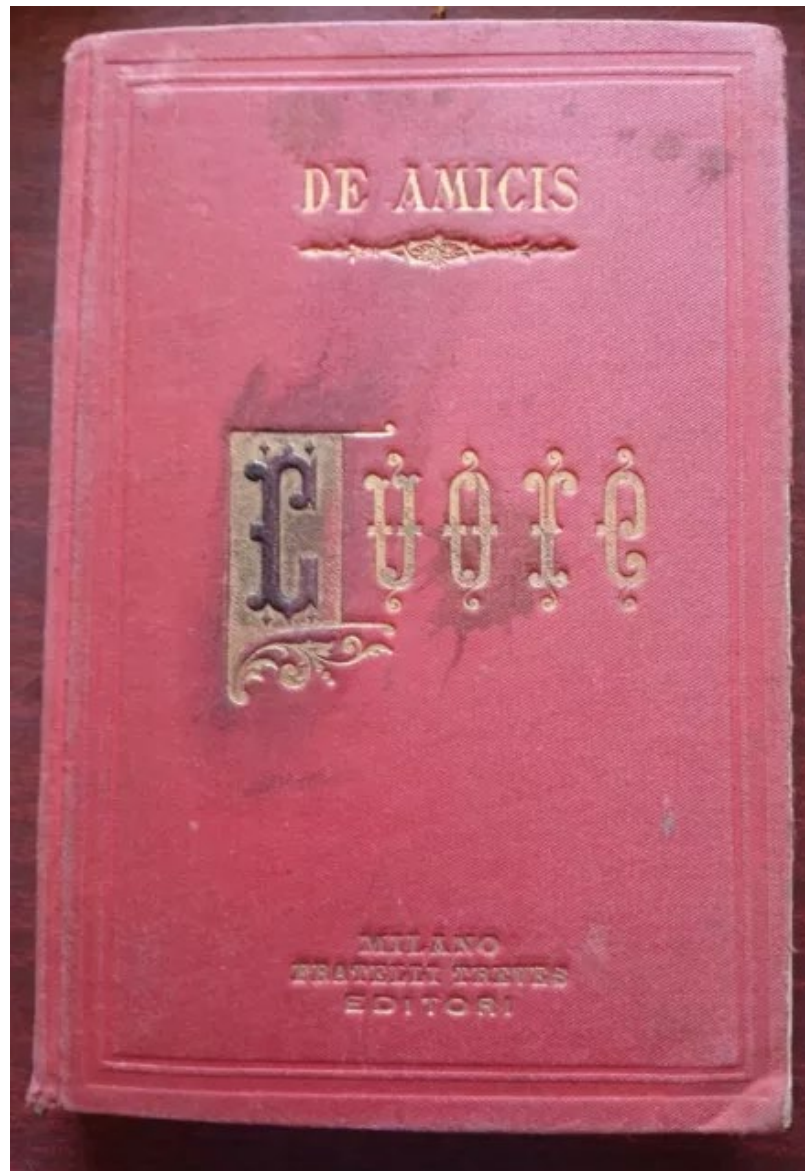
dichiara per l'evento in oggetto:

che nell'ultimo biennio ha avuto fonti di finanziamento e rapporti con i seguenti soggetti portatori di interessi commerciali in ambito sanitario (intesi come supporti per ricerche o consulenze scientifiche):

- Nono Nordisk, Astrazeneca, Eli Lilly, Bruno, SPAFarma MSD



The Hospitalized Patient with DM2



La gestione della persona con diabete ricoverata per altra patologia

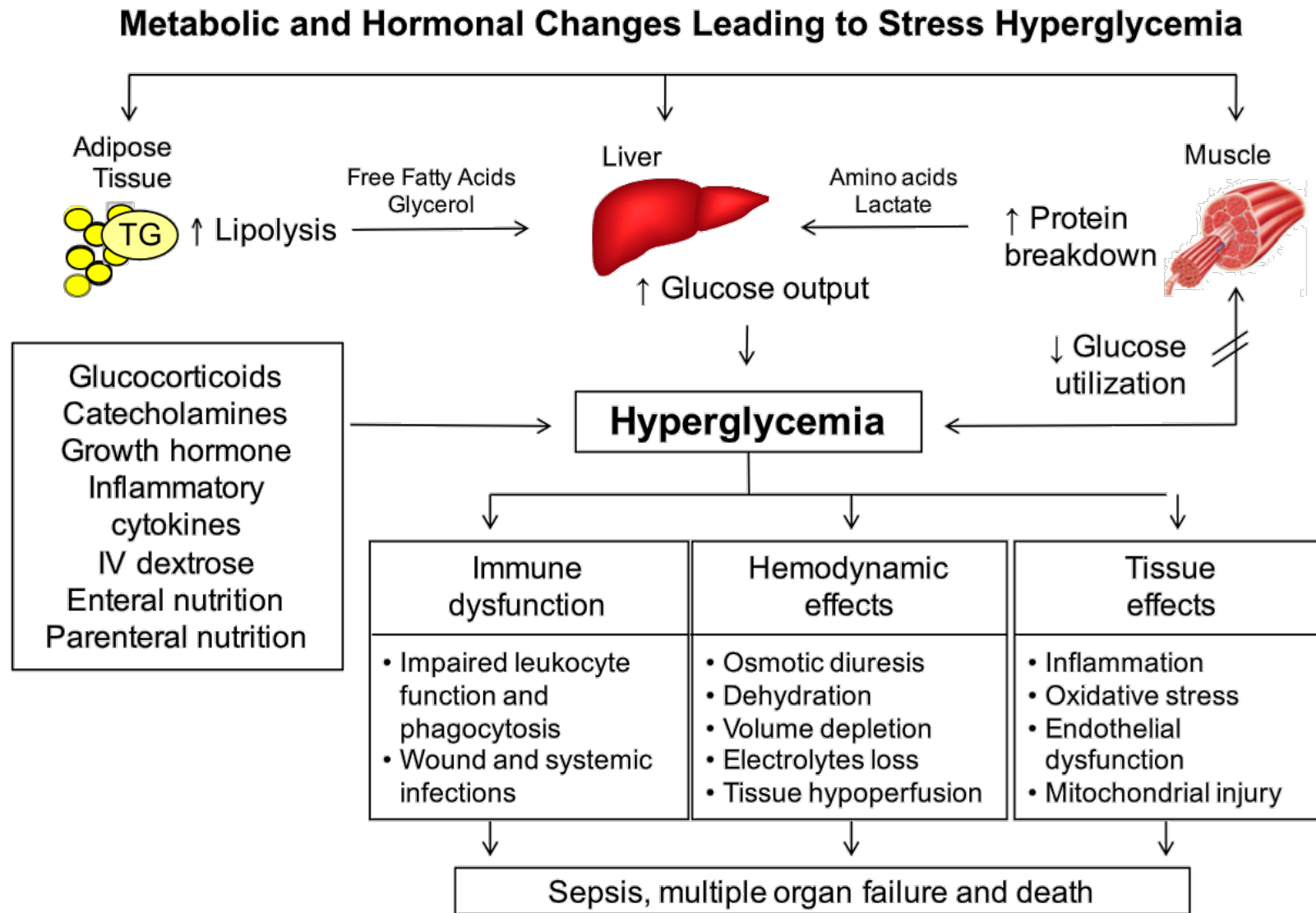
a cura di Daniela Bruttomesso e Laura Sciacca



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Pathophysiology of inpatient hyperglycemia

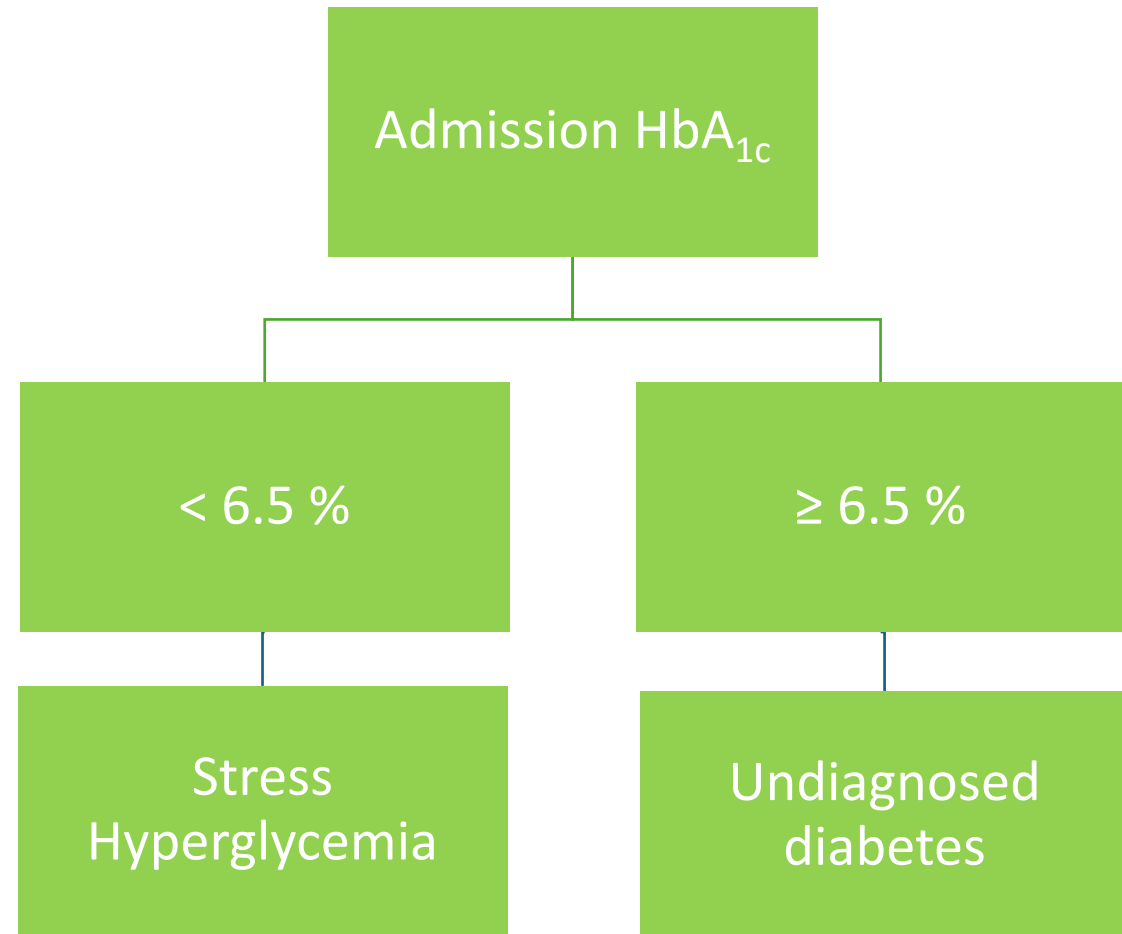


Hospital Care Delivery Standards

- 16.1** Perform an **A1C test on all patients with diabetes or hyperglycemia** (blood glucose >140 mg/dL [7.8 mmol/L]) admitted to the hospital if not performed in the prior 3 months. **B**
- 16.2** **Insulin** should be administered using validated written or computerized protocols that allow for predefined **adjustments in the insulin dosage based on glycemic fluctuations.** **C**

Considerations on Admission

- No history of diabetes:



Glycemic Targets in Hospitalized Patients

16.4 **Insulin** therapy should be **initiated** for treatment of persistent hyperglycemia starting at a **threshold ≥ 180 mg/dL** (10.0 mmol/L). Once insulin therapy is started, a target glucose range of 140–180 mg/dL (7.8– 10.0 mmol/L) is recommended for the majority of critically ill patients and noncritically ill patients. **A**

16.5 More stringent goals, such as 110–140 mg/dL (6.1–7.8 mmol/L), may be appropriate for selected patients if they can be achieved without significant hypoglycemia. **C**



Glucose-lowering Treatment in Hospitalized Patients

- 16.6** Basal insulin or a basal plus bolus correction insulin regimen is the preferred treatment for noncritically ill hospitalized patients with poor oral intake or those who are taking nothing by mouth. **A**
- 16.7** An insulin regimen with basal, prandial, and correction components is the preferred treatment for noncritically ill hospitalized patients with good nutritional intake. **A**
- 16.8** ~~Use of only a sliding scale insulin regimen in the inpatient hospital setting is strongly discouraged.~~ **A**
- 16.11** For people with type 2 diabetes hospitalized with heart failure, it is recommended that use of a sodium–glucose cotransporter 2 inhibitor be initiated or continued during hospitalization and upon discharge, if there are no contraindications and after recovery from the acute illness. **A**



LINO BANFI
in

L'ALLENATORE NEL PALLONE

GIGI
Sammarchi

ANDREA
Roncato

REGIA DI SERGIO MARTINO

EDU

5 - 5 - 5

Materiali e metodi

Criteri di inclusione:

- Pazienti di età ≥ 75 anni;
- Ricovero per riacutizzazione di scompenso cardiaco cronico

Criteri di esclusione:

- Demenza severa;
- Patologia oncologica terminale;
- Impossibilità alla VMG

PARAMETRI DEMOGRAFICI
Età
Sesso

PARAMETRI LABORATORISTICI		
AZOTEMIA	CREATININA	eGFR (C&G)

FATTORI CLINICO-ANAMNESTICI
Tipo di ICC
Frazione di eiezione (%)
Diabete mellito tipo 2
Tipo di Glifozina (Dapaglifozin, Empaglifozin)

VALUTAZIONE MULTIDIMENSIONALE GERIATRICA		
Residency status	Fragilità (criteri di Fried)	Tono dell'umore (GDS)
Social status	Stato funzionale (ADL e IADL)	Stato nutrizionale (MNA)
Numero di caregiver	Rischio di caduta (≥ 1 nei 6 mesi antecedenti)	Comorbidità (CIRS-C)
Numero di assunzioni farmacologiche		Stato cognitivo (Moca)

Risultati

Laboratorio

	T0	T1	P
CREATININA (M ± SD)	1,44 ± 0,41	1,36 ± 0,24	< 0,0001
AZOTEMIA (M ± SD)	90,93 ± 38,89	88,65 ± 0,24	0,03
eGFR (M ± SD)	36,61 ± 27,57	39,20 ± 29,69	0,02

VMDG

	T0	T1	P
NUMERO DI "CARGIVER" (M ± SD)	1,20 ± 0,79	1,17 ± 0,12	
NUMERO ASSUNZIONE FARMACI (M ± SD)	8,53 ± 4,35	6,87 ± 1,14	< 0,0001
CRITERI DI FRIED (M ± SD)	2,88 ± 1,84	2,66 ± 0,47	
ADL (FUNZ. CONSERV.) (M ± SD)	3,30 ± 0,70	3,66 ± 0,94	0,01
IADL (FUNZ. CONSERV.) (M ± SD)	2,51 ± 1,70	2,58 ± 3,82	
GDS (M ± SD)	4,38 ± 1,41	4,23 ± 0,16	
MNA (M ± SD)	20,16 ± 5,08	20,75 ± 1,23	
CIRS-c (M ± SD)	2,82 ± 1,49	2,82 ± 0,58	
MOCA (M ± SD)	25,69 ± 0,92	27 ± 0,70	0,0145
MPI (M ± SD)	0,53 ± 0,16	0,49 ± 0,10	0,0008



Management of Hyperglycemia in Hospitalized Adult Patients in Non-Critical Care Settings: An Endocrine Society Clinical Practice Guideline

Initial Therapy:

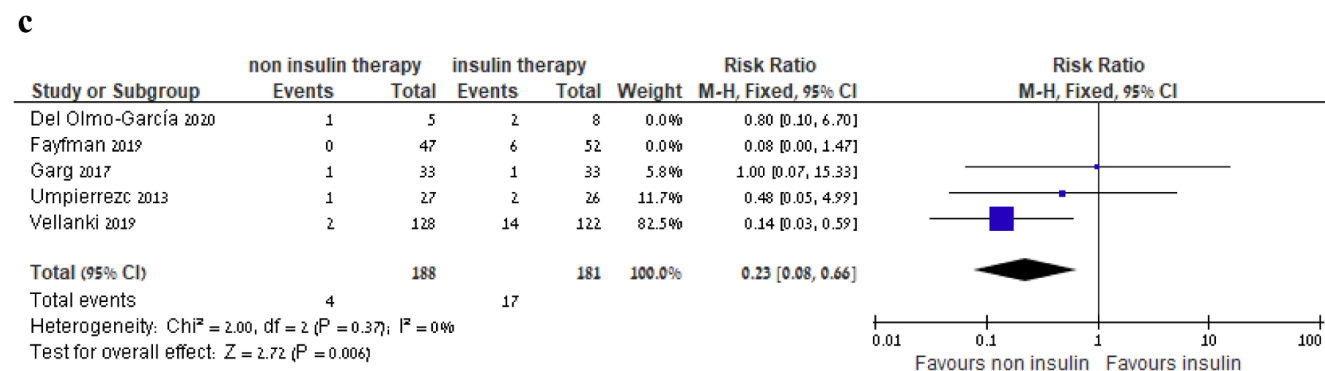
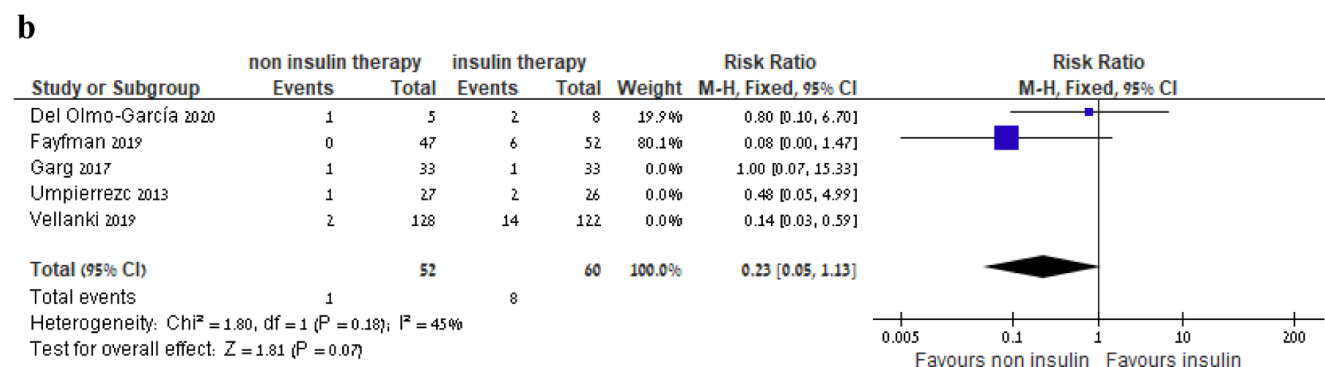
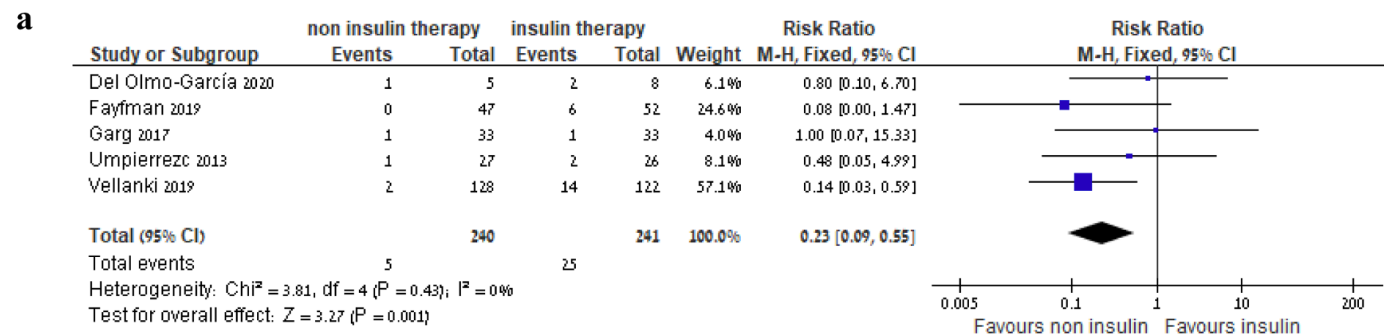
Non-insulin therapies (e.g., DPP-4 inhibitors) may be considered only in patients with well-controlled type 2 diabetes and mild hyperglycemia.

Use of DPP-4 Inhibitors:

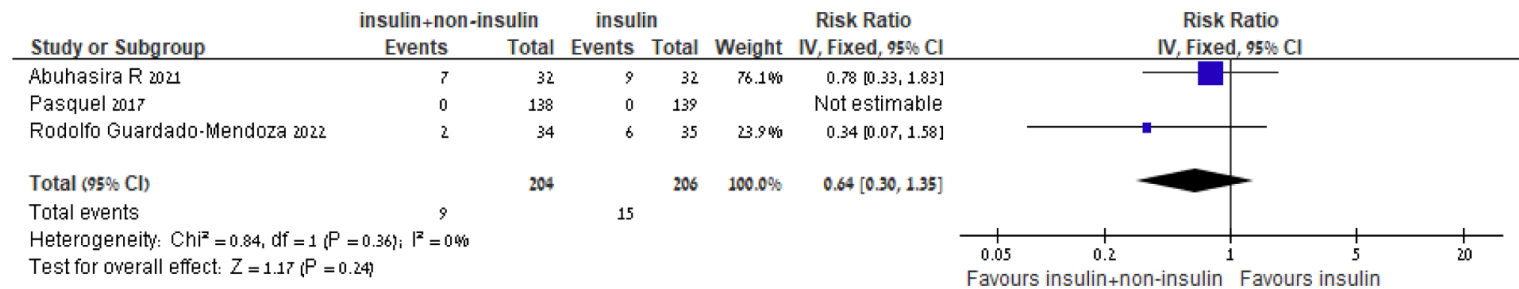
- Recommended for patients with type 2 diabetes and mild hyperglycemia, as an adjunct to correctional insulin therapy.

Korytkowski MT, et al. (2022). 107(8):2101–2128. doi:10.1210/clinem/dgac278

Hypoglycemic events, insulin vs non-insulin therapy.



30-day mortality, insulin vs insulin + non- insulin



Continuous Glucose Monitoring

16.9 In people with diabetes using a personal continuous glucose monitoring (CGM) device, the use of **CGM should be continued during hospitalization** if clinically appropriate, with **confirmatory point-of-care (POC) glucose measurements** for insulin dosing decisions and hypoglycemia assessment, if resources and training are available, and according to an institutional protocol. B

16.10 For people with diabetes using an automated insulin delivery (AID) system along with CGM, **the use of AID and CGM should be continued** during hospitalization if clinically appropriate, with confirmatory POC blood glucose measurements for insulin dosing decisions and hypoglycemia assessment, if resources and training are available, and according to an institutional protocol. C

Consensus Considerations and Good Practice Points for Use of Continuous Glucose Monitoring Systems in Hospital Settings

<https://doi.org/10.2337/dci24-0073>

Table 4—Summary of performance characteristics in published reports on the use of CGM systems in hospitalized patients

CGM system	Comparator method; sample type	Population(s) evaluated (n)	Accuracy (n paired measurements)	Reference no.
FreeStyle Libre Pro	Nova StatStrip and Roche Accu-Chek Inform II POCT meters; capillary specimens measured within 5 min	Non-ICU with type 2 diabetes (97)	EMGD 12.8 mg/dL (95% CI 8.3–17.2), Clarke EGA 98.8% in zones A and B (1,829)	18
Dexcom G6	No details provided on specific meter; POC capillary glucose	ICU (11)	MARD 12.6%, Clarke EGA 98.2% in zones A and B (493)	22
Dexcom G6	Nova StatStrip, Roche Accu-Chek Inform II, Abbott Precision Xceed Pro POCT meters; capillary specimens measured within 5 min	Non-ICU with type 1 and type 2 diabetes (218)	MARD 12.8%, Clarke EGA 98.7% in zones A and B (4,067)	20
Dexcom G6	No details on method provided; laboratory serum glucose within 5 min	Pediatric ICU, following TPIAT (25)	EMGD 14.7 mg/dL, MARD 13.4%, Clarke EGA 100% in zones A and B (183)	60
FreeStyle Libre	Blood glucose method details not provided	COVID-19 ICU (17)	Consensus EGA 97.1 in zones A and B Clarke EGA 97.7% in zones A and B	61
Dexcom G6	Roche Accu-Chek Inform II POCT meter, Beckman Coulter DxC 8000; lab glucose measurement within 5 min	COVID-19 ICU, non-ICU with type 1 diabetes or steroid-induced hyperglycemia (28)	MARD, non-ICU, 14%; MARD, ICU, 12.1%; MARD, lab, 10.9%; MARD, POCT, 13.9%; SEG, non-ICU, 98.7%; SEG, ICU, 99.7%; SEG, lab, 99.5%	52
FreeStyle Libre	GlucoDr AGM-400 POC meter, Radiometer ABL800 blood gas analyzer, Beckman Coulter AU640 Chemistry Analyzer; serum glucose measured within 15 min	Pediatric ICU (16)	EMGD, lab, 13.9 mg/dL; EMGD, blood gas, 31.6 mg/dL; EMGD, POCT, 25.2 mg/dL; MARD, lab, 19%; MARD, blood gas, 28.3%; MARD, POCT, 25.2%; SEG, lab, 95.5%; SEG, blood gas, 92%; SEG, POCT, 94.7%	62
FreeStyle Libre Pro	Vitros 5600; serum glucose measured within 15 min	Pediatric HSCT (29)	EMGD 24.4 mg/dL, MARD 20%, Clarke EGA 99% in zones A and B (893)	63

Here, accuracy is reported including the following performance measures: estimated mean glucose difference (EMGD), mean absolute relative difference (MARD), point accuracy as percentages of measurements with acceptability criteria (15%, 15 mg/dL; 20%, 20 mg/dL; 30%, 30 mg/dL), and consensus error grid analysis (EGA) showing the total percentage in clinically accurate zones. Surveillance error grid (SEG) data are summarized as a combination of the percentages of measurements for risk zones: 0 (none), 1 (slight, lower), and 2 (slight, higher). HSCT, hematopoietic stem cell transplant; lab, laboratory; TPIAT, total pancreatectomy with islet autotransplantation.

BREAKING
NEWS



To reduce surgical risk in people with diabetes, the following approach may be considered:

- A **preoperative** risk assessment should be performed for people with diabetes who are **at high risk for ischemic heart disease** and those with **autonomic neuropathy or renal failure**.
- The A1C target for elective surgeries **should be <8%** (63.9 mmol/L) whenever possible.
- The **target** range for blood glucose in the perioperative period should be **100–180 mg/dL (5.6–10.0 mmol/L) within 4 h of the surgery**.
- Metformin should be **held on the day of surgery**.



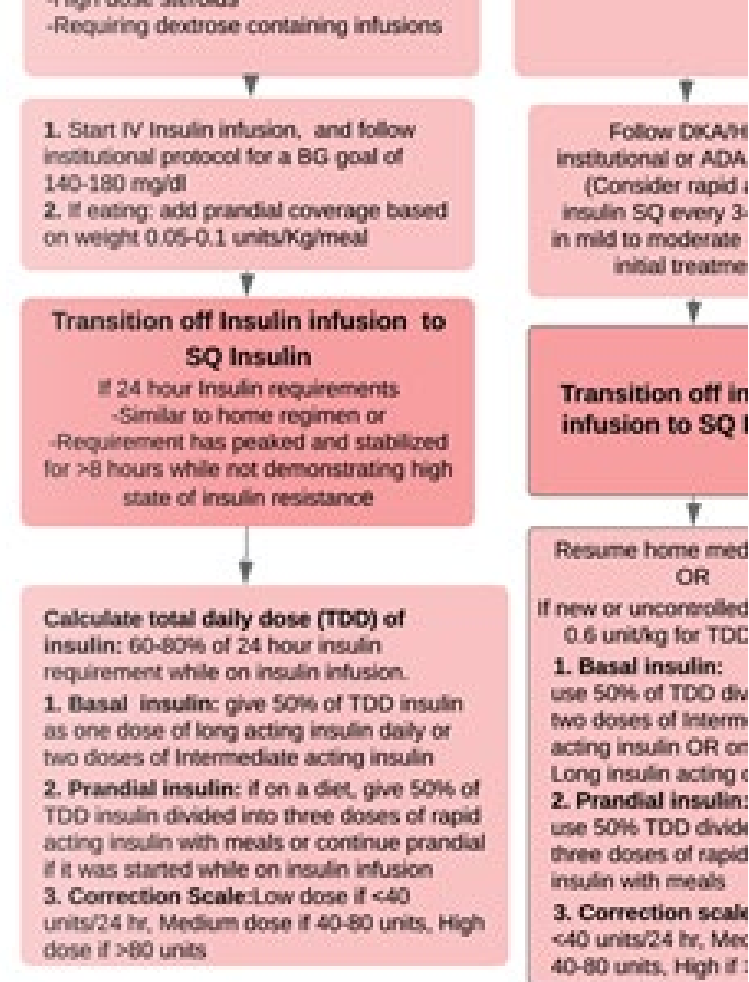
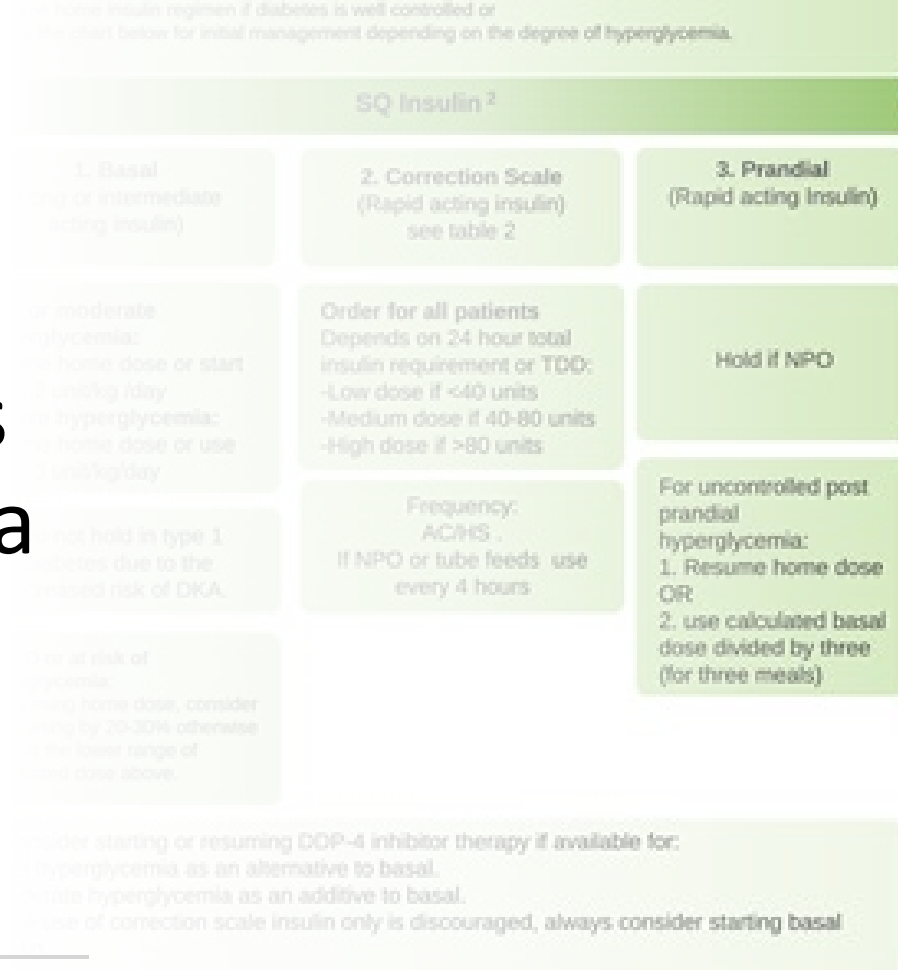
To reduce surgical risk in people with diabetes the following approach may be considered:

- **SGLT2** inhibitors must be **discontinued 3–4 days** before surgery.
- **Hold any other oral glucose-lowering agents the morning** of surgery or procedure and **give** half of NPH dose or **75–80% doses of long-acting analog** or insulin pump basal insulin based on the type of diabetes and clinical judgment.
- **Monitor** blood glucose at least **every 2–4 h** while the individual takes nothing by mouth and dose with short- or rapid-acting insulin as needed.
- There are no data on the use and/or influence of glucagon-like peptide 1 receptor agonists or ultra-long-acting insulin analogs on glycemia in perioperative care.

DOTTORE ALLORA QUANTA INSULINA GLI FACCIAMO?



Inpatient Diabetes and Hyperglycemia Management Protocol in the COVID-19 Era



Maintenance

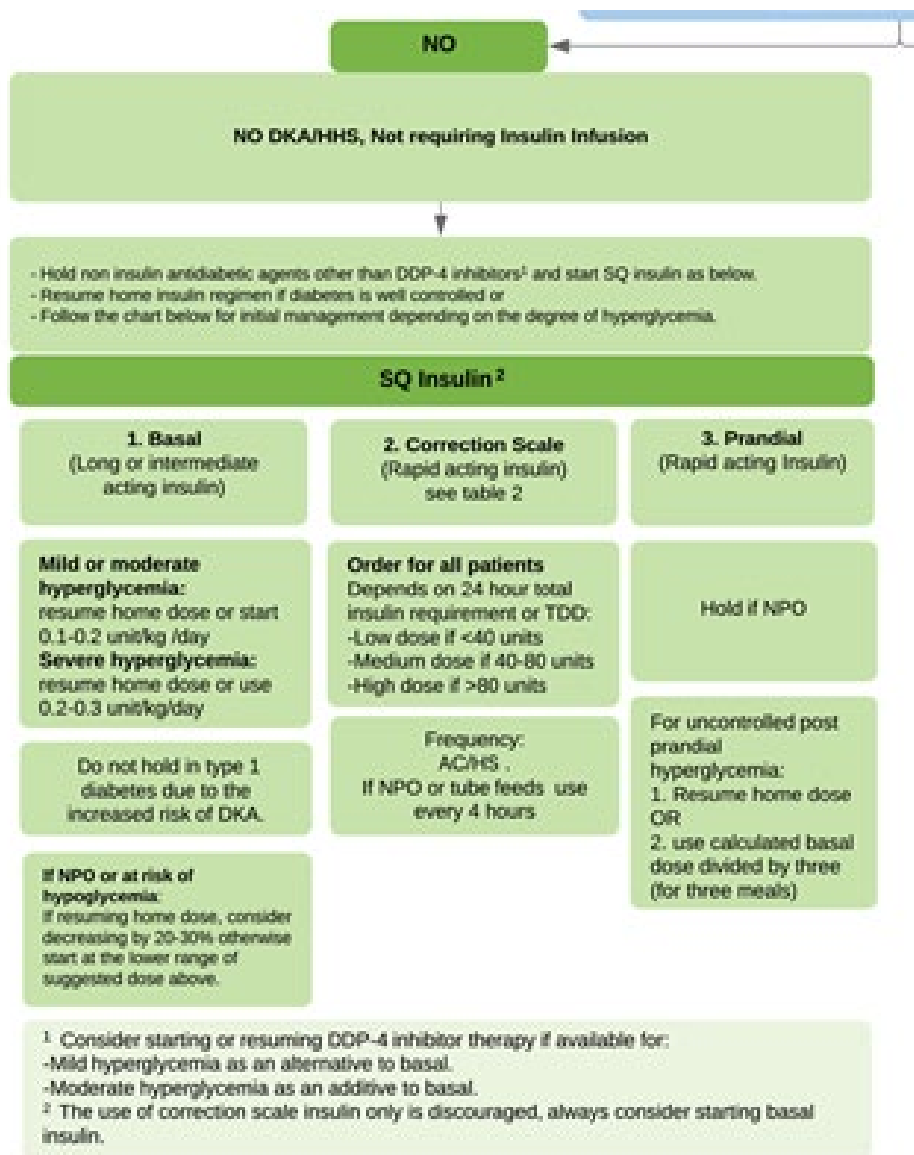
For non-critically ill patients, the goal of blood glucose should be BG 140-180 mg/dL (pre-meal <140 mg/dL and random <180 mg/dL) while avoiding hypoglycemia. The plan needs to be reviewed at least daily.

Uncontrolled fasting BG

Reflects basal insulin. If fasting BG 100-140 mg/dl and no hypoglycemia: no change. If fasting BG 141-180 mg/dl and no hypoglycemia: increase basal insulin by 10%. If fasting BG >181 mg/dl and no hypoglycemia: increase basal insulin by 20%. If fasting BG 70 - 99 mg/dl and no hypoglycemia: decrease basal insulin by 10%. If hypoglycemia (BG <70 mg/dL): decrease basal insulin by 20%.

Uncontrolled post prandial BG

Reflects prandial insulin. Start prandial and maintain correction. If pre-lunch not at goal: adjust breakfast bolus. If pre-dinner not at goal: adjust dinner bolus. If bedtime not at goal: adjust dinner bolus. If BG 100-180 mg/dl and no hypoglycemia: no change. If BG 181-220 mg/dl: increase prandial insulin by 1-2 units. If BG >221 mg/dl: increase prandial insulin by 2-3 units. If BG 70-99 mg/dl: decrease prandial insulin by 1-2 units. If hypoglycemia (BG <70 mg/dL): decrease prandial insulin by 20%.



TDD for insulin	Patient group
0.4 unit/kg body weight	- Normal BMI if admission BG 140–200 mg/dl and A1c is controlled
0.5 unit/kg body weight	- Normal BMI if admission BG >200 mg/dl and/or A1c is not controlled - BMI 25–30
0.2–0.3 unit/kg body weight	Conditions with high risk of hypoglycemia: - Age >70 - Liver disease (AKI or CKD especially on HD) - Liver disease - Underweight
0.6 unit/kg body weight	Conditions associated with Insulin resistance: - BMI>30 - Glucocorticosteroids therapy.

Transition from the Hospital Setting to the Ambulatory Setting

16.12 There should be a structured discharge plan tailored to the individual patient with diabetes. **B**

Diabetes Care December 2023, Vol.47, S1-S4. doi:<https://doi.org/10.2337/dc24-SINT>

Discharge **

Look at A1C and current hospital regimen compared to the home regimen.

-Provide updated antidiabetic agents instructions with refills and ensure close follow up after discharge.

-Make sure there are no contraindications before resuming home medications

A1c <7%

Restart outpatient treatment regimen

A1c 7-9%

Restart outpatient non insulin antidiabetic agents and
add once daily basal at 50% of hospital dose

A1c >9%

Start basal-bolus at same hospital dose .

Alternative: Restart outpatient non insulin anti-diabetic agent
and add on once daily basal at 80% of hospital dose.

It is recommended that the following areas of knowledge be reviewed and addressed prior to hospital discharge:

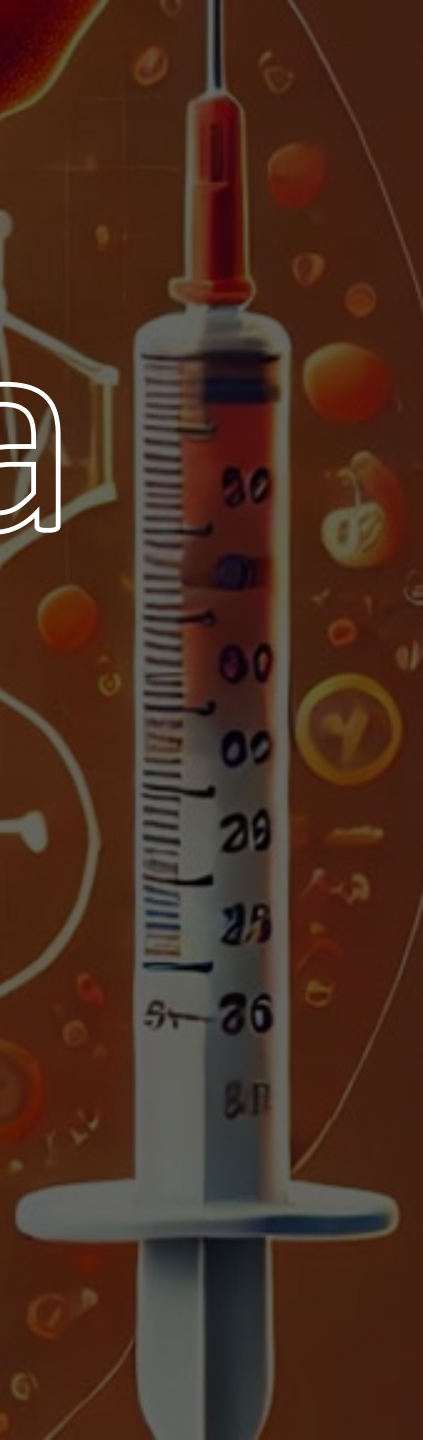
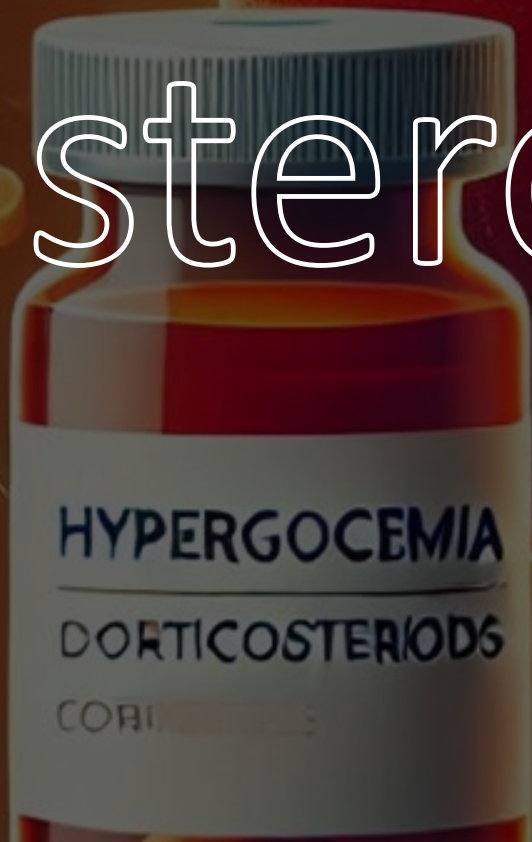
- **Identification of the health care provider** who will provide diabetes care after discharge.
- Level of **understanding** related to the diabetes diagnosis, self-monitoring of blood glucose, home blood glucose goals, and when to call the provider.
- Definition, recognition, treatment, and **prevention of hyperglycemia and hypoglycemia**.
- Information on making **healthy food choices** at home and referral to an outpatient RD/RDN to guide individualization of meal plan, if needed.
- If relevant, **when and how** to take blood glucose–lowering medications, including insulin administration.
- **Sick-day management**.
- Proper use and disposal of **needles and syringes**.

COME TI SENTI QUANDO SEI DI GUARDIA



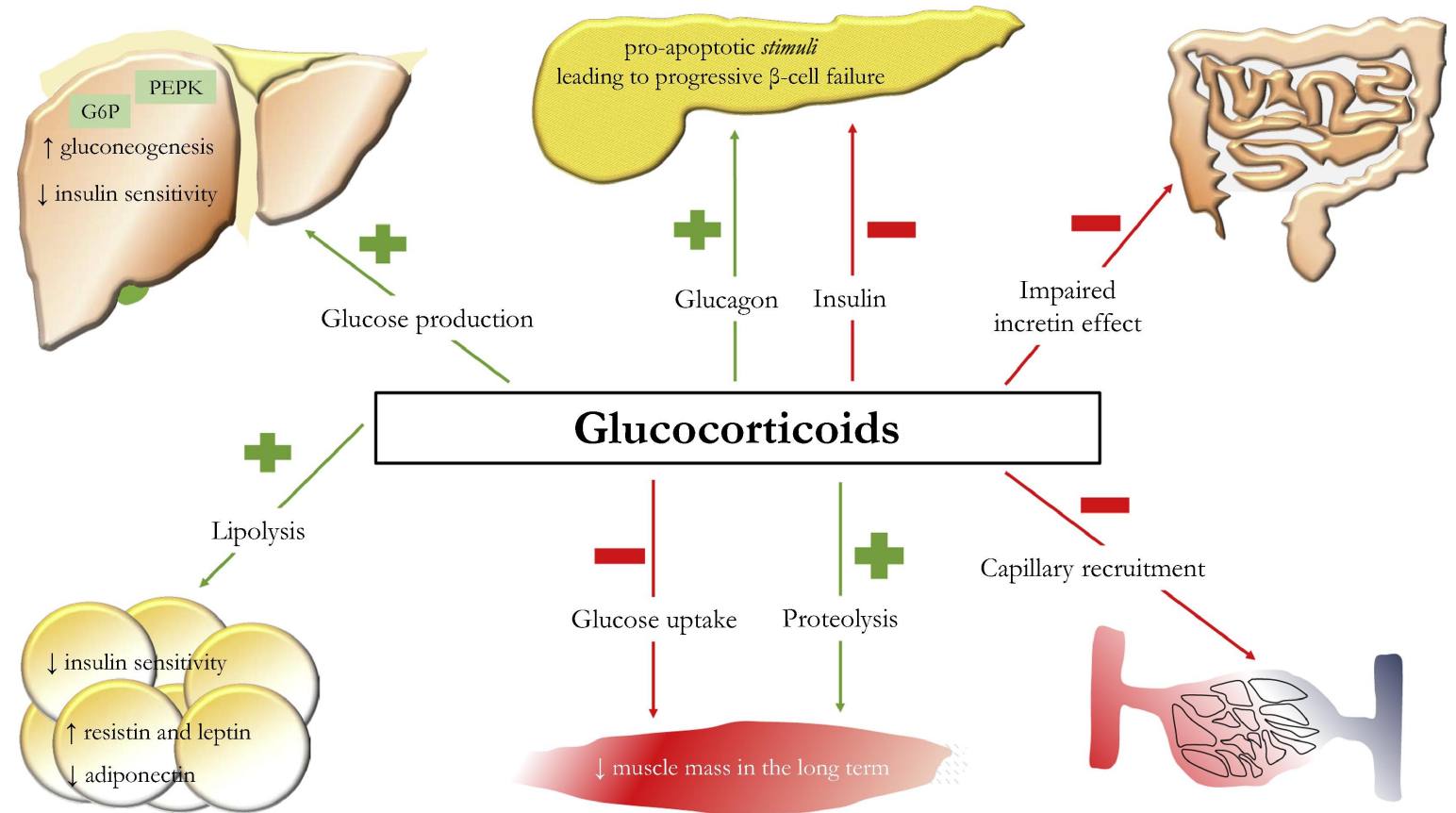
E IN TERAPIA TROVI SCRITTA INSULINA ACTRAPID

Iperglicemia da steroidi

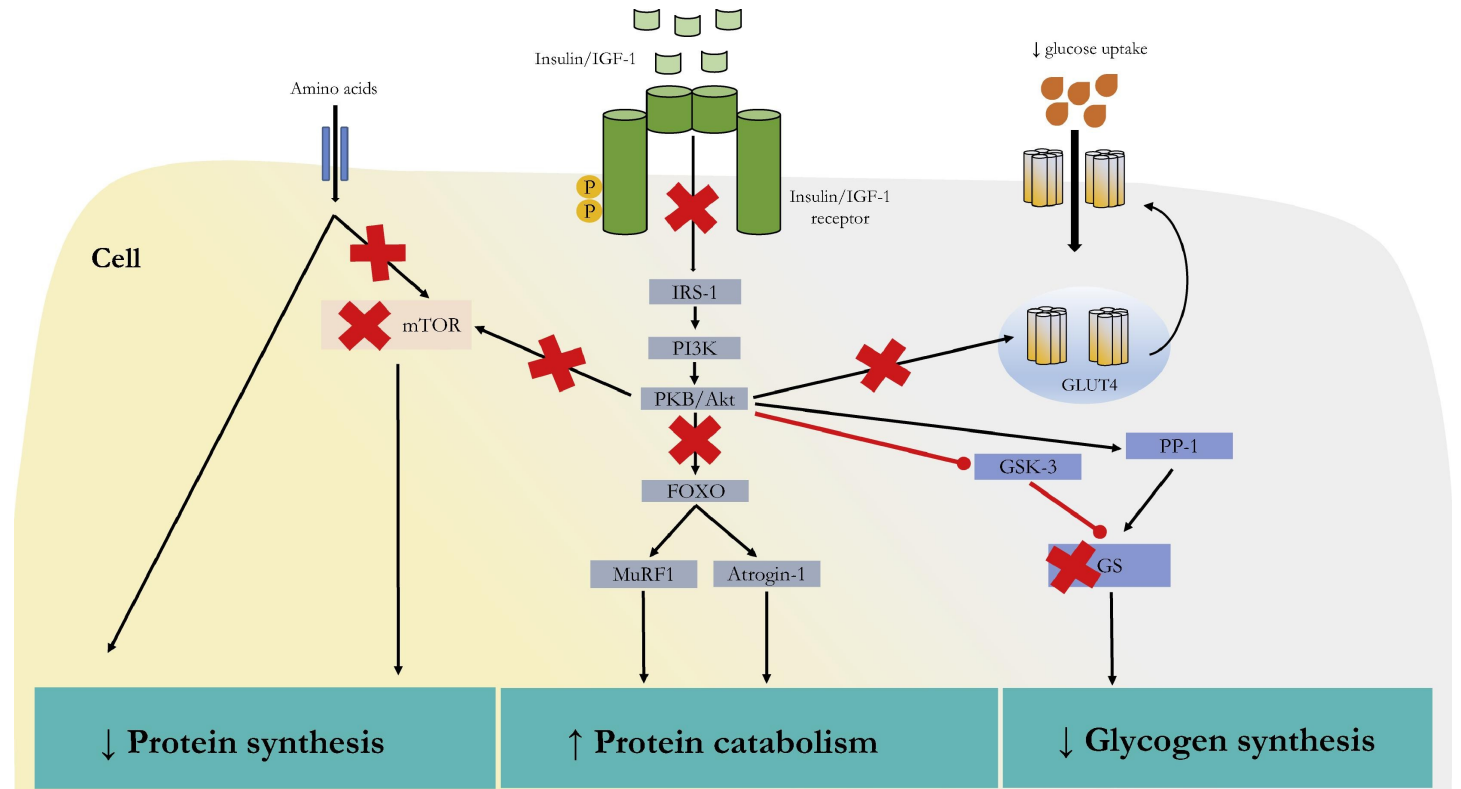


Drug Induced Diabetes Mellitus	Mechanism	Incidence of Hyperglycemia/Diabetes (%)	Reversibility
Androgen Deprivation therapy	Insulin resistance	20–30%	Yes
Somatostatin Analogues	Inhibition of insulin secretion	30%	Yes
Glucocorticoids	Decreased insulin secretion and increased gluconeogenesis	65%	Yes
mTOR inhibitors/Immunosuppressive Agents	Decreased insulin secretion and action	10–50%	Yes
Tyrosine Kinase Inhibitors	Insulin resistance	20–36%	Yes
Diuretics	Decreased insulin secretion and sensitivity	11%	Yes
Statins	Decreased insulin secretion and action	9–12%	Yes
Antipsychotics	Decreased insulin secretion and action due to antagonism at peripheral Muscarinic receptors.	22% [22]	Yes
Anti Retroviral therapy	Lipodystrophy, insulin resistance and mitochondrial dysfunction	0.5%	No
Interferon α	Immunological beta cell destruction	0.34%	No

Mechanisms of glucocorticoid-induced hyperglycemia



Molecular pathways of glucocorticoid effects on glycemic and protein metabolism.



Risk factors for steroid-induced diabetes mellitus

Dosage and type of the drug

Duration of treatment

Posology (e.g. continuous vs. bolus treatment)

Age ≥ 65 years old

Male sex

BMI^{*} > 25 kg/m²

African American ethnic group

eGFR[°] < 40 mL/min/1.73 m²

HbA1c[§] $\geq 6.0\%$

History of gestational diabetes

Family history of diabetes mellitus

Concomitant use of mycophenolate mofetil and calcineurin inhibitors

Previous history of IFG[§] and/or IGT[¶]

*BMI: body mass index. °eGFR: estimated glomerular filtration rate. § HbA1c: glycated hemoglobin. § IFG: impaired fasting glucose. ¶ IGT: impaired glucose tolerance.

Half-life of different glucocorticoids



Drug	Half-life (h)
<u>Short-acting</u>	
Cortisone	8–12
Hydrocortisone	
<u>Intermediate-acting</u>	
Prednisolone	12–16
Prednisone	
Methylprednisolone	
Deflazacort	12–24
Fludrocortisone	
Triamcinolone	
<u>Long-acting</u>	
Betamethasone	20–36
Dexamethasone	

Insulin Management Protocols for Steroid Use



Basal-Bolus Regimen: Starting Dose: 0.3–0.6 U/kg/day, adjusted per patient needs.



Prandial Insulin: Tailor to meals and peak steroid effect, e.g., higher doses in the afternoon for intermediate steroids.



Alternative Approaches: Basal Insulin Alone: For high-dose steroids, administer in the morning to cover peaks.



Weight-Based Adjustments: Modify prandial doses based on patient weight and BG readings.



Monitoring and Adjustments: Frequent BG Monitoring: Essential within the first 48 hours of steroid initiation.



Post-Discharge: Create a structured plan for outpatient follow-up, especially for patients on high-dose or long-term steroids.

An anatomical illustration of a kidney, viewed from the front, with a glowing, reddish-orange collecting system. The background is a dark, textured brown with faint, light-colored outlines of the renal vasculature. Various medical icons are scattered throughout, including a test tube with red liquid, a pulse line, and a molecular structure.

Iperglicemia nella insufficienza renale

CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)				Albuminuria categories		
				Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–299 mg/g 3–29 mg/mmol	≥300 mg/g ≥30 mg/mmol
GFR categories (mL/min/1.73 m ²) Description and range	G1	Normal or high	≥90	Screen 1	Treat 1	Treat and refer 3
	G2	Mildly decreased	60–89	Screen 1	Treat 1	Treat and refer 3
	G3a	Mildly to moderately decreased	45–59	Treat 1	Treat 2	Treat and refer 3
	G3b	Moderately to severely decreased	30–44	Treat 2	Treat and refer 3	Treat and refer 3
	G4	Severely decreased	15–29	Treat and refer* 3	Treat and refer* 3	Treat and refer 4+
	G5	Kidney failure	<15	Treat and refer 4+	Treat and refer 4+	Treat and refer 4+

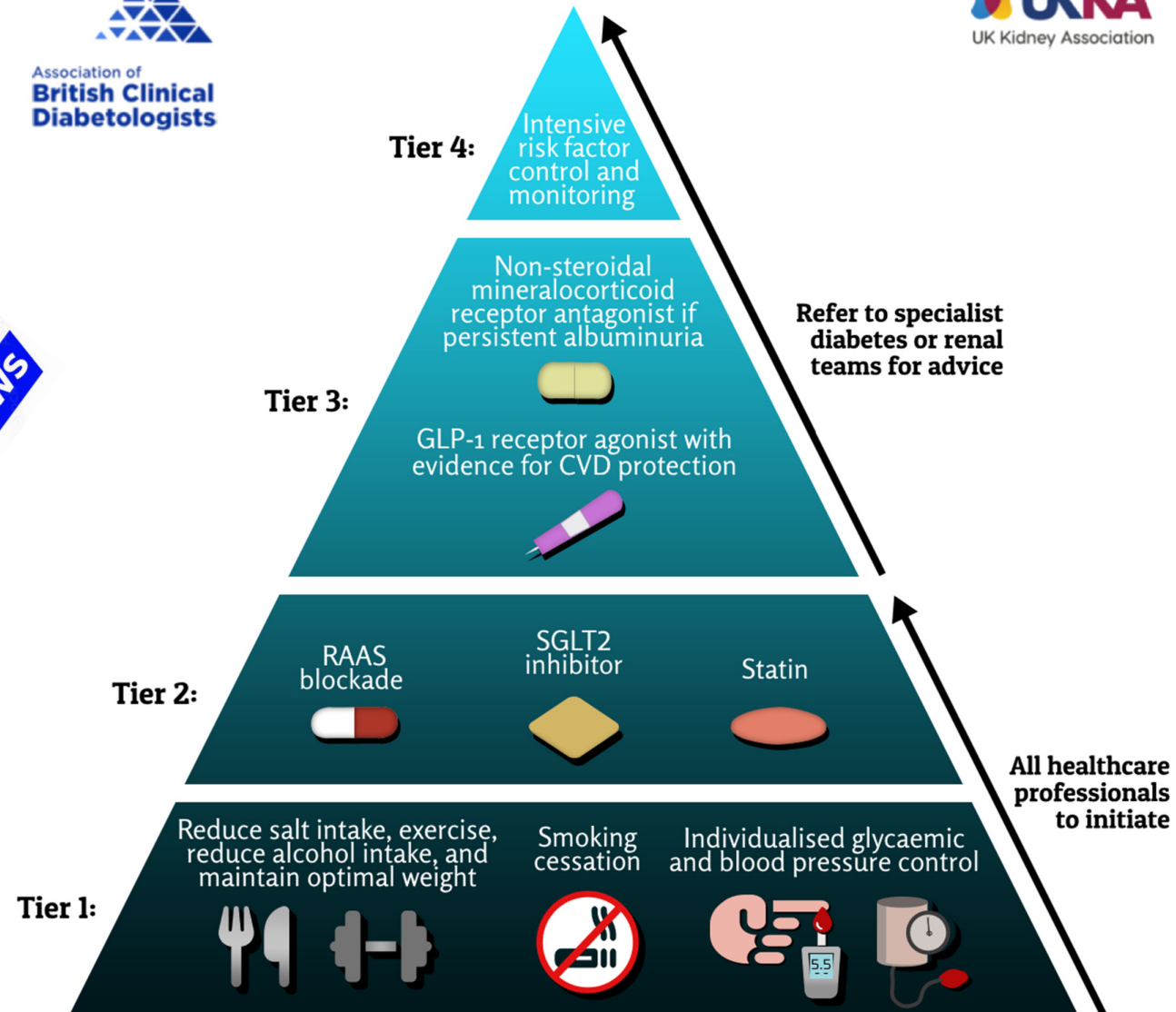
Low risk (if no other markers of kidney disease, no CKD)

Moderately increased risk

High risk

Very high risk

BREAKING
NEWS



Factors resulting in high HbA1c

Mechanism

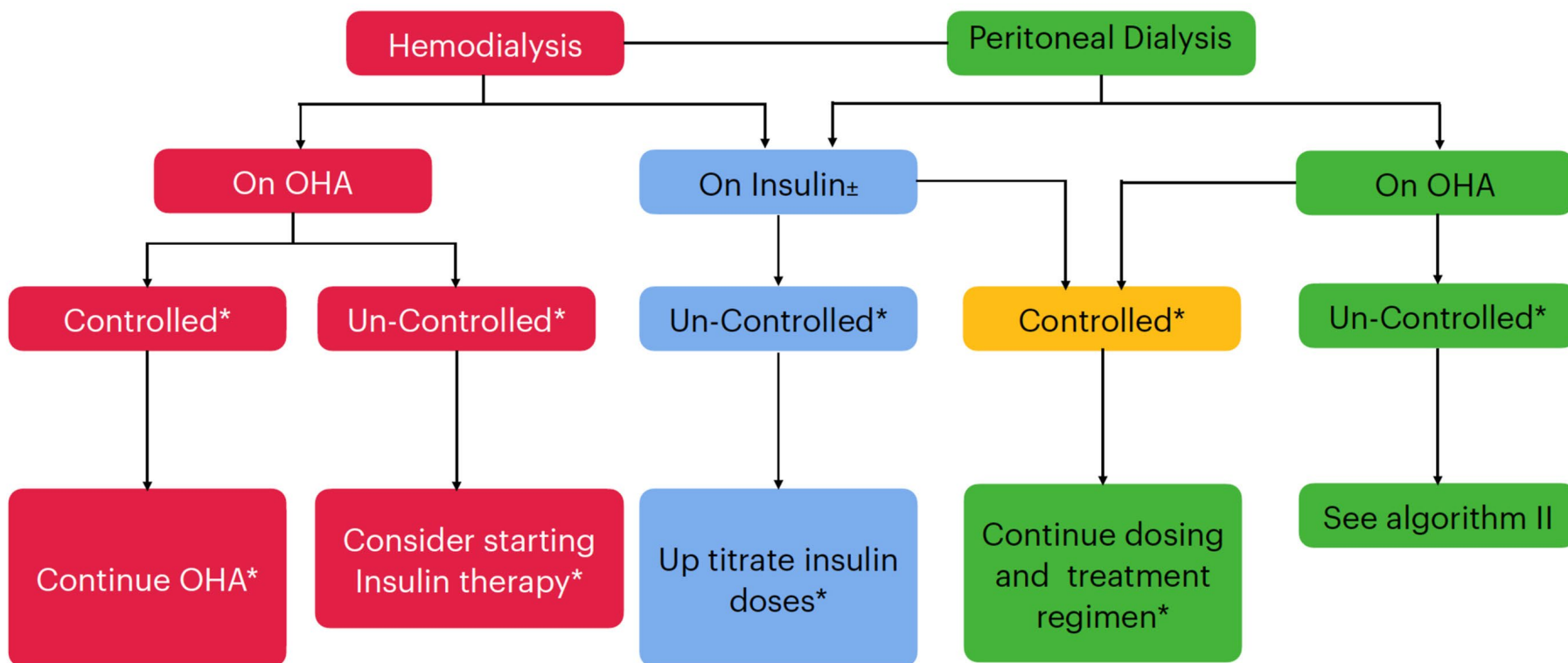
1. Iron deficiency anemia	Iron deficiency leads to increased HbA1c, which is reversed by iron replacement therapy [52].
2. High urea	Exposure to high urea concentrations promotes the formation of carbamylated haemoglobin, which cannot be distinguished from glycated haemoglobin in some assays (e.g., electric charge-based assays). In contrast, analytically specific methods such as boronate-agarose affinity chromatography and thiobarbituric acid methods provide a more reliable measurement of HbA1c in dialysis patients [61,64,65].
3. Metabolic Acidosis	Acidosis has been involved in carbamylation; hence it influences HbA1c levels, but its impact on the estimation of HbA1c is unknown [52].

Factors resulting in low HbA1c

Mechanism

1. Anemia	Might cause an increased red blood cell turnover and therefore may lead to underestimation of HbA1c
2. Malnutrition	
3. Blood transfusions	A cause of HbA1c underestimation.
4. Splenic dysfunction	A frequent finding in dialysis patients leading to increase the removal of RBCs from the circulation
5. Shortened erythrocyte life span or hemolysis	Erythrocyte fragility due to uraemia, Erythrocyte lysis due to the dialysis procedure itself, Chronic blood loss due to residual blood left in the dialyzers, repeated vascular access punctures, occasional blood leaks, phlebotomy for routine testing and clotted dialyzers may all lead to reduced red cells lifespan. This decreases the time available for glycosylation of haemoglobin chains and underestimation during long-term blood glucose monitoring.
6. Erythropoietin	Stimulating agents in dialysis patients also falsely lower HbA1c levels by accelerating erythropoiesis in the preceding two months and increase the proportion of young circulating erythrocytes that have a limited time for haemoglobin glycosylation.

Diabetes management upon starting dialysis

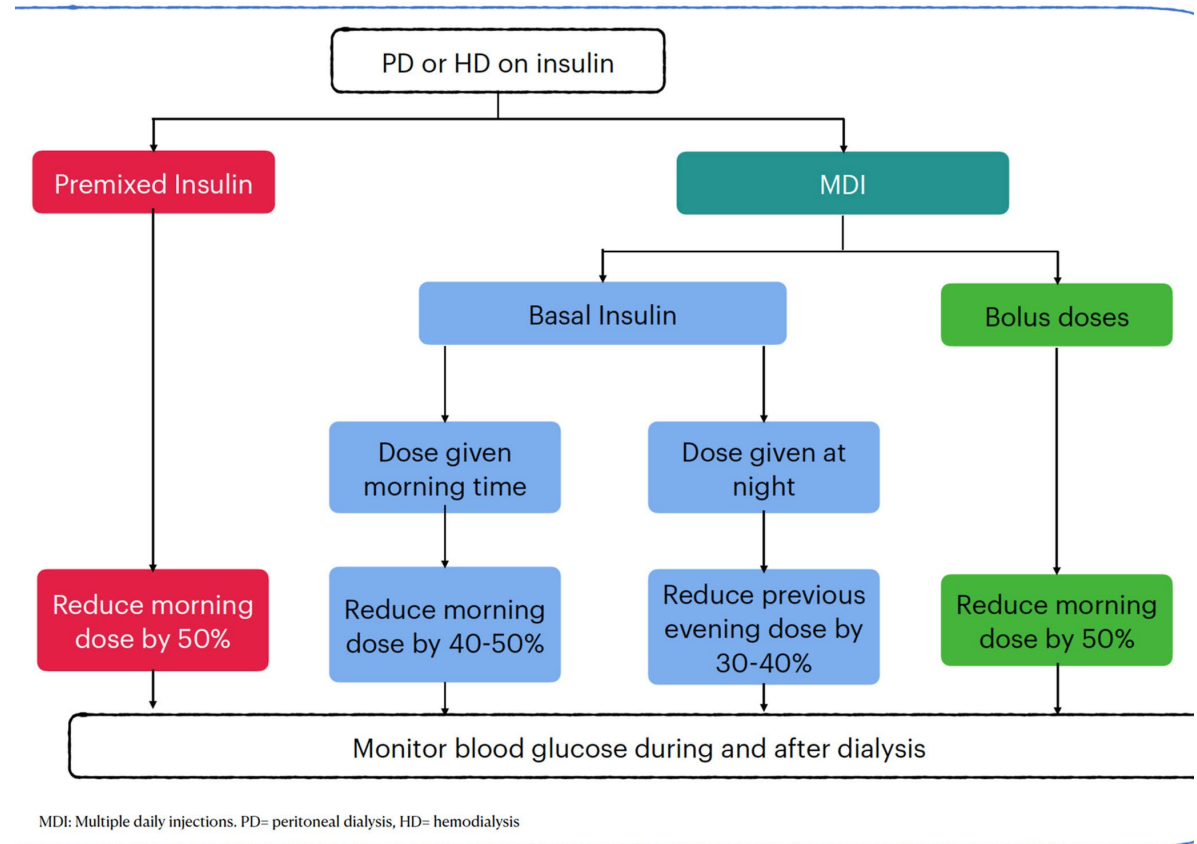


± Reduce the pre-dialysis dose by 30-50% based on glycemic targets and risk of hypoglycemia

* monitoring glycemic control by HbA1c and SMBG (Self Monitoring of Blood Glucose)

OHA= Oral hypoglycemic agents

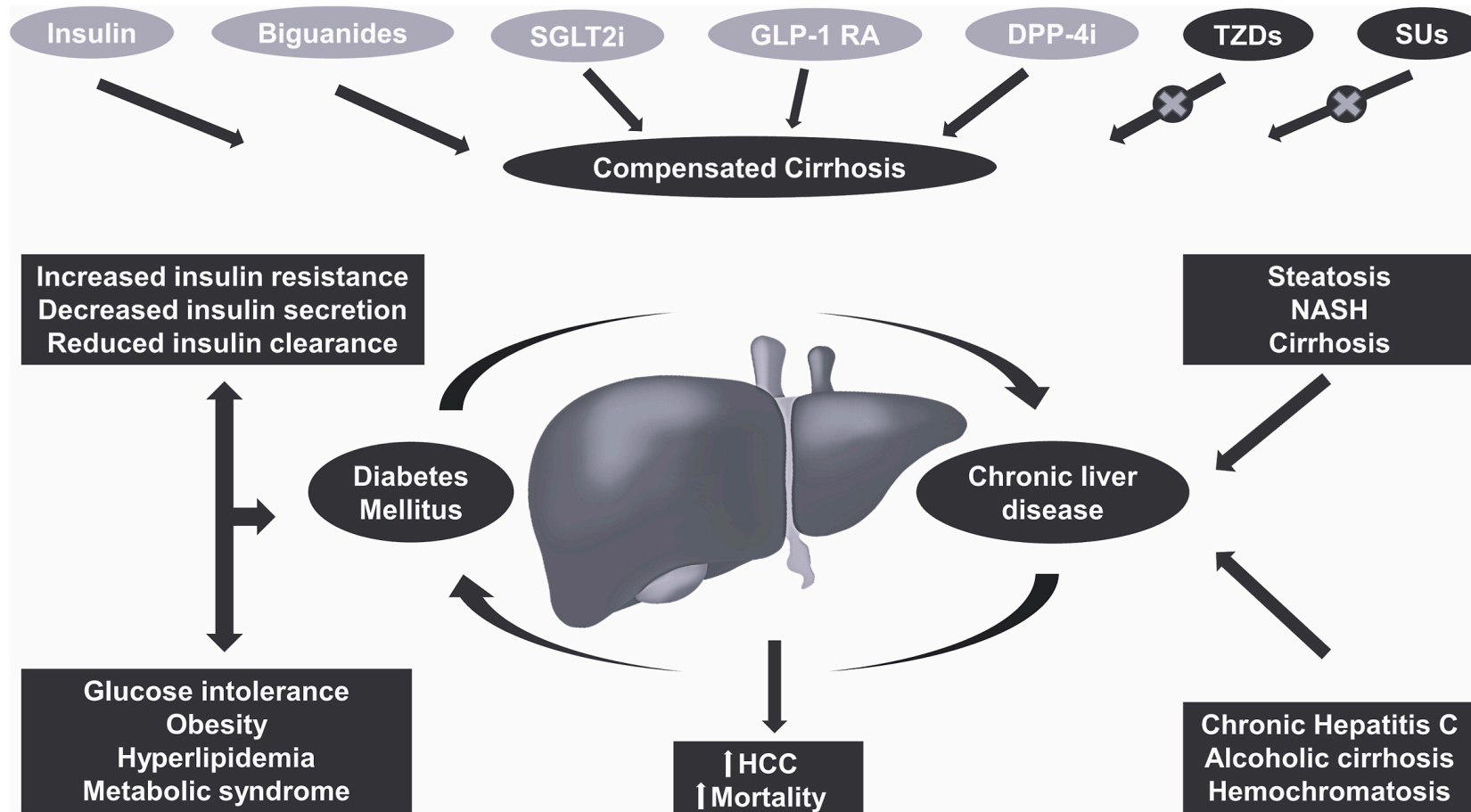
Insulin adjustment before dialysis.



Iperglicemia nella malattia epatica



The two-way relationship between type 2 diabetes and chronic liver disease



Summary of available treatment options for the management of type 2 diabetes in patients with compensated cirrhosis

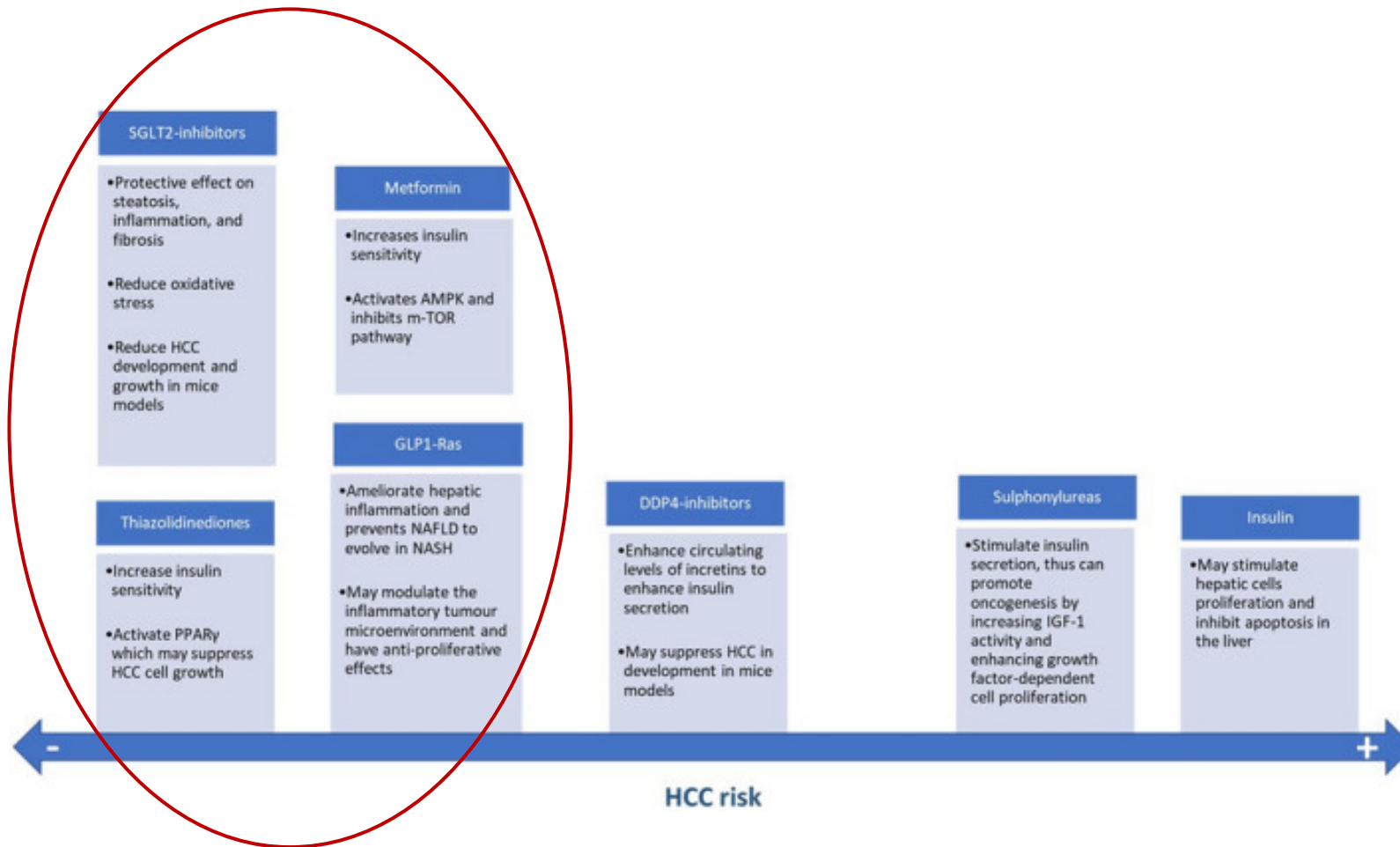
Drug Class	Mechanism of action	Efficacy	Clinical usefulness in compensated cirrhosis	Potential risks	Hypoglycemia	Weight effect	GI side-effects
Biguanides	They increase insulin sensitivity and reduce hepatic gluconeogenesis	High	High	MALA, acute renal dysfunction	No	Neutral or loss	Frequent
Thiazolidinediones	Insulin sensitization via PPAR-γ agonist effect	High	Limited data	Weight gain, fluid retention, osteoporosis	No	Gain	Neutral
Alpha-glucosidase Inhibitors	They decrease carbohydrate absorption in the gut	Intermediate	High	High withdrawal rates, hyperammonemia	No	Neutral	Frequent
Sulphonylureas and meglitinides	Stimulation of insulin release from pancreatic islet cells	High	Low	Concerns about CVD and HCC risk	Yes	Gain	Neutral
SGLT2i	They inhibit SGLT2 expressed in the early portion of the proximal renal tube	Intermediate	Potentially high	DKA, genitourinary tract mycotic infections	No	Loss	Neutral
GLP-1 RA	They increase insulin sensitivity, suppress glucagon production, delay gastric emptying, and reduce appetite	High	Potentially high	Dehydration	No	Loss	Frequent
DPP-4i	They inhibit DPP-4, thus raising endogenous GLP-1	Intermediate	Intermediate	Hepatic decompensation, variceal bleeding	No	Neutral	Rare
Insulin	Replacement of insulin deficiency	Highest	High	Carefully adjust dosing, increase in all-cause mortality	Yes		

SGLT2i: sodium-glucose cotransporter 2 inhibitors; GLP-1: glucagon-like peptide-1; GLP-1 RA: glucagon-like peptide-1 receptor agonists; DPP-4: dipeptidyl peptidase-4; DPP-4i: dipeptidyl peptidase-4 inhibitors; MALA: metformin-associated lactic acidosis; HCC: hepatocellular carcinoma; CVD: cardiovascular disease; GI: gastrointestinal; DKA: diabetic ketoacidosis; PPAR-γ, peroxisome proliferator-activated receptor-γ.

BREAKING
NEWS

company announcement

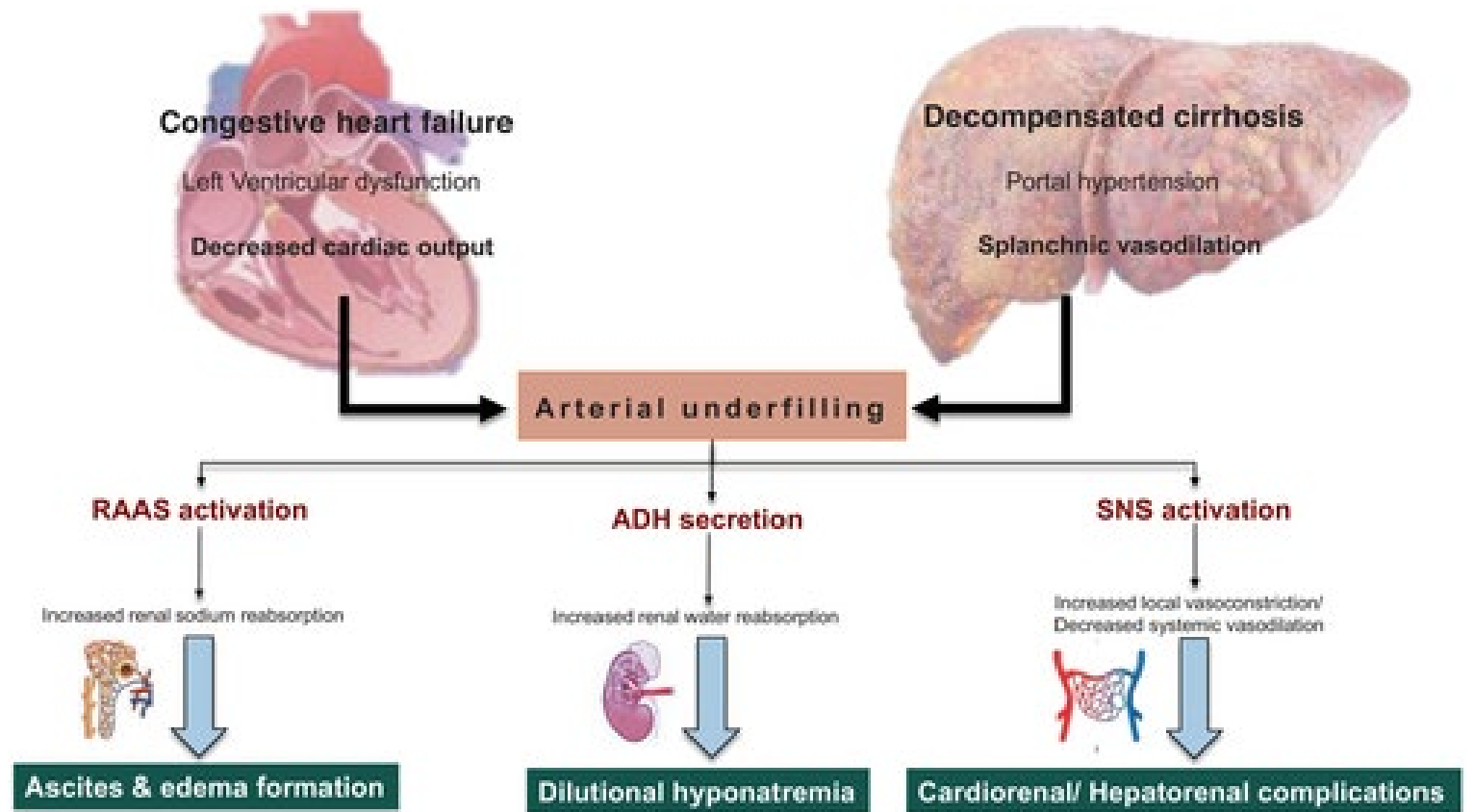
Semaglutide 2.4 mg demonstrates superior improvement in both liver fibrosis and MASH resolution in the ESSENCE trial

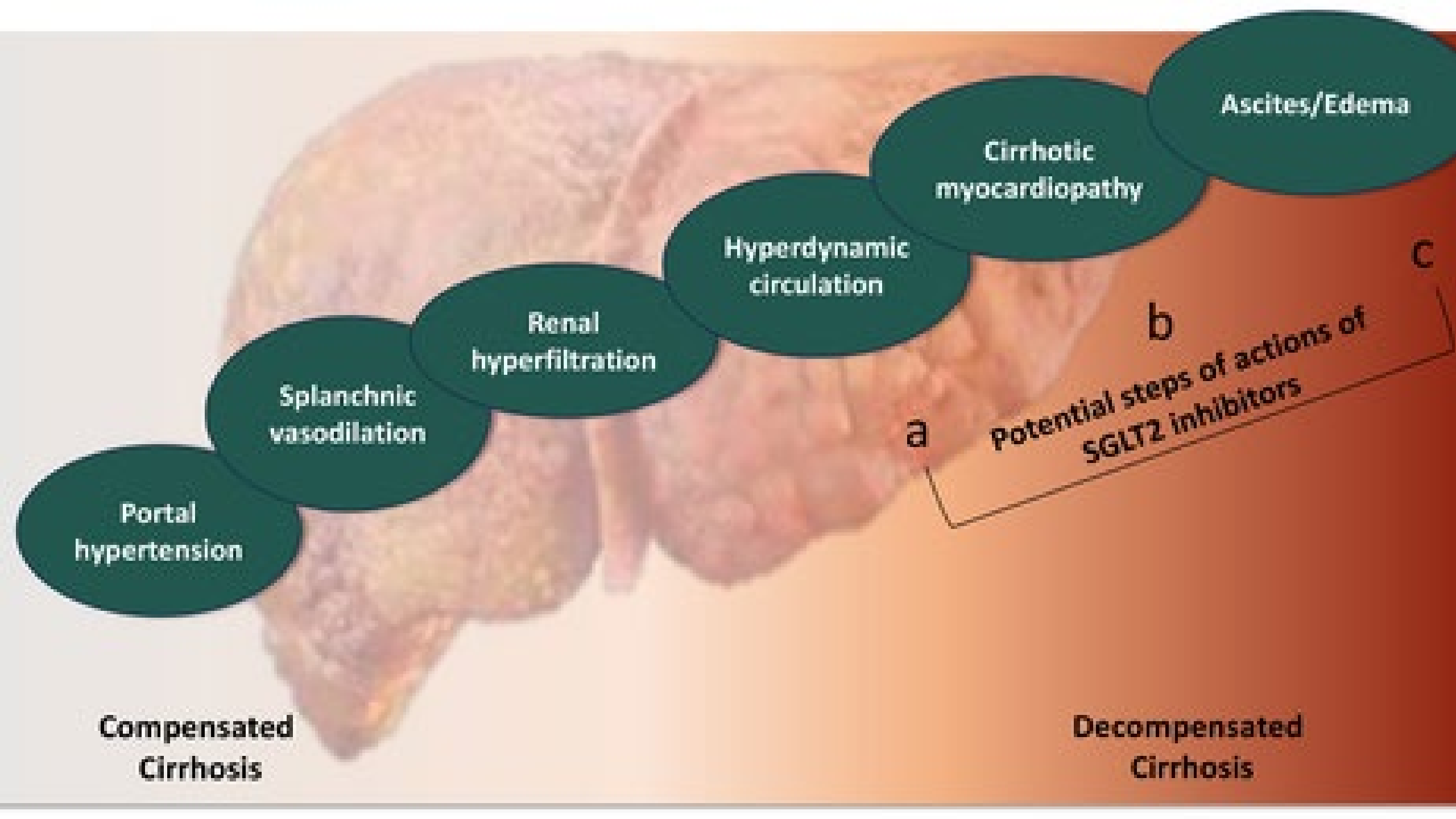


Diabetes medications and risk of HCC

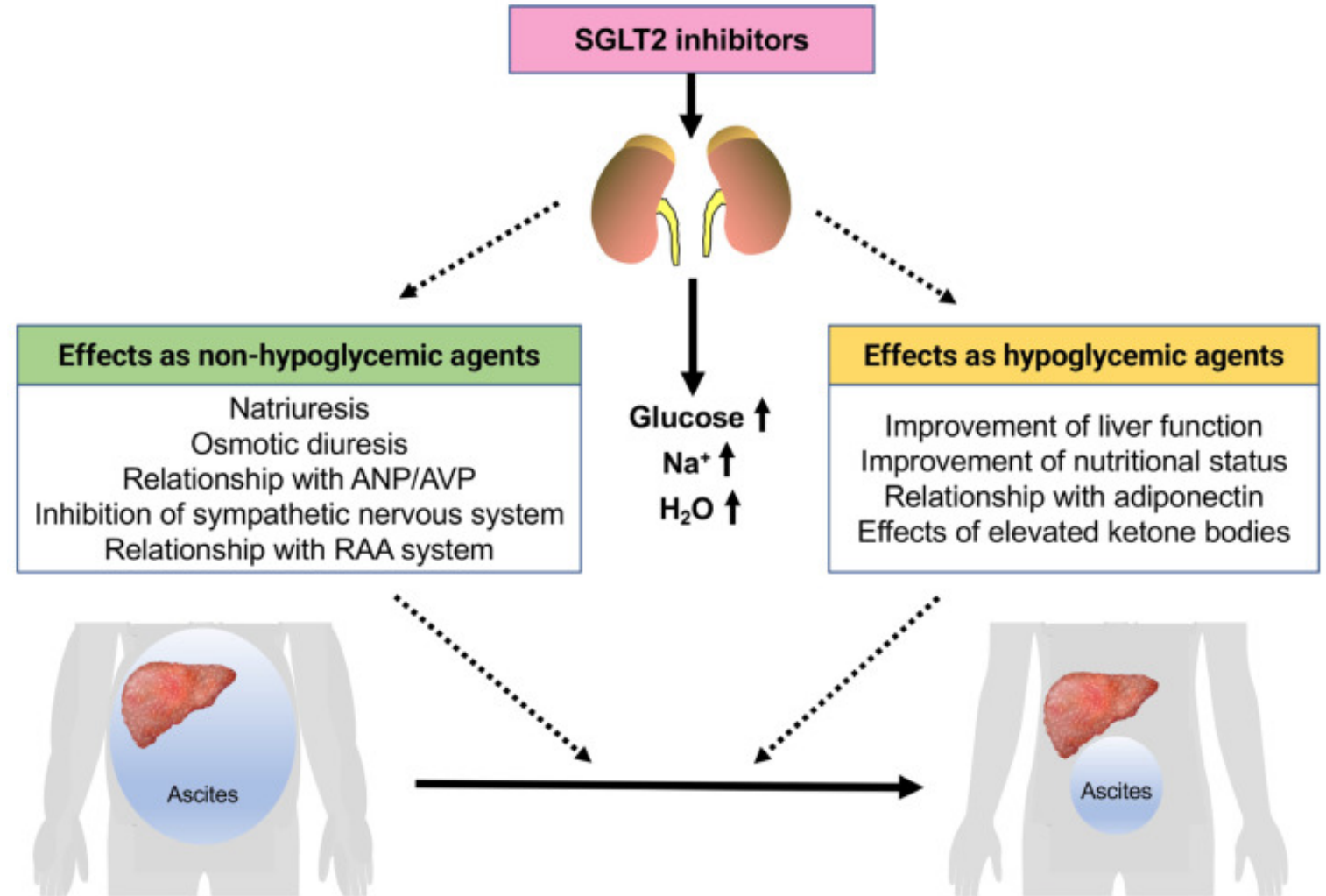
Hepatology . 2022 Dec;76(6):1880-1897. doi: 10.1002/hep.32439.

Common pathophysiological mechanisms of congestive heart failure and decompensated cirrhosis.

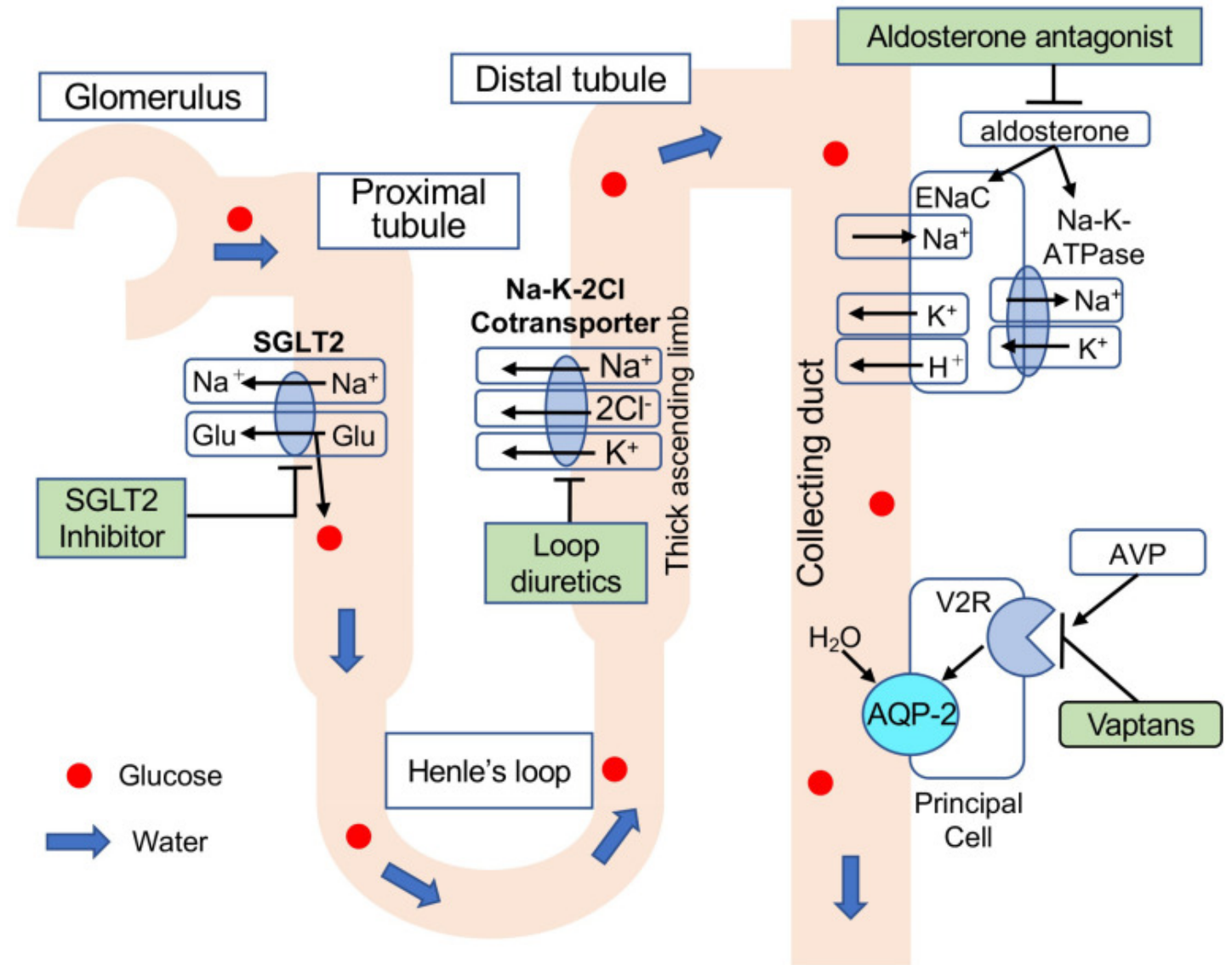




Mechanism of the effects of SGLT2 inhibitors on liver cirrhosis patients with refractory ascites.

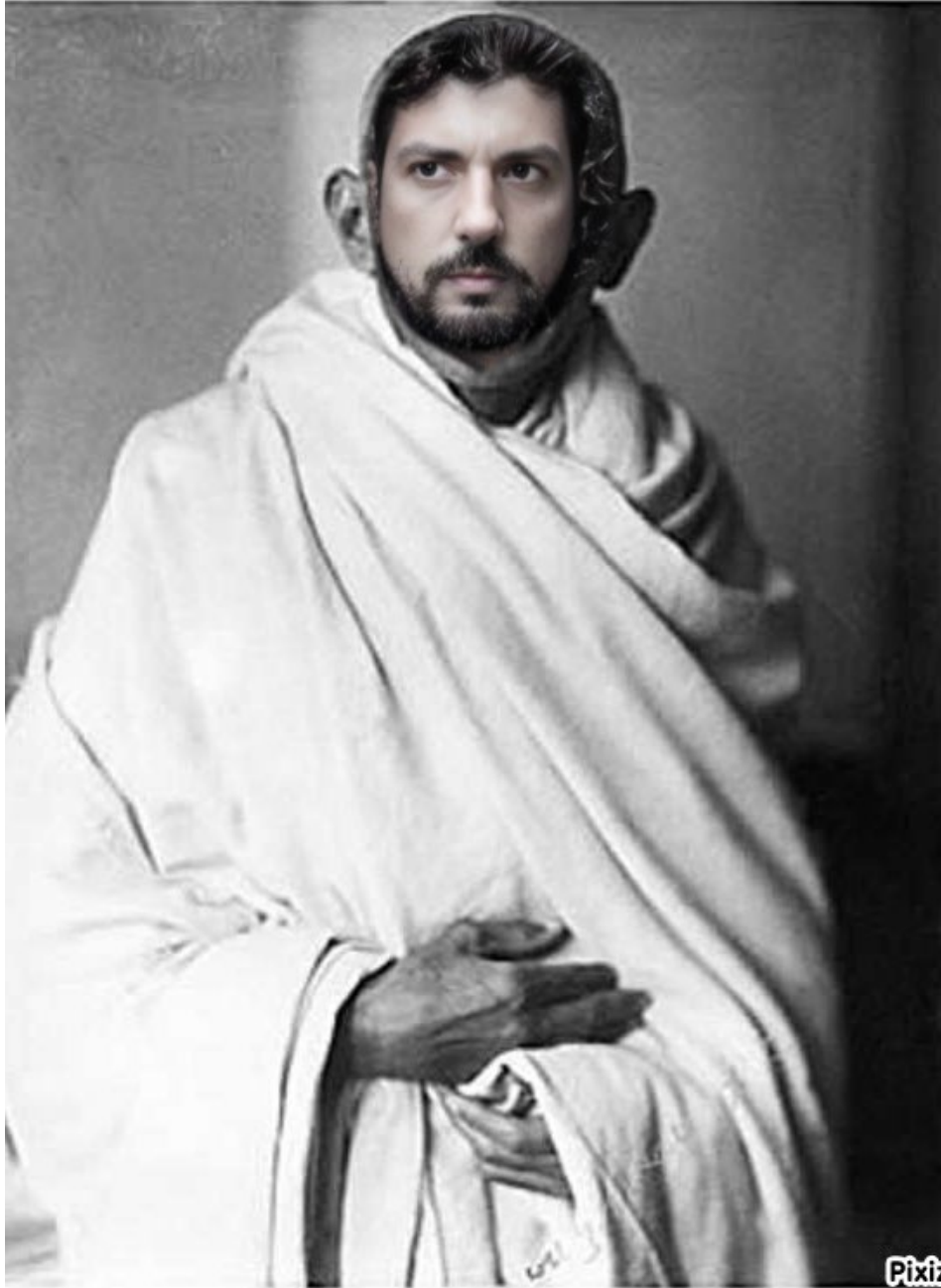


SGLT2 Inhibitors as Diuretics



Conclusioni

- **Gestione Personalizzata:** L'iperglicemia nei pazienti ospedalizzati con insufficienza epatica, renale o in terapia con steroidi richiede un trattamento specifico che tenga conto delle condizioni cliniche particolari. **Superare l'Approccio Insulinocentrico:** L'insulina rimane un pilastro essenziale, ma il suo utilizzo deve essere bilanciato per evitare ipoglicemia, soprattutto in pazienti con condizioni critiche.
- **L'integrazione di nuovi farmaci** (SGLT2 inibitori, GLP-1 agonisti, DPP-4 inibitori) può migliorare la sicurezza e l'aderenza del paziente.
- **Insufficienza Renale:** Necessita di frequenti aggiustamenti delle dosi insuliniche e monitoraggio attento per evitare ipoglicemia.
- **Insufficienza Epatica:** Richiede un approccio cauto con monitoraggio stretto per gestire l'accumulo farmacologico.
- **Terapia con Steroidi:** Strategia insulinica adattata con regime basal-bolus per gestire le variazioni glicemiche.
- **TAKE HOME MESSAGE:** L'uso di terapie non insuliniche, integrate in un piano personalizzato e sicuro, migliora gli outcome clinici nei pazienti ospedalizzati con comorbidità complesse.



"Educhiamoci, impegnamoci e lavoriamo insieme per un futuro cardiometabolicamente sano."

- Mahatma Gandhi

Download all references

