Welcome
Unmet needs in T2D

Dr. Bernhard Ludvik
Goals of diabetes care

• Improve cardiometabolic health
• Prevent complications
• Improve health-related quality of life
Where do we stand in optimal diabetes care?

“After more than a decade of progress from 1999 to the early 2010s, glycemic and blood-pressure control declined in adult NHANES participants with diabetes, while lipid control leveled off.”

Twin epidemics of T2D and obesity

**T2D:** 9.8% → → → 14.3%

**Obesity (BMI > 30):** 30.5% → → → 42.4%

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Diagnosed, undiagnosed, and total diabetes


https://www.cdc.gov/nchs/products/databriefs/db360.htm#fig4

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The obesity and diabetes syndemic

- Sleep disorders
- Inability to be active
- Stigma and impaired mental health

Social environment
- Disadvantage
- Sociocultural barriers
- Income inequality

Promotes 2 core defects
- Insulin resistance
- β-cell decompensation

T2D

Physical environment
- Food
- Physical activity
- Safety
- Alcohol

- Medication-induced weight gain
- Neuropathy and decreased activity
- Hypoglycemia and stimulation of food intake
- Stigma and impaired mental health
Moderate weight loss has benefits — greater weight loss is associated with greater benefits

- Measures of glycemia\(^1\)
- Triglycerides and HDL cholesterol\(^1\)
- Systolic and diastolic blood pressure\(^1\)
- Progression from prediabetes to diabetes\(^1\)
- Hepatic steatosis (measured by MRI)\(^2\)
- Measures of feeling and function
  - Symptoms of urinary stress incontinence\(^1\)
  - Measures of sexual function\(^3\)
  - Quality of life measures (IWQOL)\(^4\)
- NASH activity score (measured by biopsy)\(^1\)
- Apnea-hypopnea index\(^1\)
- Reduction in CV events, mortality, remission of T2D\(^5,6\)

Treatment for T2D to minimize weight gain/promote weight loss

Pharmacologic Treatment of Hyperglycemia in Adults with Type 2 Diabetes

First-Line Therapy depends on comorbidities, patient-centered treatment factors, including cost and access considerations, and management needs and generally includes metformin and comprehensive lifestyle modification.

- Consider additional comorbidities, patient-centered treatment factors, and management needs in choice of therapy, as below:

  PHARMACOTHERAPY

  **Preferably**
  - GLP-1 RA with good efficacy for weight loss
  - SGLT2i
  - Combination therapy

  **If A1C above target**
  - Consider adding GLP-1 RA, SGLT2i, or metformin

  **CONSIDER COST AND ACCESS**
  - Assess in generic form of lowest cost
  - Use GLP-1 RA and cost

  **If A1C above target**
  - Consider adding GLP-1 RA and cost

  **MINIMIZE HYPOGLYCEMIA**
  - For GLP-1 RA, consider incorporating SGLT2i and cost

  **MINIMIZE WEIGHT GAIN**
  - For SGLT2i, consider adding GLP-1 RA and cost

  **MULTIPLE DRUG THERAPY**
  - Combination therapy

  **CONSIDER COST AND ACCESS**
  - Assess in generic form of lowest cost

Used with permission from: Standards of Medical Care in Diabetes — 2022 Abridged for Primary Care Providers. Clin Diabetes. 2022;40:10-38. doi:10.2337/cd22-as01
Social determinants of health

Social Determinants of Health (SDoH).
Available at: https://www.medisked.com/solutions/social-determinants-of-health-sdoh/

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Emergence of new classes of T2D therapeutics

- **GLP-1 RAs (2007)**
  - Favorable weight loss profiles (liraglutide and semaglutide)
  - CV risk reduction (liraglutide, dulaglutide, semaglutide)

- **SGLT2 inhibitors (2015)**
  - Favorable weight loss and blood pressure profile
  - CV risk reduction, renal and cardiac protection

- **Dual GIP/GLP-1R agonist (expected 2022)**
  - Novel, once-weekly GIP and GLP-1 dual receptor agonist – new class
  - Enhanced glycemic control and weight loss benefits
Delineating The Incretin Effect and The Roles Of GLP-1 and GIP

Potential Benefits of Agonism of Multiple Receptors: Mechanism of Action of Unimolecular Dual Agonists

Dr. Michael Nauck
The incretin effect in healthy subjects

A: Plasma glucose [mg/dl]
B: Insulin [μU/ml]
C: GLP-1 [pmol/l]
D: Glucose infusion rate [mg/kg/min]
E: C-peptide [nmol/l]
F: GLP-1 total [pmol/l]

The incretin effect in T2D

GIP and GLP-1 administered as single agents or in combination in T2D patients

GLP-1 stimulates insulin secretion and reduces plasma glucose, but GIP has no effect

The traditional view

A reduced incretin effect in T2D indicates an inability of GIP to stimulate insulin secretion
Is GIP the obesity hormone?

The traditional view: GIP receptor stimulation promotes obesity.
Recent findings on GIP receptor agonism and body weight in animal studies

GIP receptor stimulation leads to reduced food intake and weight loss
GIP reduces food intake and body weight by interacting with CNS-GIPR

However, in human studies: The reduction in energy intake with GLP-1 is confirmed. GIP alone was without effect. The combination with GLP-1 showed a reduced effect*.

Acyl, acylated (free fatty acid residue attached); mGIPR ko, mouse GIP receptor knockout
GIP and GLP-1: The two incretin hormones

GIP and GLP-1 are the gut hormones interacting in the post-prandial stimulation of insulin secretion
The influence of GIP and GLP-1 on postprandial glucose tested by use of specific receptor antagonists in human subjects

Clinical Implications of Dual Agonist Efficacy and Safety data

Dr. Juan Pablo Frias
Tirzepatide: A novel GIP and GLP-1 receptor agonist

Molecular Attributes

• Tirzepatide is a multi-functional peptide engineered from the native GIP peptide sequence, modified to bind to both GIP and GLP-1 receptors¹
• 39 amino acid linear peptide and includes a C20 fatty diacid moiety¹

Pharmacokinetics and pharmacodynamics

• Mean half-life of ~5 days (116.7 hours), enabling once-weekly dosing¹
• Enhances 1st and 2nd phase insulin secretion and reduces glucagon levels, both in a glucose-dependent manner¹,²
• Concentrations in people with renal and hepatic impairment do not differ versus healthy people³,⁴

Single agent possessing activity at 2 pharmacologic targets

GIP = glucose-dependent insulino tropic polypeptide; GLP-1R = glucagon-like peptide-1.
<table>
<thead>
<tr>
<th>Monotherapy</th>
<th>2-Drug Combination</th>
<th>2-3 Drug Combinations</th>
<th>2-4 Drug Combinations</th>
<th>Combination With Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>SURPASS-1 vs placebo&lt;sup&gt;1&lt;/sup&gt; Drug-naïve or washout from any OAM</td>
<td>SURPASS-2 vs semaglutide&lt;sup&gt;2&lt;/sup&gt; Add-on to metformin</td>
<td>SURPASS-3 vs insulin degludec&lt;sup&gt;3&lt;/sup&gt; Add-on to metformin with or without SGLT-2i</td>
<td>SURPASS-4 vs insulin glargine&lt;sup&gt;4&lt;/sup&gt; Add-on to ≥ 1 and ≤ 3 OAMs (metformin, SGLT-2i, or SU)</td>
<td>SURPASS-5 vs placebo&lt;sup&gt;5&lt;/sup&gt; Both with insulin glargine with or without metformin</td>
</tr>
<tr>
<td>SURPASS-CVOT vs dulaglutide&lt;sup&gt;7&lt;/sup&gt; (ongoing)</td>
<td></td>
<td></td>
<td></td>
<td>SURPASS-6 vs insulin lispro (TID)&lt;sup&gt;6&lt;/sup&gt; Both with insulin glargine with or without metformin (ongoing)</td>
</tr>
</tbody>
</table>


OAM = oral antihyperglycemic medication; SU = sulfonylurea; TID = three times daily
SURPASS trial design: tirzepatide 5, 10, and 15 mg versus active comparator or placebo

Primary Objective
Superiority and/or noninferiority of TZP 5 mg and/or 10 mg and/or 15 mg vs placebo or active comparator in mean change in A1c from baseline at 40 or 52 weeks

Tirzepatide at all doses significantly reduced A1c versus placebo or active comparators

**SURPASS-1**
(N=478)
40 weeks
7.9%
Monotherapy

**SURPASS-2**
(N=1,878)
40 weeks
8.3%
MET

**SURPASS-3**
(N=1,437)
52 weeks
8.2%
MET ± SGLT-2i

**SURPASS-4**
(N=1,995)
52 weeks
8.5%
1-3 OAMs (MET/SU/SGLT-2i)

**SURPASS-5**
(N=475)
40 weeks
8.3%
Basal insulin ± MET

Mean Change in A1c, % (SE)

-3 -2 -1 0 1 2 3

TZP 5 mg  TZP 10 mg  TZP 15 mg  Placebo  Semaglutide  Degludec  Glargine

Treatment regimen estimand.
Tirzepatide significantly reduced HbA1c and more patients achieved A1c targets compared with semaglutide 1 mg

Overall mean baseline A1c = 8.3%

Data are LSM (SE); mITT (efficacy analysis set) ANOVA analysis (week 0) and MMRM analysis (week 40). Arrows indicate when the maintenance dose of tirzepatide 5 mg, 10 mg and 15 mg and semaglutide 1 mg are achieved.

*p<0.001 vs. semaglutide 1 mg
Tirzepatide decreased weight (kg and %) more than comparators in SURPASS trials

**SURPASS-1**
(N=478)
- Treatment duration: 40 weeks
- Baseline weight (kg): 85.9
- Add-on to: Monotherapy

**SURPASS-2**
(N=1,878)
- Treatment duration: 40 weeks
- Baseline weight (kg): 93.7
- Add-on to: MET

**SURPASS-3**
(N=1,437)
- Treatment duration: 52 weeks
- Baseline weight (kg): 94.3
- Add-on to: MET ± SGLT-2i

**SURPASS-4**
(N=1,995)
- Treatment duration: 52 weeks
- Baseline weight (kg): 90.3
- Add-on to: 1-3 OAMs (MET/SU/SGLT-2i)

**SURPASS-5**
(N=475)
- Treatment duration: 40 weeks
- Baseline weight (kg): 95.2
- Add-on to: Basal insulin ± MET

**Mean Change in Weight**
(kg) [SE] (% change)

**Color changes**
- TZP 5 mg
- TZP 10 mg
- TZP 15 mg
- Placebo
- Semaglutide
- Degludec
- Giargine

**Treatment regimen:***
Superiority vs placebo or active comparator:
* p < 0.05; ** p < 0.001

No plateau in body weight loss at 40 and 52 weeks (SURPASS 2 and 3)

**SURPASS-2**
Overall mean baseline weight = 93.8 kg

**SURPASS-3**
Overall mean baseline weight = 94.5 kg

**Mean Change in Weight (kg) [SE]**

More participants achieved ≥10% weight loss with all doses of tirzepatide vs comparators

<table>
<thead>
<tr>
<th>Treatment duration</th>
<th>Surpass-1 (N=478)</th>
<th>Surpass-2 (N=1,878)</th>
<th>Surpass-3 (N=1,437)</th>
<th>Surpass-4 (N=1,995)</th>
<th>Surpass-5 (N=475)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Add-on to</td>
<td>40 weeks Monotherapy</td>
<td>40 weeks MET</td>
<td>52 weeks MET ± SGLT-2i</td>
<td>52 weeks 1-3 OAMs (MET/SU/SGLT-2i)</td>
<td>40 weeks Basal insulin ± MET</td>
</tr>
</tbody>
</table>

% of Patients Achieving Body Weight Loss ≥ 10%

<table>
<thead>
<tr>
<th>Treatment Regimen Estimand</th>
<th>Surpass-1</th>
<th>Surpass-2</th>
<th>Surpass-3</th>
<th>Surpass-4</th>
<th>Surpass-5</th>
</tr>
</thead>
<tbody>
<tr>
<td>TZP 5 mg</td>
<td>27*</td>
<td>34**</td>
<td>57**</td>
<td>50**</td>
<td>21**</td>
</tr>
<tr>
<td>TZP 10 mg</td>
<td>34**</td>
<td>47**</td>
<td>58**</td>
<td>50**</td>
<td>2**</td>
</tr>
<tr>
<td>TZP 15 mg</td>
<td>0.1</td>
<td>24</td>
<td>35**</td>
<td>32**</td>
<td>2**</td>
</tr>
<tr>
<td>Placebo</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Semaglutide</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Degludec</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glargine</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Superiority vs placebo or active comparator: *p < 0.05; ** p < 0.001

Up to 60% of participants on tirzepatide achieved composite endpoint compared to 22% on once-weekly semaglutide 1 mg (SURPASS-2)

Prespecified composite endpoint
- A1c ≤6.5%, and
- Weight loss ≥10%, and
- No Level 2 (<54 mg/dL [3.0 mmol]) or Level 3 (severe) hypoglycemia

**Tirzepatide safety and tolerability**

- Side effect profile similar to that of selective GLP-1 receptor agonists
- Most common adverse events were gastrointestinal in nature and occurred primarily during dose escalation

<table>
<thead>
<tr>
<th>Preferred Term, %</th>
<th>TZP 5 mg (N=470)</th>
<th>TZP 10 mg (N=469)</th>
<th>TZP 15 mg (N=470)</th>
<th>Sema 1 mg (N=469)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any GI TEAE</td>
<td>40.0</td>
<td>46.1</td>
<td>44.9</td>
<td>41.2</td>
</tr>
<tr>
<td>Nausea</td>
<td>17.4</td>
<td>19.2</td>
<td>22.1</td>
<td>17.9</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>13.2</td>
<td>16.4</td>
<td>13.8</td>
<td>11.5</td>
</tr>
<tr>
<td>Vomiting</td>
<td>5.7</td>
<td>8.5</td>
<td>9.8</td>
<td>8.3</td>
</tr>
<tr>
<td>Dyspepsia</td>
<td>7.2</td>
<td>6.2</td>
<td>9.1</td>
<td>6.6</td>
</tr>
<tr>
<td>Constipation</td>
<td>6.8</td>
<td>4.5</td>
<td>4.5</td>
<td>5.8</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>3.0</td>
<td>4.5</td>
<td>5.1</td>
<td>5.1</td>
</tr>
</tbody>
</table>
Incidence of nausea over time through 40 weeks (SURPASS-2)

Most cases of nausea were mild to moderate, transient, and occurred during the dose-escalation period in all groups

## Low incidence of hypoglycemia in SURPASS trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>Duration</th>
<th>Treatment</th>
<th>Hypoglycemia*</th>
<th>Severe Hypoglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SURPASS-1 (40 weeks)</strong></td>
<td></td>
<td>Monotherapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 5 mg (N=121)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 10 mg (N=119)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 15 mg (N=120)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Placebo (N=115)</td>
<td>0.9</td>
<td>0</td>
</tr>
<tr>
<td><strong>SURPASS-2 (40 weeks)</strong></td>
<td></td>
<td>Metformin</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 5 mg (N=470)</td>
<td>0.9</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 10 mg (N=469)</td>
<td>0.2</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 15 mg (N=470)</td>
<td>1.7</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Semaglutide (N=469)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>SURPASS-3 (52 weeks)</strong></td>
<td></td>
<td>Metformin ± SGLT-2i</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 5 mg (N=356)</td>
<td>1.4</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 10 mg (N=360)</td>
<td>1.1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 15 mg (N=359)</td>
<td>2.2</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Degludec (N=358)</td>
<td>7.3</td>
<td>0</td>
</tr>
<tr>
<td><strong>SURPASS-4 (52 weeks)</strong></td>
<td></td>
<td>± Metformin ± SU ± SGLT-2i</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 5 mg (N=329)</td>
<td>8.8</td>
<td>0.30</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 10 mg (N=328)</td>
<td>6.1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 15 mg (N=338)</td>
<td>8.0</td>
<td>0.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Glargine (N=1,000)</td>
<td>19.1</td>
<td>1.10</td>
</tr>
<tr>
<td><strong>SURPASS-5 (40 weeks)</strong></td>
<td></td>
<td>Basal insulin ± Metformin</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 5 mg (N=116)</td>
<td>15.5</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 10 mg (N=119)</td>
<td>19.3</td>
<td>1.68</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TZP 15 mg (N=120)</td>
<td>14.2</td>
<td>0.83</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Placebo (N=120)</td>
<td>12.5</td>
<td>0</td>
</tr>
</tbody>
</table>

Pooled tirzepatide vs pooled comparator effect on time to first MACE-4

![Graph showing time to first MACE-4](image)

**SURPASS-CVOT is estimated to complete in 2024**

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**Planned follow-up period**

- **GPGB**: (30 weeks)
- **SURPASS-1, -2 and -5**: (44 weeks)
- **SURPASS-3 and J-mono**: (56 weeks)
- **SURPASS-4**: (56–108 weeks)

**Cumulative number of events: number of patients at risk**

<table>
<thead>
<tr>
<th></th>
<th>Pooled tirzepatide</th>
<th>Pooled comparator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time from first dose (weeks)</td>
<td>0.4887 15.4813 28.4726 43.4477 53.2477 62.9620 68.832 69.515 72.188</td>
<td>0.2328 13.2292 19.2250 28.2118 36.1438 52.914 62.794 67.496 69.172</td>
</tr>
</tbody>
</table>

MACE-4, CV death, MI, stroke, and hospitalized unstable angina. P values were based on the Wald chi-square test.

Tirzepatide indication in EU and key prescribing information from US label

**Indication**

Tirzepatide is indicated for the treatment of adults with insufficiently controlled type 2 diabetes mellitus as an adjunct to diet and exercise

- as monotherapy when metformin is considered inappropriate due to intolerance or contraindications
- in addition to other medicinal products for the treatment of diabetes

**Dosing and Administration**

- Single-dose prefilled pen with hidden needle
- 2.5 mg, 5 mg, 7.5 mg, 10 mg, 12.5 mg, and 15 mg per 0.5 mL
- The recommended starting dosage is 2.5 mg SC once weekly
- After 4 weeks, increase to 5 mg SC once weekly
- If additional glycemic control is needed, increase the dosage in 2.5-mg increments after at least 4 weeks on the current dose
- The maximum dosage is 15 mg SC once weekly
- Administer once weekly at any time of day, with or without meals

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Tirzepatide: contraindications, limitations of use, and warning/precautions in the US label

Contraindications

- Personal or family history of medullary thyroid carcinoma or patients with MEN2
- Known serious hypersensitivity to tirzepatide or any of the excipients

Warnings and Precautions

- Pancreatitis
- Hypoglycemia with concomitant use of insulin secretagogues or insulin
- Hypersensitivity reactions
- Acute kidney injury
- Severe gastrointestinal disease
- Diabetic retinopathy complications in patient with a history of diabetic retinopathy
- Acute gallbladder disease

Limitations of Use

- Has not been studied in patients with a history of pancreatitis
- Is not indicated for use in patients with type 1 diabetes


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Summary and conclusions

- Tirzepatide is a unimolecular dual GIP and GLP-1 receptor agonist
- Based on pre-clinical and clinical studies, its mechanism of action is likely due to both synergistic and complementary effects of agonism of GIP and GLP-1 receptors
- In Phase 2 and Phase 3 clinical trials, significant reductions in A1c and body weight were demonstrated versus placebo and active comparators across the spectrum of type 2 diabetes
- Tirzepatide was recently approved in the US for treatment of people with T2D and received a positive opinion from the Committee for Medicinal Products for Human Use (CHMP) in the EU
- It is currently being studied for other therapeutic indications, including the treatment of overweight and obesity

Questions & Answers
Case Studies
Comorbidities, treatment intensification, shared decision making

Dr. Bernhard Ludvik
Patient communication is key to weight management success

Patients prefer the terms “excess body weight,” “BMI,” “above ideal body weight,” and “maintaining a healthy weight”

They dislike the terms “excess fat,” “obese,” and “obesity”

Seek the patient’s permission to discuss weight

- “As we get your glucose under control, do you have additional goals concerning your weight?”
- “What kind of help from me would you like regarding your weight?”

Case 1: Rudolph, 63 years

T2D since 2017

- Struggling with obesity (BMI 31 kg/m²)
- Treated with metformin and empagliflozin
- HbA1c - 8.1%, no evidence of CV disease (yearly screening with exercise test)
- Insulin was originally recommended because of failure of oral glucose-lowering therapy
Case 1: Rudolph, 63 years

Treatment

- Treatment with a GLP-1 receptor agonist or GIP/GLP-1 receptor agonist (tirzepatide) was considered as an alternative to basal insulin
  - Achievement of HbA1c goal without risk of hypoglycemia and weight gain

- Tirzepatide was chosen because of expected superiority over a GLP-1 receptor agonist (SURPASS-2) as well as basal insulin regarding glucose control and weight loss, as suggested by the results of SURPASS-3 study with tirzepatide 15 mg:
  - HbA1c -2.1%,
  - Body weight -11.3 kg

What can we expect from the study evidence for Rudolph after 1 year?

HbA1c: 6.0%, BMI: 28.6 kg/m²

Case 2: Monica, 69 years

T2D since 2013

Obese (BMI 31 kg/m²)

Treated with metformin, empagliflozin, and semaglutide 1 mg QW

Current HbA1c 7.4%, no evidence of CV disease, Evidence of NASH

Addition of basal insulin was considered to improve diabetes control
Case 2: Monica, 69 years

Treatment

- Switch from semaglutide to a GIP/GLP-1 receptor agonist (tirzepatide) was considered as an alternative to basal insulin
  - Achievement of HbA1c goal (<7.0%) and weight loss
- Tirzepatide was chosen because of expected superiority over semaglutide regarding glucose control and weight loss, as suggested by the results of SURPASS-2 with tirzepatide 15 mg (treatment difference HbA1c -0.4%, body weight -5.5 kg)

What can we expect from the study evidence for Monica after 1 year?

HbA1c: 6.7%, BMI: 29.2 kg/m²
Ask Me Anything
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