DISCLOSURE OF RELATIONSHIPS

Premier Health and Wright State University endorse the ACCME Standards of Commercial Support. We require that the planners of continuing medical education activities and the speakers at these activities disclose relationships with commercial interests. Having an interest or affiliation with a commercial interest does not prevent a speaker from participating in a continuing medical education activity, but the relevant financial relationship must be made known to the audience prior to the start of the activity. A relevant financial interest is a financial benefit the speaker, a spouse or a partner has received in the past twelve (12) months. Significant relationships include receiving research grants, consultancies, honoraria and travel or other benefits from a commercial interest. Disclosure of a relationship is not intended to suggest or condone bias in any presentation but is made to provide participants with information that might be of potential importance to their evaluation of a presentation.

Disclosure of the discussion of off-label drug use is also required for speakers. The following Planning Committee members and Speakers report no actual or potential conflict in relation to this activity:

- Joseph Mauro, MD
- Mark Oxman, DO
- Dana Mackert
- India Myers

Accreditation

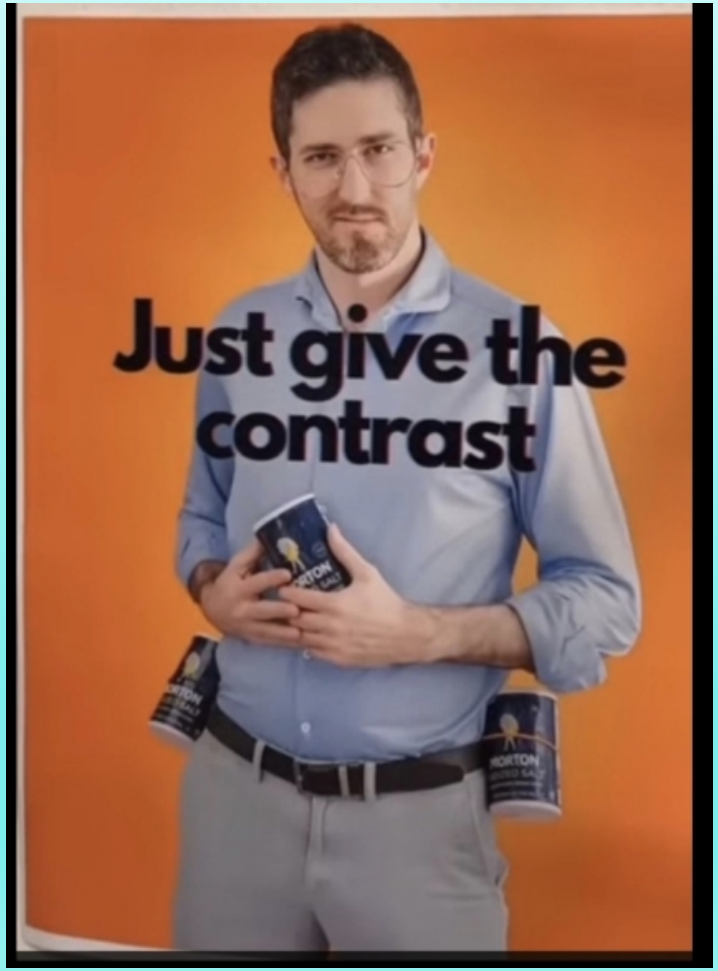
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of Wright State University (WSU) and Premier Health. WSU is accredited by the ACCME to provide continuing medical education for physicians.

WSU designated this online virtual activity for a maximum of 1.0 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Contrast Induced Nephropathy The Facts and The Myths

Mark Oxman,DO FACOI

Contrast Nephropathy



OR NOT

Contrast patient

- 78 year old female presents with shortness of breath. No CXR performed but CT PE protocol and CT of abdomen and pelvis performed. No acute findings noted
- Pt discharged home, suffers a fall
- Returns to ER; CT with contrast of chest and CT of abdomen and pelvis with contrast repeated. No CXR done
- CT revealed pneumonia
- Pt admitted, developed AKI and while pneumonia improved on Day 2, renal failure did not and pt required 7 days in hospital. Had 4 contrast studies in the span of 16 hours. Short of breath from pneumonia; no CXR ever done

Contrast Patient

- Past hx included Diabetes, Hypertension
- Labs included a creatinine of 1.8 with GFR of 32
- Hx of CHF and EF or 35 % with some moderate MR
- Previous smoker
- Other HPI include fever and chills as well as a productive cough
- Better Choice than CT PE protocol ?

Contrast patient

- 67 year old female with known hx of CHF; EF 15% and severe MR, presents to ER with shortness of breath and pulmonary edema on CXR. CT PE protocol performed. Pt develops AKI and required dialysis for 2 weeks in the hospital and eventually recovered function. No other nephrotoxic exposures noted
- Creatinine 2.3 on admission and GFR of 28
- 3 months later, pt presents with same symptoms and CXR findings. CT PE protocol done again. Pt developed AKI and did not recover. Died 3 months later from her end stage heart but required HD until she died
- Why was CT PE protocol necessary with an obvious cause of shortness of breath

Occurrence

- Third leading cause of hospital acquired kidney failure, decreased renal perfusion being the number one cause is various forms of shock.
- Actual incidence unclear as rate varies depending on type of contrast used, amount, preexisting risk factors and the timing of follow up labs
- The controversy over the existence of contrast nephropathy was based on a retrospective study of 17,934 emergency patients from 2009-2014 and it was found that there was no difference in contrast nephropathy in pts receiving contrast vs non contrast CT pts regardless of underlying baseline renal function. This was a single center study and excluded pts with a creatinine of greater than 4
- In addition, this and similar studies are retrospective and do not take into account biases as to why contrast was given or not given and any prophylactic measures, as well as underrepresentation of pts with lowest GFR

Definition of Contrast Nephropathy

Defined as either a 25% increase in creatinine from baseline or 0.5% mg/dl increase in the absolute value

Needs to be acute occurring within in first 2-7 days(depending on author) and should not be attributable to any other cause of renal insufficiency

Generally peaks in 2-5 days and returns to normal in 14 days

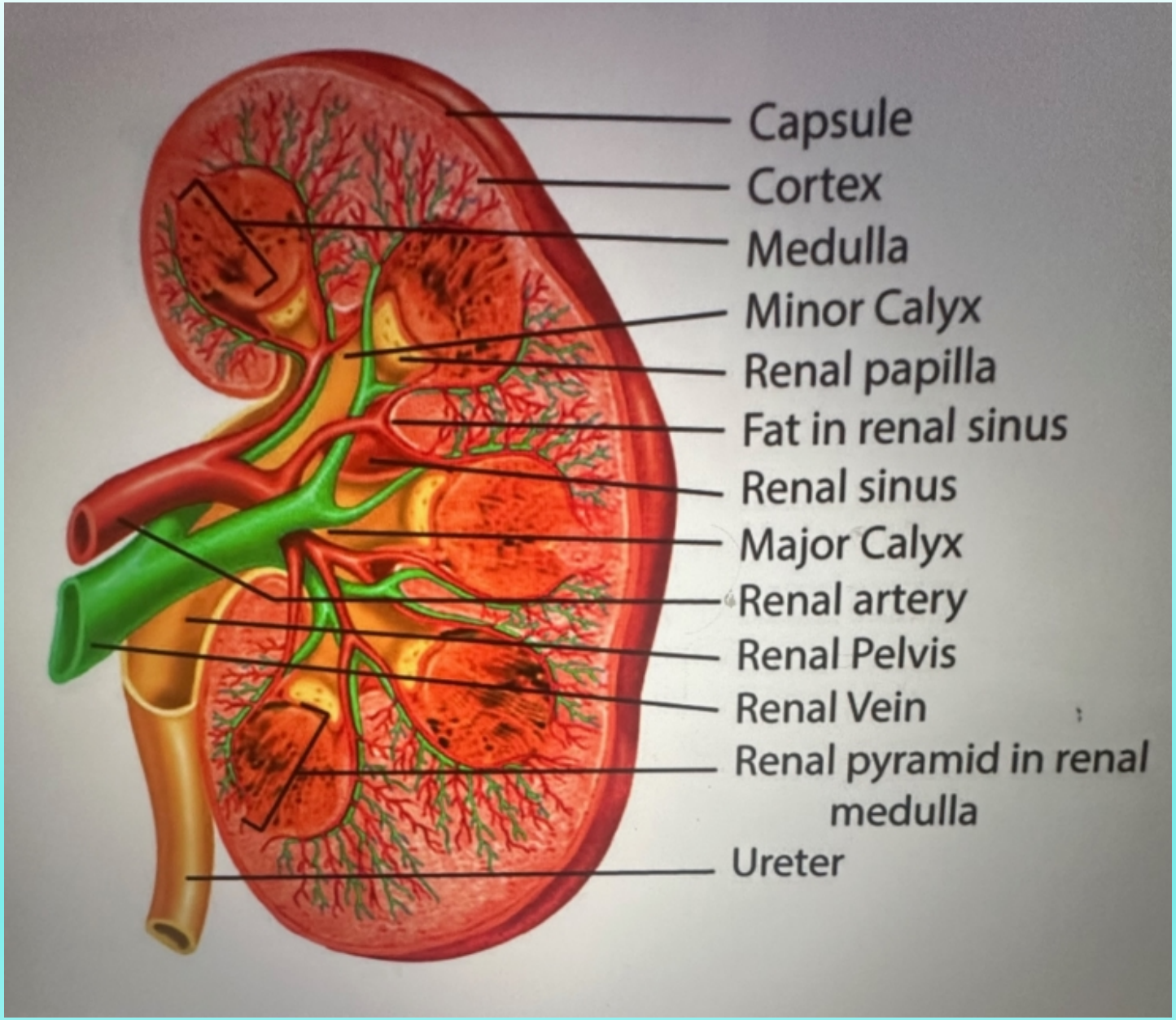
Pre-existing chronic kidney disease remains a risk factor

Risk factor for in hospital mortality and 1 year mortality even in pts who did not require dialysis

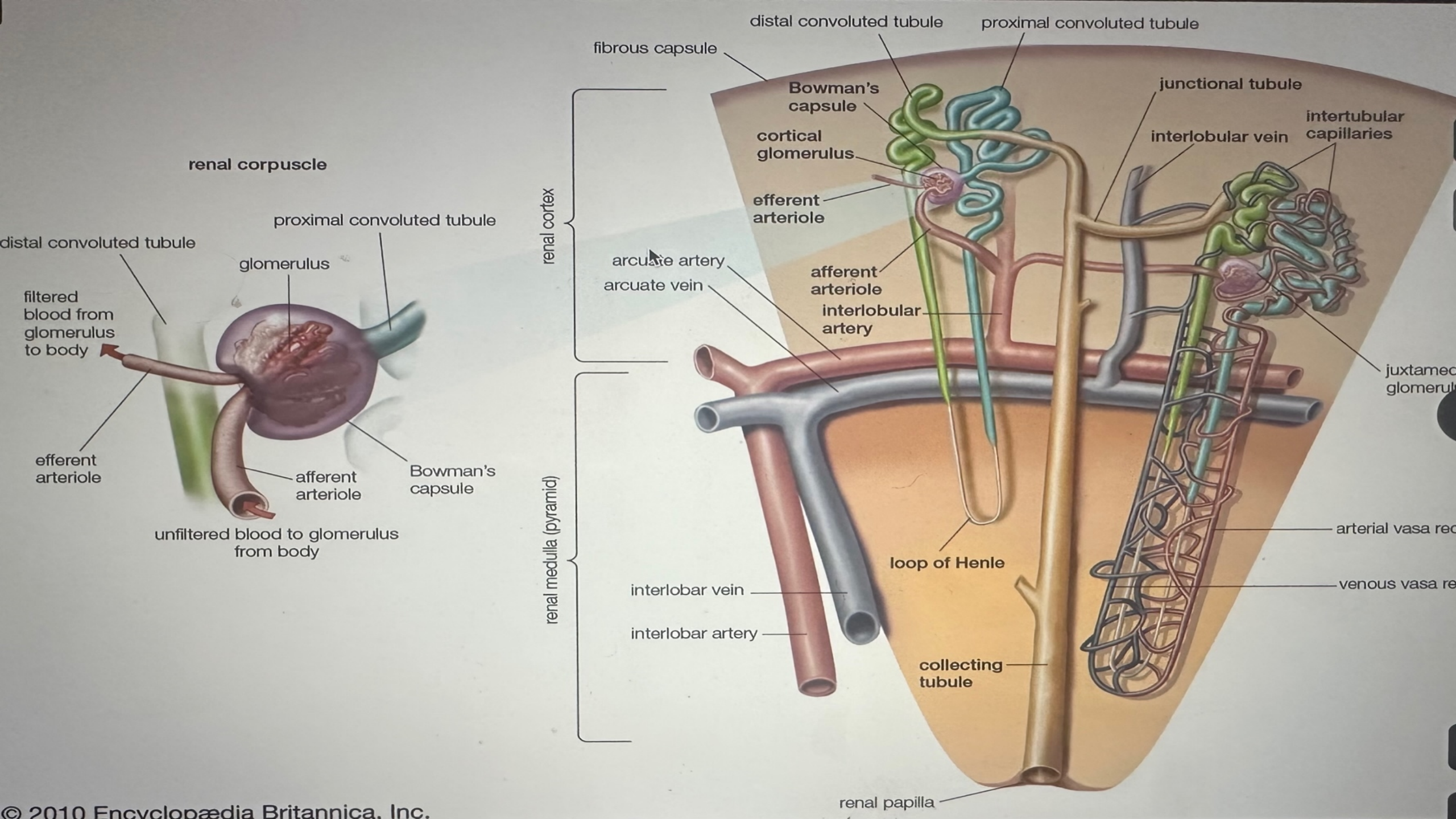
Incidence likely an underestimation as rise in creatinine at day 3 frequently not measured as pts do not remain in the hospital

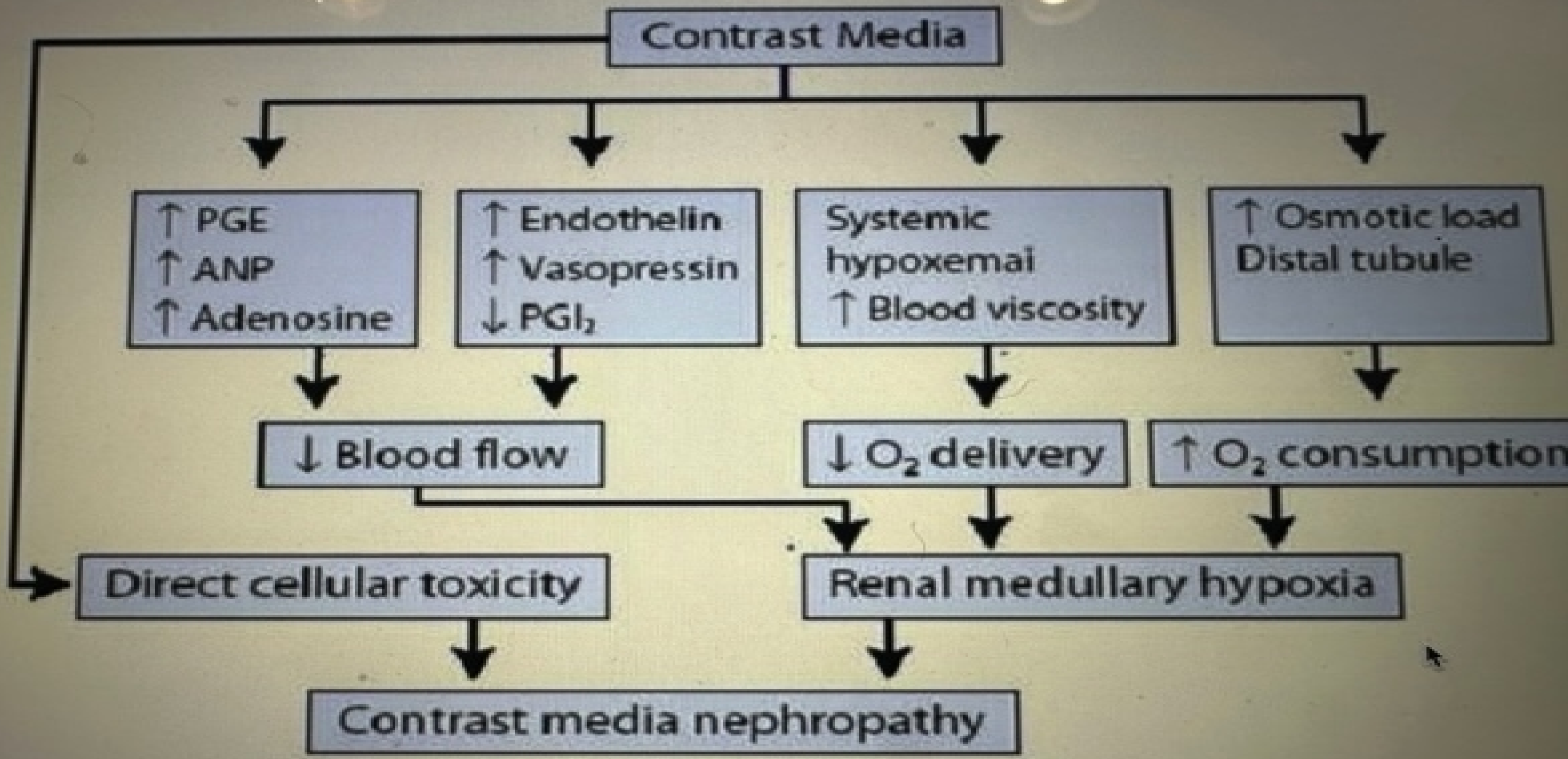
KDIGO Working Group

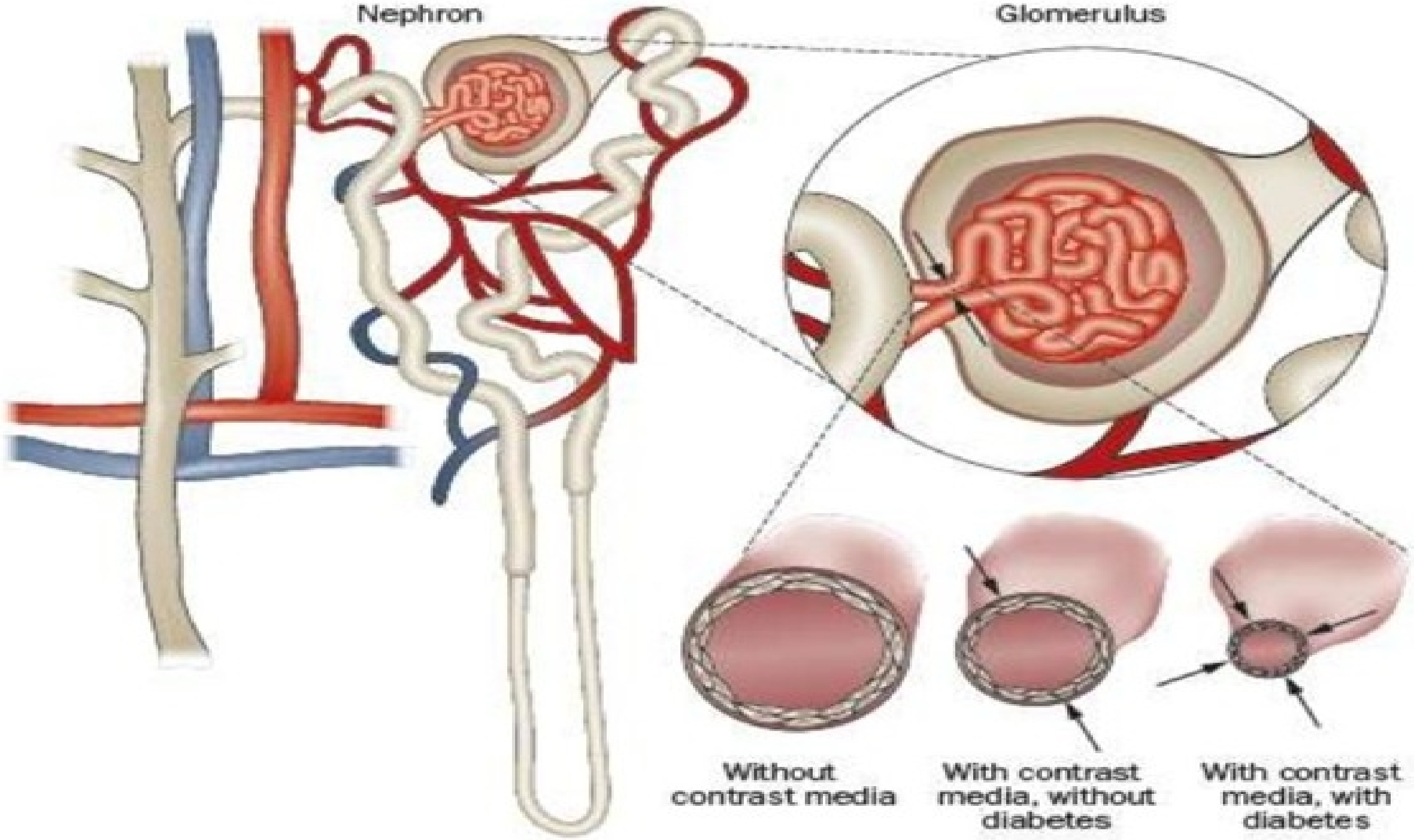
- Kidney Disease Improving Global Outcomes proposed the term contrast induced acute kidney injury with the definition of plasma creatinine that increased by 1.5X within 7 days of exposure, rise of greater than 0.3 mg/dl with 48 hours of exposure or urinary output of less than 0.5 ml per kg of body weight per hour that occurs at least 6 hours after exposure
- Plasma creatinine felt to have poor specificity with good sensitivity as creatinine levels will fluctuate based on fluid shifts, medications etc. Since medications, atheroemboli, and hypotension can precipitate acute kidney injury after exposure to contrast medium the term “contrast-associated acute kidney injury” has gained some favor

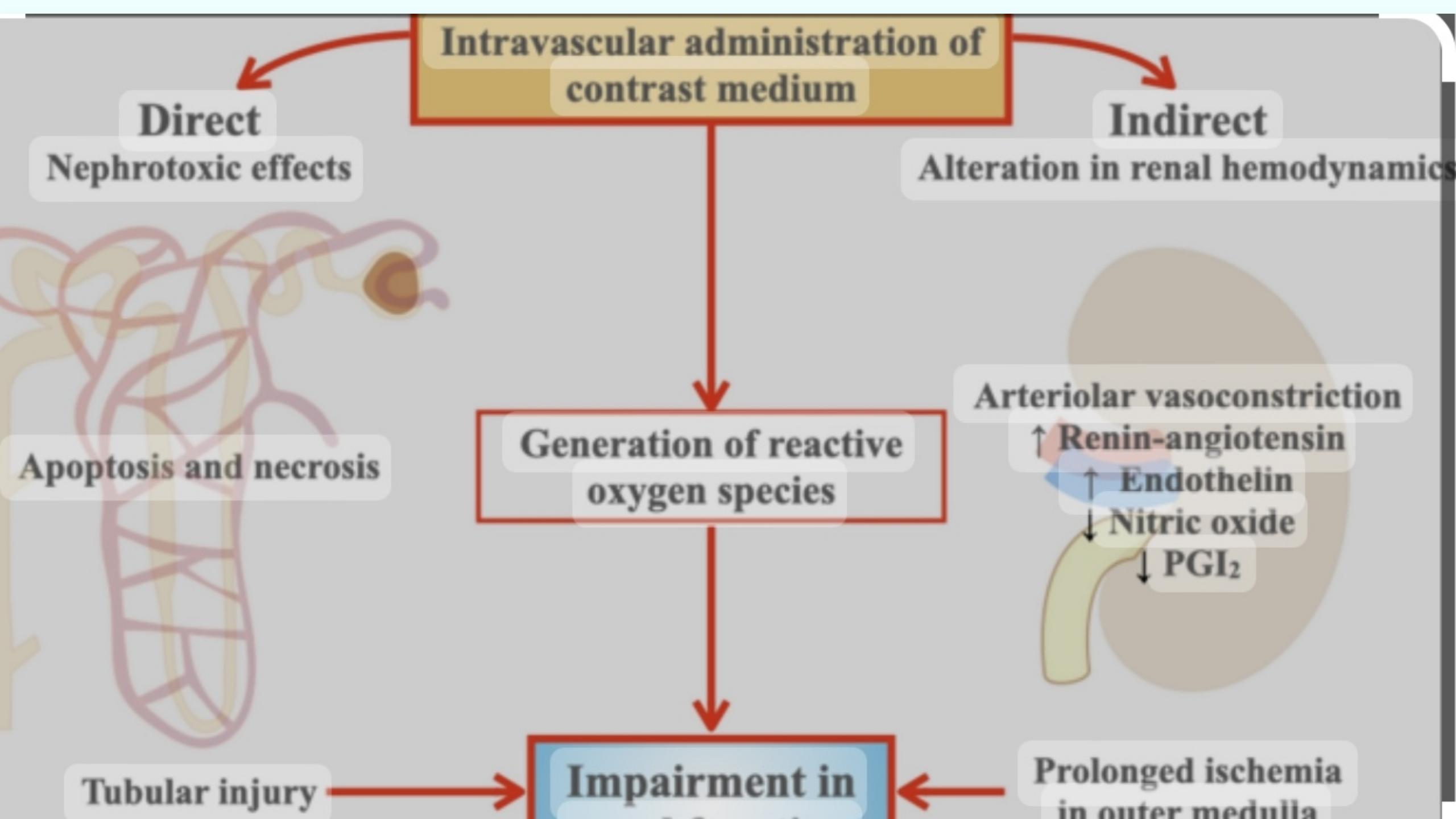


Renal Anatomy




Contrast Media
↑ PGE
↑ ANP
↑ Adenosine
↑ Endothelin
↑ Vasopressin
↓ PGI_2
Systemic hypoxemai
↑ Blood viscosity
↑ Osmotic load
Distal tubule
↓ Blood flow
↓ O_2 delivery
↑ O_2 consumption
Direct cellular toxicity
Renal medullary hypoxia
Contrast media nephropathy


Nephron
Glomerulus
Without contrast media
With contrast media, without diabetes
With contrast media, with diabetes


Intravascular administration of contrast medium
Direct
Nephrotoxic effects
Indirect
Alteration in renal hemodynamics
Apoptosis and necrosis
Generation of reactive oxygen species
Arteriolar vasoconstriction
↑ Renin-angiotensin
↑ Endothelin
↓ Nitric oxide
↓ PGI_2
Tubular injury
Impairment in
Prolonged ischemia
in outer medulla

Etiology

- Has multiple adverse effects on the kidney
- Direct cytotoxic effect on proximal tubular cells, increases resistance to renal blood flow leading to medullary hypoxemia and exacerbated by pts with underlying chronic kidney disease

Comparison of Contrast Agent Nephropathy Potential

- Agents are classified as high, low or iso-osmolar agents
- Low osmolarity agents 600-900 mosm vs high 1500 mosm in high agents
- Iohexol Cooperative Study double blinded study comparing low vs high agents; risk of CIN 3.3 times higher with high agents but difference seen only in pts with preexisiting kidney disease with creatinine 1.5 or higher(Barrett BJ, Carlisle EJ, Radiology 1993)
- Other risk factors in this study were diabetes mellitus, male sex, higher contrast volume
- NEPHRIC study found that risk of contrast nephropathy 9X higher in low osm agents vs iso-osmolar agents but subsequent studies did not duplicate the findings
- Some suggestion in other studies that iodixanol(Visipaque) may cause a smaller increase in creatinine than other agents especially in diabetes and pts with CKD

Risk Factors for Contrast Nephropathy: Patient Related

- Older age
- CKD
- Diabetes Mellitus
- Hypertension
- Nephrotic Syndrome
- Multiple Myeloma
- Kidney Transplant
- Hypovolemia and decrease effective circulating volume
- Hypotension

Risk Factors for Contrast Nephropathy- Continued

- Preexisiting kidney disease is greatest risk factor. Analysis of 985,737pt undergoing PCI confirmed underlying CKD as greatest risk factor for CIN
- Diabetes not an independent risk factor but magnifies susceptibility in pts with underlying CKD
- Any prerenal state is risk factor for CIN. This includes cirrhosis, nephrotic syndrome, CHF especially if EF less than 35%, dehydration etc

Contrast Nephropathy- Risk Factors

- Arteriography thought to be higher than than CT given concentrated material delivered to the kidneys and higher overall risk factors in this patient population
- As noted previously underlying CKD is the greatest independent risk factor for development of CIN and probably the best determinant as to risk of development of issues post procedure
- Prognostic Nutritional Index uses the serum albumin and peripheral blood lymphocyte count. Meta-analysis of 17,590 pts use in predicting CIN in pts receiving arteriography or PCI showed higher risk in pts with low PNI vs higher PNI
- Bottom line is to assess pt for risk factors **before** contrast procedure performed

Risk Stratification Strategies

- The presence of 2 or more rise factors appears additive and likelihood of CIN increases sharply as the number of risk factors increases
- Studies have been primarily studied in PCI; Examples:

Mehran in 2004 used a scoring system based on points awarded to the following:

Hypotension- 5 points

IABP- 5 points

Creatinine greater than 1.5- 4 points

Anemia- 3 points

Diabetes- 3 points

Contrast volume 1 point for every 100 ml used

Mehran calculated score

- Low risk- score less than or equal to 5: CIN rate 7.5%, dialysis 0.04%
- Moderate risk- score 6-10: CIN rate 14%, dialysis 0.12%
- High risk- score 11-15: CIN rate 26.1%, dialysis in 1.09%
- Very high risk- score greater than 16- CIN 57.3%, dialysis in 12.6%

Represents a complex scoring system back in 2004 but risk factors remain unchanged as far as risks to consider

Simpler Risk Scoring System Lin et al 2017

- Each risk factor is assigned 1 point:

Age greater than 75

Baseline creatinine greater than 1.5

Hypotension

IABP use

Low risk 0 points; 1.0%

Moderate risk- 1-2 points; incidence 13.4%

High risk- 3 points or more; incidence **90%**

Risk Factors for Contrast Nephropathy

Patient Characteristics *Nephrotoxic Agents*

Age>75	Nephrotoxic Agents
Diabetes Mellitus	Aminoglycosides
Hypertension	Vancomycin
Hypotension	Amphotericin B
CHF	Ace Inhibitors/ARB agents
Decreased EF less than 35%	Diuretics
Cardiogenic Shock	Metformin
Acute MI	NSAIDS
IABP	Type of Contrast Media
Pre-exisiting Renal Failure	
Dehydration	

Contrast Nephropathy

Intravenous CT Contrast Media and Acute Kidney Injury: A Multicenter Emergency Department–based Study

AKI, 48–72h	Contrast Event/total (%)	Non-contrast Event/total (%)	Adjusted OR‡ [95% CI]	P_{int}
Overall	1,105/10,143 (10.9)	981/11,921 (8.2)	1.16 [1.04, 1.30]**	.048
≥90	170/3,487 (4.9)	129/2,577 (5.0)	0.96 [0.73, 1.26]	
60-89	268/3,303 (8.1)	150/2,533 (5.9)	1.19 [0.93, 1.52]	
45-59	202/1,600 (12.6)	132/1,463 (9.0)	1.16 [0.88, 1.52]	
30-44	185/1,103 (16.8)	200/1,751 (11.4)	1.35 [1.06, 1.73]*	
<30	158/650 (24.3)	636/3,597 (17.7)	1.36 [1.09, 1.70]**	
AKI, 48h–1w				
Overall	1,966/31,103 (6.3)	2,126/37,584 (5.7)	1.00 [0.93, 1.08]	.017
≥90	438/11,766 (3.7)	338/8,990 (3.8)	0.99 [0.84, 1.17]	
60-89	460/10,253 (4.5)	365/8,827 (4.1)	0.95 [0.81, 1.12]	
45-59	301/4,517 (6.7)	314/4,888 (6.4)	0.86 [0.72, 1.04]	
30-44	284/3,017 (9.4)	432/5,231 (8.3)	1.02 [0.86, 1.21]	
<30	272/1,550 (17.5)	1,261/9,648 (13.1)	1.49 [1.27, 1.74]***	

Association between contrast media exposure and the likelihood of acute kidney injury.

- In a retrospective study of 68 687 patients who underwent intravenous contrast-enhanced CT had a higher risk of acute kidney injury (AKI) within 48–72 hours after exposure (odds ratio [OR], 1.16; *P* = .007).
- Among patients with a pre-CT eGFR < 30 mL/min/1.73 m^2, intravenous contrast media exposure was associated with a higher risk of AKI (OR, 1.36–1.49; *P* < .001 to *P* = .007) and further hemodialysis within 1 month (OR, 1.36; *P* = .008).

Su TH and Hsieh CH et al. Published Online: October 12, 2021
https://doi.org/10.1148/radiol.2021204446

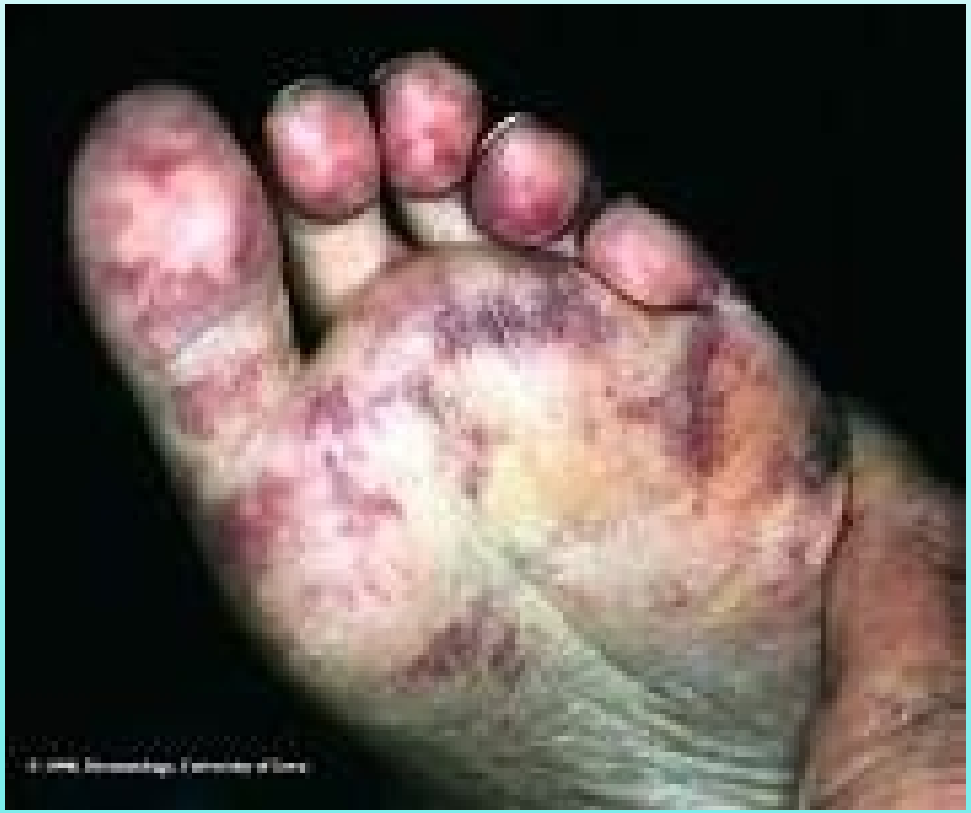
Radiology

History and Physical Findings

- History compatible with onset 24-72 hours after contrast exposure
- Generally non-oliguric(500-600ml/day)
- Concomitant risk factors
- Medications
- Physical exam- useful in ruling out other causes of AKI such as cholesterol emboli, drug induced AIN, dehydration, decompensated heart failure, cirrhosis, and nephrotic syndrome

Differential Diagnosis

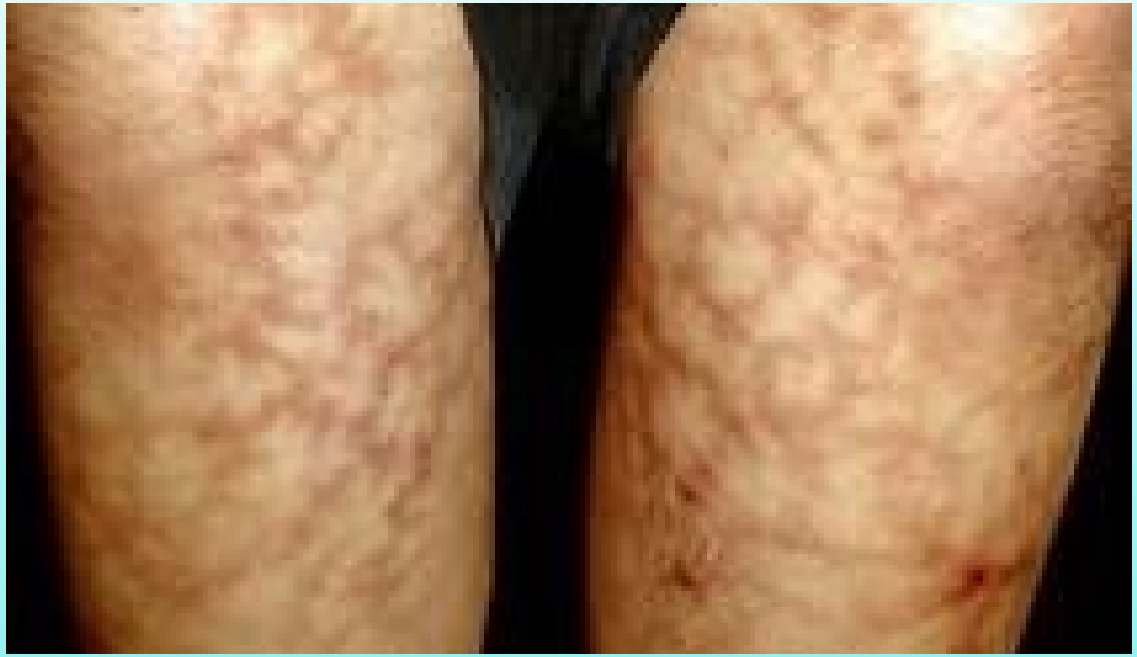
- Atheroembolic renal disease- blue toes, livedo reticularis, eosinophilia, eosinphiluria, and tends to occur 1 week after procedure with continued rise in creatinine and prolonged course of AKI with or without recovery. Cholesterol deposits on renal biopsy
- Other causes of AKI including overdiuresis, other prerenal causes, as well as post renal causes with obstruction
- Acute interstitial nephritis with possible triad of fever, skin rash, and eosinophilia as well as possible eosinophiluria. Many drug induced AIN including certain antibiotics and NSAIDS which tend not cause eosinophilia
- Acute tubular necrosis- can be seen with other nephrotoxins including pigment nephropathy(rhabdomyolysis), chemo agents, organic solvents, heavy metals, etc



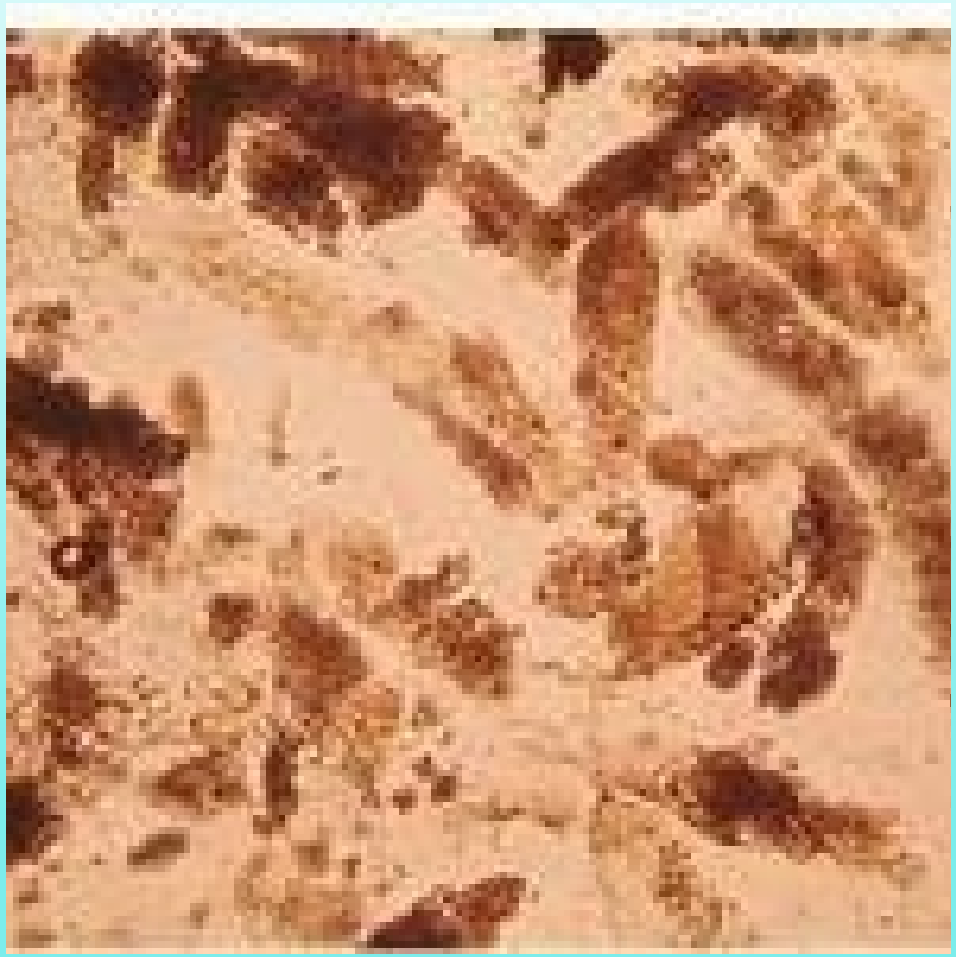
Cholesterol Emboli

- Showering of Emboli to Small Vessels

Cholesterol Emboli



Livedo reticularis



Muddy Brown Casts

Workup

- Really relies on history and physical examination
- Creatinine begins to rise within 24 hours, peaks at day 3-5 and returns to baseline with in 10 days
- Urine may show muddy brown casts, RTE cells, debris but findings non descript
- Urine osmolarity usually less than 350mOsm/kg with variable FeNa
- Again history and physical remains the most important diagnostic tool
- Rule out other causes by analyzing other potential causes of acute kidney injury

Treatment

- Prevention remains the cornerstone of treatment of contrast nephropathy
- Multiple treatments have been proposed including:

Hydration

Sodium Bicarbonate

N-acetycysteine

Statins

Theophylline

Diuretics

Dialysis

Preventive Measures

- Fluids- the only tried and true preventative measure for contrast nephropathy
- Outpatient:

Give 3ml/kg over one hour pre-procedure and 1-1.5 ml/hr for 4-6 hours post procedure so at least 6ml/kg infused overall

Inpatient:

1ml/kg to be given 6-12 hours before the procedure and continue for 6-12 hours after

IV volume expansion remains the standard of care despite absence of randomized studies demonstrating the benefit

One trial the AMACING found no benefit but the study was limited by the fact that 65% of pts had GFR 46-59%

Pts with chronic heart failure should be given fluid but at reduced dose

N-acetylcysteine

- Initially felt to be beneficial in preventing contrast nephropathy in conjunction with hydration
- Conflicting data in regards to benefit
- Most recent study in 2018 in NEJM PRESERVE Trial Group looked at 5177 high risk pts.
- Pts received either sodium bicarbonate or saline and 5 days of NAC or placebo. Primary composite endpoint of death, the need for dialysis, or persistent increase of creatinine of at least 50% above baseline at 90 days. Showed no difference in pts at high risk for renal failure
- Some other studies with mixed data. No harm using oral NAC but no longer considered standard of care

Statins

- Felt to have favorable effects on endothelin and thrombus formation, plaque stabilization, and anti-inflammatory properties.
- No prospective studies but meta-analysis seemed to suggest that statins might reduce the risk of contrast nephropathy
- Short term rouvastatin use in pts with DM and CKD seemed to reduce risk of contrast renal disease. Study published in 2014
- Meta-analysis has not shown statins to be effective in preventing contrast nephropathy in highest risk group of pts- those with CKD
- Further studies will be necessary to further delineate any benefit. May be difficult to tease out given widespread use of statins

Other Therapies

- Ascorbic acid- one study seemed to show 3 gram dose pre-procedure and two subsequent 2 gram doses lowered risk by 62%. A second large study did not show a benefit
- Oral sodium citrate- showed a benefit in one study
- Atrial natriuretic peptide – beneficial in animals but not humans
- Vasodilators such as calcium channel blockers, dopamine/fenoldopam, have all had mixed results
- Loop diuretics and mannitol in hope that this would dilute contrast with in the tubule and in the case of loop diuretics decrease metabolism within the medullary portion of the kidney to decrease ischemic damage have not borne out. In fact, they seem to increase the risk

Renal Replacement Therapy

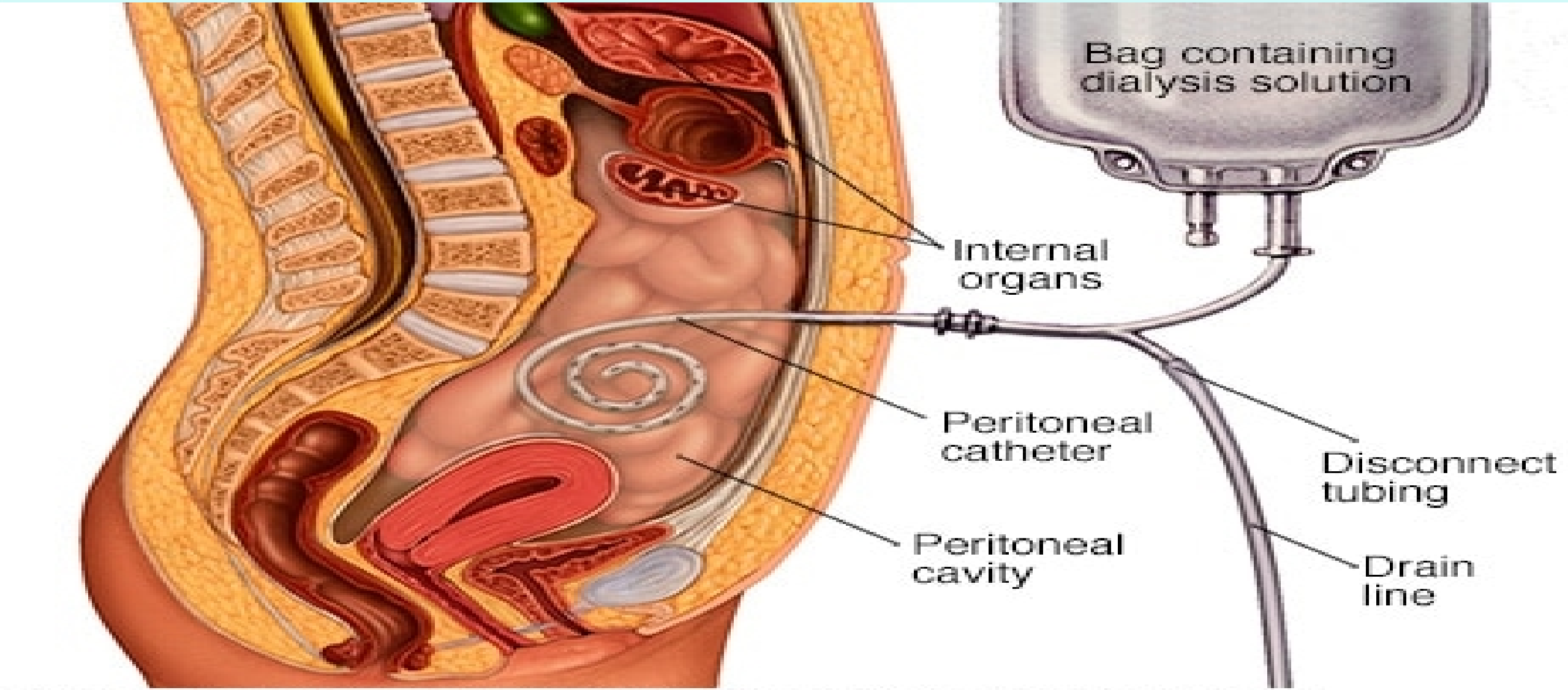
- In patients with severe kidney disease and diabetes incidence of need to dialyze pt can be as high as 12%. Those pts who require dialysis can have in-hospital mortality as high as 35% and two year survival of 19%.
- Pts with normal kidney function have the contrast excreted on first pass through glomerulus with 50% eliminated within 2 hours vs 16-84 hours in patients with renal impairment
- Greatest risk for contrast nephropathy is in CKD possibly because of contrast concentration in remaining nephrons and prolonged contrast exposure and can have implications for residual renal function in ESRD
- Hemodialysis can efficiently remove contrast but no data suggesting prophylactic dialysis can improve outcome

Contrast in Dialysis Population

- No studies supporting immediate dialysis following contrast procedure in pts already on dialysis; can wait until next scheduled dialysis
- Only concern is that of volume expansion/overload which is minimized by low and iso-osmolar contrast material
- Preservation of residual renal function very important in the ESRD population especially in pts on peritoneal dialysis which rely to some extent on residual kidney function for dialysis adequacy
- ESRD DOES NOT EQUAL “CARTE BLANCHE” TO GIVING PT IV CONTRAST. The same discussion should be undertaken in the ESRD population as in the CKD population

Home Hemodialysis

Peritoneal Dialysis



Growth of Home Dialysis

	Home Penetration %		
PHC Location	Q3'22	Q3'23	YoY Variance
DAYTON OHIO	26.6%	32.1%	5.5%
WORCESTER, MA	24.0%	27.1%	3.1%
TULSA, OK	23.6%	25.4%	1.7%
NASHVILLE, TN	21.0%	23.9%	2.9%
SAVANNAH, GA	19.8%	22.9%	3.1%
INDIANAPOLIS, IN	21.7%	21.5%	-0.2%
VIRGINIA BEACH, VA	20.8%	21.0%	0.2%
ALBUQUERQUE, NM	20.7%	20.8%	0.0%
PADUCAH, KY	20.8%	20.0%	-0.8%
SAN ANTONIO, TX	20.7%	19.9%	-0.8%
GREENVILLE, NC	21.8%	19.4%	-2.5%
WEST CHICAGO, IL	16.8%	19.1%	2.3%
SPRINGFIELD, MA	18.2%	18.9%	0.7%
JACKSON, MS	19.0%	18.7%	-0.2%
LAS VEGAS, NV	16.1%	18.2%	2.1%
TUPELO, MS	18.5%	18.0%	-0.6%
CHARLOTTE, NC	16.6%	16.8%	0.2%
ST. LOUIS, MO	15.7%	16.1%	0.4%
COLUMBIA, SC	14.8%	15.3%	0.6%
JACKSONVILLE, FL	14.8%	15.3%	0.5%
BAYTOWN, TX	15.9%	15.3%	-0.6%
ATLANTA, GA	14.2%	15.2%	1.0%
MOBILE, ALABAMA	13.8%	14.6%	0.8%
ASHLAND, KY	12.3%	14.4%	2.0%
BATON ROUGE, LA	14.3%	14.2%	-0.1%
LOUISVILLE, KY	13.2%	13.5%	0.4%
WACO, TX	12.8%	13.2%	0.4%
SAN DIEGO, CA	14.7%	12.4%	-2.4%
SOUTH MIAMI & NORTH MIAMI	11.4%	11.4%	-0.1%
CHICAGO, IL/SOUTH	11.0%	10.9%	-0.1%
PHILADELPHIA, PA	9.0%	10.2%	1.2%
DALLAS, TX	9.9%	9.9%	0.1%
CHICAGO, IL/NORTH	8.6%	9.5%	0.9%
MINNEAPOLIS, MN	8.2%	9.0%	0.8%

Use of Intravenous Iodinated Contrast Media in Patients with Kidney Disease: Consensus Statements from the American College of Radiology and the National Kidney Foundation

KDIGO AKI Staging	
Stage	Serum Creatinine Criteria
1	1.5–1.9 times baseline serum creatinine
	OR
	Increase in serum Cr ≥ 0.3 mg/dL (≥26.5 µmol/l)
2	2.0–2.9 times baseline serum creatinine
3	3.0 times baseline serum creatinine
	OR
	Increase in serum Cr to ≥ 4.0 mg/dL (≥353.6 µmol/l)
	OR
	Initiation of kidney replacement therapy
	OR
	Decrease in eGFR to < 35 mL/min/1.73 m^2 (for patients < 18 years old)

Kidney Disease Improving Global Outcomes (KDIGO) staging criteria for acute kidney injury (AKI).

- The risk of contrast-induced acute kidney injury has been estimated to be near 0% at eGFR greater than or equal to 45, 0%–2% at eGFR of 30–44, and 0%–17% at eGFR less than 30 mL/min/1.73 m^2.

- Prophylaxis for contrast-induced acute kidney injury with IV normal saline is indicated for patients with an eGFR less than 30 mL/min/1.73 m^2 who are not undergoing maintenance dialysis, or in high-risk patients with an eGFR of 30–44 mL/min/1.73 m^2.

Davenport MS et al. Published Online: January 21, 2020
https://doi.org/10.1148/radiol.2019192094

Radiology

ACR Recommendations and Recommendations from Cleveland Clinic

Consensus statement	Authors' comments
The risk of contrast-*induced* acute kidney injury is substantially less than the risk of contrast-*associated* acute kidney injury, but the actual risk remains uncertain. However, necessary contrast-enhanced CT without an alternative should not be withheld.	We believe this statement should be extrapolated to patients in whom coronary angiographic procedures are deemed necessary.
Patients at risk for contrast-induced acute kidney injury include those with recent acute kidney injury or those with eGFR < 30 mL/min/1.73 m^2 (including nonanuric dialysis patients).	Age, diabetes, hypertension, and proteinuria are absent from the risk classification. We believe patients with an eGFR < 45 mL/min/1.73 m^2, particularly those with the above noted risk factors, should also be considered at increased risk.
Prophylaxis with intravenous isotonic saline is indicated for patients with eGFR < 30 mL/min/1.73 m^2 not undergoing dialysis and in patients with acute kidney injury.	We believe that prophylaxis is also warranted in nonanuric patients on hemodialysis or peritoneal dialysis to preserve residual kidney function. Careful attention to volume status is required to avoid hypervolemia.
Prophylaxis should be individualized for high-risk patients with eGFR between 30 and 44 mL/min/1.73 m^2.	We support prophylaxis in this population, particularly in the presence of traditional risk factors (diabetes, hypertension, proteinuria).
Prophylaxis is not indicated for patients with stable eGFR ≥ 45	We concur that the risk of contrast-induced acute kidney injury in this population is

Prevention of Contrast Nephropathy

- #1 is avoidance if possible
- Hydration in patients with GFR less than 30 or high risk pts with GFR 30-44
- No need to hold Ace inhibitors or ARB agents
- Hold diuretics if risk of volume depletion
- Unclear if newer agents such as SGLT2 inhibitors need to be held although they do lead to some volume depletion
- Cirrhosis, CHF, nephrotic syndrome are prerenal conditions and can possibly increase risk of contrast nephropathy

Gadolinium Induced Nephrogenic Systemic Fibrosis

- NSF is a debilitating and often fatal disease characterized by skin thickening and organ fibrosis
- First reported in HD pts in 2000; agents available since 1980's and felt to be safe
- Initially unknown association until gadolinium was detected by electron microscopy on skin biopsy
- Disease was well established by 2009
- Gadolinium agents with linear shape were of higher risk
- Risk greater in GFR less than 30
- Dialysis was recommended in pts who needed these studies and GFR less than 30 as Gad is removed by hemodialysis

Gadolinium Agent Classification

- Group I – agents associated with greatest number of NSF cases
- Group II- agents associated with few, if any cases
- Group III- agents with limited data

TABLE 3

Gadolinium-based contrast agents and risk of nephrogenic systemic fibrosis

	Cyclic	Linear
Ionic	Gadoteric acid	Gadobenate dimeglumine
		Gadofosveset Gadoxetic acid
		Gadopentetate dimeglumine
Nonionic	Gadoteridol Gadobutrol	Gadodiamide Gadoversetamide

Red—group I agents: associated with the greatest number of cases of nephrogenic systemic fibrosis

Green—group II agents: associated with few cases

Yellow—group III agents: data are limited, but few unconfirmed cases have been reported

Kidney function	Recommendation ACR	Clinic Authors
CKD 1 or 2	No increased risk of developing NSF	No case reports
CKD 3	Minimal risk of developing NSF	No definite cases
CKD 4 or 5 not on dialysis	Group I agents contraindicated If study needed use Group II	Use only Group II agents given increased risk
ESRD on hemodialysis	ACR favors contrasted CT over MRI. Use only group II agents Need to time with dialysis and do dialysis as soon as possible	CT contrast may affect needed residual renal function Use Group II agents and perform single dialysis ASAP rather than two
ESRD on peritoneal dialysis	ACR favors CT over MRI but in MRI only Group II agents. PD pts may have some increased NSF risk	Again need to concern with CT with residual kidney function Group II agents only and consider single HD session especially in pts with no residual kidney function
Acute Kidney Injury	Group II agents only	If on dialysis perform HD session If not on dialysis and nonoliguric treat as above with CKD Oliguric kidney failure, avoid and if needed use Group II and follow with single dialysis session

ACR Manual on Contrast Media and Nephrogenic Systemic Fibrosis and Cleveland Clinic Recommendations

Contrast Nephropathy –Summary

- Yes it does exist, some patients at higher risk than other patients. Preexisting kidney disease remains the greatest risk factor with Diabetes and other chronic disease states especially Pre-renal states magnifying the risk
- Generally safe in patients whose GFR is greater than 45, however risk is not zero as incidence tends to be underreported in this group. Other disease as above may increase risk even in this population. If another option or cause of illness ie CHF etc noted then may not be necessary to give contrast
- IV hydration remains the only definitive preventive measure other than avoidance
- With the growing home dialysis population, dialysis itself does not equal ”Cart Blanche” to giving contrast

Gadolinium Based Procedures

- Newer agents certainly safer than first generation agents
- In the dialysis population, a single dialysis should be performed immediately after contrast even in Group II agents and in Peritoneal population with no residual function need to consider a single dialysis treatment if feasible
- In acute oliguric renal failure should consider dialysis after Gadolinium procedure
- BOTTOM LINE- INDIVIDUALIZE NEED FOR CONTRAST PROCEDURES WHETHER IODINATED OR GADOLINIUM AND RESPECT POSSIBILITY OF ADVERSE OCCURENCES. HISTORY, PHYSICAL AND UNDERLYING DISEASE STATES SHOULD HELP DIRECT THERAPY RATHER THAN USING STUDIES THAT MAY OR MAY NOT BE NEEDED,ESPECIALLY IN HIGH RISK POPULATIONS

The End

Go Eagles

SAVE THE DATE!

NEXT GRAND ROUNDS, THURSDAY, JANUARY 4TH!

"The Controversies of CBD"

Presented by
Cynthia Sheppard Solomon, BSPharm, RPh, FASCP, CTTS, NCTTP
Clinical Associate Professor, Department of Internal Medicine at Wright State University
and
Glen D. Solomon, MD, MACP, FRCP (London)
Professor and Chair, Department of Internal Medicine at Wright State University

Registration and other information will be shared next week!

Evaluation

Using the **QR code** or the **link** placed in the chat on the **Teams Live Event** please complete the evaluation to receive the CME credit being offered.

Evaluation will close on 12/22 – CME certificates will be emailed by 1/4.

Contact Dana Mackert, dlmackert@premierhealth.com, for any questions.