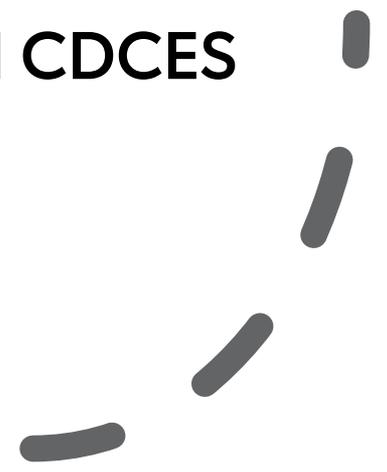


# Improving Outcomes Across the CKM Continuum: The Importance of Comprehensive Ophthalmic Care & Collaboration

Melissa Magwire MSN RN CDCES



# DISCLOSURES

None to report

## OBJECTIVES

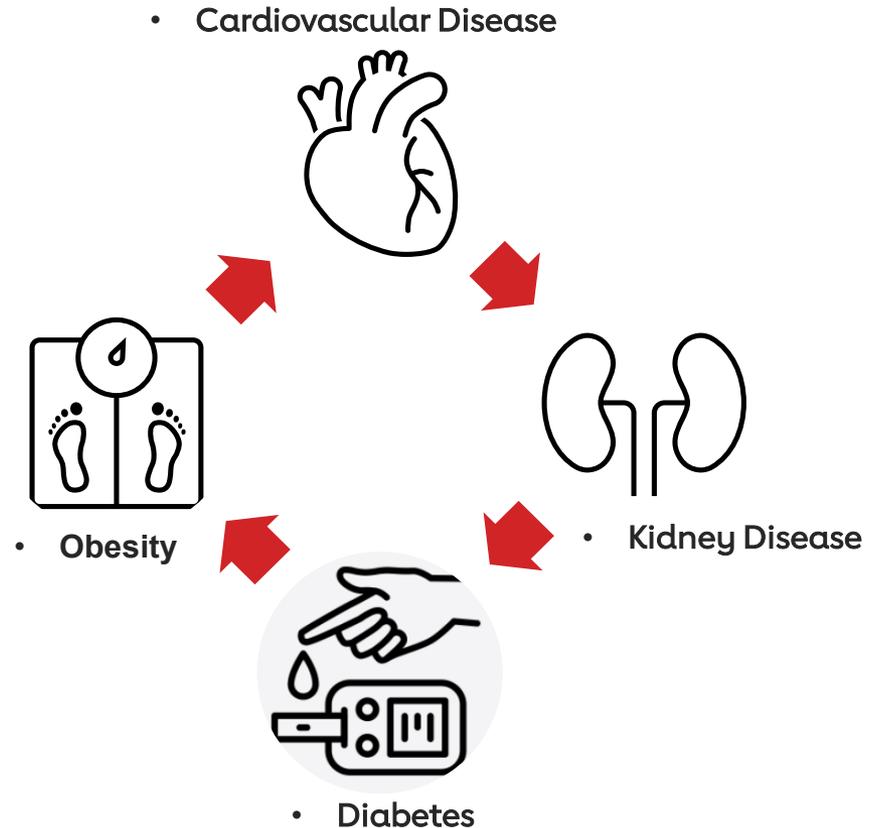
- ❖ Describe the systemic link between CKM Syndrome and ocular manifestations.
- ❖ Apply knowledge of AHA CKM staging to ophthalmic practice
- ❖ Evaluate the ocular safety and implications of GLP-1 RA and SGLT2i therapies.
- ❖ Formulate a proactive screening strategy for CKM syndrome.
- ❖ Express and understanding of the importance of an interdisciplinary approach and outline specific strategies for collaboration.
- ❖ Provide patient-centered education on CKM risks and the connection/impact to their eye/vision health.

# MAKING THE CKM CONNECTION

Estimated that 1 in 3 US adults has at least 3 risk factors contributing to CKM:

- Extra body fat, especially around the waist
- Prediabetes or Diabetes
- High cholesterol
- High triglycerides
- High blood pressure

Having more than one CKM condition puts you at risk for developing others





## QUESTION

Which of the following describes the most significant consideration for an ophthalmologist when managing a patient with CKM syndrome who is starting a new GLP-1 receptor agonist?

- a) An immediate change in vision.
- b) The increased cardiovascular benefits of GLP-1 RAs negate the need for proactive ophthalmic screening.
- c) A stable blood glucose level ensures there will be no risk of developing or worsening diabetic retinopathy.



## QUESTION

Which of the following represents the most effective proactive screening strategy for CKM syndrome in an ophthalmology setting?

- a) Only screening for retinopathy in patients with a known diagnosis of type 2 diabetes.
- b) Routinely performing optical coherence tomography angiography (OCTA) scans on all patients
- c) Incorporating risk factor assessments into routine ophthalmic exams to identify patients at risk for CKM and recommend further systemic evaluation when indicated.

# October 2023 – Cardiovascular Kidney Metabolic Health: A Presidential Advisory From the AHA

CKM syndrome is a systemic disorder characterized by pathophysiological interactions among metabolic risk factors, CKD, and the cardiovascular system leading to multiorgan dysfunction and a high rate of adverse cardiovascular outcomes. CKM syndrome includes both individuals at risk for CVD due to the presence of metabolic risk factors, CKD, or both and individuals with existing CVD that is potentially related to or complicates metabolic risk factors or CKD. The increased likelihood of CKM syndrome and its adverse outcomes is further influenced by unfavorable conditions for lifestyle and self-care resulting from policies, economics and the environment.

**CKM syndrome is a health disorder due to connections among heart disease, kidney disease, diabetes and obesity leading to poor health outcomes.**

# CURRENT STATISTICS AND TRENDS



**United States:** About **80% of adults** have some form of CKM syndrome

**Global:** Approximately **40–46% of the world’s adult population** meets criteria for metabolic syndrome, which is a major component of CKM.

**Regional Trends:** Asia and Africa see increases due to economic shifts; North America and Europe face high rates linked to obesity.

**Affected Populations:** Middle-aged and older adults are mostly affected, but younger populations show rising incidence.

**Contributing Factors:** Urbanization, sedentary lifestyles, unhealthy diets, and socioeconomic disparities worsen CKM trends.



# FINANCIAL IMPACT ON HEALTH SYSTEMS

## Direct Healthcare Costs

Direct costs include hospitalizations, medications, and outpatient care for managing CKM-related cardiovascular and metabolic conditions.

## Indirect Economic Burden

Lost productivity, disability, and premature mortality significantly increase the indirect financial impact of CKM syndrome globally.

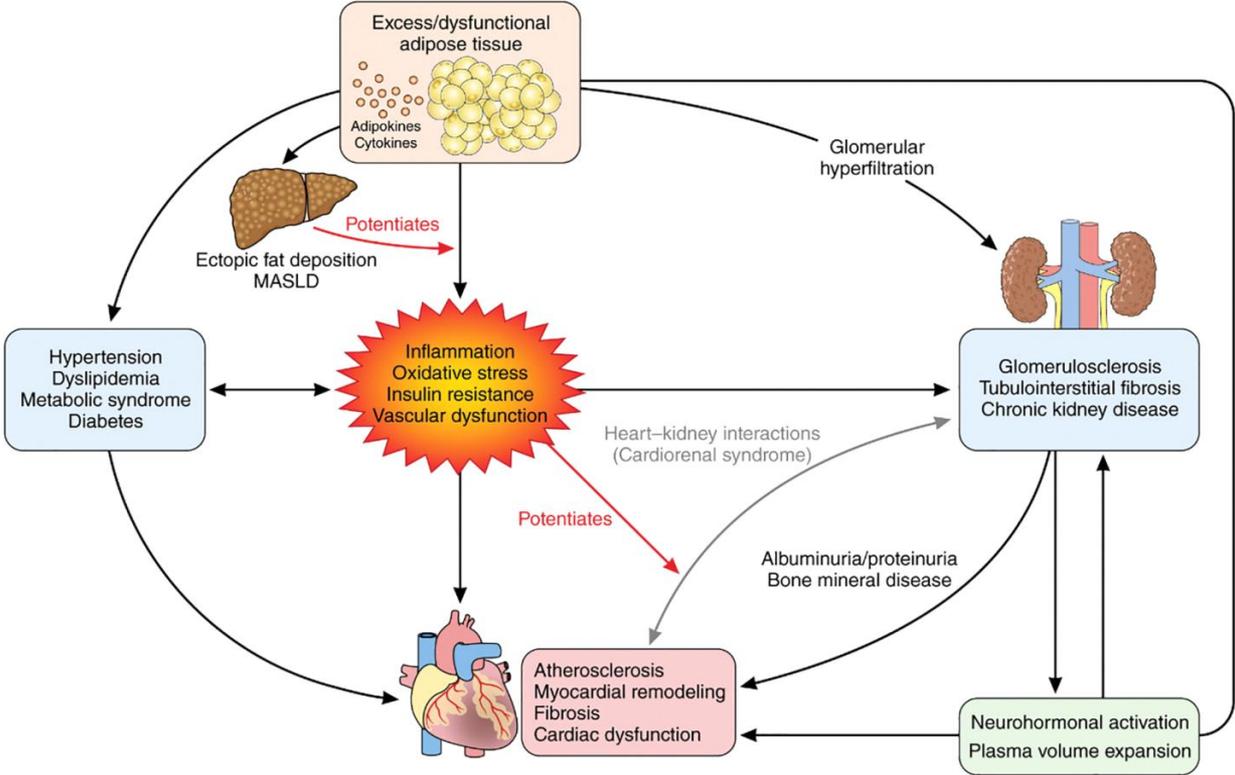
## Challenges in Developing Countries

Limited healthcare resources in developing countries are strained by CKM complications, affecting other essential health services.

## Prevention and Early Intervention

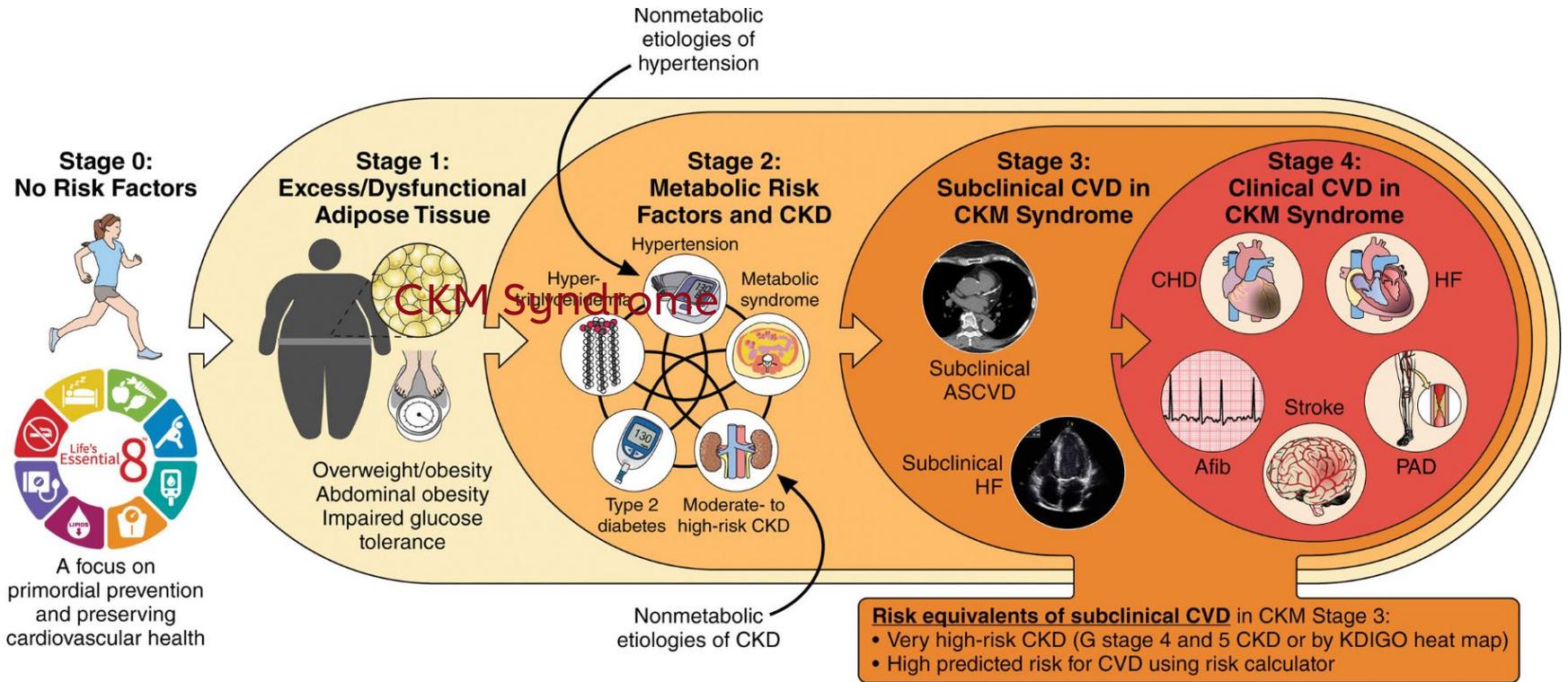
Early detection and lifestyle interventions reduce long-term costs compared to treating advanced disease stages.

# UNDERLYING PATHOPHYSIOLOGY OF CKM SYNDROME

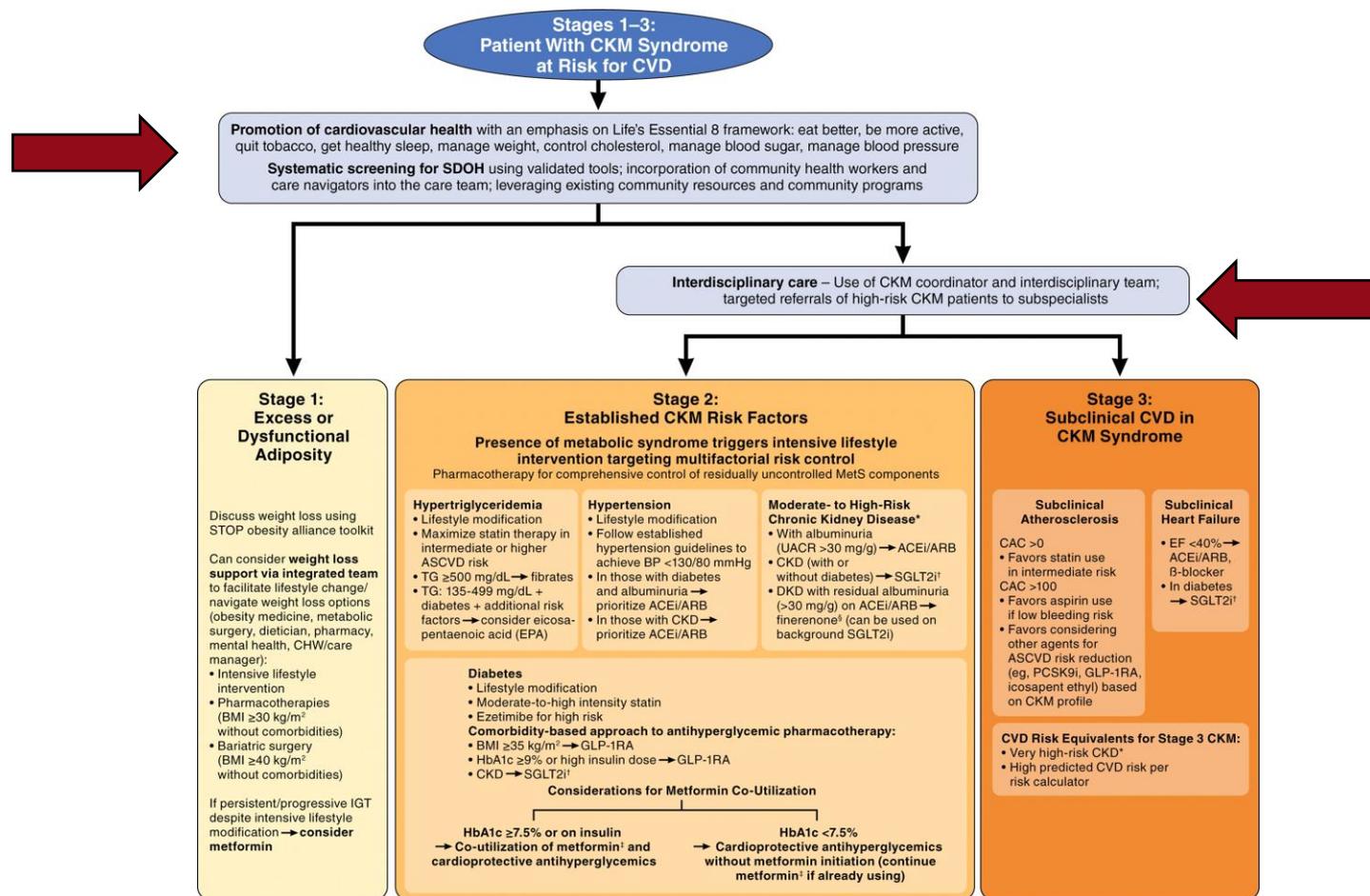


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# CKM SYNDROME



# CKM SYNDROME STAGES 1-3 THOSE AT RISK FOR CVD



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# CKM STAGES 0-2

## STAGE 0<sup>79,86,196,197</sup>

- Attaining and maintaining ideal CVH linked to decreased CVD and mortality
- Multilevel school-based and family-based interventions increase likelihood of ideal CVH
- Avoiding weight gain with aging decreases likelihood of developing CKM risk factors

## STAGE 1<sup>125-128,197,198</sup>

- Nonjudgmental weight loss counseling increases the likelihood of weight loss attempts
- A comprehensive lifestyle intervention is most effective for sustained behavioral changes
- 5%–10% weight loss associated with improved BP, glycemia, and lipids
- ≥10% weight loss associated with lower CVD event rates
- Incretin analogues\* induce >15% weight loss and improve metabolic risk factors
- Bariatric surgery associated with reductions in metabolic risk factors, CVD events, and mortality

## STAGE 2<sup>39,66,71,76,77,129,133,135,138,139,142,143,145,146,151,159,168</sup>

### Hypertension

- BP control reduces risk for multiple CVD outcomes; goal of <130/80 mmHg
- Pharmacotherapy for those with diabetes, CKD, age ≥65 y or ≥10% CVD risk; ACEi/ARB if CKD or diabetes with albuminuria

### Hypertriglyceridemia

- Initial focus on lifestyle changes and addressing secondary causes; use statins if intermediate or higher ASCVD risk
- In those with diabetes + risk factors + triglycerides ≥135 mg/dL, icosapent ethyl lowers CVD risk

### MetS

- Lifestyle changes/weight loss improve MetS components and other pathophysiologic features
- Lifestyle changes accompanied by targeted pharmacotherapy for risk factor control lowers CVD event rates

### Diabetes

- Statins lower CVD event rates; ezetimibe helps achieve 50% LDL-C reduction and further lowers ASCVD risk
- SGLT2i decrease adverse kidney events, HF events, and MACE/CVD mortality
- GLP-1RA reduce weight, glycemia, MACE, and CVD mortality
- Metformin in concert with SGLT2i useful for achieving glycemic targets if HbA1c ≥7.5%

### CKD

- ACEi/ARB in albuminuric CKD decrease adverse kidney and CVD events
- SGLT2i in CKD with eGFR >20 mL/min/1.73 m<sup>2</sup> decrease adverse kidney and CVD events
- Finerenone in CKD with diabetes with eGFR >25 mL/min/1.73 m<sup>2</sup> reduces adverse kidney and CVD events

# CKM STAGES 3-4

## STAGE 3<sup>100,149-151,154-156</sup>

### Subclinical ASCVD

- Presence of CAC favors statin therapy in those with borderline-intermediate ASCVD risk
- CAC score  $\geq 100$  indicates greater net benefit from aspirin and other preventive therapies

### Subclinical HF

- In asymptomatic LV systolic dysfunction, ACEi and  $\beta$ -blockers associated with less HF/CVD mortality
- In diabetes, SGLT2i decrease the risk for incident HF

## STAGE 4<sup>39,59,60,65,71,72,76,77,103,125,129,135,147,151,158,161,163,164,169,170,177,203</sup>

- All ASCVD: Aspirin or P2Y12i + high-intensity statin indicated to reduce ASCVD events; use of additional LDL-C-lowering agents based on the presence of high risk ASCVD and LDL-C thresholds.
- All HF: 4 pillars of GDMT ( $\beta$ -blockers, ARNi, MRAs, SGLT2i) to improve HF outcomes/mortality, particularly for HFrEF

### Obesity and CVD

- Nonjudgmental approach to weight loss discussion improves effectiveness
- Exercise training in obesity and HFpEF improves functional status
- Integrated weight management teams facilitate patient-centered approach
- Incretin analogues induce  $>15\%$  weight loss, improve QOL, and reduce recurrent CVD events
- Bariatric surgery reduces recurrent CVD events and mortality

### Hypertriglyceridemia and CVD

- Statin therapy modestly reduces triglycerides (10%–30%) and lowers ASCVD risk
- Icosapent ethyl reduces CVD events and mortality

### Hypertension and CVD

- BP control reduces recurrent CVD events and mortality; goal  $<130/80$  mmHg
- ACEi/ARB in CVD with CKD or diabetes; in African American patients with HFrEF, hydralazine/isosorbide after 4 pillars of GDMT

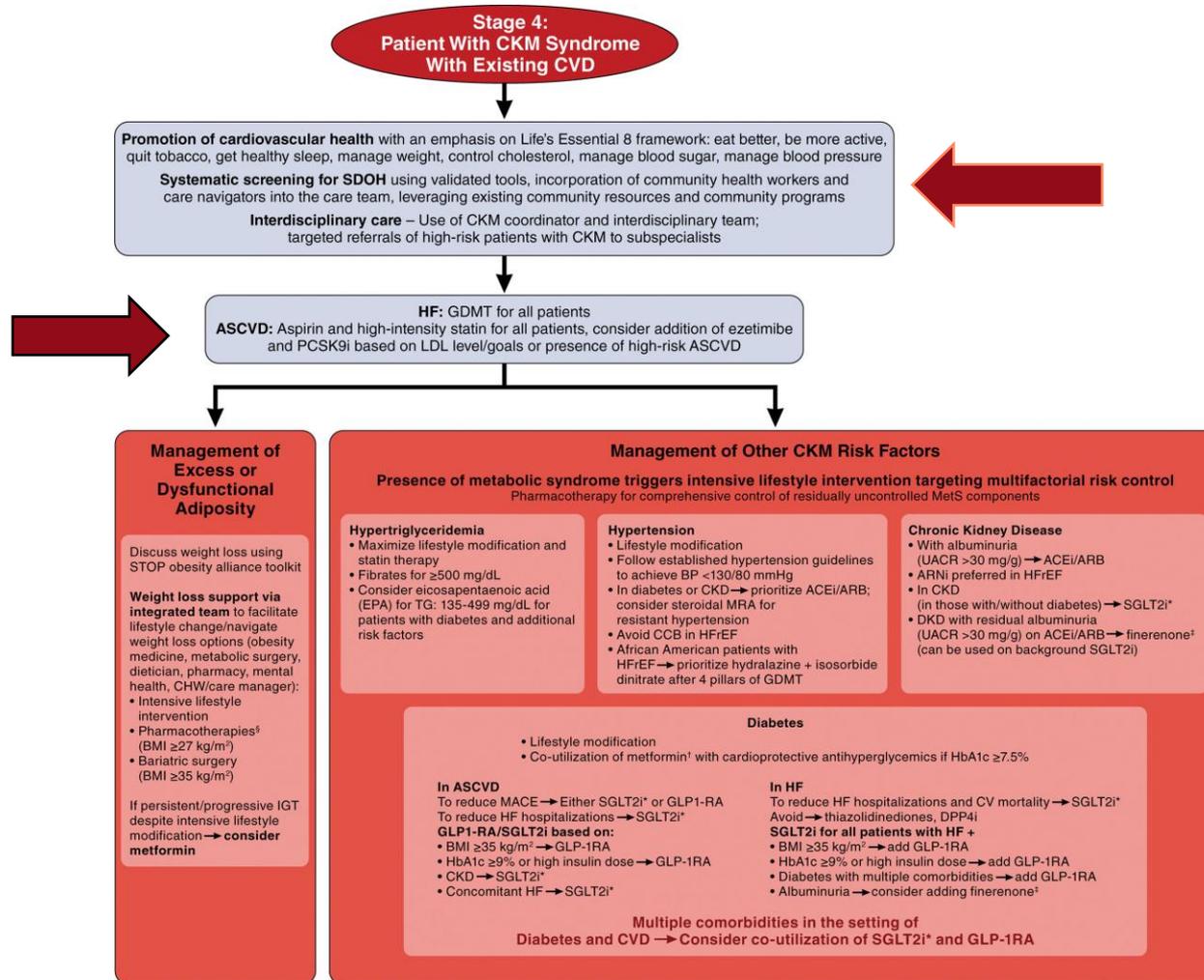
### Diabetes and CVD

- Lifestyle modification improves control of glycemia/risk factors and QOL
- In HF, SGLT2i improve QOL, reduce HF hospitalizations, and reduce mortality risk
- In ASCVD, SGLT2i reduce MACE and HF hospitalizations
- In ASCVD, GLP-1RA reduce weight, glycemia, and MACE

### CKD and CVD

- Statin continuation recommended for reducing recurrent ASCVD events
- ACEi/ARB reduce adverse kidney events and reduce morbidity and mortality rates in CVD
- SGLT2i in CKD with eGFR  $>20$  mL/min/1.73 m<sup>2</sup> reduce adverse kidney events, HF hospitalizations, MACE, and CVD mortality
- Finerenone in CKD with diabetes with eGFR  $>25$  mL/min/1.73 m<sup>2</sup> reduces adverse kidney events and CVD events
- ARNi reduces adverse kidney events, HF hospitalizations, and CV death in HF

# STAGE 4: CKM WITH EXISTING CVD



# CKD - CLASSIFICATION AND DIAGNOSIS

<b>CKD is classified based on:</b> <ul style="list-style-type: none"> <li>• Cause (C)</li> <li>• GFR (G)</li> <li>• Albuminuria (A)</li> </ul>				<b>Albuminuria categories</b> 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
<b>GFR categories (mL/min/1.73 m<sup>2</sup>)</b> 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)

High risk

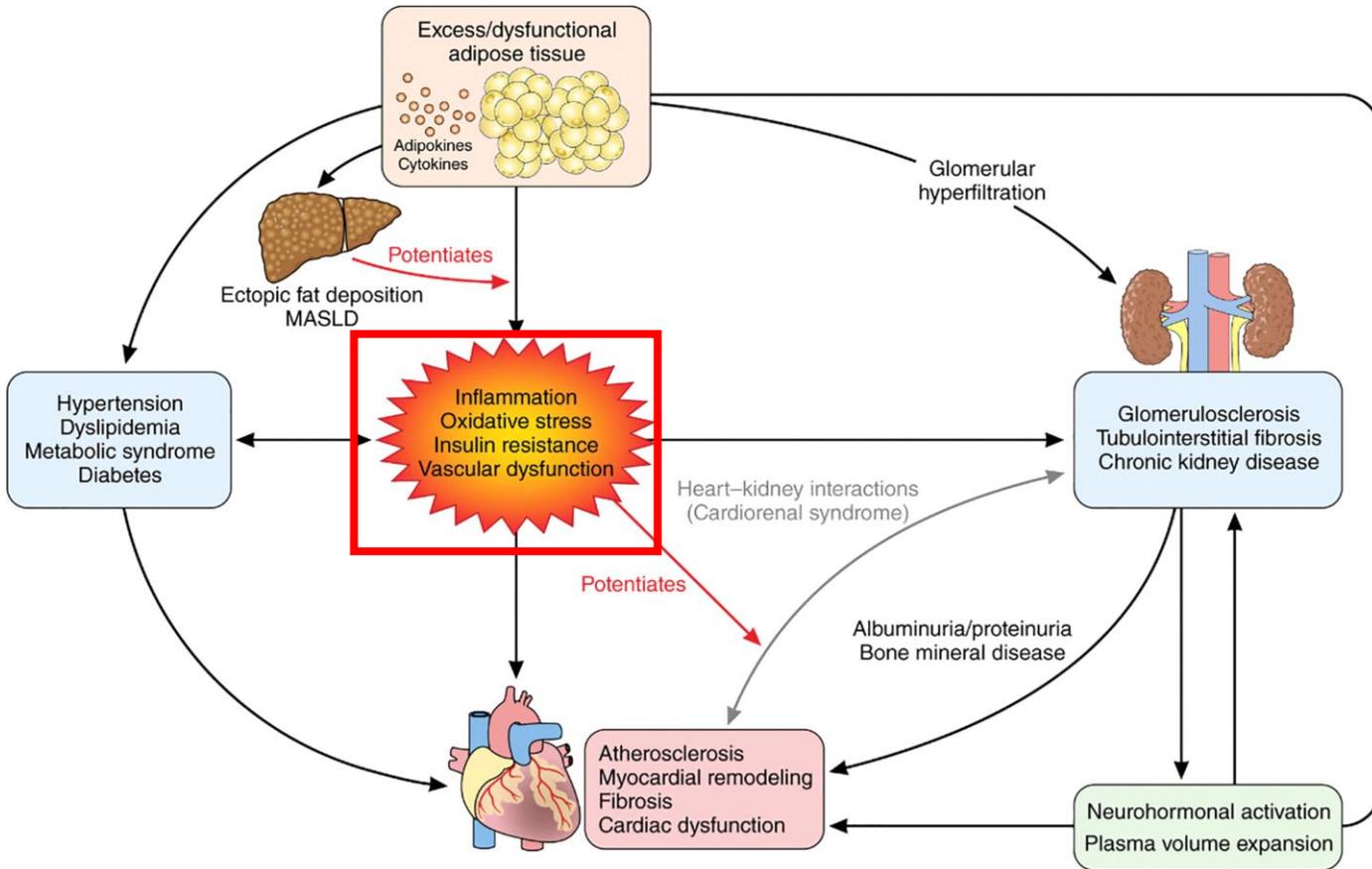
Moderately increased risk

Very high risk



**PATHOPHYSIOLOGICAL  
MECHANISMS IN CKM SYNDROME  
AND OCULAR HEALTH**

# UNDERLYING PATHOPHYSIOLOGY OF CKM SYNDROME



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# INFLAMMATION AS A COMMON PATHWAY

## Inflammation in CKM Syndrome

- Chronic low-grade inflammation in CKM Syndrome results from metabolic issues and promotes vascular injury

## Ocular Inflammatory Impact

- Inflammation disrupts the blood-retinal barrier causing leakage and edema in diabetic and hypertensive retinopathy

## Shared Inflammatory Effects

- Systemic and ocular inflammation accelerate microvascular damage and impair tissue repair

## Therapeutic Importance

- Targeting inflammation is key to managing CKM Syndrome and preventing vision loss

# **OXIDATIVE STRESS AND CELLULAR DAMAGE**

## **Role of Reactive Oxygen Species**

- Excessive reactive oxygen species production causes damage to lipids, proteins, and DNA in CKM Syndrome

## **Ocular Vulnerability**

- Retinas high oxygen consumption and light exposure increase susceptibility to oxidative stress and damage

## **Clinical Manifestations**

- Oxidative stress contributes to diabetic retinopathy and age-related macular degeneration with photoreceptor loss

## **Therapeutic Approaches**

- Antioxidant therapies and lifestyle changes help reduce oxidative damage and improve ocular health in CKM Syndrome.

## **MICROVASCULAR DYSFUNCTION AND ENDOTHELIAL INJURY**

### **Systemic Microvascular Dysfunction**

- CKM Syndrome causes impaired nitric oxide production, vascular stiffness, and capillary loss that reduce tissue blood flow

### **Retinal Microvascular Injury**

- Endothelial dysfunction in retinal vessels leads to ischemia, capillary dropout, and abnormal new vessel growth causing vision loss

### **Therapeutic Strategies**

- Endothelial protection, glycemic control, and anti-angiogenic treatments help preserve cardiovascular and eye health in CKM

# OPHTHALMIC CONNECTION TO PREDICTING SYSTEMIC CKM DISEASE PROGRESSION

## Retina as Microvascular Window

- The retina reflects microvascular health and reveals early signs of systemic vascular dysfunction in CKM.

## Advanced Retinal Imaging

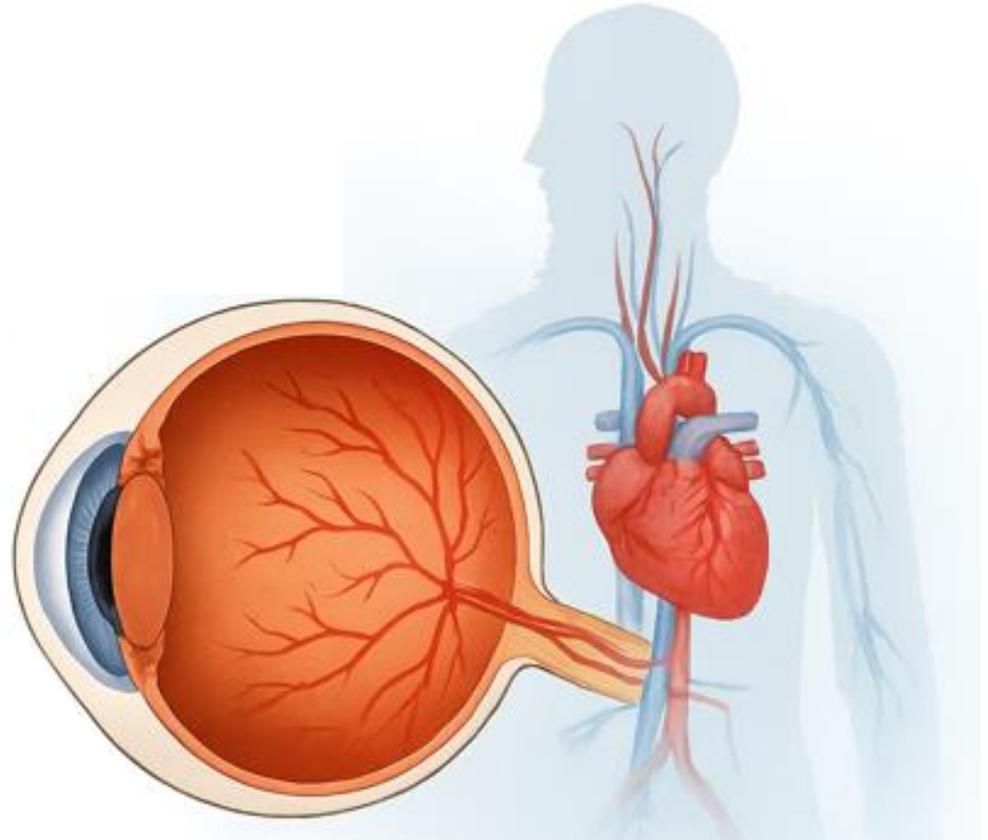
- Techniques like OCT and fundus photography enable detection of subtle retinal vessel changes linked to systemic disease.

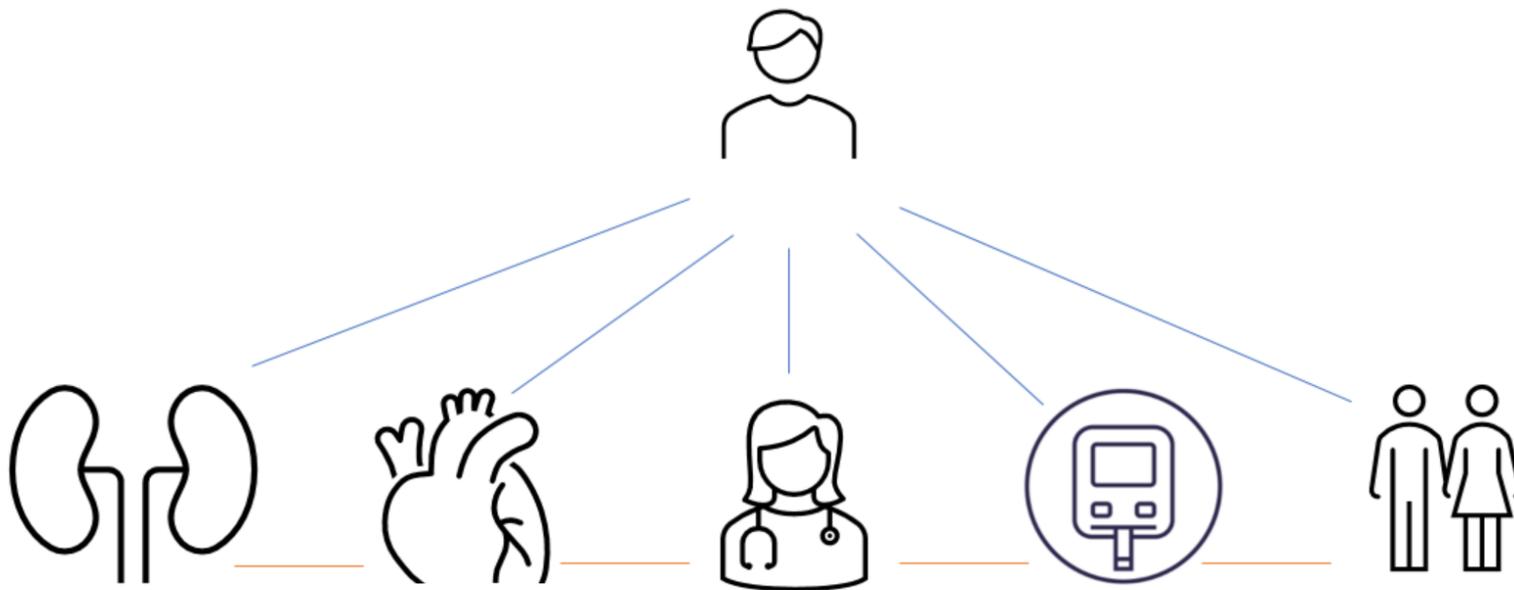
## Indicators of Cardiometabolic Syndrome

- Narrowing arterioles and widened venules correlate with hypertension and insulin resistance in CKM patients.

## Ocular Signs of Systemic Dysfunction

- Microaneurysms and hemorrhages reveal oxidative stress and inflammation mirroring systemic endothelial issues.

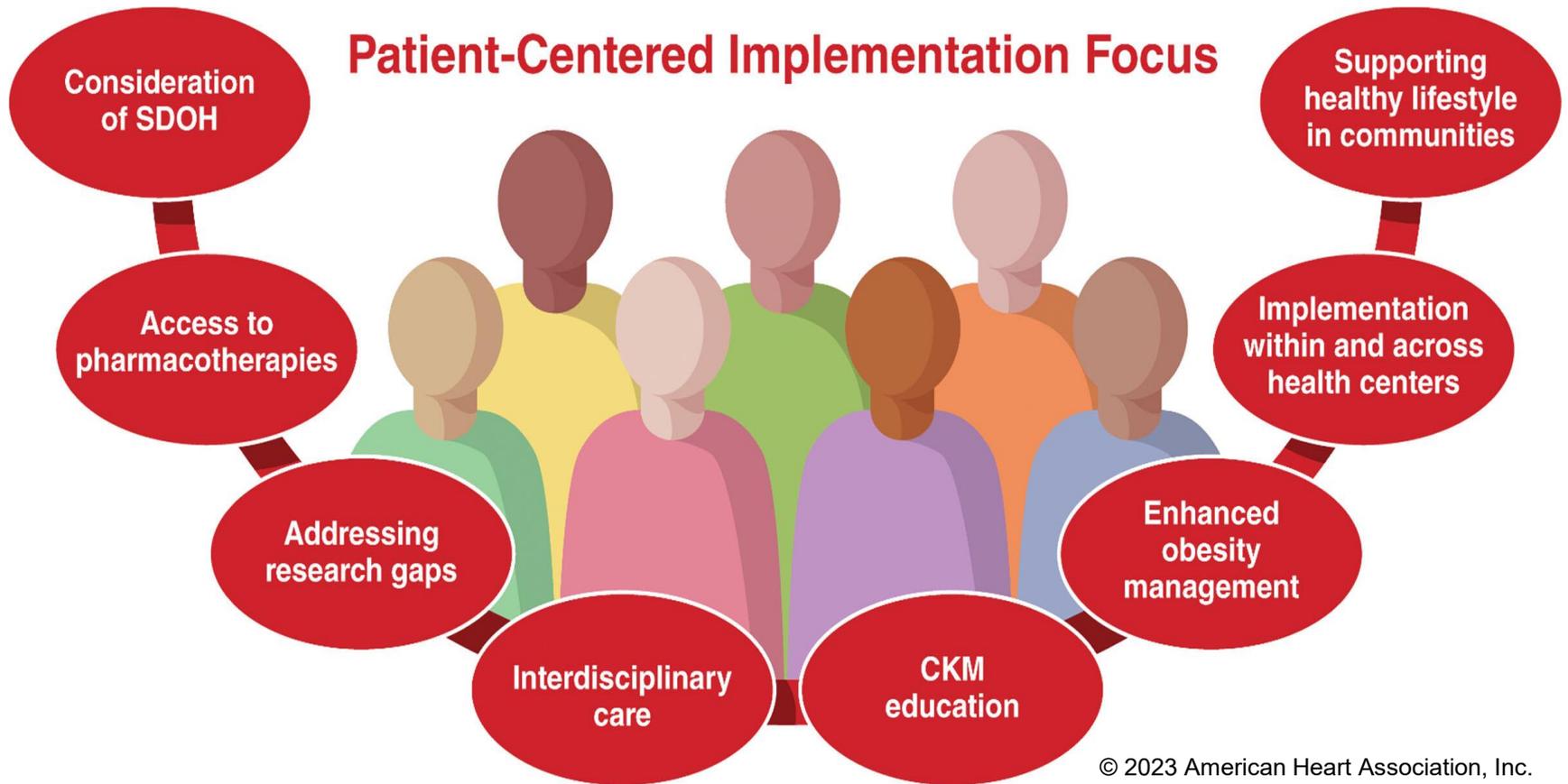




## THE STRAIN OF CHRONIC DISEASE

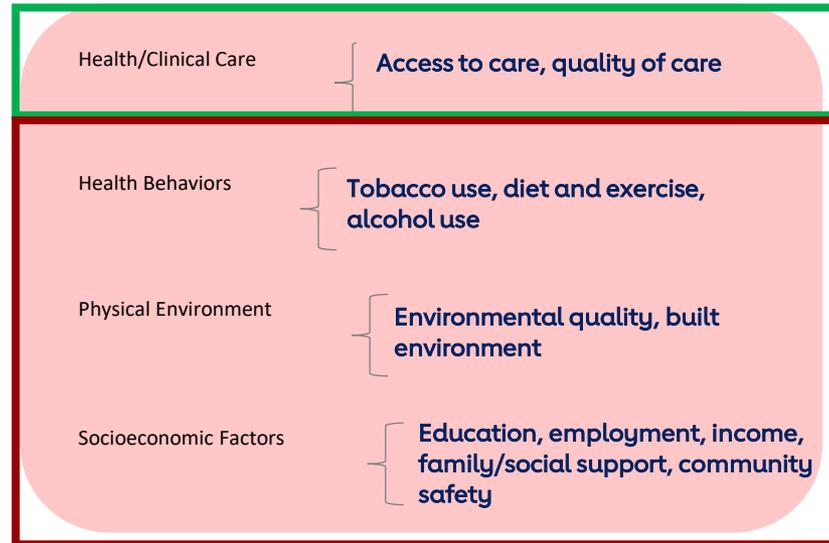
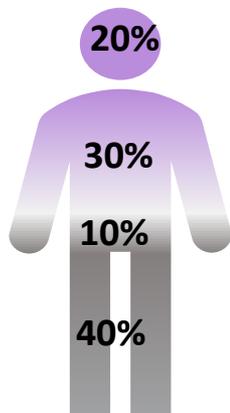
- Large chronically ill populations are straining health care systems
- Need for care coordination
- Decision –making occurring in a vacuum
- No one practitioner is taking the lead
- Coordination of care is left up to ill prepared patients and family members

## Patient-Centered Implementation Focus



# BUILDING THE CASE FOR CARE COORDINATION

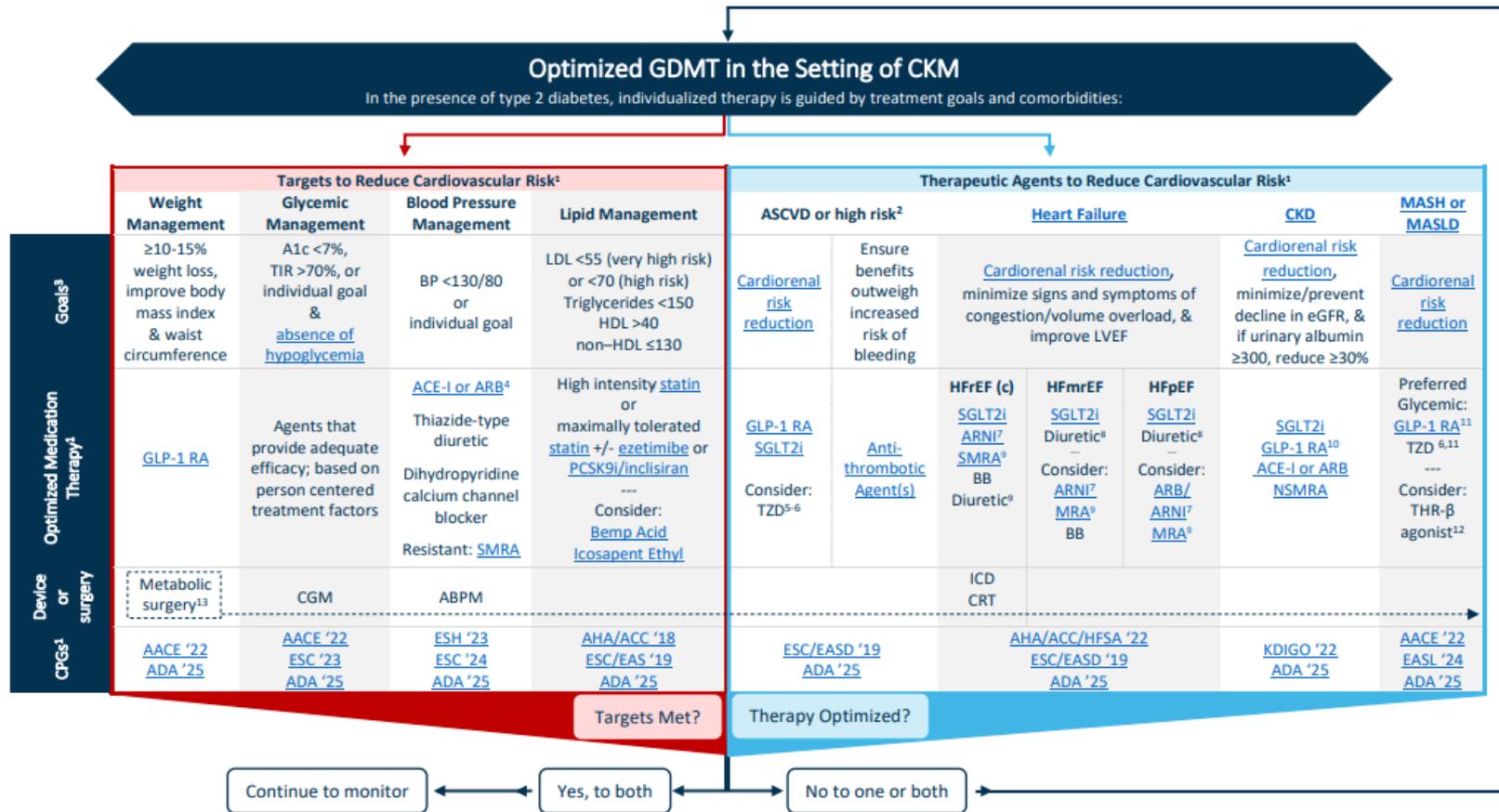
*80% of what influences health is related to social determinants of health—patients' behaviors, socioeconomic conditions and other factors that can be challenging to address in clinical visits*



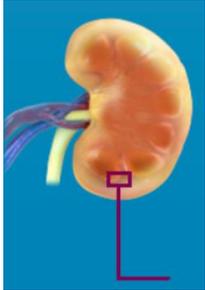


**GUIDELINE DIRECTED MEDICAL  
THERAPY CONSIDERATIONS**

# OPTIMIZED GDMT IN THE SETTING OF CKM



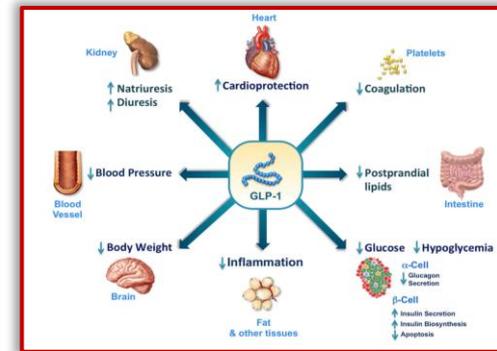
# Different MOAs Providing Overlapping Cardiometabolic Benefits



## SGLT2 Inhibitors

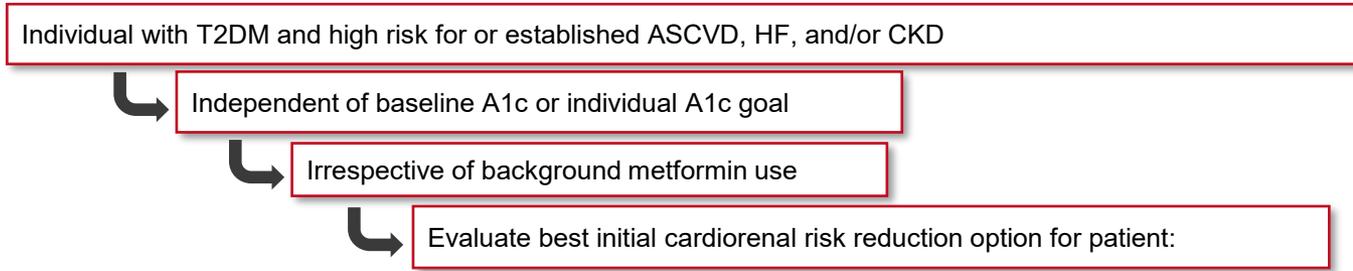
- Glucose filtration
- Reduced glucose and sodium reabsorption
- Decrease in intracellular sodium concentration
- Increased urinary excretion of excess glucose
  
- Diuresis
- Natriuresis
- HbA1c reduction
- Weight loss
- SBP reduction

## GLP1 receptor agonists



- Increased insulin
- Decreased glucagon
- Decreased gastric emptying
  
- Weight loss
- Reduced blood pressure
- Improvement in lipids
- Decrease inflammation

# Selecting T2DM Medication for Cardiorenal Risk Reduction



High risk for ASCVD:  $\geq 55$  years of age with two or more additional risk factors (e.g., obesity, hypertension, smoking, dyslipidemia, or albuminuria)

	SGLT2i	GLP-1 RA
<b>CV Mortality</b>	++	++
<b>Total Mortality</b>	++	++
<b>MACE</b>	++	++
<b>Heart Failure/HHF</b>	+++ (any/all EF)	++ (HFpEF)
<b>CKD/DKD</b>	+++	++
<b>Improve Glycemic Control</b>	++	+++
<b>Obesity</b>	-	+++
<b>MASH/MASLD</b>	-	++
<b>OSA</b>	-	++

Realizing that combination therapy is often indicated, therapies may be started in tandem or sequentially.

ADA (2025) states that the pharmacologic regimen should include agents that reduce cardiorenal risk (e.g., SGLT2i, GLP-1ra), with or without metformin use; ESC (2019) indicates that SGLT2i and/or GLP-1ra may be prioritized over metformin based on the presence of CVD and CV risk.

# Selecting T2DM Medication for Cardiorenal Risk Reduction

## SGLT2i

Patient Preference: Daily oral administration

Reasons to consider exclusion or delay initiation:

- Initiation not recommended if eGFR <20; once initiated, continue until dialysis or transplant
- History of prior amputation, severe PAD, or active diabetic foot ulcer
- History of recent genital mycotic infection
- History of diabetic ketoacidosis

and/or

## GLP-1 RA

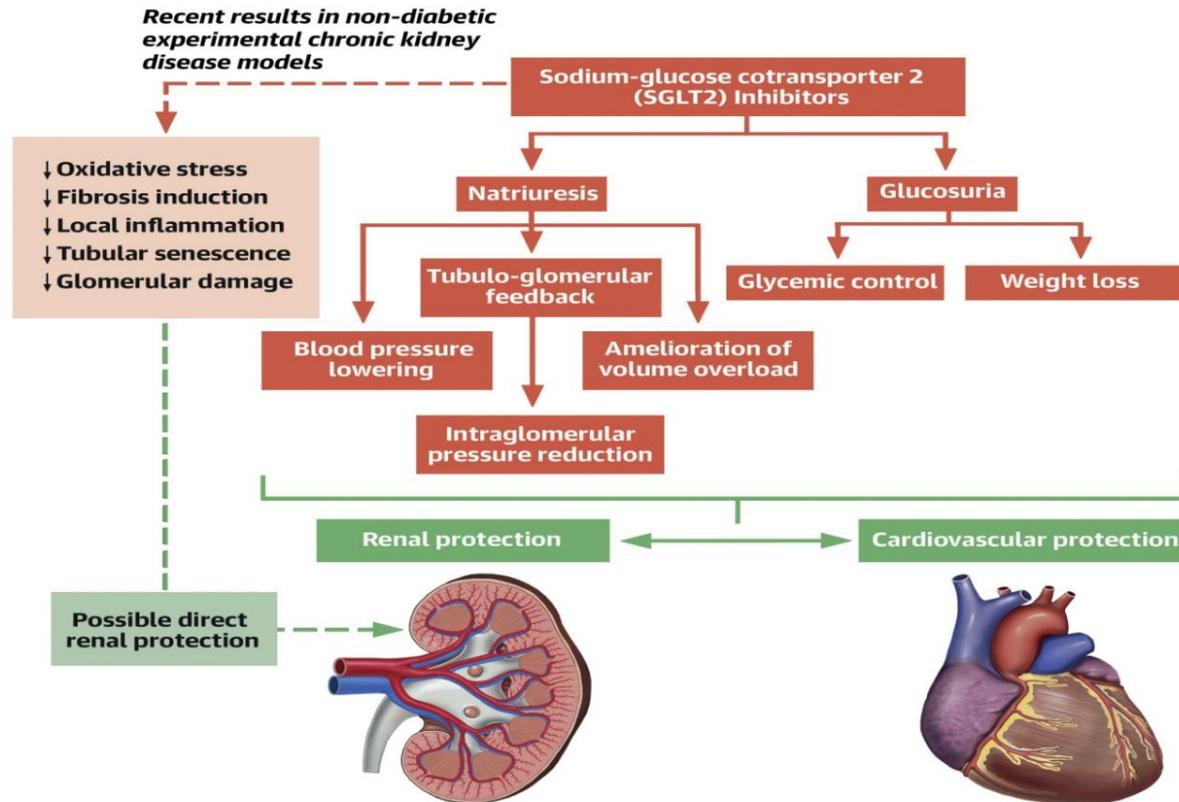
Patient Preference: Once weekly administration

Reasons to consider exclusion:

- Persistent nausea despite dietary changes and lower doses
- History of gastroparesis
- Active, symptomatic gallbladder disease
- Active or recent pancreatitis
- History of MEN2 or medullary thyroid cancer
- History or active proliferative retinopathy, consider risk/benefit especially if A1c poorly controlled

# SGLT2 Inhibitors

## CENTRAL ILLUSTRATION: Sodium-Glucose Cotransporter 2 Inhibitor Cardiorenal Protection Mechanistic Overview



Zelniker, T.A. et al. J Am Coll Cardiol. 2020;75(4):422-34.

# SGLT2 INHIBITOR

	Bexagliflozin	Canagliflozin	Dapagliflozin	Empagliflozin	Ertugliflozin	Sotagliflozin <sup>e</sup>	Indication <sup>a</sup>
Population	T2DM	T2DM	T2DM	T2DM	T2DM		Improve Glycemic Control
		T2DM + DKD					Reduce Risk of Doubling of Serum Creatinine, ESKD
			CKD <sup>b</sup>	CKD <sup>b</sup>			Reduce Risk of Sustained eGFR Decline, ESKD
		T2DM + DKD	T2DM + CVD <sup>c</sup> HF <sup>b</sup> CKD <sup>b</sup>	HF <sup>b</sup>		HF <sup>b</sup> T2DM + CKD + CV RFs	Reduce Risk of HF Hospitalization <sup>d</sup>
				CKD <sup>b</sup>			Reduce Risk of Hospitalization
		T2DM + DKD	HF <sup>b</sup> CKD <sup>b</sup>	T2DM + CVD HF <sup>b</sup> CKD <sup>b</sup>		HF <sup>b</sup> T2DM + CKD + CV RFs	Reduce Risk of CV Mortality
		T2DM + CVD				Reduce Risk of MACE	

<sup>a</sup> Indications apply to the specific population for which the medication is approved

<sup>b</sup> Specified population with or without type 2 diabetes (T2DM)

<sup>c</sup> Either established cardiovascular disease (CVD) or multiple risk factors (RFs)

<sup>d</sup> Dapagliflozin and sotagliflozin have an indication to reduce the risk of urgent heart failure (HF) visits in addition to hospitalizations

<sup>e</sup> Sotagliflozin is an inhibitor of SGLT2 and SGLT1

# SGLT2I USE & CONCOMITANT MEDICATIONS

Prevention of Hypoglycemia	Prevention of Volume Depletion
<ul style="list-style-type: none"> <li>Risk for Hypoglycemia is only increased in the setting of concomitant use of insulin secretagogues &amp;/or insulin</li> <li>Patients should be advised of the risk of potential hypoglycemia when adding new therapies</li> <li>Advise patients to self-monitor blood glucose closely for 3 to 4 weeks after SGLT2i initiation</li> </ul>	<ul style="list-style-type: none"> <li>In combination with loop diuretics there is a potential for additional natriuretic effects when SGLT2i are added</li> <li>Monitor for signs of volume depletion</li> <li>Advise patients to report dizziness, lightheadedness upon standing , monitor blood pressure</li> </ul>
<p style="text-align: center;"><b><u>Should be INDIVIDUALIZED</u></b></p> <ul style="list-style-type: none"> <li>Up to a 50% reduction in oral secretagogues/or up to 50% of the maximum recommended dose</li> <li>20% reduction in total daily dose of insulin*</li> <li>*ensure T2DM</li> </ul>	<p style="text-align: center;"><b><u>Should be INDIVIDUALIZED</u></b></p> <ul style="list-style-type: none"> <li>Potential decrease in diuretics</li> </ul>

# SGLT2I.....WHAT PATIENTS NEED TO KNOW

Taking an SGLT2i may improve heart and kidney health even if blood sugar (A1c) is in goal.



## Who may benefit from taking an SGLT2i?

People with:

- Type 2 diabetes
- Heart failure
- Kidney disease
- Heart and blood vessel disease (ASCVD)
- High risk for heart failure or ASCVD



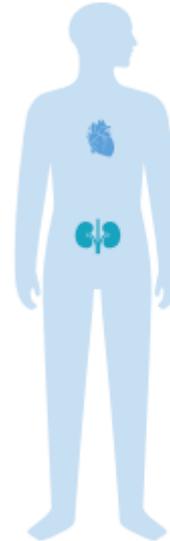
## Where does my SGLT2i work?

**Kidneys:** Works with my kidneys to flush sugar out of my urine\* and may improve my heart's ability to pump blood to my body



## When should I take my SGLT2i?

Take in the morning with or without food



## Why should I take my SGLT2i?



**To lower** risks of heart failure worsening, heart attack, stroke, and dying from heart disease



**To reduce** the risk of worsening kidney function



**To decrease\*** blood sugar with minimal risk of low blood sugar

## Additional benefits



**To promote** modest weight loss



**To help decrease** blood pressure



## Possible side effects and how to prevent them

**Infection:** Itching, irritation, or discharge in the genital area; may include genital yeast or urinary tract infections

- Usually mild and treatable
- Risk of infection decreases with improved blood glucose levels
- Keep genital area clean and dry

**Dehydration:** Dizziness, thirst, fast heart rate, lightheadedness (especially standing)

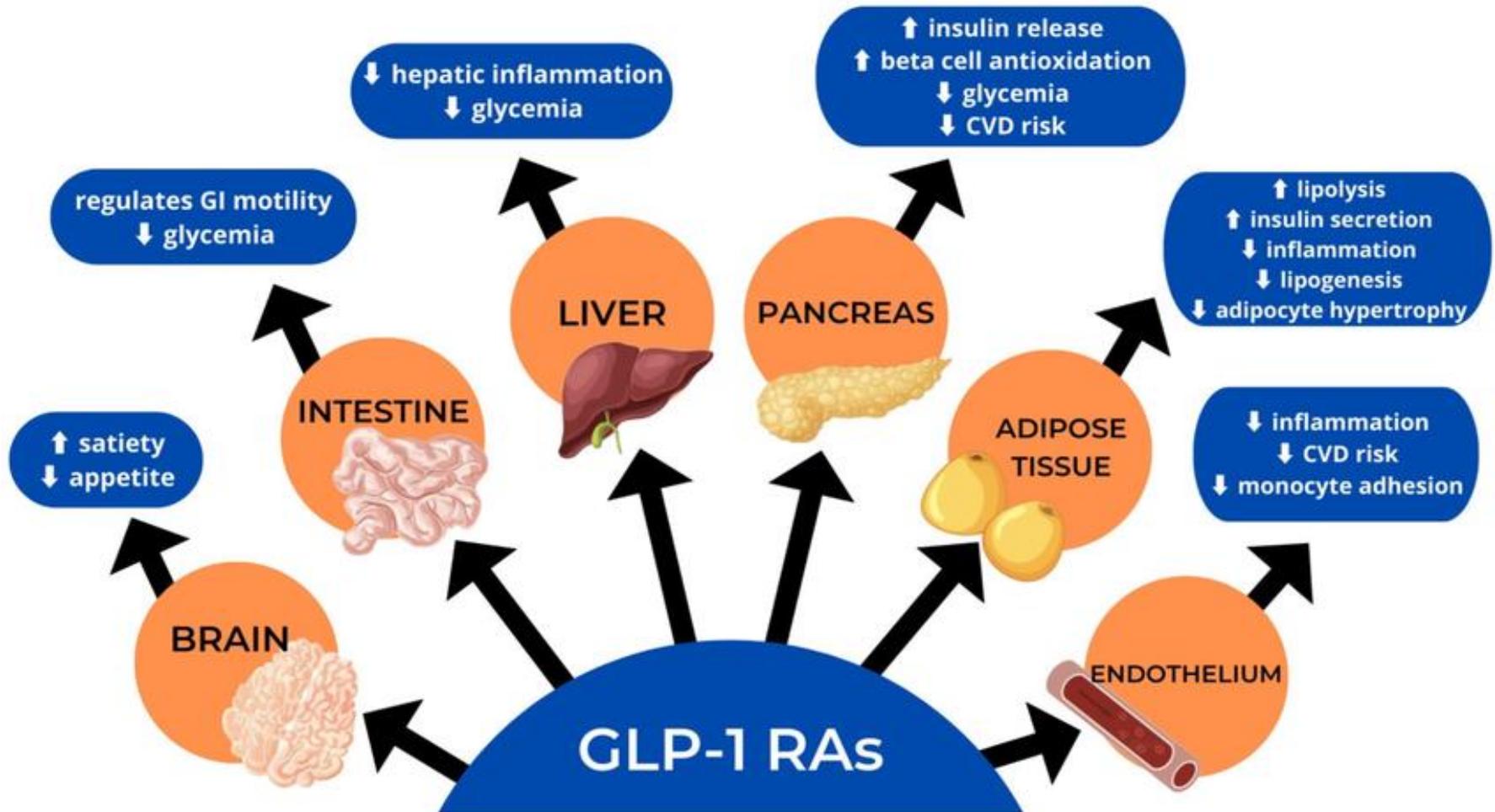
- Stay well hydrated, avoiding alcohol and caffeine
- Use caution when active or in hot weather
- Monitor blood pressure more often

**Low blood sugar\*:** Shakiness, sweateness, dizziness, fatigue, irritability

- Avoid skipping meals
- Time medications based on meals
- Monitor blood sugar more often
- If needed, discuss changes to other diabetes medication with my doctor

**Less common side effects:** Ketoacidosis (increased ketones in blood or urine—higher acid levels)

# Therapeutic Activity of GLP1ra



# Indications by Agent

Indications	Dulaglutide	Exenatide	Liraglutide	Lixisenatide	Semaglutide INJ	Semaglutide oral	Tirzepatide**
<b>Glucose Lowering in T2DM</b>	Yes	Yes	Yes	Yes	Yes	Yes	Yes
<b>Obesity</b>	-	-	Yes	-	Yes	-	Yes
<b>Reduction of MACE</b>	Yes*	-	Yes*	-	Yes*	-	-
<b>Reduction of eGFR Decline, ES-CKD, &amp; CV Death</b>	-	-	-	-	Yes*	-	-
<b>Obstructive Sleep Apnea</b>	-	-	-	-	-	-	Yes*

\*Established or multiple risk factors

\*\*Tirzepatide is a dual GIP/GLP-1.

# Precautions for Use: Mitigating Risk

## Gastroparesis

Since GLP-1 RA agents are not recommended in the presence of gastroparesis, clinical confirmation (via positive radiologic gastric emptying study) is recommended as symptoms of gastroparesis can mimic other benign gastrointestinal conditions.

## Pancreatitis

Patients with active pancreatitis are not candidates for GLP-1 RA therapy; those with a history of resolved pancreatitis and clearly identified precipitating factor (unrelated to a GLP-1 RA) may still be candidates for treatment depending on the etiology.

## Nausea

If prior intolerance due to nausea, thoroughly assess patient's previous medication experience and adherence to meal reduction plan before excluding from future use.

# Precautions for Use: Mitigating Risk

## Proliferative Retinopathy

In patients with history of proliferative retinopathy GLP-1 RA may be associated with worsening retinopathy. However, this association appears to be strongly correlated with rapid improvement in glycemic control that often occurs when an intensive glucose lowering therapy is initiated. Slower titration of the GLP-1 RA and monitoring of symptoms of retinopathy is highly encouraged.

## Anesthesia

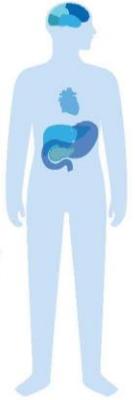
Patients undergoing anesthesia and concurrently taking a GLP-1 RA are theoretically at risk of aspiration due to delayed gastric emptying. According to ASA guidelines, most can continue therapy as metabolic disruption risk may outweigh risk of aspiration. Rather than hold therapy, those at high risk for GI problems should follow liquid diet prior to procedure.

# GLP1RA.....WHAT PATIENTS NEED TO KNOW

**Taking a GLP-1 RA may improve heart health even if blood sugar (A1c) is in goal.**

**Who may benefit from taking a GLP-1 RA?**  
 People with:

<input type="checkbox"/> Type 2 diabetes	<input type="checkbox"/> Obesity
<input type="checkbox"/> Heart and blood vessel disease (ASCVD)	<input type="checkbox"/> Chronic kidney disease
<input type="checkbox"/> High risk for ASCVD	<input type="checkbox"/> Obstructive sleep apnea
	<input type="checkbox"/> Metabolic liver disease (MASH)



**Why should I take my GLP-1 RA?**

- To lower risks of heart attack, stroke, and dying from heart disease
- To decrease blood sugar
- To promote significant weight loss
- To improve sleep
- To reduce the risk of worsening kidney function
- To improve liver function

**Additional benefits**

- To reduce the need for insulin with minimal risk of low blood sugar
- To help decrease blood pressure

**Where does my GLP-1 RA work?**

- **Muscles:** Makes my body more sensitive to insulin
- **Liver and pancreas:** Lowers my blood sugar levels after meals
- **Stomach:** Slows my stomach emptying
- **Brain:** Decreases my appetite and cravings

**When should I take my GLP-1 RA?**  
**Injectable:** Any time of day, with or without food

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**Possible side effects and how to prevent them**

<p><b>Nausea:</b> Feeling too full, stomach discomfort, urge to vomit</p> <ul style="list-style-type: none"> <li>• Reduce meal size by 50%</li> <li>• Begin meal with protein</li> <li>• Avoid meals with portions that are larger than fist size</li> <li>• Stop eating when full, even if meal is not finished</li> </ul>	<p><b>Constipation:</b> Less frequent bowel movements, stools are hard to pass or painful, stools are dry and hard</p> <ul style="list-style-type: none"> <li>• Increase fiber and water in diet</li> <li>• Avoid alcohol and caffeine</li> <li>• Consider a stool softener, if needed</li> </ul>	<p><b>Low blood sugar:</b> Shakiness, sweateness, dizziness, fatigue, irritability</p> <ul style="list-style-type: none"> <li>• Avoid skipping meals</li> <li>• Time medications based on meals</li> <li>• Monitor blood sugar more often</li> <li>• If needed, discuss changes to other diabetes medication with your doctor</li> </ul>
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**Less common side effects:** Diarrhea, abdominal pain, gastric reflux

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**Decrease my risk of complications**

Foot care 
 Eye care 
 Dental care 
 Kidney health 
 Avoid tobacco use

Eat more heart-healthy foods 
 Increase physical activity 
 Work toward my weight loss goals

**My goal numbers:**

Blood sugar range	A1c	Cholesterol	Blood pressure
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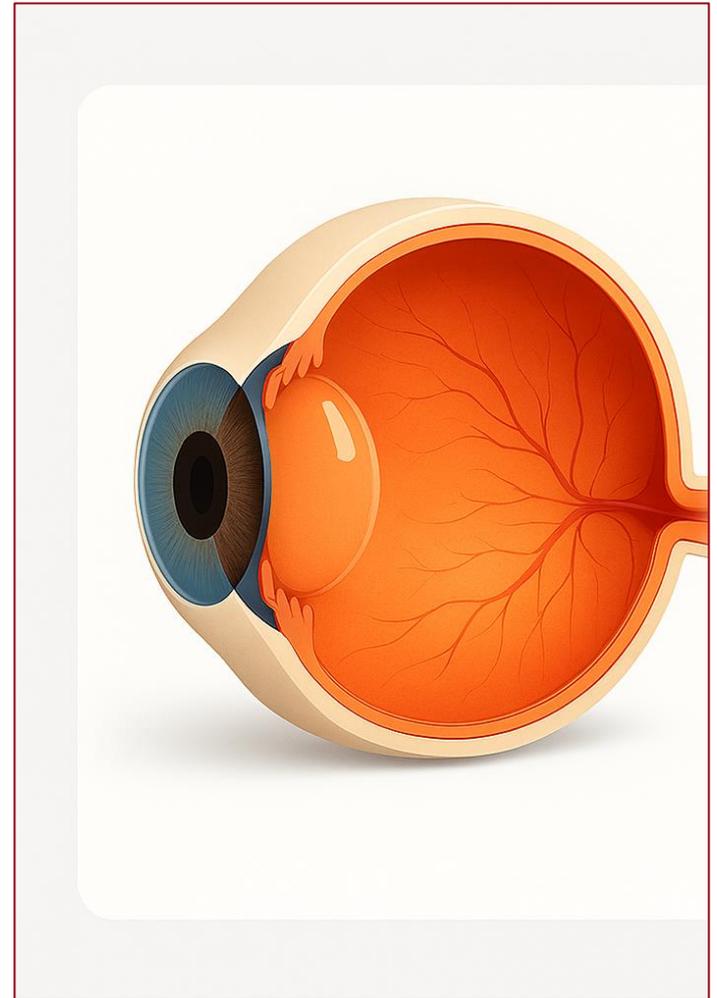
# Diabetic Retinopathy

## CLINICAL TRIAL EVIDENCE

SUSTAIN-6: HR 1.76 for DR complications.

Meta-analysis: risk linked to rapid HbA1c drop.

Real-world: HR 1.07 for incident DR.



# NON-ARTERITIC ANTERIOR ISCHEMIC OPTIC NEUROPATHY



NAION: very rare (~1 in 10,000)



Case reports: optic neuropathies, papillitis



JAMA cohort: no significant NAION increase

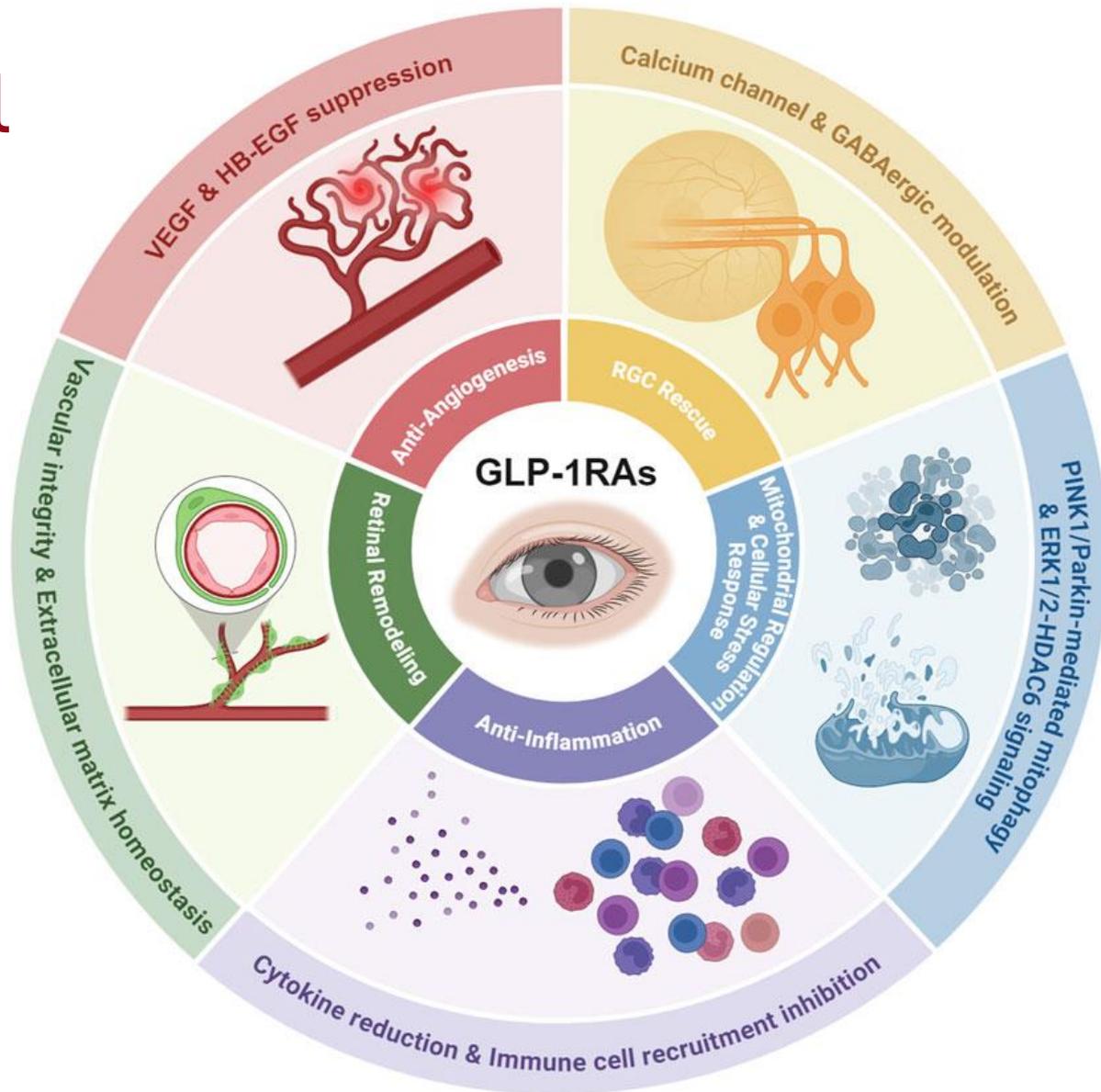
# ADULT MACULAR DEGENERATION

Study	Population	AMD Type	Association with GLP-1RAs
Ahuja et al. (2025), JAMA Ophthalmology	Non-diabetic, 55+ adults	Nonexudative	Reduced risk (RR $\approx$ 0.09 at 10 years)
Albanese et al. (2025), Diabetology Review	Broad clinical/lab data	Nonexudative & Exudative	Conflicting evidence— signals of both benefit and risk

Ahuja, A., et al. (2025). Glucagon-like peptide-1 receptor agonists and age-related macular degeneration. *JAMA Ophthalmology*, 143(2), 123–131. <https://doi.org/10.1001/jamaophthalmol.2024.5678>

Albanese, G., et al. (2025). Ocular effects of GLP-1 receptor agonists: A review of current evidence and safety concerns. *Diabetology*, 6(10), 117. <https://doi.org/10.3390/diabetology6100117>

# GLP1RA Potential Impact



# GUIDELINES & SCREENING



Baseline eye exam before GLP-1RA  
initiation



Close follow-up for moderate/severe DR



Multidisciplinary care recommended

# CLINICAL MANAGEMENT



Gradual titration to avoid rapid HbA1c drop.



Educate patients on visual symptoms.



Coordinate care

# FUTURE DIRECTIONS



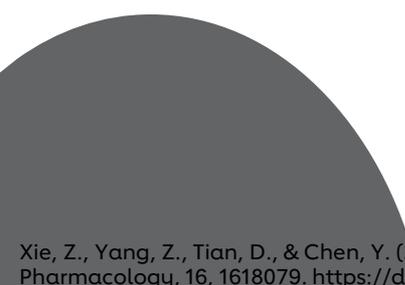
Explore neuroprotective effects

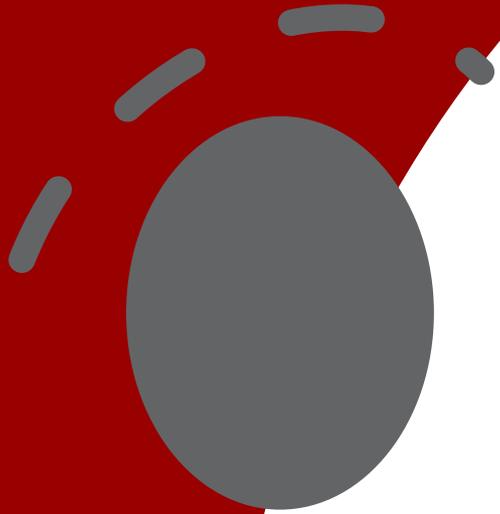


Long-term registry data needed



Identify patient-level risk stratifiers





# **THE NEED FOR A COLLABORATIVE APPROACH**

# NECESSARY COLLABORATION

## The Problem:

Fragmented care sets the stage for missed diagnosis and an increased risk for complications

## The Goal:



TIMELY  
INTERVENTION



EARLY DETECTION

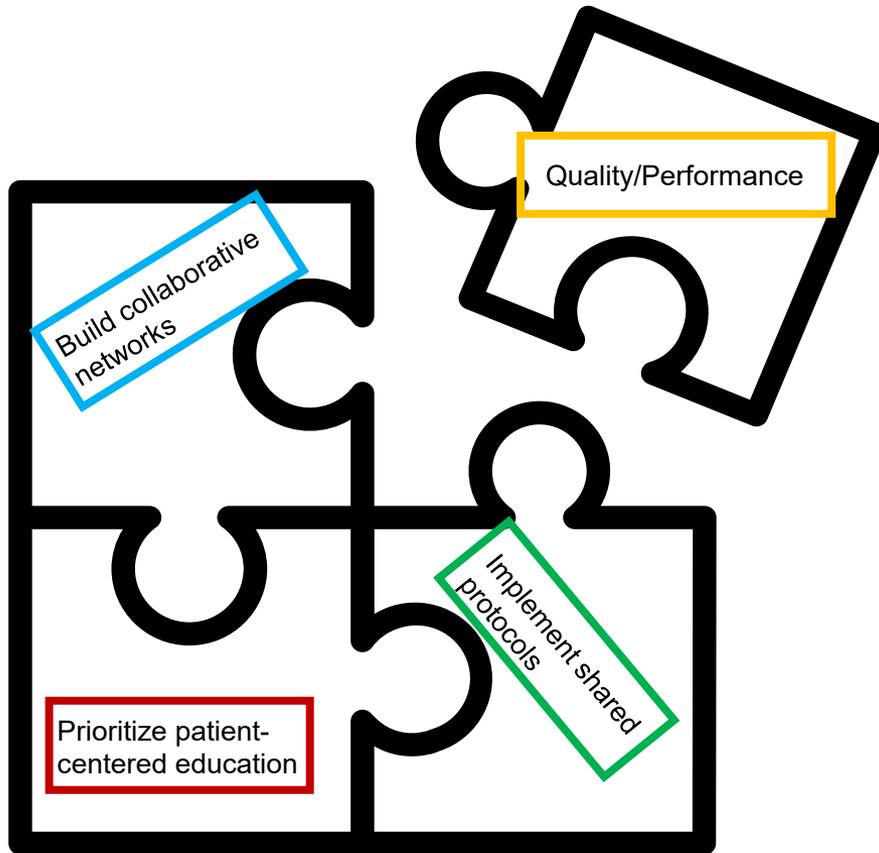


COLLABORATIVE  
CARE



IMPROVED PATIENT  
OUTCOMES

# Putting the pieces together



- Eye health reflection of systemic health
- Multiple conditions undiagnosed without proactive screening

- Ensures appropriate ,timely referrals
- Reduction in complications
- Compliance with quality measures (MIPS etc. )

- Challenges – siloed systems, lack of standardized reporting
- Solutions- EMR integration, secure messaging, collaborative protocols

- Link between eye health and systemic health
- Early detection = improved outcomes
- Importance of routine exams

# CKM related quality measures... its all relative

## MIPS

Quality (30%)

Promoting Interoperability (25%)

Improvement Activities ( 15%)

Cost ( 30%)

Measure 117: Diabetes: Eye Exam  
Measure 130: Documentation of Current Medications in the Medical Record  
Measure 236: Controlling High Blood Pressure

Measure 238: Use of High-Risk Medications in Older Adults  
Measure 317: Preventive Care and Screening for High Blood Pressure and Follow-Up Documented  
Measure 374: Closing the Referral Loop: Receipt of Specialist Report

Improvement Activities: adoption of processes that demonstrated improvement in efficiency, quality and outcomes

# CONCLUSION

- 👁️ **CKM Syndrome and Eye Health Are Interconnected**

Ocular manifestations often reflect systemic cardiometabolic dysfunction, making ophthalmic care a critical component of CKM management.

- ❤️ **GLP-1 RA Therapy: Benefits and Vigilance**

While GLP-1 receptor agonists offer significant cardiometabolic advantages, rapid glycemic improvement can pose ocular risks—necessitating proactive monitoring.

- 🔍 **Early Detection Improves Outcomes**

Routine eye exams and advanced imaging can serve as non-invasive predictors of systemic disease progression.

- 🤝 **Collaboration Is Essential**

Interdisciplinary care—linking ophthalmology, endocrinology, cardiology, and primary care—ensures timely intervention and reduces complications.

- 🧩 **Patient-Centered Education**

Empowering patients with knowledge about CKM risks and vision health fosters adherence and better long-term outcomes.

**THANK YOU**

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