Helping Your Patients with Diabetes
Slowing the Progression of Diabetic Kidney Disease in T2D
Part 1 – Screening, Diagnosis & Monitoring

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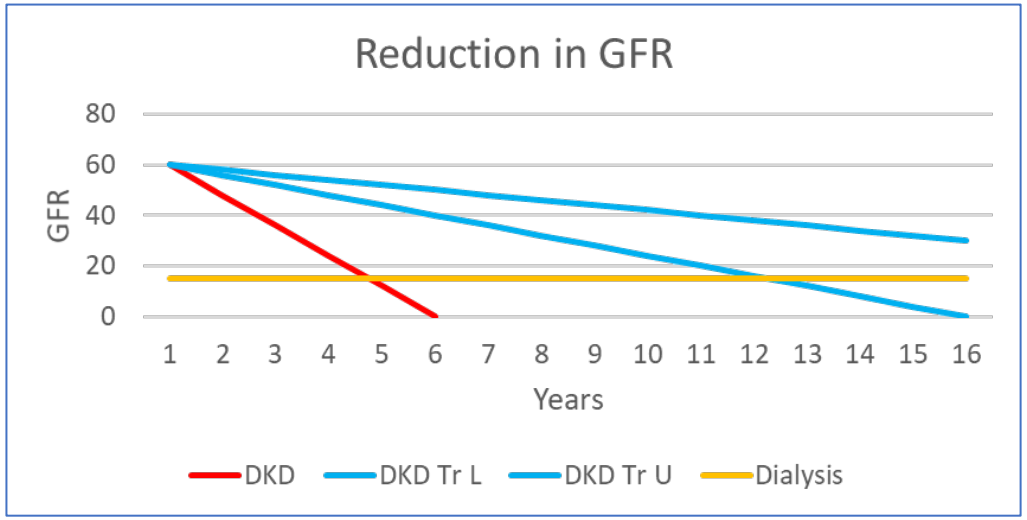
Objectives for Part 1

- Overarching objective - Improve health outcomes for your patients with T2D and diabetic kidney disease (DKD)
  - Review the ADA standards of care for detection of and monitoring of diabetic kidney disease
  - Recognize the impact of albuminuria on patient outcomes
  - Identify contributors to acute kidney injury
  - Differentiate BP results from standardized measurement vs routine measurement techniques

Background – Glomerular Filtration Rate

- Normal GFR >90 ml/min/1.73M2
  - Hyperfiltration >120
  - Chronic Kidney Disease (CKD) <60 for =/>3 months
- GFR declines with normal aging (>30) – average 0.75-0.8 ml/min/year (GFR 50-60 by age 80)
- Rate of decline of GFR in Diabetic Kidney Disease (DKD) – average 12 ml/min/year
- Decline in GFR associated with structural & functional changes in the kidney → complications of CKD (anemia, bone, electrolytes, hypoglycemia, etc.)
  - At GFR<15 – require renal replacement
- Treatment of DKD can slow decline in GFR from average of ***12 ml/min/year*** to an average of ***2-4 ml/min/year*** – thus delaying or avoiding ESRD (ESKD) – and at same time help reduce CVD/HF risk



Diabetic Kidney Disease (DKD)
Chronic Kidney Disease (CKD) attributed to Diabetes

- Occurs in 20-40% of patients with diabetes (<10% are aware) (>90% unaware)
  - In type 1 diabetes, usually develops after diabetes duration of 10 years
  - In type 2 diabetes, may be present at diagnosis
- Can **lead to end-stage renal disease (ESRD)** requiring dialysis or kidney transplantation
  - Is the **leading cause of ESRD (ESKD)** in the US (HTN is second leading cause)
- Markedly **increases cardiovascular risk** and **health care costs**
  - ***Even in early stages***, CKD has been associated with an ***increased cardiovascular morbidity and mortality***
  - Therefore, *early detection of CKD* is essential to
    - retard the progression of kidney disease
    - reduce cardiovascular morbimortality
    - reduce the economic burden caused by kidney disease.

Screening for DKD

American Diabetes Association Standards of Care

Recommendations

- 11.1a At ***least annually*** , urinary albumin (e.g., spot urinary albumin-to-creatinine ratio (**UACR**)) and estimated glomerular filtration rate (**eGFR**) should be assessed
  - in patients with type 1 diabetes with duration of =/>5 years and
  - in all patients with **type 2 diabetes** regardless of treatment (at diagnosis) B
- 11.1b Patients with diabetes and urinary albumin >300 mg/g Cr and/or an estimated GFR 30-60 ml/min/1.73 m2 should be monitored ***twice annually*** to guide therapy. B

A = clear evidence from well conducted trials

B = supportive evidence from well conducted trials

C = supportive evidence from poorly controlled or uncontrolled studies

ADA Standards of Care – Screening for Nephropathy

- *"Screening for albuminuria can be most easily performed by **urinary albumin-to-creatinine ratio (UACR)** in **random spot urine** collection."*
  - Measurement of spot urine sample for *albumin alone* without simultaneously measuring urine creatinine (Cr) is less expensive but susceptible to false-negative and false-positive determination
  - Timed or *24-h collections* are more burdensome & add little to accuracy

- Normal UACR is defined as <30 mg/g Cr (high UACR is defined as =/>30 mg/g Cr; very high =/>300 mg/g)
  - Because of high biologic variability in urinary albumin excretion, **two of three UACR specimens** collected within a ***3-to-6-month period*** should be abnormal before considering a patient to have high or very high albuminuria.
  - UACR can be *elevated independently of kidney damage* by
    - Exercise within 24-hour
    - Infection
    - Fever
    - Congestive Heart Failure
    - Marked hyperglycemia
    - Marked hypertension
    - Menstruation

2 out of 3 abnormal

Causes of False Positive UACR

Fever Infection
CHF Menstruation
Exercise (within 24h)
Marked hyperglycemia
Marked hypertension

ADA Standards of Care – additional comments re screening

- An **eGFR** persistently <60 ml/min/1.73 m2 is considered abnormal
  - For older adults, optimal thresholds for clinical diagnosis are *debated* (age related decline vs CKD – some suggest <45 ml/min/1.73m2)
    - Still may need to adjust medications for renal dosing for age related GFR decline
  - If eGFR reduced, confirm chronic over 3 month or more (consider evaluate for AKI)

- Updated May 31, 2022 – ADA *Living* Standards of Care Addendum to Section 11, "Chronic Kidney Disease and Risk Management"
  - "Traditionally, *eGFR is calculated from serum creatinine* using a validated formula. The **Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation refit without the race variable** is preferred..."
  - "Increased use of **cystatin C** (released from all nucleated cells not just muscle), especially to confirm estimated GFR in adults who are at risk for or have chronic kidney disease
    - Use when concerns about accuracy of Cr (muscle mass concerns w/ body building, amputation, frailty, significant weight loss), etc.
  - ***Combining filtration markers (creatinine and cystatin C)*** is more accurate and would support better clinical decisions than either marker alone."

ADA Standards of Care – additional comments re screening

- DKD is a *clinical* diagnosis
  - Based on the presence of albuminuria &/or reduced eGFR in the absence of signs or symptoms of other primary causes of kidney damage
  - Typical presentation included
    - long-standing duration of diabetes (but can be present at diagnosis),
    - albuminuria without hematuria (reduced eGFR without albuminuria increasingly common in DKD)
    - retinopathy (but can be present without retinopathy, especially in T2D)
    - gradual progressive loss of eGFR
- Presentations suggestive of an *alternative or additional cause* of kidney disease (consider referral to a nephrologist for further diagnosis including possible kidney biopsy)
  - An active urinary sediment (containing red or white cells or cellular casts)
  - Rapidly increasing albuminuria or nephrotic syndrome
  - Rapidly decreasing eGFR
  - Absence of retinopathy in T1D

People with diabetes can have other causes for kidney disease – instead of or superimposed on DKD

Referral to Nephrology (ADA Standards of Care)

- 11.10 Patients should be **referred** for **evaluation by a nephrologist** if they have an **eGFR <30 mL**/min/1.73 m2. A

- 11.11 Promptly **refer** to a physician experienced in the care of kidney disease for
  - uncertainty about the etiology of kidney disease
  - difficult management issues* (anemia & secondary hyperparathyroidism may occur earlier in DKD vs other causes of CKD)
  - rapidly progressing kidney disease. A

*__Difficult management issues__
- Anemia
- Metabolic Bone Disease
- Secondary Hyperparathyroidism
- Resistant Hypertension
- Electrolyte Disturbances
- Volume overload

ADA Standards of Care – additional comments re screening

- *Both albuminuria & eGFR* must be *quantified to guide treatment decisions*
  - eGFR levels are essential to modify drug dosages or restrictions of use
    - Risk for complications/management issues (low BG, anemia, bone disease, volume overload, etc.)
  - At any eGFR, the degree of **albuminuria** is associated with risk of
    - Cardiovascular disease (CVD)
    - CKD progression
    - Mortality

At any eGFR, the Degree of Albuminuria is associated with Risk of Cardiovascular Disease (CVD)

Table 2. Risk Assessment for CVD in CKD

CKD Stages	GFR	10-29	30-299	>300
1	90+			
2	89-60			
3A	59-45			
3B	44-30			
4	29-15			
5	< 15			

CVD, cardiovascular disease; CKD, chronic kidney disease; GFR, glomerular filtration rate.
Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group[9]

Risk of CKD Progression, Morbidity & Mortality

GFR Stages				Albuminuria categories		
				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
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-90			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

Key to Figure:
Colors: Represents the risk for progression, morbidity and mortality by color from best to worst.
Green: Low Risk (if no other markers of kidney disease, no CKD)
Yellow: Moderately Increased Risk
Orange: High Risk
Red: Very High Risk
Deep Red: Highest Risk

The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI) CKD classification

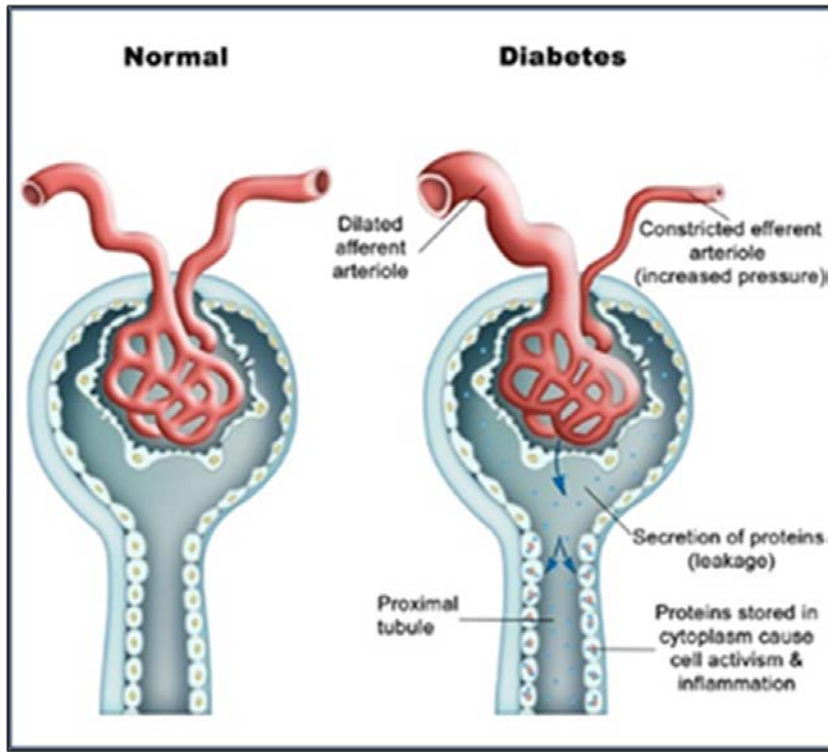
New ADA Standard regarding Treatment Goal for Albuminuria

- 11.3d In patients with chronic kidney disease who have =/>300 mg/g urinary albumin, a ***reduction of 30% or greater in mg/g urinary albumin*** is recommended to slow chronic kidney disease progression B
  - Monitor for reduction over 6-12 months

"In clinical trials of ACEI or ARB therapy in T2D, reducing albuminuria to levels <300 mg/g Cr or by >30% from their baseline has been associated with ***improved renal & cardiovascular outcomes****"*

What about Glomerular Hyperfiltration?

- Glomerular hyperfiltration can be caused by ***afferent arteriolar vasodilation*** as seen in patients with diabetes and/or by ***efferent arteriolar vasoconstriction*** owing to activation of the renin-angiotensin-aldosterone system, thus leading to ***glomerular hypertension***.
    - A *physiological state* of glomerular hyperfiltration occurs during pregnancy and after consumption of high-protein meals.
- The various *diseases* that have been associated with glomerular hyperfiltration include:
    - diabetes mellitus
    - polycystic kidney disease
    - secondary focal segmental glomerulosclerosis
    - sickle cell anemia
    - high altitude renal syndrome
    - obesity



High Intraglomerular Pressure
from afferent arteriolar vasodilation and efferent vasoconstriction

- The afferent arteriole dilates
    - in response to vasodilatory factors such as hyperglycemia and high blood levels of amino acids.
    - in response to decreased delivery of sodium chloride to the distal tubular macula densa via tubuloglomerular feedback (because of a high filtered load of glucose, reabsorption of glucose and sodium chloride is increased in the proximal tubule)
- The efferent arteriole vasoconstricts
    - in response to high local production of angiotensin II.

Is hyperfiltration [itself] associated with the future risk of developing diabetic nephropathy?

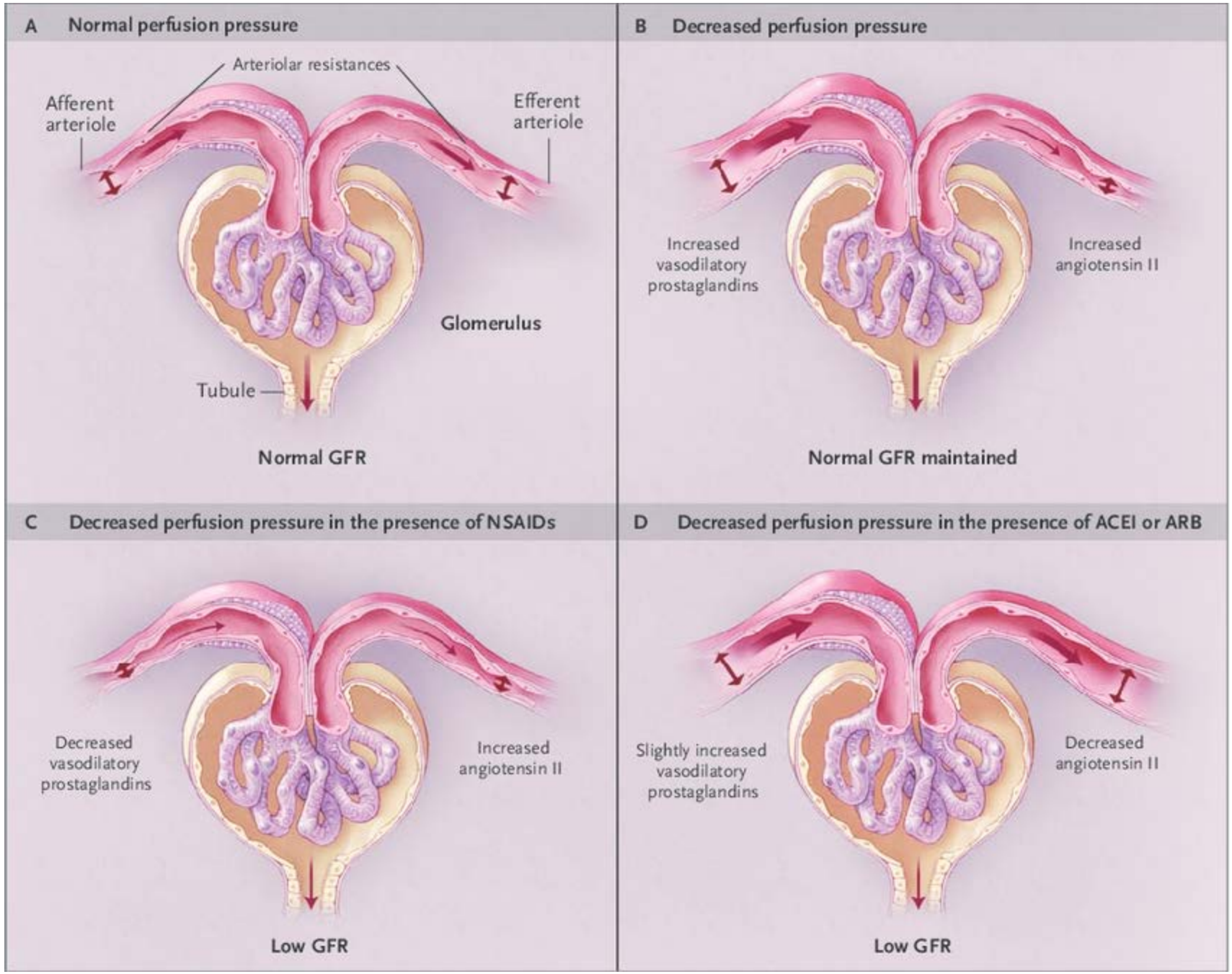
- A meta-analysis (2009)- Conclusions: **patients with hyperfiltration are 2.7 times more likely to progress to incipient nephropathy** than those with normo-filtration

- Glomerular Hyperfiltration in Diabetes: Mechanisms, Clinical Significance, and Treatment (2017)
  - Accumulating **evidence suggests a prognostic and pathogenic role of glomerular hyperfiltration in the initiation and progression of DKD.**
  - However, ***studies are needed to confirm whether targeting hyperfiltration improves clinically relevant end points***
  - Several antihyperglycemic (including SGLT2i and GLP1 RA) and nonhyperglycemic interventions are associated with ameliorated hyperfiltration.
    - ***Whether these treatments add benefit*** in the ongoing search for renal risk reduction in diabetes is ***worth investigating***

ADA Standards of Care – additional comments re screening

- **Acute Kidney Injury (AKI)** – diagnosed by a 50% or greater increase in serum creatinine (reflected as a rapid decrease in eGFR) over a short period of time.
  - **AKI is associated with increased risk of CKD and accelerated progression of existing CKD & poor health outcomes – *timely identification & treatment is critical***
  - Risk factors for AKI
    - Diabetes
    - Preexisting CKD
    - Use of medications that cause kidney injury
      - e.g., *non-steroidal anti-inflammatory drugs, iodinated contrast agents*
    - Use of medications that alter renal blood flow & intrarenal hemodynamics
      - e.g., *diuretics, ACE inhibitors, ARBs*
      - studies show SGLT2i meds *protect* against, not cause AKI
      - increased risk with the combination of ACEI/ARB, diuretic & NSAID ("Triple whammy")

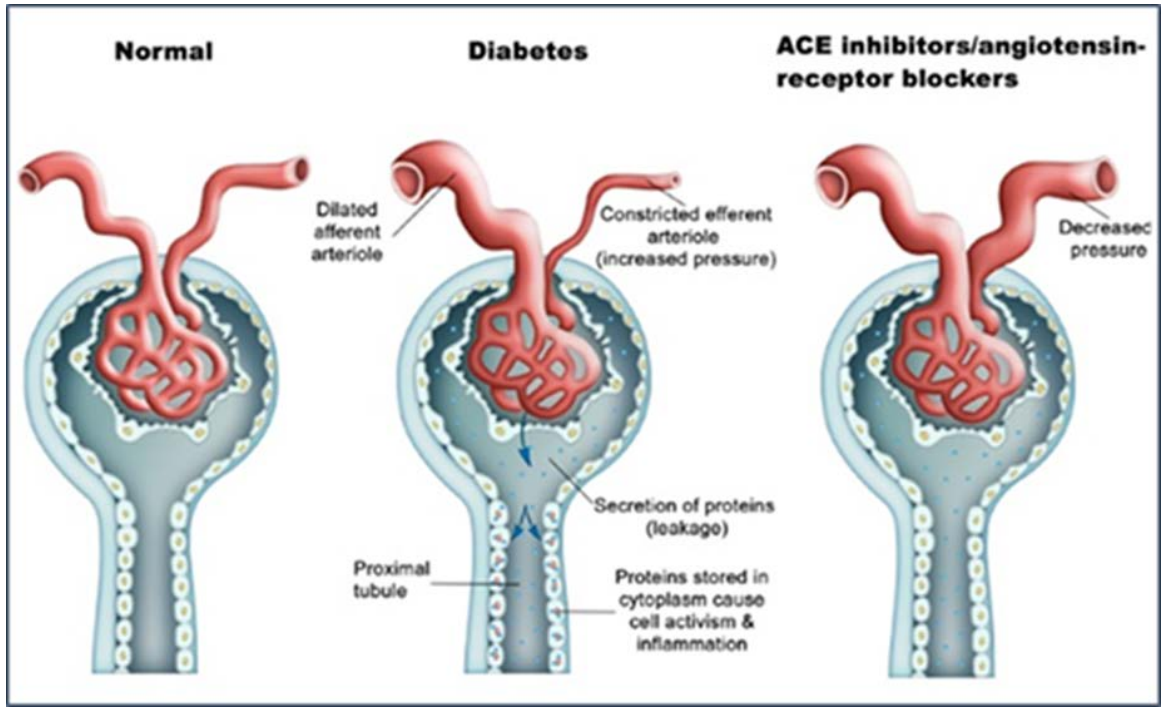
Caution -avoid diuretics for edema from CCBs (vascular dilation, not volume overload) (this does not apply to ESRD with volume overload when diuretics reduce renal congestion)


A Normal perfusion pressure
Arteriolar resistances
Afferent arteriole
Efferent arteriole
Glomerulus
Tubule
Normal GFR
B Decreased perfusion pressure
Increased vasodilatory prostaglandins
Increased angiotensin II
Normal GFR maintained
C Decreased perfusion pressure in the presence of NSAIDs
Decreased vasodilatory prostaglandins
Increased angiotensin II
Low GFR
D Decreased perfusion pressure in the presence of ACEI or ARB
Slightly increased vasodilatory prostaglandins
Decreased angiotensin II
Low GFR

ADA Standards of Care – additional comments re screening

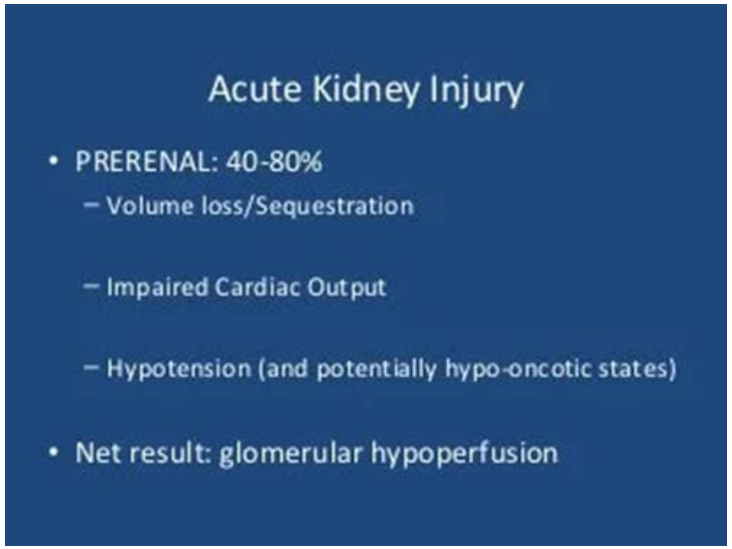
- **Small elevations of serum Cr** (***up to 30%*** from baseline) with RAS blockers (ACEIs & ARBs) must ***not*** **be confused with AKI** –
  - usually begins 3-5 days after start of ACEI or ARB therapy
  - Studies found a strong association between acute increases in serum creatinine of up to 30% that stabilize within the first two months of ACE-inhibitor therapy and ***long-term preservation*** of renal function (*reduce intraglomerular hypertension*)

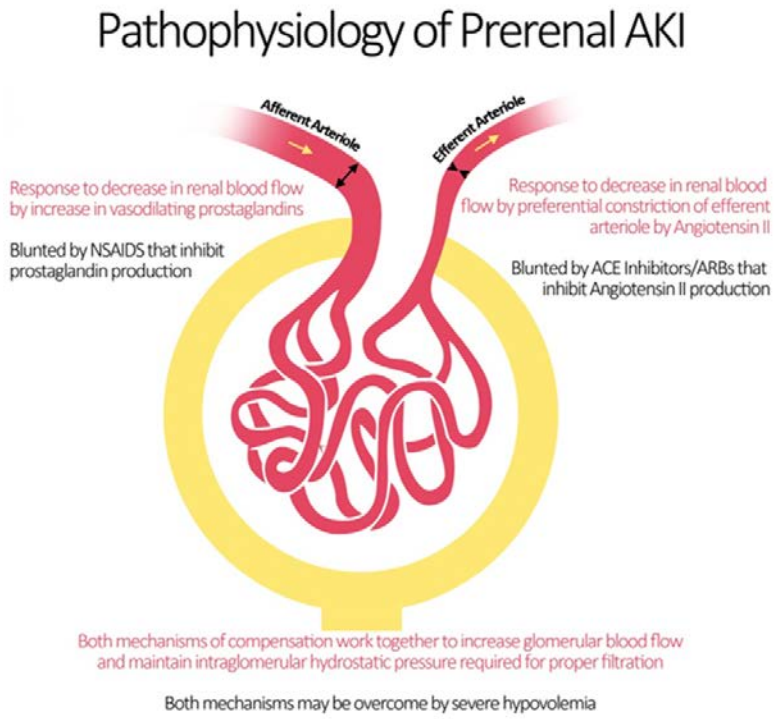
11.5 Do not discontinue renin-angiotensin system blockade for minor increases in serum creatinine (</=30%) in the absence of volume depletion. A



ADA Standards of Care – additional comments re screening

- A **more severe acute decline** in renal function can be seen *in patients with already reduced intrarenal perfusion pressure-*
  - such as from bilateral renal artery stenosis or congestive heart failure or volume depletion (*intraglomerular hypotension*)
  - **dose reduction or withdrawal of ACE-inhibitor therapy only with a rise in serum creatinine >30% from baseline in the first two months of medication use**

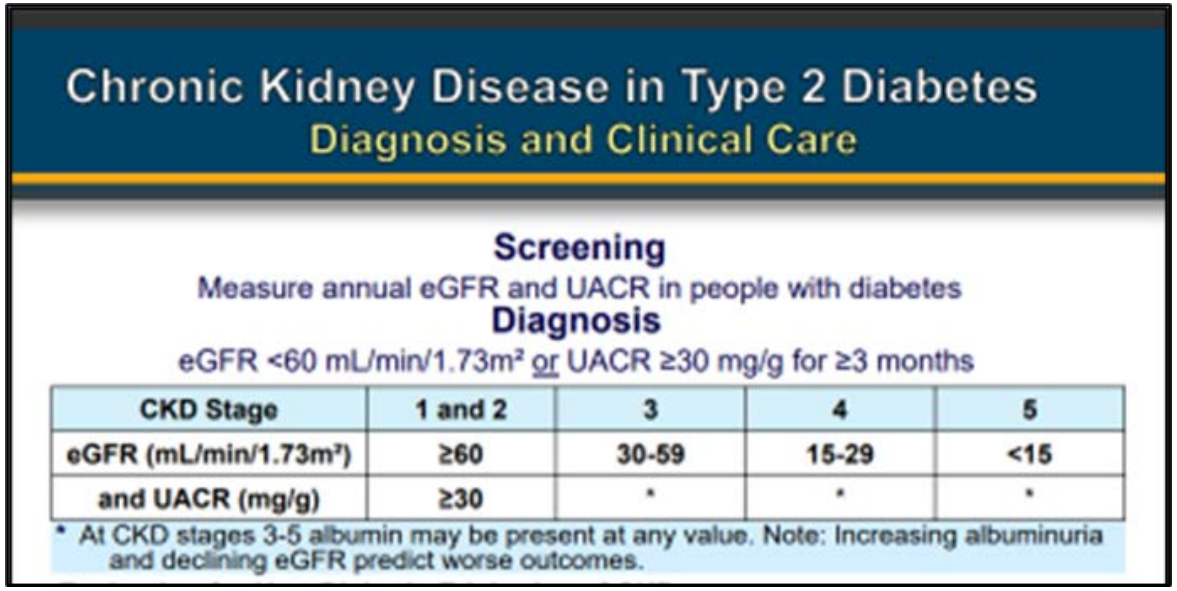




Diagnosis of DKD: ADA Standards of Care & Indian HS

- Diabetic kidney disease (DKD) is defined by persistent albuminuria (increased uACR ≥30 mg/g) and/or reduced eGFR in the setting of diabetes and the absence of signs or symptoms of other primary causes of kidney damage

- Staging of Chronic Kidney Disease
  - Stages 1–2 CKD have been defined by evidence of high albuminuria with eGFR ≥60 mL/min/1.73 m2, while stages 3–5 CKD have been defined by progressively lower ranges of eGFR

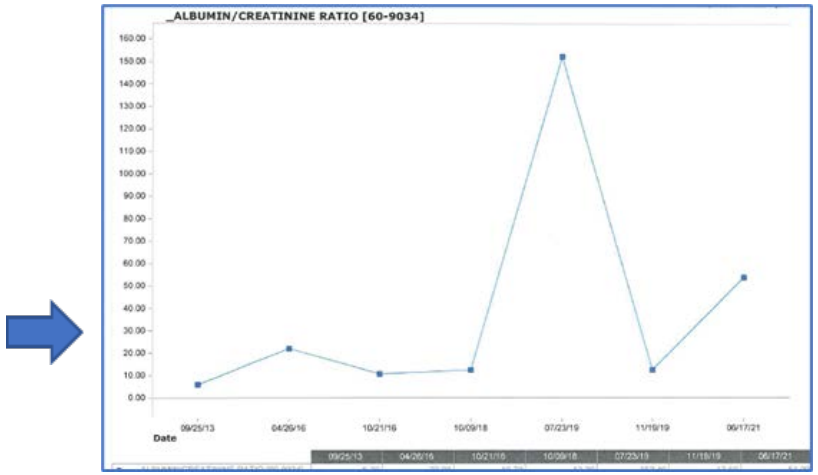


ADA Standards of Care – additional comments re screening

- **Regular monitoring (surveillance) of albuminuria & eGFR** allows
  - Timely diagnosis of CKD (and begin treatment to slow progression)
  - Monitoring progression of CKD (and response to treatment)
  - Detect superimposed kidney disease, including Acute Kidney Injury
  - Assess risk of CKD complications (often earlier onset in DKD than other causes of CKD)
    - Elevated Blood Pressure >140/90
    - Glucose management issues (increased risk for hypoglycemia & harm from hypoglycemia)
    - Volume overload
    - Electrolyte abnormalities
    - Metabolic acidosis
    - Anemia
    - Metabolic bone disease
    - Secondary hyperparathyroidism
  - Dose drugs appropriately ("renal dosing") – including diabetes med choices/doses
  - Determine whether a nephrology referral is needed

Patient #1: “Valerie”

- 38-year-old female diagnosed with T2D 2007
- Height 65”, weight 236#, BMI 39; central obesity pattern; BP 112/78
- no tobacco use; single, works at a childcare program
- A1c level often >10% – but occasionally <8%; TC 170,LDL 75, HDL 33, TG 385, TSH 1.15
- Cr 0.54 with eGFR >60 (>120); UACR 54 mg/g
- ED 7x/12 months N&V, twice with AKI (acute kidney injury)
- Meds:
  - Canagliflozin 300 mg QD
  - Degludec insulin 32u QD
  - Metformin 1g BID
  - Lisinopril 10 mg QD



- Clinical Question from her Primary care clinician
  - “Concern about how to preserve her kidneys
    - When to refer?
    - Should she be taken off SGLT2i (due to AKI risk)?
    - What am I missing in this patient?”

Patient #1: “Valerie”

- 38-year-old female diagnosed with T2D 2007 (Dx age ~23 –early onset – 15-year hx) – BMI 39
  - A1c level often >10% – but occasionally <8%
- **UACR** 54 mg/g x 1
- Cr / eGFR shows ***hyperfiltration***
- Recurrent bouts of **AKI** – (associated with presentation to ED with N&V)
- Meds:
  - Canagliflozin 300 mg QD
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  - Metformin 1g BID
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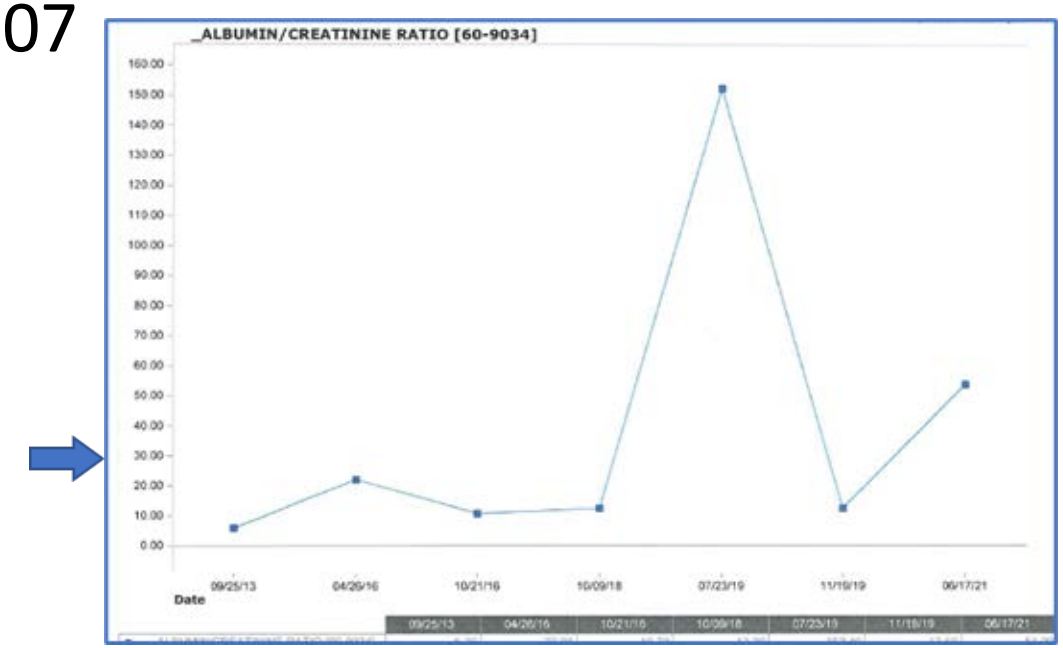

Patient #1: “Valerie” – sorting things out

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- Is the SGLT2i the likely cause of her bouts of AKI (acute kidney injury)?

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- Any thoughts on what might be the cause of or contributing to AKI?

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- Any thoughts on what might be the cause of or contributing to AKI?
  - Add THC/ Cannabinoids to list of drugs that can cause AKI

Cannabinoid hyperemesis syndrome (CHS)

- CHS is an episodic syndrome with **vomiting episodes that last for 24–48 h** at a time ***separated by asymptomatic periods*** that may last weeks or months.
  - During episodes, patients suffer **severe nausea and intractable vomiting** often accompanied by **abdominal pain** for which conventional antiemetic therapy offers no relief but ***relief with hot showers/bathing*** (*diagnostic*)
  - Protracted periods of *vomiting (as often as 20 times a day)* leaves some patients **dehydrated**, disoriented, and in electrolyte imbalance.
  - Long *hot showers* can contribute to **dehydration & AKI**

"The unique combination of intractable vomiting and constant hot showers *seems to put CHS patients at significant risk of* ***severe dehydration and prerenal failure,*** *a common and distinct entity we suggest be termed* ***cannabinoid hyperemesis acute renal failure (CHARF)."*** (acute renal failure (ARF) = acute kidney injury (AKI))

Accurate measurement of BP is critical

- Most adults with hypertension in the United States do not have their hypertension under control (92.1 million).
  - Hypertension is a critical *promotor of progression* of DKD
  - In the United States - Hypertension accounts for more *cardiovascular disease (CVD*) deaths than any other modifiable risk factor and is second only to cigarette smoking as a preventable cause of death for any reason.
  - Worldwide -Hypertension is the leading cause of *death and disability*.
- Over-treatment of Hypertension can result in Hypotension
  - Hypotension can trigger AKI (under-perfused kidneys)
  - Hypotension is a cause of falls & injuries

HTN Trial Outcomes & Guideline Recommendations are based on Standardized BP measurements and results

Routine BP measurement

Standardized BP measurement

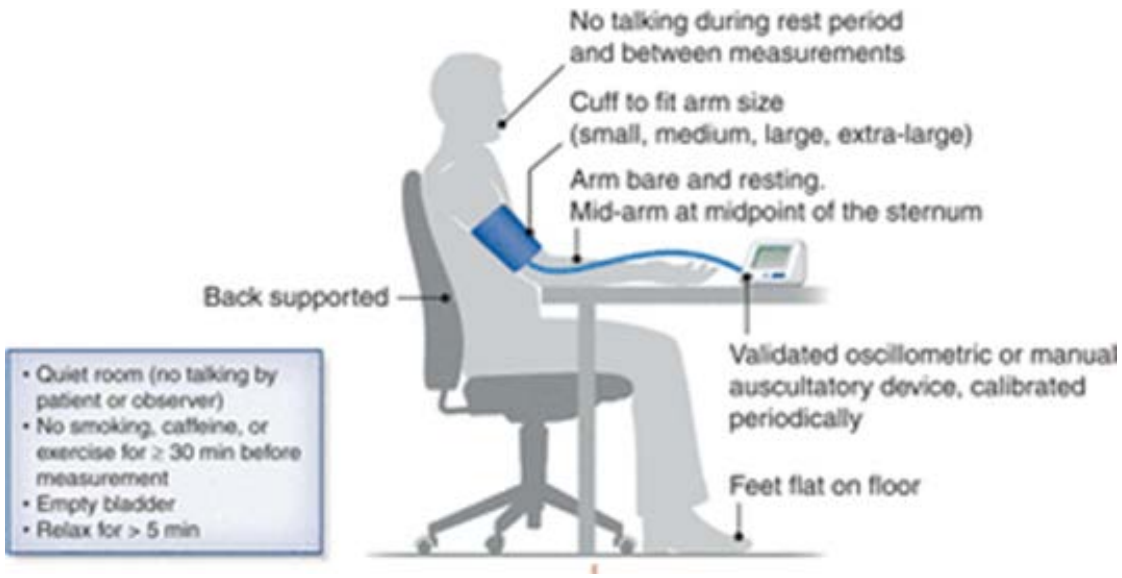
Standardized BP vs Routine BP

On average:

- 20% of standardized BP results are higher than routine BP results
  - Undertreat based on routine BP measurement
- 40% of standardized results are lower than routine BP results
  - Overtreat based on routine BP measurement

Getting it Right (accurate)

Technique for Standardized BP Measurement

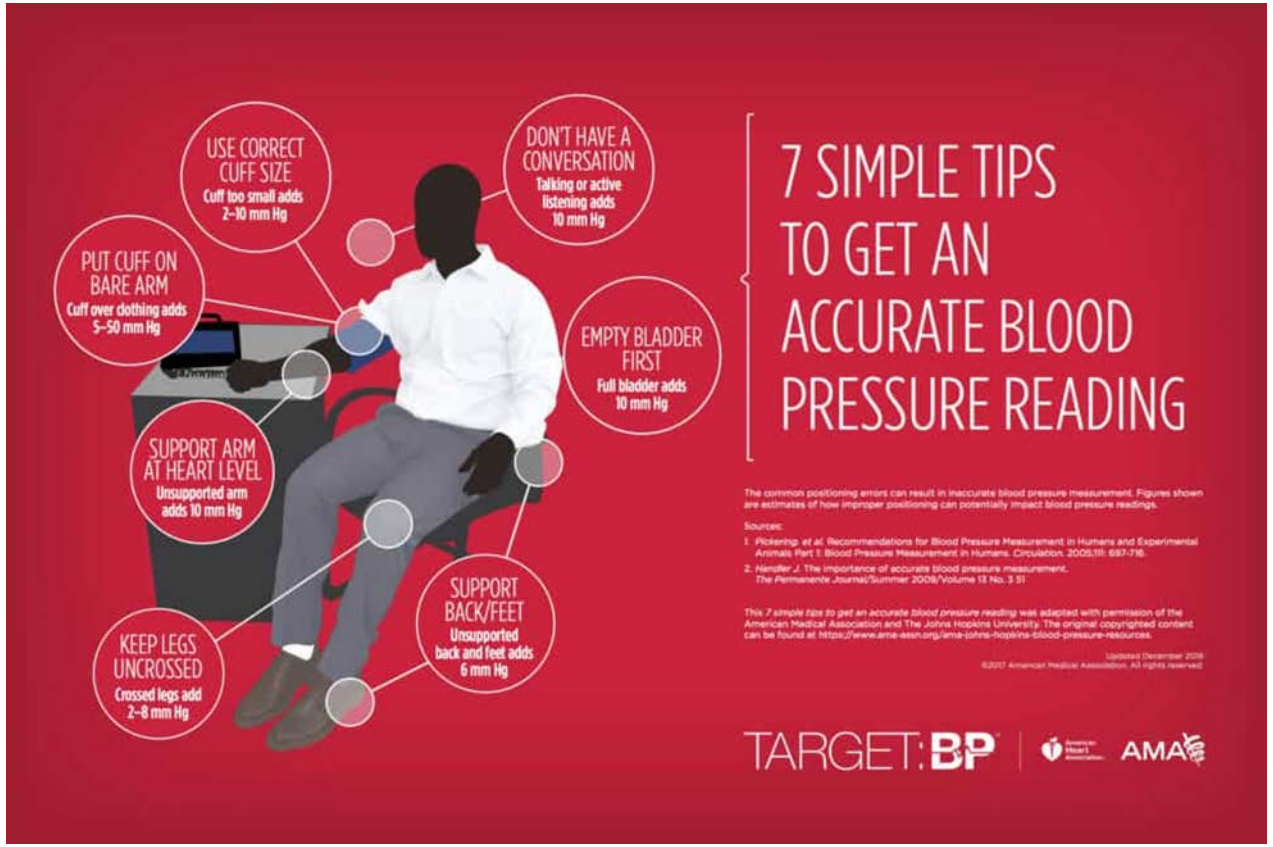


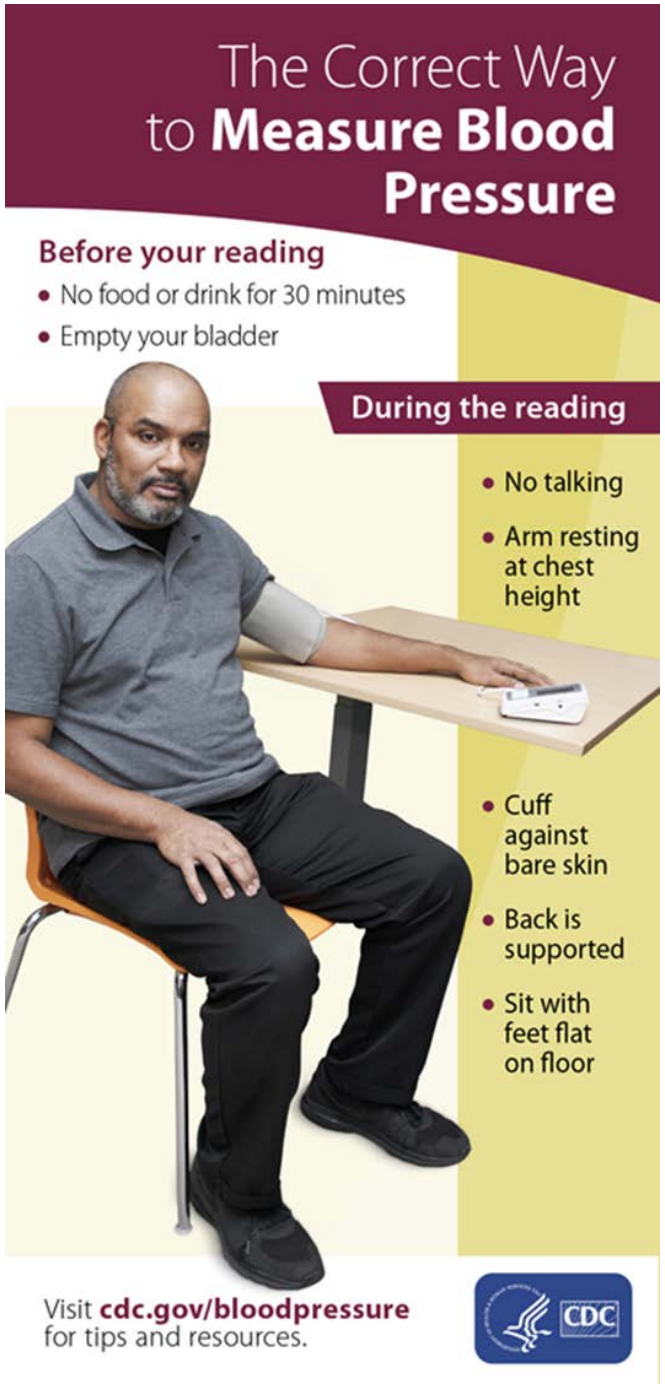
Common Causes of Error resulting in Inaccurate BP Readings

- Technique errors
  - Measuring BP in an arm positioned *above heart level* will provide a *falsely low* BP readings
  - Positioning the arm *below heart level* will provide *falsely high* BP readings
  - Cuff too small can falsely increase the BP reading by 5 to 20 mm Hg
    - Cuff too large will provide a falsely low BP result
  - Crossing the legs can increase the BP reading by up to 8 points
    - This is the reason we have someone who feels faint cross their legs to help keep them from passing out
  - See table for impact of additional patient prep and positioning factors

Factor	Magnitude of systolic/diastolic blood pressure discrepancy (mm Hg)
Talking or active listening	10/10
Distended bladder	15/10
Cuff over clothing	5–50/
Cuff too small	10/2–8
Smoking within 30 minutes of measurement	6–20/
Paralyzed arm	2–5/
Back unsupported	6–10/
Arm unsupported, sitting	1–7/5–11
Arm unsupported, standing	6–8/

Utilize posters to help remind patients of the proper positioning & technique required for accurate BP measurements





Key Points

- Screening & surveillance is essential
  - Early identification & treatment reduces risk of ESKD, CV & HF events, morbidity & mortality (improves patient outcomes [& demands on the clinician & system] and reduces costs)
  - Confirm abnormal screening tests (eGFR, UACR) over 3-6 months
  - Ensure labs use new CKD-EPI equation to calculate eGFR/ use cystatin C if unreliable muscle mass, etc.
- Consider AKI (acute kidney injury) & help prevent it
- At any eGFR, the *degree of albuminuria* is associated with risk of DKD progression, cardiovascular disease (CVD), and mortality
  - New recommended treatment goal for albuminuria (reduce uACR by >/=30% or to <300 mg/g)
- Hypertension is a critical promotor of progression for DKD & CVD
  - Accurate BP measurement is critical for diagnosing, monitoring & managing hypertension
  - Utilize *standardized* BP measurements / can include home BP measurements

Workflow Issues & Ideas

- How to get that uACR & repeat it if abnormal….
- How to get standardized BP measurement in clinic…
  - Starting point is room set up


Questions?
Comments?
Suggestions?

Extra Slides & References

AKI and CKD

- History of AKI is a strong risk factor for both new-onset CKD and accelerated progression of existing CKD
- https://www.youtube.com/watch?v=USpDtPxAzBw
- https://www.youtube.com/watch?v=USpDtPxAzBw

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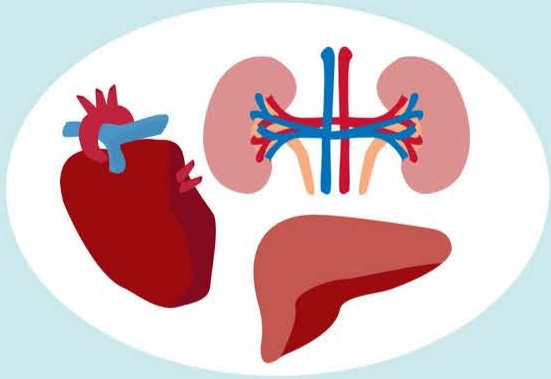
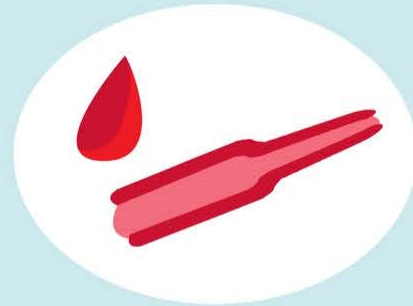
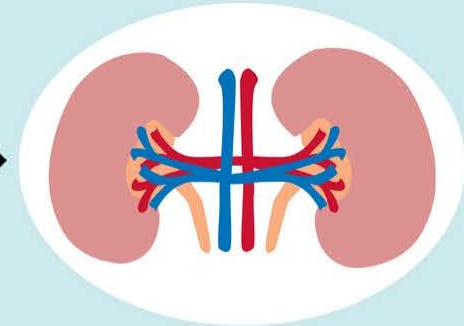


NSAIDs

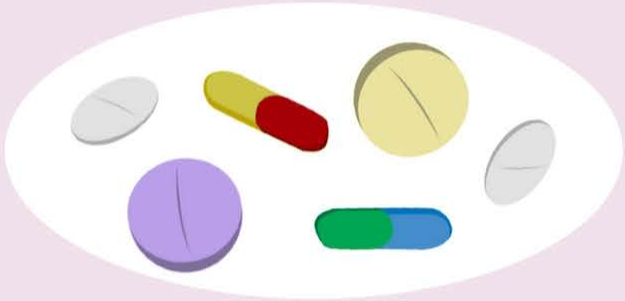
HEMODYNAMIC DISTURBANCES



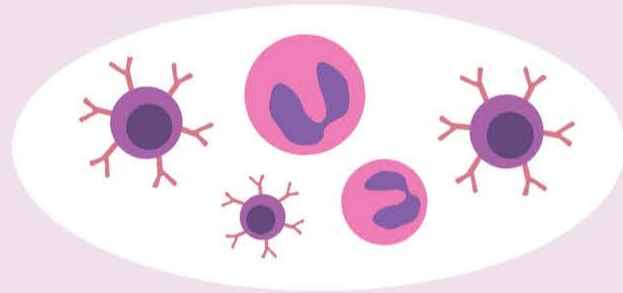
AKI



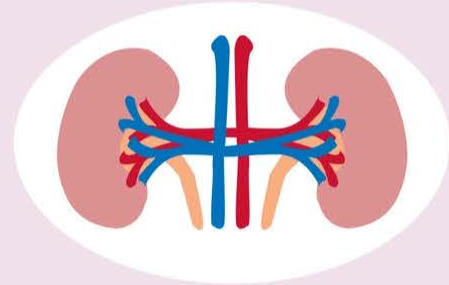
PROLONGED EXPOSURE TO NSAIDs



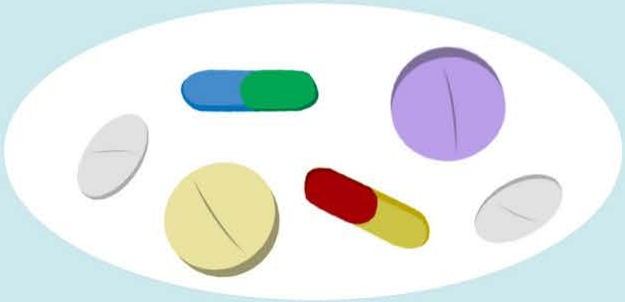
HYPERSENSITIVITY REACTION



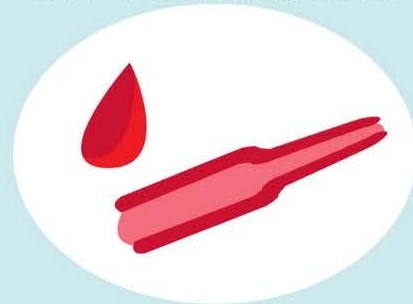
TIN



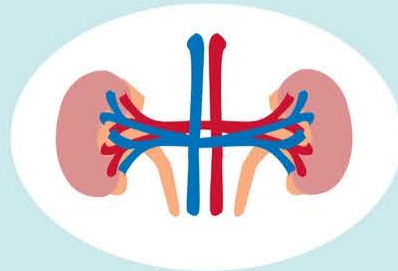
CHRONIC USE OF NSAIDs



HEMODYNAMIC DISTURBANCES



CKD



Prognosis of CKD by GFR and albuminuria category

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

GFR categories (ml/min/ 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	**A2**	**A3**
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red, very high risk.

CKD in T2DM: Screening Frequency and Referral

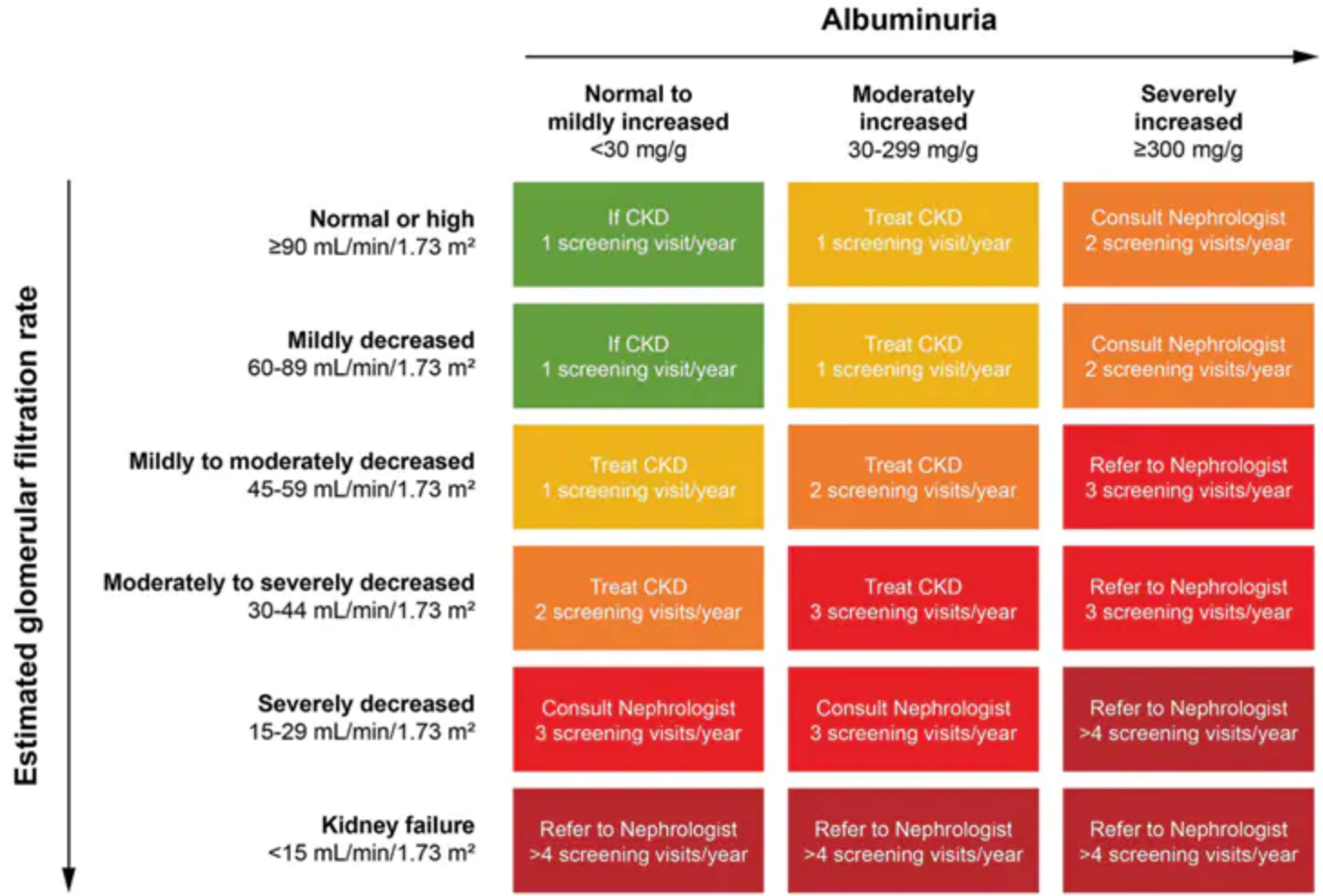


Fig. 1 Risk of CKD progression, frequency of visits, and referral to a nephrologist according to glomerular filtration rate (GFR) and albuminuria. The GFR and albuminuria grid depicts the risk of progression, morbidity, and mortality by color, from best (green) through yellow, orange, and red to worst (dark red). *CKD* Chronic kidney disease. Modified from Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group [61], copyright 2013, with permission from Elsevier

Skolnik NS, *Style AJ. Diabetes Therapy*. 2021 Apr 29:1-8.

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- ADA Standards of Care 2022: https://diabetesjournals.org/care/issue/45/Supplement_1
- In Depth Article (also available as 2-hour Webinar) Expert Insights for Primary Care Physicians in Managing Chronic Kidney Disease in T2DM (medscape.org)
- Article on eGFR using Cr and/or cystatin https://www.aacc.org/cln/articles/2016/april/cystatin-c-and-creatinine-complementary-markers-of-gfr-expert-john-c-lieske-md
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Cannabinoid Hyperemesis Syndrome

- Prim Care Companion CNS Disord. 2016; 18(1): 10.4088/PCC.15l01847.PMCID: PMC4874760 PMID: 27247840 Cannabinoid Hyperemesis Syndrome Associated With Compulsive Showering and Acute Kidney Injury Priya Srihari, MS, Mengyang Liu, BS,a Steven Punzell, MS,a Shady S. Shebak, MD,a and William S. Rea, Mda

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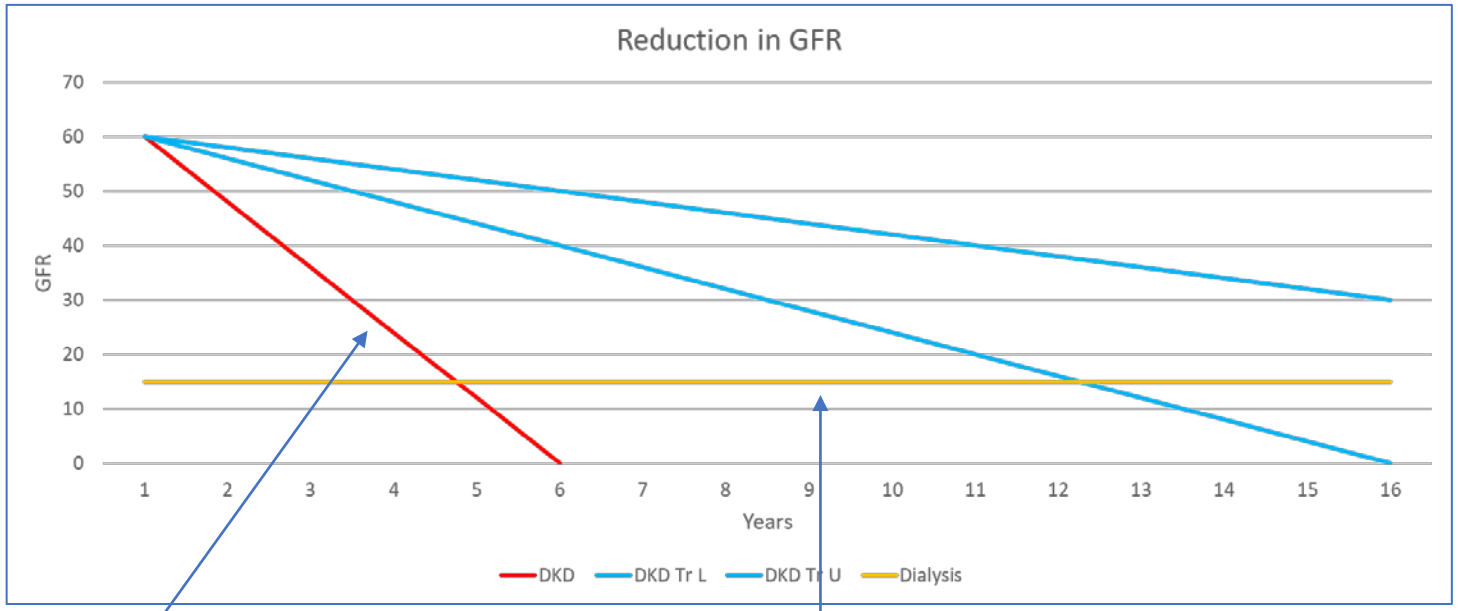
- Effective detection and early intervention in DN can help to slow renal function decline. It can also prevent complications, thus improving survival and quality of life in type 2 diabetics.
- Annual decline in glomerular filtration rate (GFR) in a person varies widely depending on various factors such as ethnicity, age, underlying medical problems, the etiology of chronic kidney disease (CKD), and the presence of comorbidities.
- Kidney Disease: Improving Global Outcomes (KDIGO) guidelines define rapid progression as rate of eGFR declines > 5 mL/min per 1.73m2 per year. The classical progression of DN is deterioration of renal function over decades with typical rate of GFR decline ranges from between 2 to 20 mL/min per 1.73m2 per year with a median of 12 mL/min per year.
- In our report, heavy amount of proteinuria, beside the traditional risk factors like poor glycemic control, hypertension are the leading risk factors for rapid progression of renal disease.

For Patients & Families

- https://www.kidneyfund.org/kidney-disease/chronic-kidney-disease-ckd/stages-of-chronic-kidney-disease/

- 11-10-1813_abe_patbro_gfr_b.pdf (kidney.org)

Slowing Down the Loss of Kidney Function with Diabetes Kidney Disease



This is how quickly your kidneys will lose function without any treatment

The yellow line is the level where you need to start dialysis

The aqua lines show how much treatment can reduce the rate at which your kidneys lose function

Treatment includes

- Optimal blood pressure, using an ACEI or ARB medication along with other meds if needed
- An SGLT2 inhibitor medication (and/or a GLP1 RA or MRA)
- Blood glucose levels in individualized safe range
- Avoiding drugs that hurt your kidneys such as NSAIDs (ibuprofen, Advil, Motrin, etc.)
- Keeping your muscles strong, avoiding being too sedentary
- Healthy food choices – especially, avoid excess animal protein & fats