Bracketology

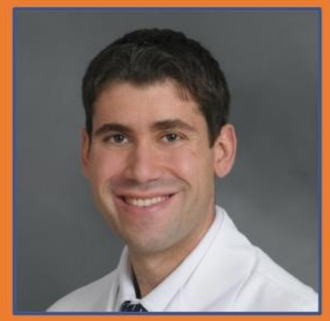
www.AJKDBlog.org
March 1-31, 2025

Slides Prepared by the NephMadness Executive Committee

- Matthew Sparks
- Anna Burgner
- Anna Vinnikova
- Elena Cervantes
- Jeffrey Kott
- Ana Catalina Alvarez-Elías
- Dia Waguespack
- Krithika Mohan








What to do

- Review the Regions & Teams on *AJKD*Blog
- Submit your picks by March 31, 2025, on Tourneytopia
- Discuss on social media using #NephMadness
- Claim CME/MOC credit through NKF PERC

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The BRP Has Chosen the Winning Bracket

To win, predict what the BRP thinks will bring the most practice change to nephrology!

- Roger Rodby
- Deidra Crew
- Kirk Campbell
- Samira Farouk
- Tom Oates
- Meredith Atkinson
- Linnys Alcántara Quiroga
- Clodagh Sweeney
- David Rush

NEPH MADNESS 2025

Resistant Hypertension

Writer:
Stephanie Torres Rodriguez

Expert:
Jordana Cohen

Region Execs:
Matthew Sparks
Elena Cervantes

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Novel Drugs for Hypertension

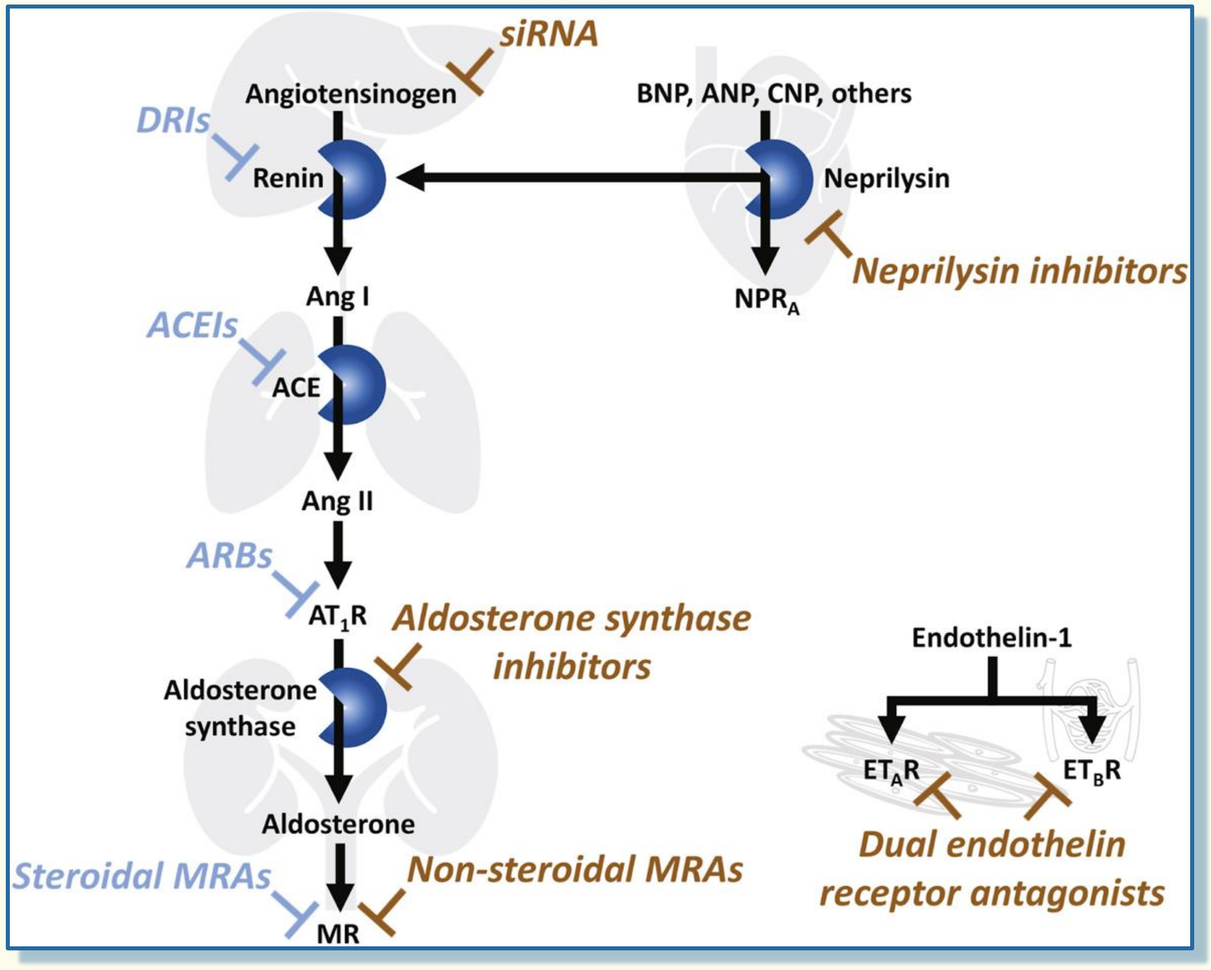
Novel Drugs for Hypertension

Definition

- Blood pressure remains above goal despite concurrent use of ≥3 anti-hypertensive agents of different classes
- If tolerated – one of the agents should be a diuretic
- All agents should be prescribed at maximum recommended (or maximally tolerated) anti-hypertensive doses

OR

- Blood pressure controlled with ≥4 medications – also is considered resistant



Evaluate for other contributors to hypertension

- Untreated obstructive sleep apnea
- Hormone dysregulation
- Hyperaldosteronism
- Thyroid disease (hypo/hyperthyroidism)
- Pheochromocytoma / paraganglioma
- Hypercortisolism (Cushing syndrome)
- Apparent mineralocorticoid excess
- Volume overload
- Aortic coarctation
- Renal artery stenosis
- Fibromuscular muscular dysplasia – 10-25% of RAS
- Atherosclerotic disease

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Novel Drugs for Hypertension

- Dual endothelin receptor antagonist: **aprocitentan**

BP Effect ~4 mmHg

FDA Approved

Adverse effects ~10-15% fluid retention

Route PO Daily

- Aldosterone synthase (CYP11B2) inhibitor: **baxdrostat & lorundrostat**

BP Effect ~10 mmHg

Adverse effects ~2% hyperkalemia

PO Daily

- Small interfering RNA (siRNA) for angiotensinogen: **zilebesiran**

BP Effect ~4 mmHg

Adverse effects ~6-7% hyperkalemia
~10% injection site reaction

SQ q 6 months

- Glucagon-like peptide-1 receptor agonist (GLP-1 RA): **semaglutide**

BP Effect ~5 mmHg ↓ anti-HTN meds

FDA Approved

Adverse effects ~30% nausea
~10% diarrhea

SQ q week

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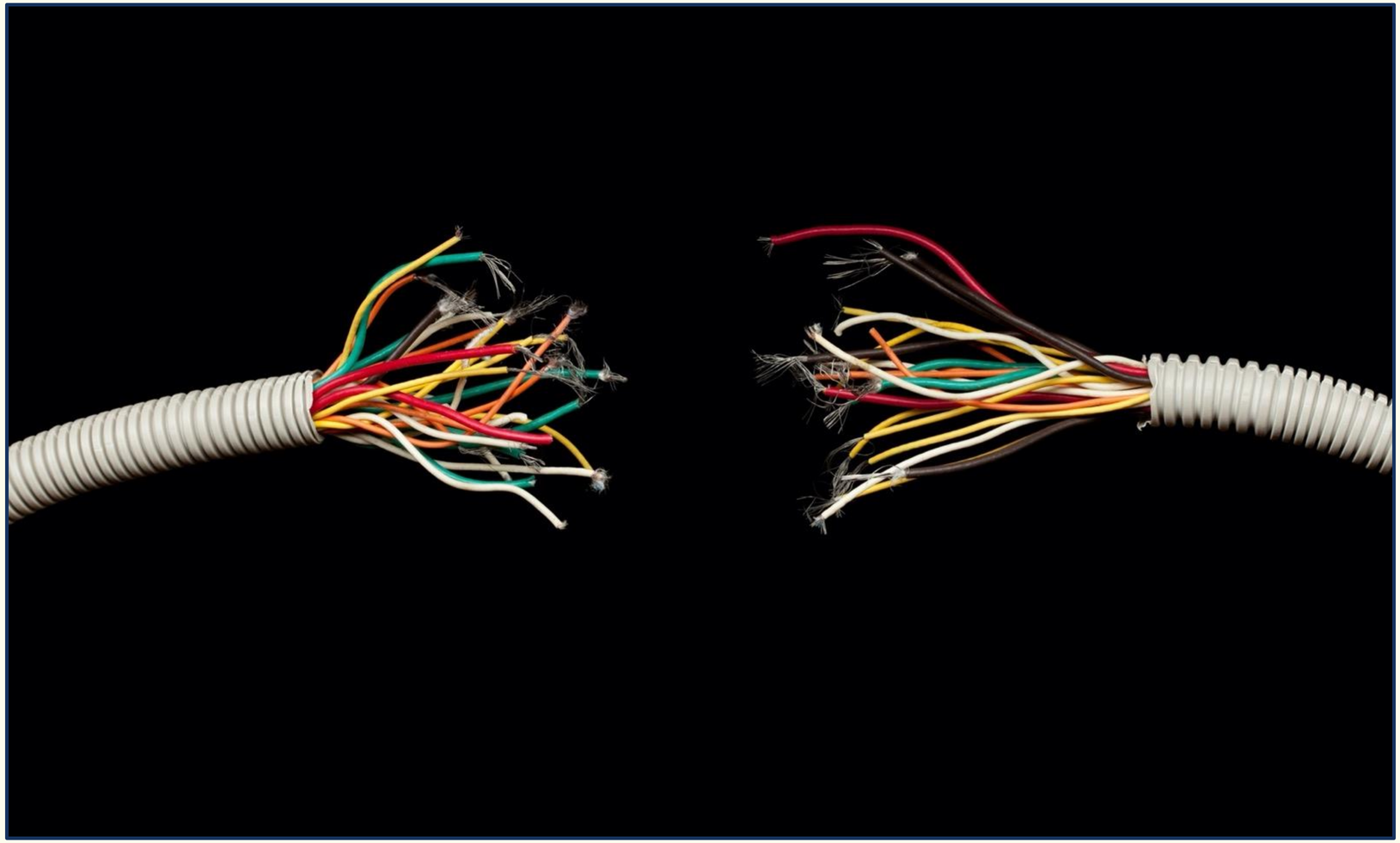
Renal Denervation



What is the role of the renal nerves in the pathophysiology of hypertension and other diseases?

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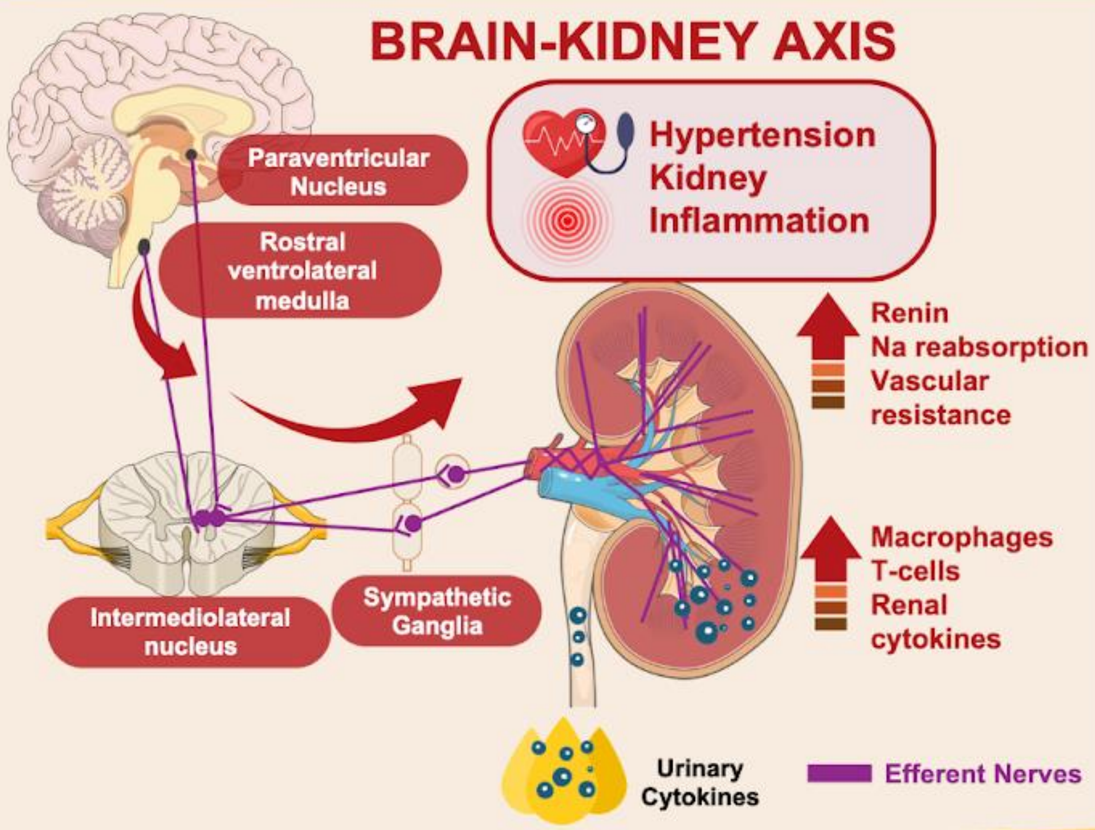

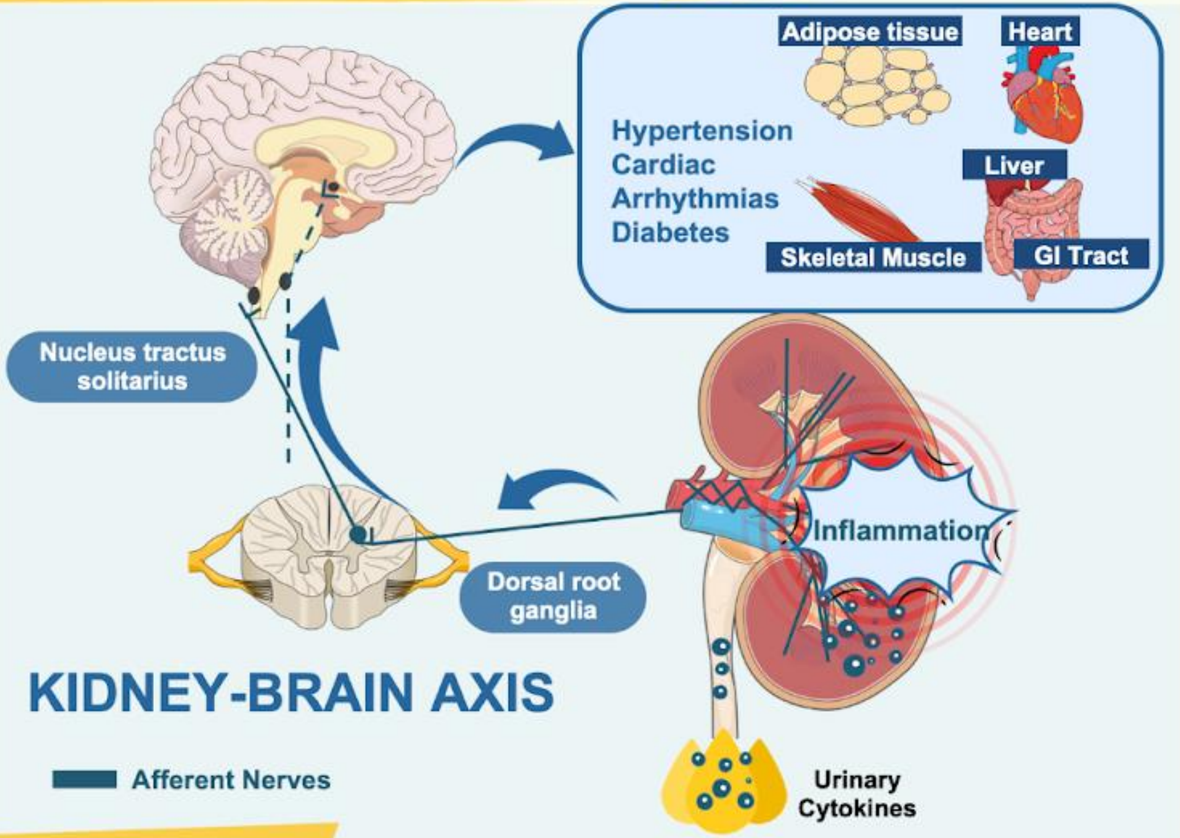

CONCLUSION: The contribution of the brain-kidney axis (efferent renal nerves) and kidney-brain axis (afferent renal nerves) in disease. The brain-kidney axis transmits signals from the central nervous system to the kidneys and can result in increased renin release, sodium reabsorption, and renal vascular resistance. Recent studies suggest that this pathway also promotes the infiltration of macrophages and T cells, which release inflammatory cytokines in the kidney that are also detected in the urine. The kidney-brain axis sends afferent signals from the kidneys (secondary to inflammation) to the central nervous system, which increases sympathetic nervous system output to other organs, resulting in hypertension, cardiac arrhythmias, diabetes, and possibly other conditions.

Reference Osborn et al. **Function of Renal Nerves in Kidney Physiology and Pathophysiology.** Annual Review of Physiology. 2021

Visual Abstract by @hellokidneyMD

Can Simplicity radiofrequency-based renal denervation reduce blood pressure?

Conclusions from SPYRAL trials

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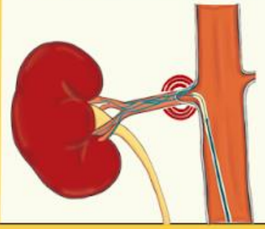

	SPYRAL HTN-OFF MED	SPYRAL HTN-ON MED	
		PILOT	EXPANSION
	Multicenter, randomized, sham-controlled, single-blind		
Denervation method	Simplicity G3 or SPYRAL (multi-electrode); Main + branch accessory >3mm		
Population	331 patients 166 renal denervation, 165 sham Not on antihypertensive medication	80 patients 32 renal denervation, 42 sham On 1-3 antihypertensive drugs	337 patients 206 renal denervation, 131 sham On 1-3 antihypertensive drugs
Follow up	3 months	6 months	6 months
Characteristics	66% male, 29% smokers, 20% diabetes, 24% obstructive sleep apnea	87% males, 21% smokers, 13% diabetes, 5% obstructive sleep apnea	80% males, 25% smokers, 22% diabetes, 23% obstructive sleep apnea
BP reduction (24 h SBP)	-3.9 mmHg Bayesian CI: -6.2 to -1.6	-7.4 mmHg 95% CI: -12.5 to -2.3	-1.9 mmHg 95% CI: -4.4 to -0.5
-Probability of RDN being superior -Primary Outcome	N/A Positive	97.5% Positive	51% Negative
Medication		Adherence: approx. 60%, varied among patients	Medication burden 17% (RDN) vs 26% (control) 16% (RDN) vs. 10% (control)

Conclusion: RDN showed stronger effects in drug-naive patients (OFF MED) but inconsistent efficacy in treated populations (ON MED). The pilot (97.5% probability of superiority) suggested benefit, but the expansion study (51%) failed to confirm it, raising doubts about its incremental value over medication, durability, and real-world applicability.

Reference: 1.Böhm M, et al. *Efficacy of catheter-based renal denervation in the absence of antihypertensive medications (SPYRAL HTN-OFF MED Pivotal): a multicentre, randomised, sham-controlled trial.* Lancet. 2020

2. Kandzari DE, et al. *Effect of renal denervation on blood pressure in the presence of antihypertensive drugs: 6-month efficacy and safety results from the SPYRAL HTN-ON MED proof-of-concept randomised trial.* Lancet. 2018

3. Kandzari DE, et al. *Safety and Efficacy of Renal Denervation in Patients Taking Antihypertensive Medications.* J Am Coll Cardiol. 2023

VA by @nephroseeker.medsky.social

Can Paradise ultrasound-based renal denervation reduce blood pressure?

Conclusions from RADIANCE trials

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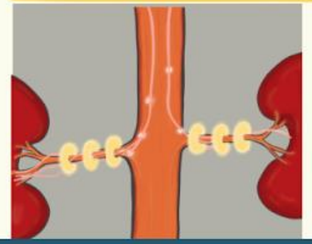


	RADIANCE-HTN SOLO N= 146	RADIANCE-HTN TRIO N= 136	RADIANCE II N= 224
Denervation method	Multicenter, randomized, sham-controlled, single-blind		
	Paradise system (ultrasound); Main renal artery, 2-3 spots 5 mm apart		
Follow up	2 months		
Blood pressure inclusion criteria	Ambulatory BP 135/85 to <170/105	Office BP ≥140/90 mmHg + ambulatory BP ≥135/85 mmHg despite triple therapy	Ambulatory BP 135/85 to <170/105
Population	41% male, 5% type 2 diabetes, 10% sleep apnea	20% male, 28% type 2 diabetes, 28% sleep apnea	28% male, 6% type 2 diabetes, 15% sleep apnea
Baseline therapy Number of antihypertensive medications at screening	Off antihypertensive medication (4-week washout of ≤2 drugs) 1.2±0.8	Standardized triple therapy (CCB + ARB + diuretic) 4.0±1.1	Off antihypertensive medication (4-week washout of ≤2 drugs) 1.0±0.8
Medication use post-randomization	None (unless BP criteria exceeded)	Continued triple therapy (addition allowed if BP exceeded threshold)	None (unless BP criteria exceeded)
Change in daytime ambulatory SBP — Sham	-2.2 mmHg (SD 10)	-3.0 mmHg (IQR -10.3 to 1.8)	-1.8 mmHg (SD 9.5)
Change in daytime ambulatory SBP — Renal denervation	-8.5 mmHg (SD 9.3)	-8.0 mmHg (IQR -16.4 to 0.0)	-7.9 mmHg (SD 11.6)
Difference 95% confidence interval	-6.3 mmHg -9.4 to -3.1	-4.5 mmHg -8.5 to -0.3	-6.3 mmHg -9.3 to -3.2

Conclusion: The RADIANCE trials confirm that ultrasound renal denervation significantly lowers BP versus a sham procedure, with similar reductions in untreated mild-to-moderate hypertension (SOLO, RADIANCE II) and a smaller effect in resistant hypertension (TRIO). RDN appears safe, but the short 2-month follow-up limits conclusions on long-term efficacy.

Reference: 1.Azizi M, et al. *Endovascular ultrasound renal denervation to treat hypertension (RADIANCE-HTN SOLO): a multicentre, international, single-blind, randomised, sham-controlled trial.* Lancet. 2018

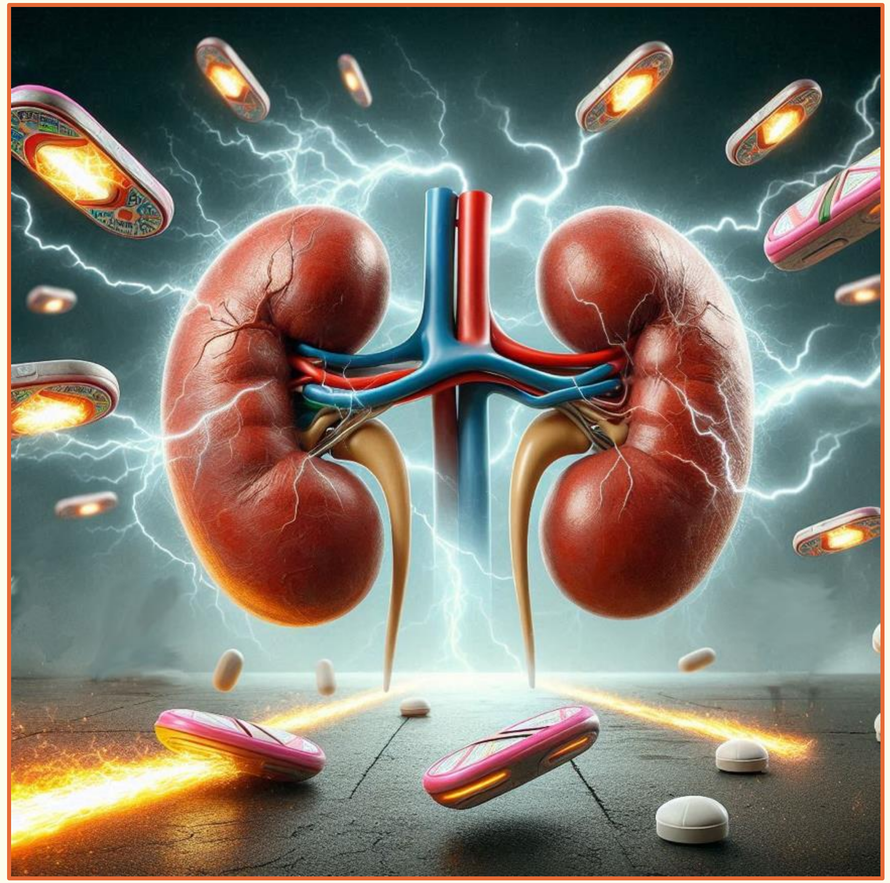
2. Azizi M, et al. *Ultrasound renal denervation for hypertension resistant to a triple medication pill (RADIANCE-HTN TRIO): a randomised, multicentre, single-blind, sham-controlled trial.* Lancet. 2021

3. Azizi M, et al. *Ultrasound Renal Denervation to Treat Hypertension: The RADIANCE II Randomized Clinical Trial.* JAMA. 2023

VA by @nephroseeker.medsky.social

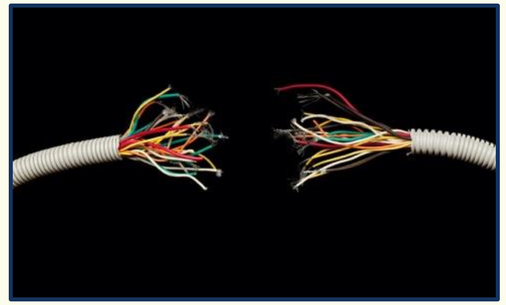
EFFLUENT EIGHT ROUND

Pick Your Champion for the Resistant Hypertension Region



Novel Rx for HTN

VS



Renal Denervation

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Disaster Nephrology

Writer:
Rasha Raslan

Expert:
Mehmet S Sever

Region Execs:
Anna Vinnikova
Ana Catalina Alvarez-Elías

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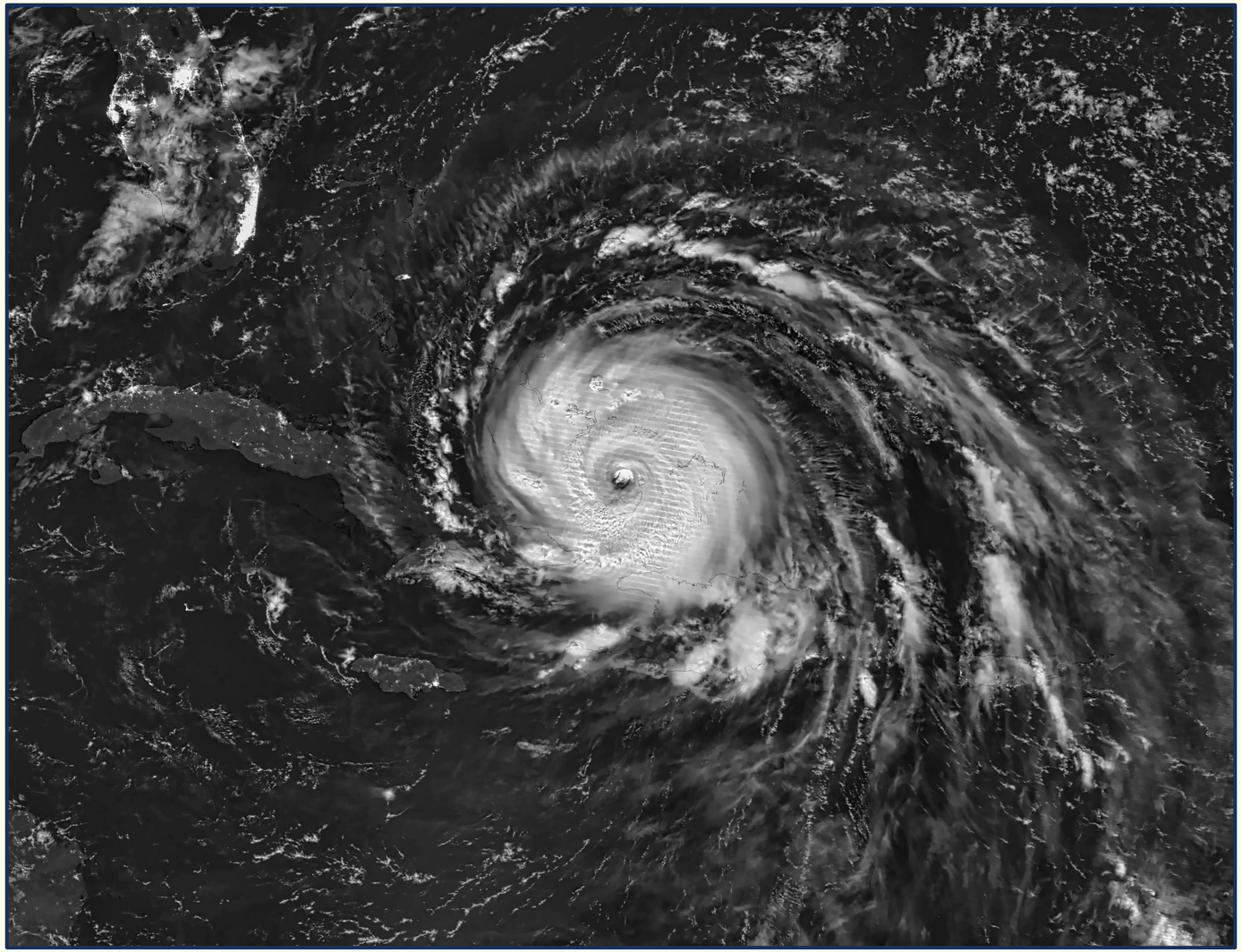
Kidney Care In Natural Disasters



Disaster Nephrology and Crush-AKI

Disaster Nephrology is a specialized field focused on coordination of care of kidney patients during a disaster. The term evolved from the earlier “Seismo-Nephrology” dedicated to managing Crush-AKI.

AKI from crush injury was first recognized during World War 1.

World War II saw the introduction of the artificial kidney by Kolff and the first ever HD treatment.

Pathophysiology of crush syndrome on the basis of traumatic rhabdomyolysis

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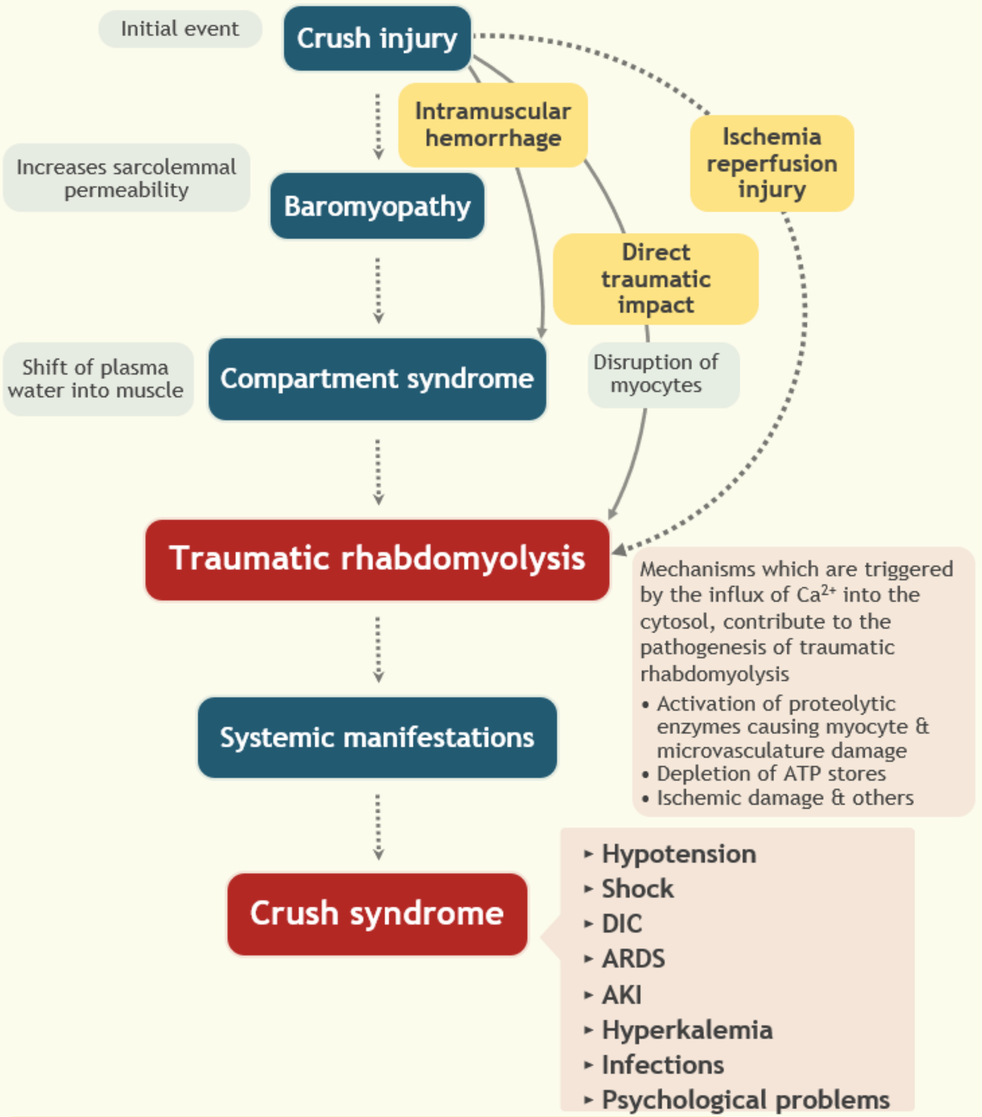

Reference: Sever MS et al., Destructive disasters, trauma, crush syndrome, and beyond. Acta Orthop Traumatol Turc, 2023 Nov;57(6):305-314

VA by @krithicism

Recommendations for Crush-AKI management

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Before extrication

A vein is sought in one of the limbs

- A vein is found
  - Initiate isotonic saline (1L/h)

- No vein can be found
  - No fluid is given

During extrication

- Continue isotonic saline (1L/h) (15-20ml/kg/hr for children)

- Extrication >2h
  - Reduce isotonic saline (<0.5L/h) (10ml/kg/hr or less for children)

After extrication

No fluid before

- Initiate isotonic saline
- Administer overall 3-6L of fluid (1.5-2L/m² for children)
- Monitor 6 hours since initiation of fluids
- Anuria
  - IV fluid 0.5-1L/day (0.4L/m²/day for children)+ presumed previous day losses

Fluid before

- Continue isotonic saline
- Administer overall 3-6L of fluid (1.5-2L/m² for children)
- Monitor 6 hours since initiation of fluids
- Urine output (+)
  - IV fluid 3-6L/day if close monitoring impossible
  - IV fluid >6L/day (3-6L/m²/day for children) with UOP monitoring

Conclusion: Consider the same principles for prevention and initial management in crush-related AKI as in AKI in general. Initiate early and rapid fluid resuscitation to ensure euvolemia in hypovolemic victims; maintain hydration in euvolemic victims with adequate urine output.

Reference: Sever MS et al, Recommendation for the management of crush victims in mass disasters, NDT, 2012 and personal communications

VA by @deniise_am

Kidney Impact in Natural Disasters

ACUTE KIDNEY INJURY

- Prerenal AKI in entrapped or bleeding victims
- ATN from prolonged shock, sepsis, and transfusion reactions
- Rhabdomyolysis and crush syndrome in crush victims

CKD AND ESKD

- Loss of access to medications
- Emergency disconnects from HD
- Complications from missed dialysis treatments
- Power and water outages
- Issues with supplies delivery

TRANSPLANT

- Disruption of transplant activities
- Loss of access to medications
- Increased infectious complications

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Kidney Care in Natural Disasters

BEFORE

Patients:

- Practice HD emergency disconnect procedures, PD manual exchanges
- Emergency kits
- Stockpile medications and supplies for 1-2 weeks

Dialysis facility:

- Develop disaster protocols
- Contracts for generators and water tankers
- Disaster preparedness training
- Tabletop drills

DURING

- Follow protocols
- Rapid triage and evacuations
- Telemedicine
- Arrange generators, water tankers as needed
- Communication with disaster relief authorities

AFTER

- Re-establish care of neglected conditions
- Mental healthcare for patients and staff
- Evaluate disaster response, debrief, lessons learned

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Kidney Care In Conflicts

Kidney Impact in Conflicts

ACUTE KIDNEY INJURY

- High AKI burden (hard to quantify)
- Pre-renal is the most common due to dehydration or hemorrhagic shock
- Nephrotoxic exposure to noxious gases and toxic agents

CHRONIC KIDNEY DISEASE

- Limited evaluation
- Difficulty with dietary and medication adherence
- Poor access to treatment and supplies
- Degradation of specialized care
- Inability to create longer term vascular access like fistulas

TRANSPLANT

- Poor functionality of transplant facilities or access to specialist
- Increased risk of rejection due to poor access to medications

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Kidney Care in Conflicts

BEFORE	DURING	AFTER
• Training patients and staff on self-security issues • Training on best practices in resource-limited setting • Medication stockpiling • Training patients on emergency dietary restrictions	• Field hospitals • Evacuations • Telemedicine • Rotating schedule for staff	• Restoration of services • Address mental health • Debriefing

Dialysis Modalities During Disasters

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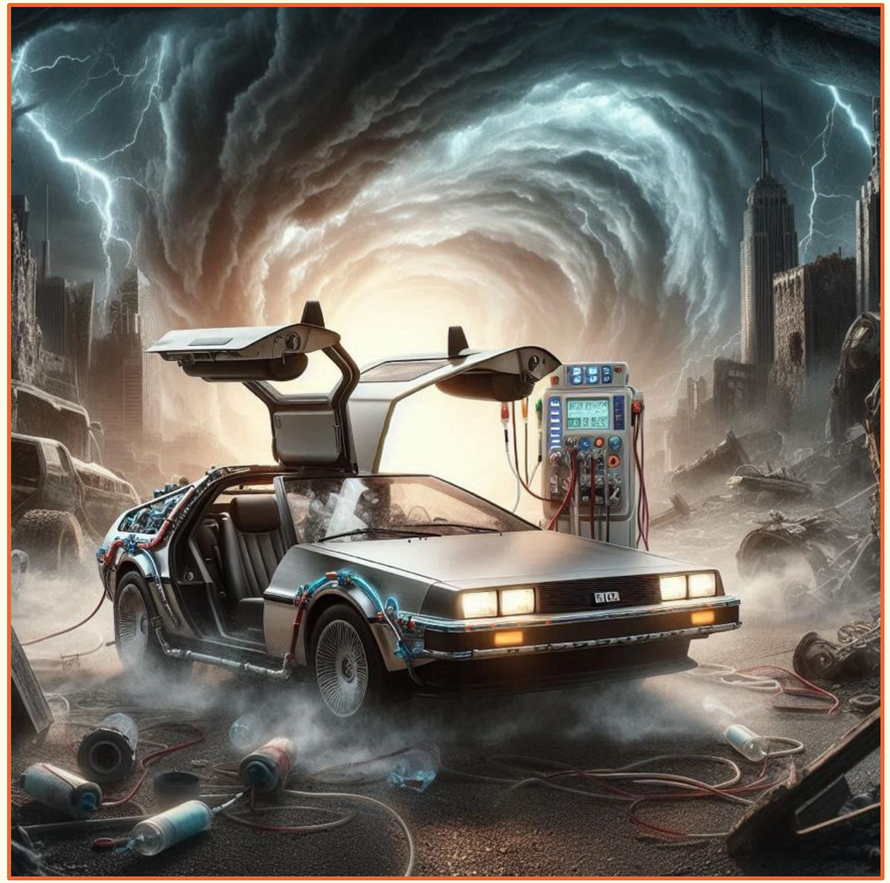
		Advantages	Drawbacks
Intermittent hemodialysis	Medical	• High clearance rate of low molecular weight solutes • Possibility to dialyze without anticoagulation	• Priming volume may induce hypotension • Risk of dialysis disequilibrium syndrome
	Logistic	• Possibility to treat several patients per day	• Need for experienced personnel and infrastructure
Slow continuous therapy	Medical	• Gradual removal of fluid hence better volume control and hemodynamic tolerance • Gradual removal of solutes hence less risk of dialysis disequilibrium • More calories/nutrition can be	• Higher need for anticoagulation • Slower removal of small solutes (eg K)
	Logistic	• Can be established rapidly	• Fewer patients per machine per day than HD • Need for experienced personnel and infrastructure • Need for high volumes of replacement fluid or dialysate
Peritoneal dialysis	Medical	• No need for vascular access • Less hemodynamic instability • Can be initiated rapidly, no risk of dialysis disequilibrium	• Lower clearance of small molecules • Difficult to perform in patients with trauma
	Logistic	• Can be performed without water and electricity	• Difficulty in maintaining sterile technique • Need for high volumes of dialysate

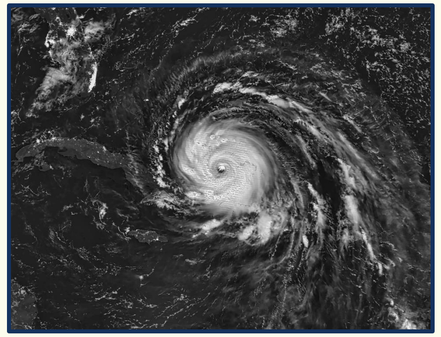
Ref: Collins, Crit Care Clin,1991; Solez et al, KI, 1993; Vanholder et al, KI, 2000; Sever et al, KI, 2002

VA by @krithicism

EFFLUENT EIGHT ROUND

Pick Your Champion for the Disaster Nephrology Region





In Natural Disasters

VS

In Conflicts

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Genetics

Writer:
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Expert:
Jordan Nestor

Region Execs:
Elena Cervantes
Matthew Sparks

NEPH 2025 MADNESS

Genetics in FSGS

Genetics in FSGS

The following are the most common genes with mutations known to cause genetic FSGS:

Gene(s)	Function & Role	Inheritance Pattern	Clinical Significance
NPHS1 & NPHS2	Podocyte signaling and slit diaphragm formation	Autosomal **recessive**	Early-onset disease, including nephrotic syndrome of the Finnish type
COL4A3/4/5	Form a heterotrimer essential for basement membrane structure in glomerulus, cochlea, and eye	Variable in Alport's: **X-linked (COL4A5, most common**), Autosomal recessive or dominant (COL4A3/A4)	Associated with Alport syndrome, thin basement membrane disease, hereditary FSGS, and auditory/ocular involvement
APOL1 (G1 & G2 risk alleles)	Lipid metabolism, apoptosis, immune regulation Innate immunity against **Trypanosoma brucei,** the parasite responsible for African sleeping sickness	APOL 1 G1 and G2 alleles - autosomal **recessive** A **secondary trigger** (infection, obesity, etc.) is required to have the disease	Linked to increased FSGS risk **Inaxaplin**, an APOL1 channel inhibitor, reduced proteinuria by 48% in Phase 2a trials; currently in Phase 3 trials
ACTN4, TRPC6, INF2	Regulate actin dynamics and calcium influx in podocytes	Autosomal **dominant**	Cause hereditary FSGS, typically via gain-of-function mutations

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APOL1 Bi- and Monoallelic Variants and Chronic Kidney Disease in West Africans

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Case - Control Study

Ghana & Nigeria

CKD Stage 3-5 (N=8355)

APOL1 Variants

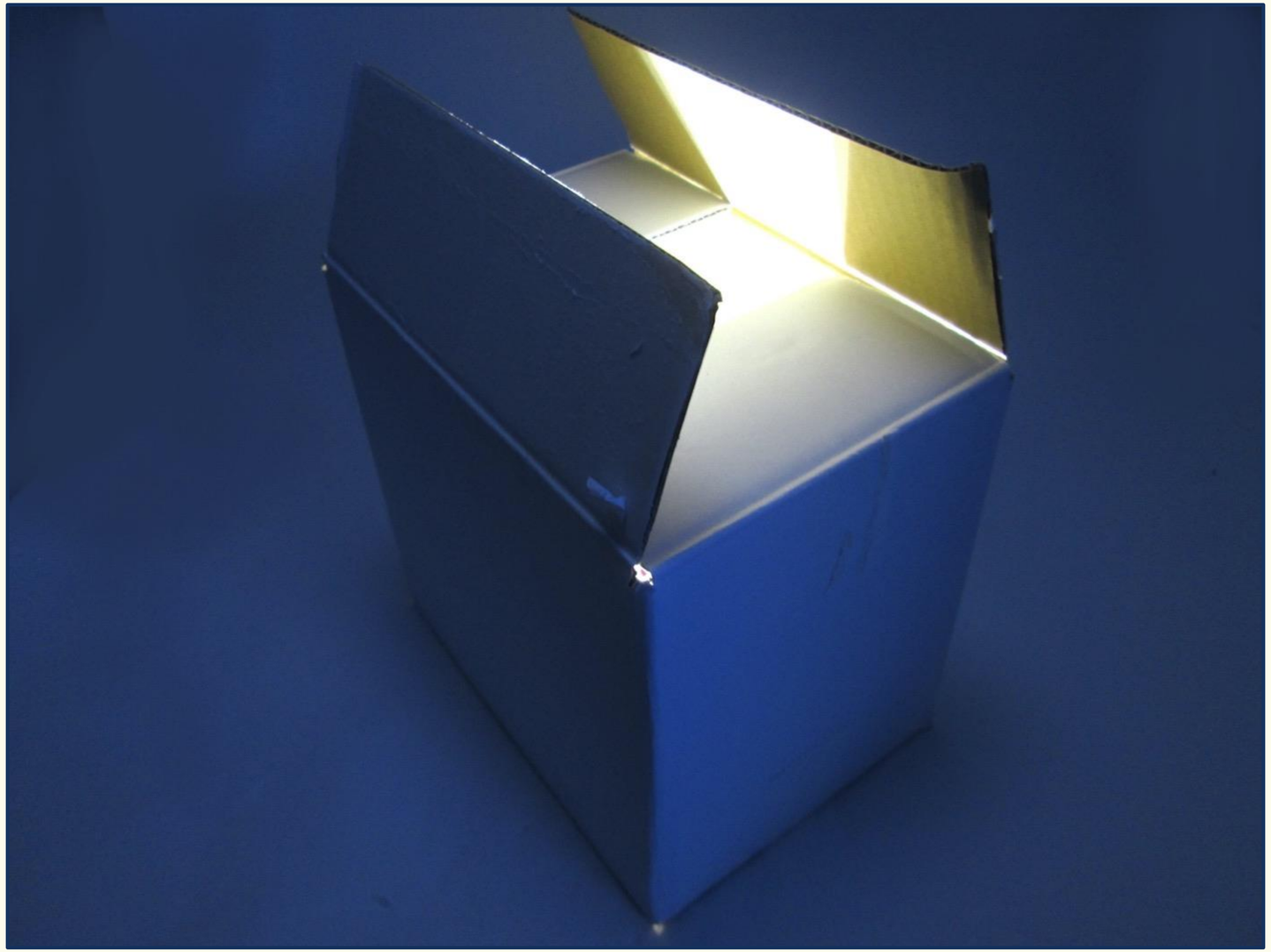
	CKD	FSGS
No risk Alleles 27.3%	Reference	Reference
Monoallelic APOL1 variants 43%	OR = 1.18 [1.04 to 1.33]	OR = 1.61 [1.04 to 2.48]
Biallelic APOL1 variants 29.7%	OR = 1.25 [1.11 to 1.40]	OR = 1.84 [1.30 to 2.61]

Conclusion: Monoallelic APOL1 variants were associated with 18% higher odds of CKD and 61% higher odds of focal segmental glomerulosclerosis; biallelic APOL1 variants were associated with 25% higher odds of CKD and 84% higher odds of focal segmental glomerulosclerosis.

Reference: Rasheed A. Gbadegesin et al, *APOL1* Bi- and Monoallelic Variants and Chronic Kidney Disease in West Africans, NEJM, 2024

VA by @nephromythri

Genetic Counseling



Genetic Counseling

- **Purpose of Genetic Testing:** It helps diagnose genetic kidney disease, assess risks in kidney donors, inform at-risk family members, and guide personalized treatments (i.e. post hoc analysis of the **DUPLEX trial** showed more proteinuria reduction with **sparsentan** vs irbesartan in genetic FSGS).

- **Testing Modalities:** There are several, but curated gene panels/targeted next-generation sequencing (NGS) - which assesses for specific sets of genes- is our go-to tool in clinical practice! **Whole exome sequencing (WES)** analyzes the entire exome and is used in research or in patients from whom NGS was non-diagnostic.

- **Informed Consent Considerations:** Patients should understand potential test implications, such as:

Findings & Clinical Impact	They may or may not change clinical management Variants of Unknown Significance can create anxiety in the patient and family
Additional Tests	May be required based on results
Costs	Insurance coverage varies depending on the specific test and patient scenario.
Risk of Discrimination	GINA protects against genetic discrimination in health insurance and employment, **BUT it does not cover disability, life, or long-term care insurance, nor does it apply to military personnel!**

- **Kidney Donation and Genetic Testing:** Genetic screening can be considered in living kidney donors- testing in ADPKD can be high yield while identifying APOL1 variants remains a complex issue.

- **Genetic Counselors:** They assist in interpreting results, managing emotional and social impacts, and guiding cascade testing for at-risk family members.

Genetic Testing in Adults with CKD: Diagnostic Yield & Clinical Utility

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Methods

60 cohort studies
10,107 CKD patients

Data Sources: PubMed & Embase

Diagnostic Yield

Overall yield: 40%
(95% CI: 33-46%)

Highest yield: Cystic kidney disease (62%)

Enhanced Yield

Positive family history

Associated extrarenal features

Impact on Diagnosis

Genetic testing reclassified 17% of diagnoses

Clinical Implications

Clinical benefits reported in six studies

Facilitated cascade testing & treatment modifications

Conclusion: Genetic testing is informative in a high proportion of clinically selected adults with CKD particularly those with family history and extra renal manifestations. Limitations include heterogeneity in reporting, testing technologies, and cohort characteristics.

Reference: Schottet al, Utility of Genetic Testing in Adults with CKD : A Systematic Review and Meta-Analysis, Clinical Journal of the American Society of Nephrology, 2025

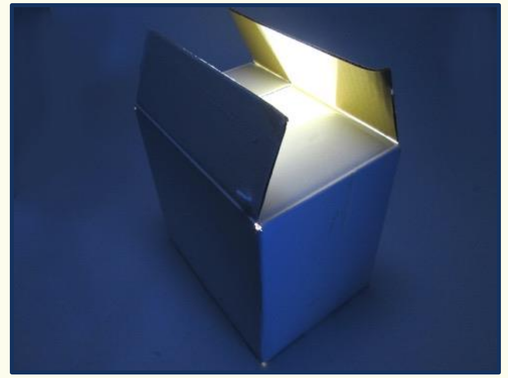
VA by @DrPSVali

EFFLUENT EIGHT ROUND

Pick Your Champion for the Genetics Region

Genetics in FSGS

VS



Genetic Counseling

NEPH MADNESS 2025

CAR-T for Kidney Disease

Writer:
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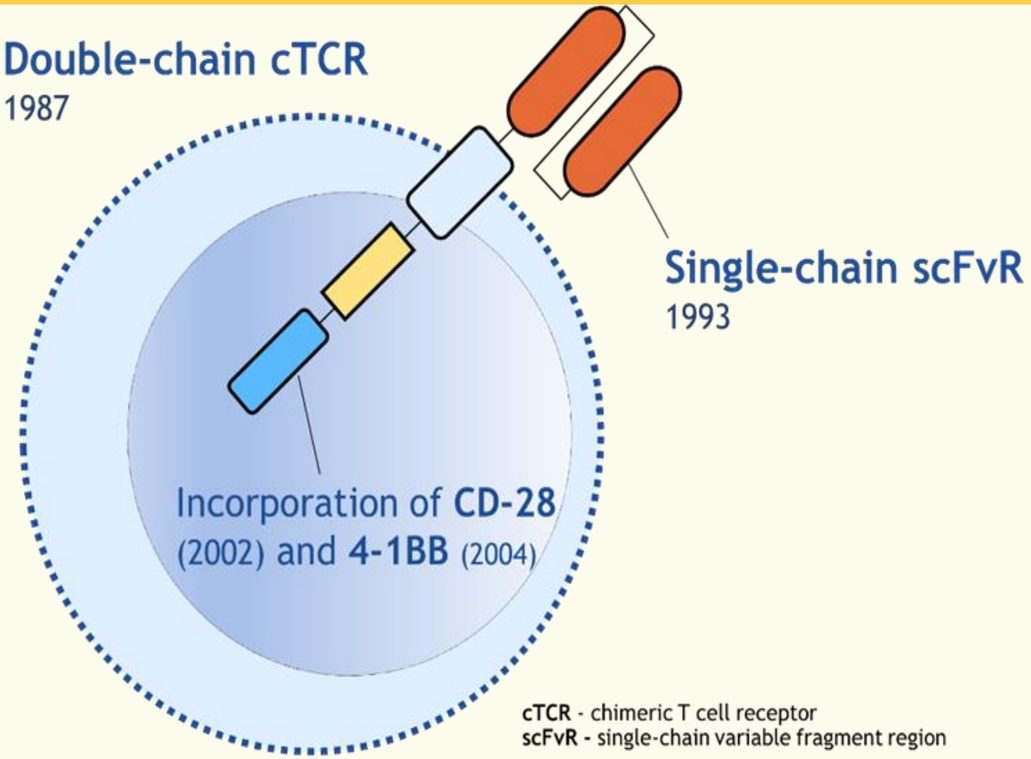
Expert:
Jeffrey Sparks

Region Execs:
Dia Waguespack
Anna Burgner

NEPH MADNESS 2025

CAR-T for Autoimmune Disease

From Idea to Impact: The CAR-T Cell Journey



cTCR - chimeric T cell receptor
scFvR - single-chain variable fragment region

2002 — Lymphodepletion prior to adsorptive cell transfer

2006 — First Trials of CAR T Therapy in patients (1st Gen CAR)

2011 — Leukemia patient achieved complete remission with CAR T Therapy (2nd Gen CAR)

2017 — Kymriah approved by FDA for leukemia treatment

2021 — Abecma approved by FDA for multiple myeloma; CAR T cell for autoimmune disease - SLE

Conclusion: CAR T cell therapy has shown remarkable progress in cancer treatment, but challenges like solid tumor targeting, cost, safety, and accessibility remain. Continued research and innovation are essential to expand its effectiveness and availability for cancer and other diseases.

Reference: Aroshi Mitra et al, From bench to bedside: the history and progress of CAR T cell therapy, Frontiers in Immunology, 2023

VA by @rnzp

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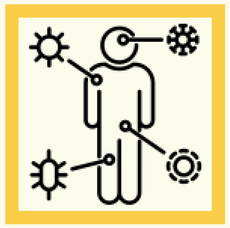

CAR-T Cell Therapy for Autoimmune Diseases

Why?

- Autoantibodies are directly pathogenic in select autoimmune diseases.
- Targeting B-cells has proven effective.
  - However, persistent B-cell infiltrates in tissue.
- Based on success in cancer therapies, use expanded to autoimmune disease.
- Murine model of lupus:
  - Effective depletion of CD19 B-cells in inflamed tissue → decreased Ab targeting dsDNA, decreased proteinuria, and increased survival.

How?

- T-cells are collected from patients through apheresis.
- CD19 CAR T-cells are generated through lentiviral transduction of the CAR encoding vector.
- Cells expanded using cytokine cocktail.
- Patient receive lymphodepleting chemotherapy:
  - Cyclophosphamide and fludarabine
- More targeted strategies (e.g. Treg in development).

NEJM 2024: Case series, 15 patients with refractory autoimmune dz (8 with SLE). Followed ~15 months. Demonstrated rapid expansion of CAR T cells and effective B cell depletion, most with B cell reconstitution at 100 days. Remained in remission off immunosuppression

*Limitations: open label, single arm small number of patients, no kidney biopsies

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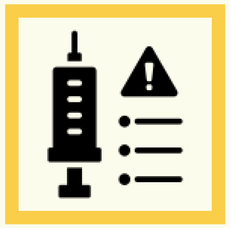
CAR-T Side Effects



CAR-T Cell Therapy - Side Effects and Potential Limitations

Short Term Side Effects

- Cytokine release syndrome (CRS):
  - Result of T-cell activation and cytokine release
  - Severity of reaction varies.
    - Symptoms: fever, respiratory failure, hypotension, possible progression to multiorgan failure, death
- Immune cell associated neurotoxicity syndrome (ICANS):
  - Mechanism not understood.
  - Severity of reaction varies.
    - Symptoms: confusion, aphasia, seizure, coma
  - Supportive treatment with steroids

Long Term Side Effects

- Cytopenias
- Hypogammaglobulinemia
- Infections
  - Potentially impaired response to vaccinations
- Malignancies
  - Due to insertional mutagenesis
  - Risk of T-cell malignancies appears to be lower than those undergoing traditional chemotherapy.
  - Require lifelong monitoring.

The small number of patients treated with CAR T-Cells for autoimmune dz, severe toxicities have thus far been rare.

Accessibility is limited by cost, limited treatment centers, eligibility restrictions and logistical challenges.

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Kidney Related Considerations

AKI and Electrolyte Abnormalities After CAR-T Therapy

Setting & Participants	Findings	
Case Series (2017-2019) 78 hospitalized patients in 2 cancer centers Diffuse large B-cell lymphoma Chimeric antigen receptor T-cell therapy	Acute kidney injury	19%
	Cytokine release syndrome	85%
	↓Na (<135 mEq/L)	75%
	↓K (<3.5 mEq/L)	56%
	↓PO_4 (<2.5 mg/dL)	51%

CONCLUSION: Cytokine release syndrome, AKI, hyponatremia, hypokalemia, and hypophosphatemia are common after CAR-T therapy

Shruti Gupta, Harish Seethapathy, Ian Strohbehn, et al (2020)
@AJKDonline | DOI: 10.1053/j.ajkd.2019.10.011
AJKD

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EFFLUENT EIGHT ROUND

Pick Your Champion for the CAR-T for Kidney Dz Region

CAR-T for Autoimmune Dz

VS

CAR-T Side Effects

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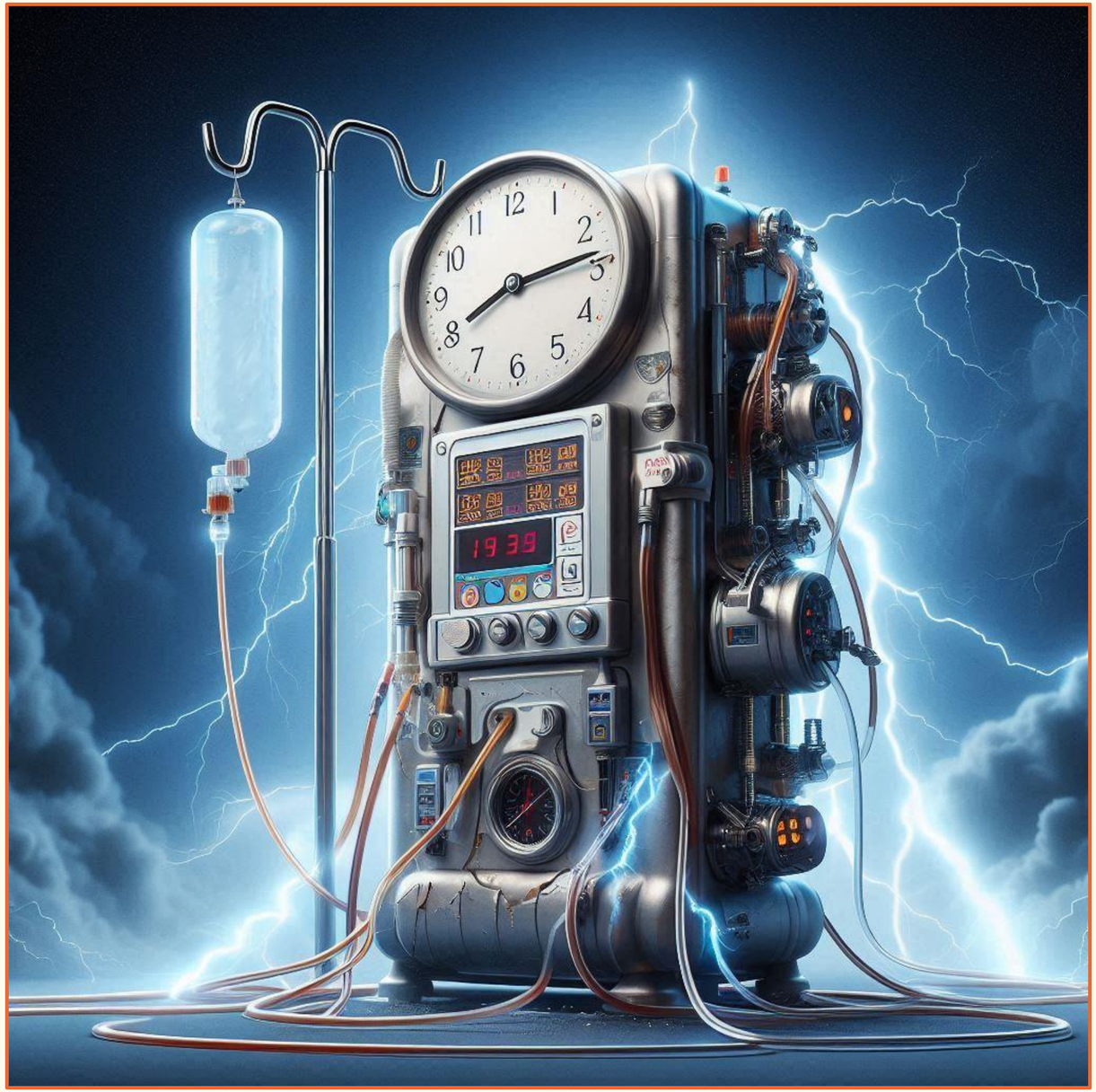

Hemodialysis

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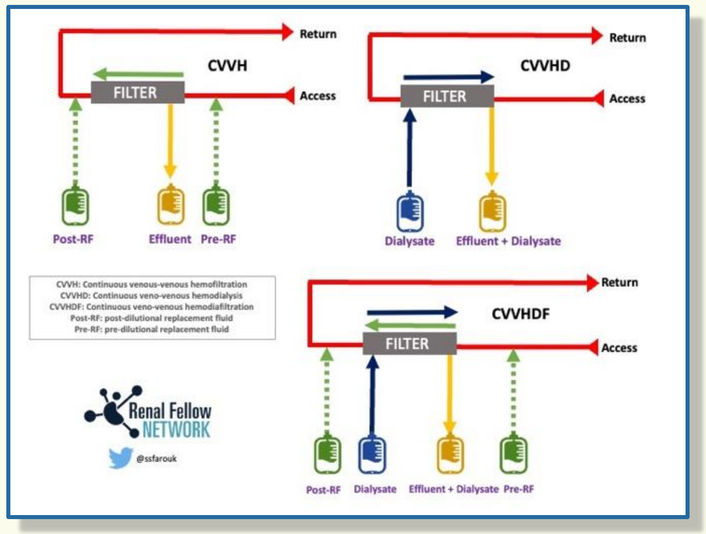
Hemodiafiltration

Hemodiafiltration

- Hemodiafiltration (HDF) combines diffusive with convective clearance to improve clearance of larger molecular weight molecules including certain proteins/uremic toxins (i.e. FGF-23, Advanced Glycation Endproducts) that contribute to increased cardiovascular (CV) mortality and impaired immune function in patients undergoing chronic dialysis.



- HDF not only uses dialysate but replacement fluid which is infused into the patient to replace the large volume of plasma water removed through ultrafiltration.

- 5 Randomized Controlled Trials have been been completed with varying results with respect to all-cause, cardiovascular, or infection related mortality
- Peters et. al., a meta-analysis of 4 RCTs (not including the Convince Trial) demonstrated a 14% reduction in all-cause mortality and a 23% reduction in CV mortality compared to HD, especially those who received higher convections volumes
- Long term follow up of the patients from 4 RCTs demonstrated stable Left Ventricular Mass and Ejection Fraction in the groups that received HDF compared to Hemodialysis
- There may be some evidence that HDF is cost effective, however the Quality-Adjusted Life Year (QALY), a measurement of patient's quality and quantity of life is limited, and further research is needed.
- In addition to cost effectiveness, there are several questions that remain to be answered regarding HDF
  - While preliminary data has demonstrated higher convection volumes may be beneficial, no direct head-to-head trial has compared the two approaches.
  - No studies have focused on individuals that have not yet started Kidney Replacement Therapy. The ideal convective volume remains unclear in this population.

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CONVINCE trial: Can hemodiafiltration vs hemodialysis reduce mortality in kidney failure?

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Pragmatic, multinational, randomized controlled trial

1360 patients on high flux hemodialysis

Median follow-up:30 months

~62 years old

~81% arterio-venous fistulas

~35% diabetes

~44% cardiovascular disease

Median convection volume: 25.3L/ session in hemodiafiltration group

	Primary outcome	Secondary outcomes		
	Death from any cause	Cardiovascular death	Non-cardiovascular death	Infection-related death Including COVID-19
High flux hemodialysis N=677	21.9%	5.5%	16.4%	8%
	0.77 0.65-0.93	0.81 0.49-1.33	0.76 0.59-0.98	0.69 0.49-0.96
High dose hemodiafiltration N=683	17.3%	4.5%	12.7%	5.6%

Hazard Ratios, 95% Confidence Intervals

Conclusion: In patients with kidney failure resulting in kidney-replacement therapy, the use of high-dose hemodiafiltration resulted in a lower risk of death from any cause than conventional high-flux hemodialysis.

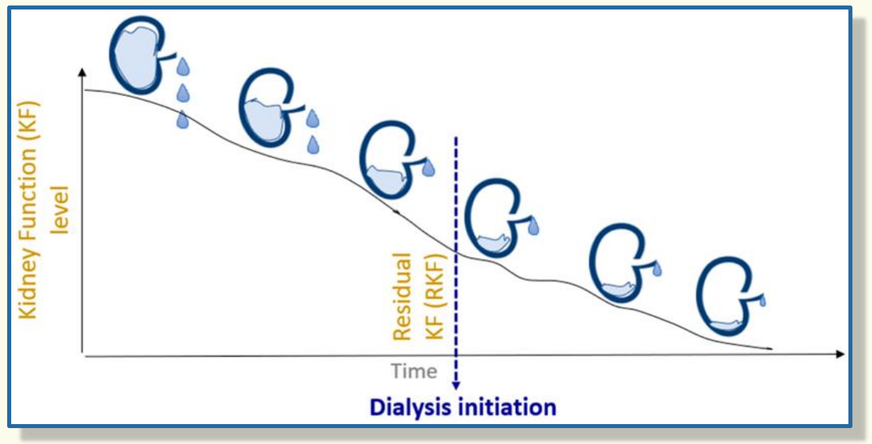
Reference: Blankestinjn PJ et al, Effect of Hemodiafiltration or Hemodialysis on Mortality in Kidney Failure, NEJM, 2023

VA by @nephroseeker.medsky.social

Incremental Dialysis

Incremental Dialysis

- Incremental Hemodialysis (HD) involves adjusting the frequency or duration of a patient's hemodialysis prescription based on their biology and symptoms and is a patient centered approach to HD compared to the traditional 3x weekly HD.
- That is in contrast with Peritoneal dialysis, where residual kidney function (RKF) is factored into a patients "dose" (Kt/V), HD prescriptions usually target a Kt/V of 1.2, not taking RKF into account.



- The decline in RKF differs based on the individual, and incremental dialysis would take into account the need for a higher dose of dialysis.

What challenges exist? And how can we overcome them?

- Uncertainty regarding patient adherence and expectations related to incremental changes in HD prescriptions.
  - Repeated Patient Education
- Increased workload for nephrologists and dialysis staff, who must monitor patient parameters more frequently and closely.
  - Automating RKF measurement and substitution of additional care typically dedicated to HD to monitoring of RKF
- Potential for reduced financial margins for dialysis stakeholders due to fewer overall HD treatments and lower financial reimbursements.
  - Potentially longer patient lifespans could ultimately lead to more dialysis treatments.

Feasibility Trials have demonstrated incremental dialysis is both feasible and safe, however larger scale, randomized trials are needed

Twice-Weekly Hemodialysis With Adjuvant Pharmacotherapy and Transition to Thrice-Weekly Hemodialysis: A Pilot Study

Setting & Participants

Randomized Controlled Trial

14 dialysis facilities in North Carolina, USA

New start on chronic HD

- eGFR ≥5 mL/min/1.73 m^2
- Urine output ≥500 mL/24 h

Intervention & Control

Incremental HD
(N= 23)
2 HD/week
+
Adjuvant pharmacotherapy
for 6 weeks, then
3 HD/week

Conventional HD
(N= 25)
3 HD/week

Mean follow-up 281.9 days

Adjuvant pharmacotherapy:
Loop diuretics, patiromer, and/or sodium bicarbonate

Results

Primary Outcome: Feasibility	
66% consent rate	
96% adhered to assigned HD protocol	
100% adhered to serial timed urine collection	
0% cross over from 3 HD/week to 2 HD/week	
9% cross over from 2 HD/week to 3 HD/week	
Secondary outcomes, mean (95% CI) *Incremental HD vs Conventional HD*	
Urine output†*	51.0 percentage points lower decline (-0.7, 102.8)
Averaged urea and creatinine clearance‡*	57.9 percentage points lower decline (-22.6, 138.4)

*Percent change, baseline to week 24; †mL/24 h; ‡mL/min/1.73 m^2

CONCLUSION: Implementation of core components of incremental HD is feasible. Larger clinical trials are indicated to determine the efficacy and safety of incremental HD.

Mariana Murea, Ashish Patel, Benjamin R. Highland , et al

@AJKDonline | DOI: 10.1053/j.ajkd.2021.12.001

AJKD

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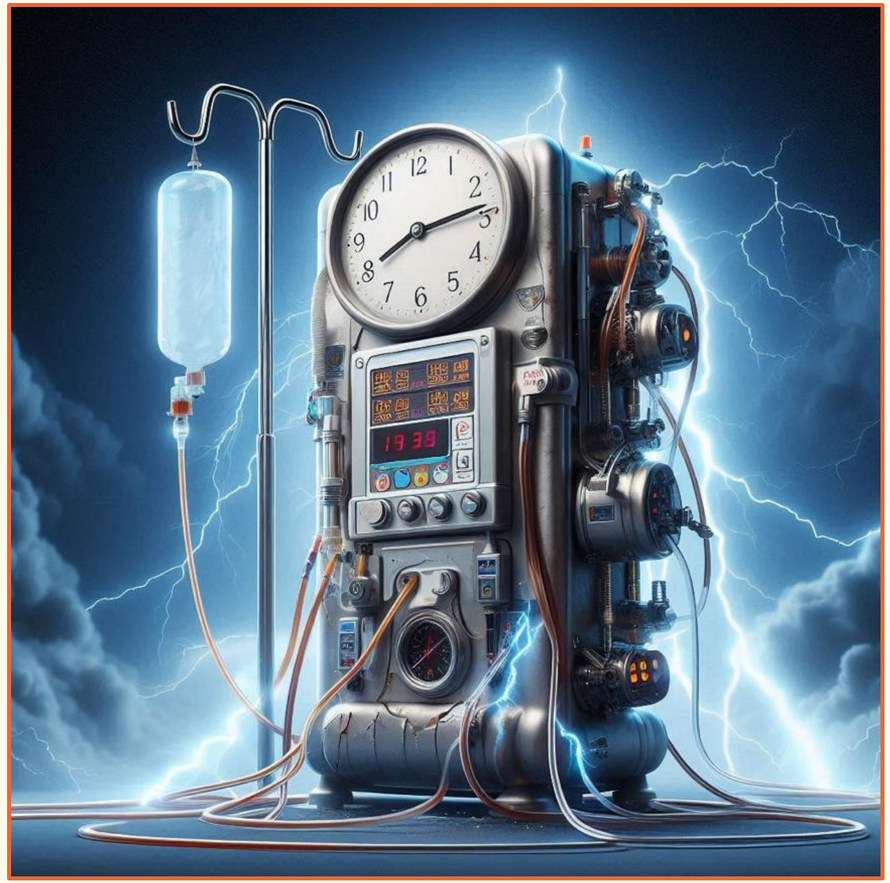
EFFLUENT EIGHT ROUND

Pick Your Champion for the Hemodialysis Region



Hemodiafiltration

VS

Incremental Dialysis

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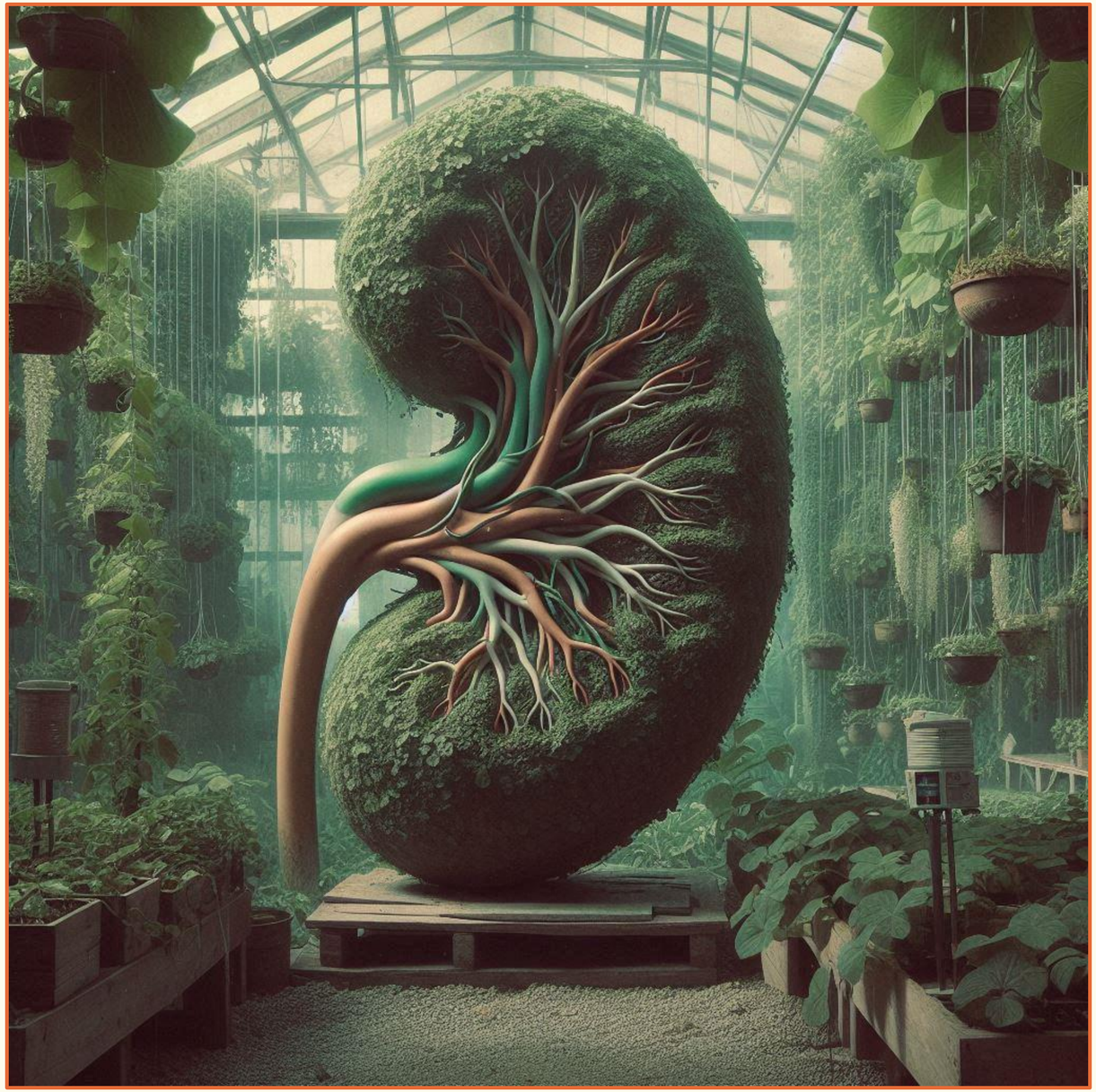

Green House

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NEPH MADNESS 2025

Tubular Toxins

Tubular Toxins

Why?

- 80% of patients in the world use herbal medicines (HMs).
- 8% of patients with CKD use HMs on NKF “avoid in CKD” list.
- HMs are not regulated or assessed for safety and efficacy.
- FDA can remove supplements only if there’s proof of harm/deceptive labeling.

How?

HMs can hurt kidneys:

- Nephrotoxicity
- Contamination
- Adulteration
- Misidentification
- Drug interactions (i.e. with immunosuppressants)

Kidneys are ‘terrific’ toxin targets

- Major site of drug excretion
- Large volume of blood processed
- Renal tubular epithelium has broad surface area and high metabolic activity relative to blood supply

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Nephrotoxicity of Herbs and Alternative Medicinal Products

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NORTH AMERICA

Alfalfa (*Medicago sativa*)
Black cohosh (*Actaea racemosa*)
Cone flower (*Echinacea*)
CKLS (colon, kidney, liver, spleen purifier
contains *Aloe vera, Cascara sagrada, Larrea tridentata* and *Arctostaphylos uva-ursi*)
Creatine
Hemlock (*Conium maculatum*)
Ma huang (*Ephedra sinica*)
Hydrazine sulfate
Noni juice (*Morinda citrifolia*)
St. John's wort (*Hypericum perforatum*)
Wormswood oil (*Artemisia absinthium*)
L-lysine
Chaparral (*Larrea tridentata*)

St. John's wort
Transplant rejection

Cranberry juice
Kidney stones

Hemlock
Acute tubular necrosis, urine bladder constriction

EUROPE

Hemlock (*Conium maculatum*)
Noni juice (*Morinda citrifolia*)
Senna fruit tea (*Sennae fructus angustifoliae*)
Germanium
Hydrazine sulfate
Willow bark (*Salix daphnoides*)
Anatloian hawthorn (*Crataegus orientalis*)

Noni juice
Hyperkalemia

Turmeric
Kidney stones

ASIA

Cyprinidae (grass carp, common carp, silver carp, black shark fish, bony-lipped barb fish)
Indian carp (*Labeo rohita*)
Mourning cypress (*Cupressus funebris*)
Snake gallbladder (*Naja naja atra*)
Star fruit (*Averrhoa carambola*)
Oduvan (*Cleisanthus collinus*)
Yellow oleander (*Thevetia peruviana*)
Djenkol beans, jering (*Pithecolobium lobatum*)

Ma huang
Hypertension

Tripterygium
Chimonanthus
Tetrandra
Menispermi
Strychnos
Wood veratry

Aconitum
Groundel
Monkhood
Bee pollen
Fish gallbladder

Aristolochia
Urinary tract carcinoma
Fanconi syndrome

Licorice
Pseudoaldosteronism
Rhabdomyolysis

Violet Tree
Acute tubular necrosis

SOUTH AMERICA

Propolis
Star fruit (*Averrhoa carambola*)
Cat's claw (*Uncaria tomentosa*)

Cat's claw
Acute interstitial nephritis

OCEANIA

Cone flower
Renal tubular acidosis
Hypokalemia

AFRICA

Wild wisteria, violet tree (*Securidaca longipedunculata*)
Paraphenylene diamine (PPD)
Takaout roumia
Sheep bile
Bird flower (*Crotalaria laburnifolia*)
Spurge (*Euphorbia matabelensis*)
Khat leaf (*Catha edulis*)
Cape aloe (*Aloe capensis*)
Impila, ox-eye daisy (*Callilepis laureola*)
Potassium dichromate

Reference

KDIGO 2024 Clinical Practice Guidelines
Bagnis et al. **Herbs and the Kidney.** AJKD. 2004

Visual Abstract by @hellokidneyMD

Famous Tubular Toxins

Aristolochia

- Originally used in slimming regimens
- Currently banned
- Causes aristolochic acid nephropathy (AAN)
  - proximal tubular dysfunction
  - rapidly progressive CKD-> ESKD
  - severe anemia
  - shrunken kidneys with fibrosis on biopsy
- Urothelial carcinoma

Licorice

- Widely used in sweets
- Alternative medicinal use as expectorant, gastroprotective and hepatoprotective agent
- Causes hypertension and hypokalemia
  - glycyrrhyzic acid blocks 11-β-HSD allowing cortisol to
  - activate MR receptors
- AKI sec to hypokalemic rhabdomyolysis or tubular toxicity

Tips To Tackle Herb Nephropathy

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- At every patient visit, complete a comprehensive medication review including use of herbal medicines
- Make it a judgment-free zone

- Use databases such as **NCCIH**[1] (free) & **NatMedPro**[2] (by subscription) to assess the known or potential nephrotoxicity of the herbal medicines
- Conduct a literature review (can use **Cochrane**[3])
- Complete a drug interaction check
- Weigh the potential for harm against the benefit of the herbal medicine

- Recommend that any herbal medicine is obtained from reputable regulated sources (for US products, look for the USP seal)
- Question possible contamination during procurement (if herbs obtained naturally)
- Question compounded products where ingredients cannot be verified

- Educate your patient on signs/symptoms of nephrotoxicity
- Refer patients to **NKF**[4] write-up about herbal medicines & kidney disease

VA by @krithicism

1 https://www.nccih.nih.gov/
2 https://naturalmedicines.therapeuticresearch.com/
3 https://cam.cochrane.org/cochrane-reviews-related-complementary-medicine
4 https://www.kidney.org/kidney-topics/herbal-supplements-and-kidney-disease

Oxalate Offenders

What is the Pathophysiology of Hyperoxaluria?

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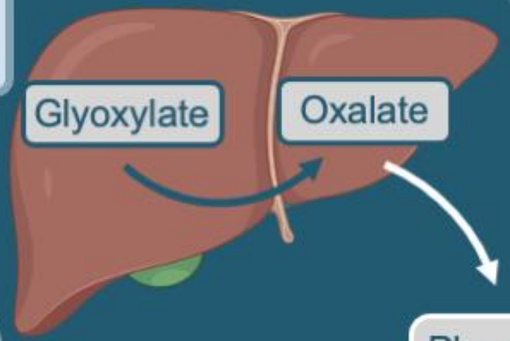
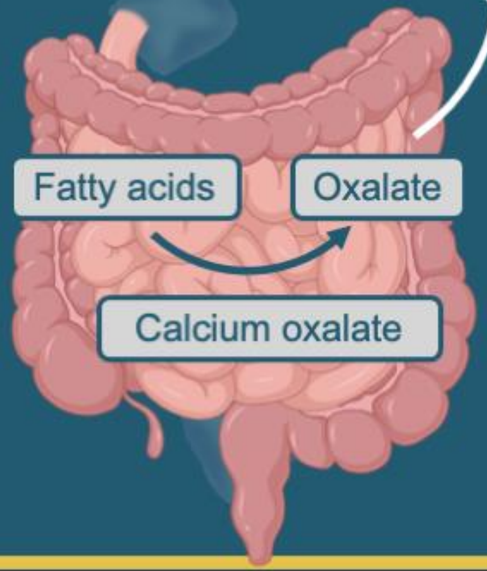
Oxalate is the metabolic end-product of glyoxylate in the liver



Only 5%-10% of ingested oxalate is absorbed normally, the remainder passes in the stool

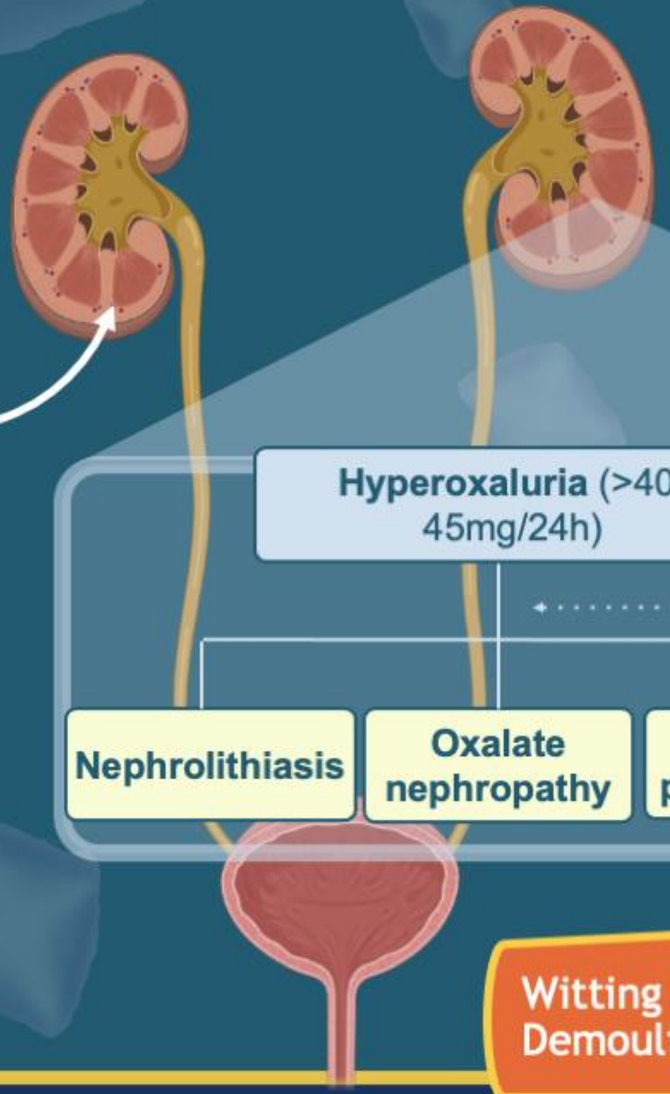
Enteric hyperoxaluria is from fat malabsorption where free fatty acids bind calcium in the large intestine, resulting in luminal unbound oxalate



Free, unbound oxalate results in ≥30% increase in intestinal reabsorption

Plasma oxalate

Oxalate is excreted by the kidneys through glomerular filtration & proximal tubular secretion



Hyperoxaluria (>40-45mg/24h)

Additional trigger: Hypovolemia, RAAS blockade, diuretic use, increased oxalate, inflammation

Nephrolithiasis

Oxalate nephropathy

CKD progression?

VA by @Sophia_kidney

Witting C et al, CJASN, 2020,
Demoulin N et al, AJKD, 2022

Oxalate Offenders

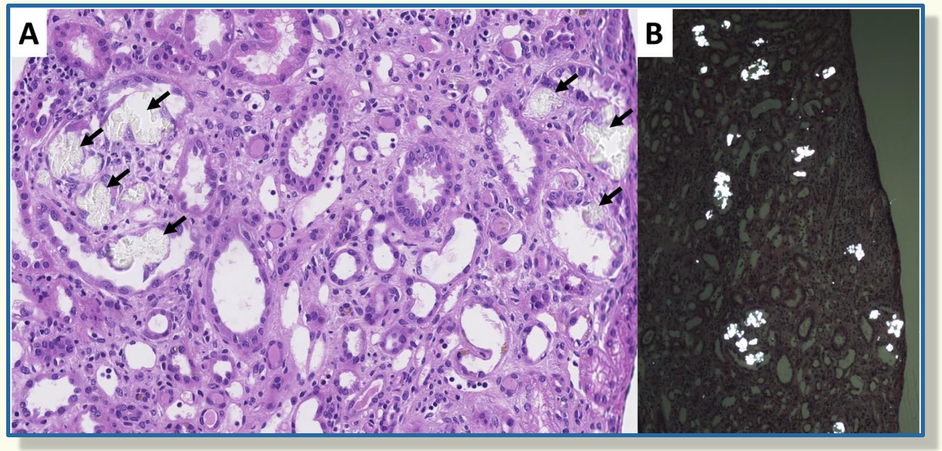
- Oxalate is an organic acid produced by plants for protection against
  - hard ions in groundwater
  - infections
  - herbivores

- Oxalate is an anti-nutrient (reduces absorption of Ca, Mg, Fe)
- Famous Oxalate Offenders: star fruit, cranberries, nuts + see the VA
- Oxalate Nephropathy:

ATN -> reabsorbed crystals initiate inflammation via NLRP3 inflammasome -> TIN -> interstitial fibrosis

A. Intratubular and interstitial deposition of translucent crystals (H&E)

B. Crystals are birefringent under polarized light



Oxalate Index: Foods that Tip the Scale

	Oxalate (mg/100g) mean	Oxalate/Ca (meq)
Group 1		
Rhubarb, stewed	260	9.3
Sorrel	500	5.6
Beetroot	275	5.1
Spinach	970	4.0
Coffee	100	3.7
Cashew	231	4.5
Cocoa	700	2.5
Group 2		
Potato	80	1.6
Tea	1150	1.1
Group 3		
Apple	15	0.7
Tomato	20	0.6
Parsley	170	0.3
Cabbage	60	0.1
Lettuce	12	0.1

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VA by @Sophia_kidney

Noonan SC et al, Oxalate content of foods and its effect on humans, Asia Pac J Clin Nutr, 1999

Oxalate Offenders

Management of dietary hyperoxaluria
(extrapolated from Primary Hyperoxaluria)

- Decrease enteric oxalate absorption
  - Low oxalate diet
  - Adequate dietary Ca
- Binders
  - Lanthanum
  - Magnesium
- Removal/excretion
  - Hemodialysis
  - High fluid intake
  - Citrate to alkalinize urine
- Metabolism
  - *Oxalobacter formigenes*
  - Oxalate decarboxylase

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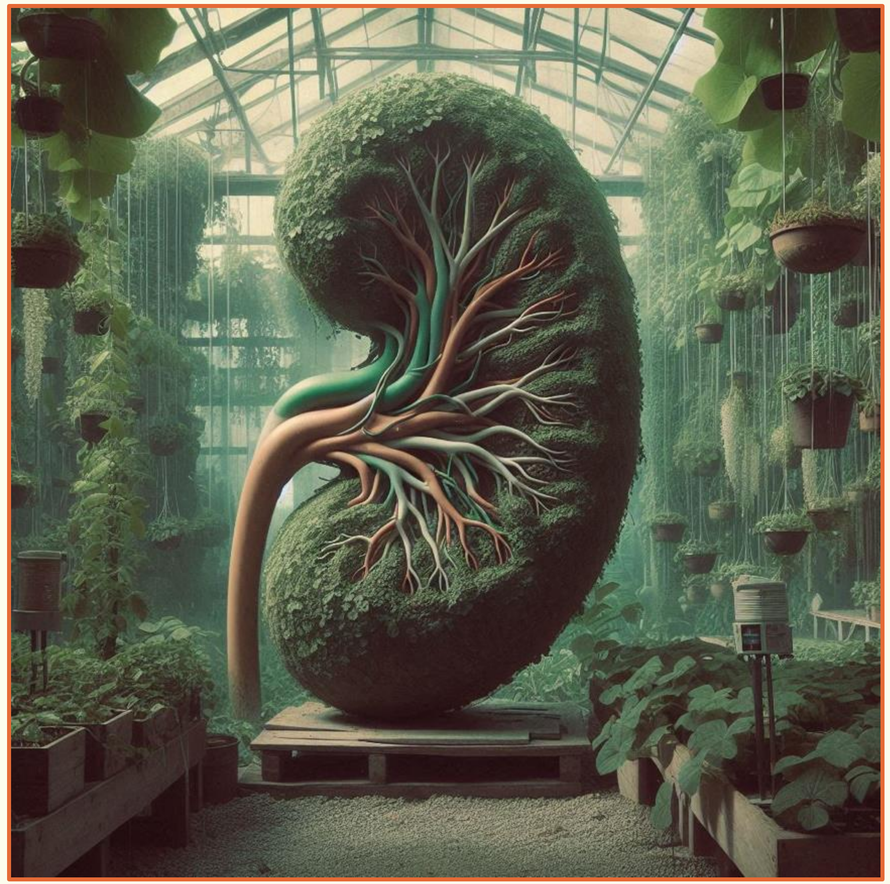
EFFLUENT EIGHT ROUND

Pick Your Champion for the Green House Region



Tubular Toxins

VS

Oxalate Offenders

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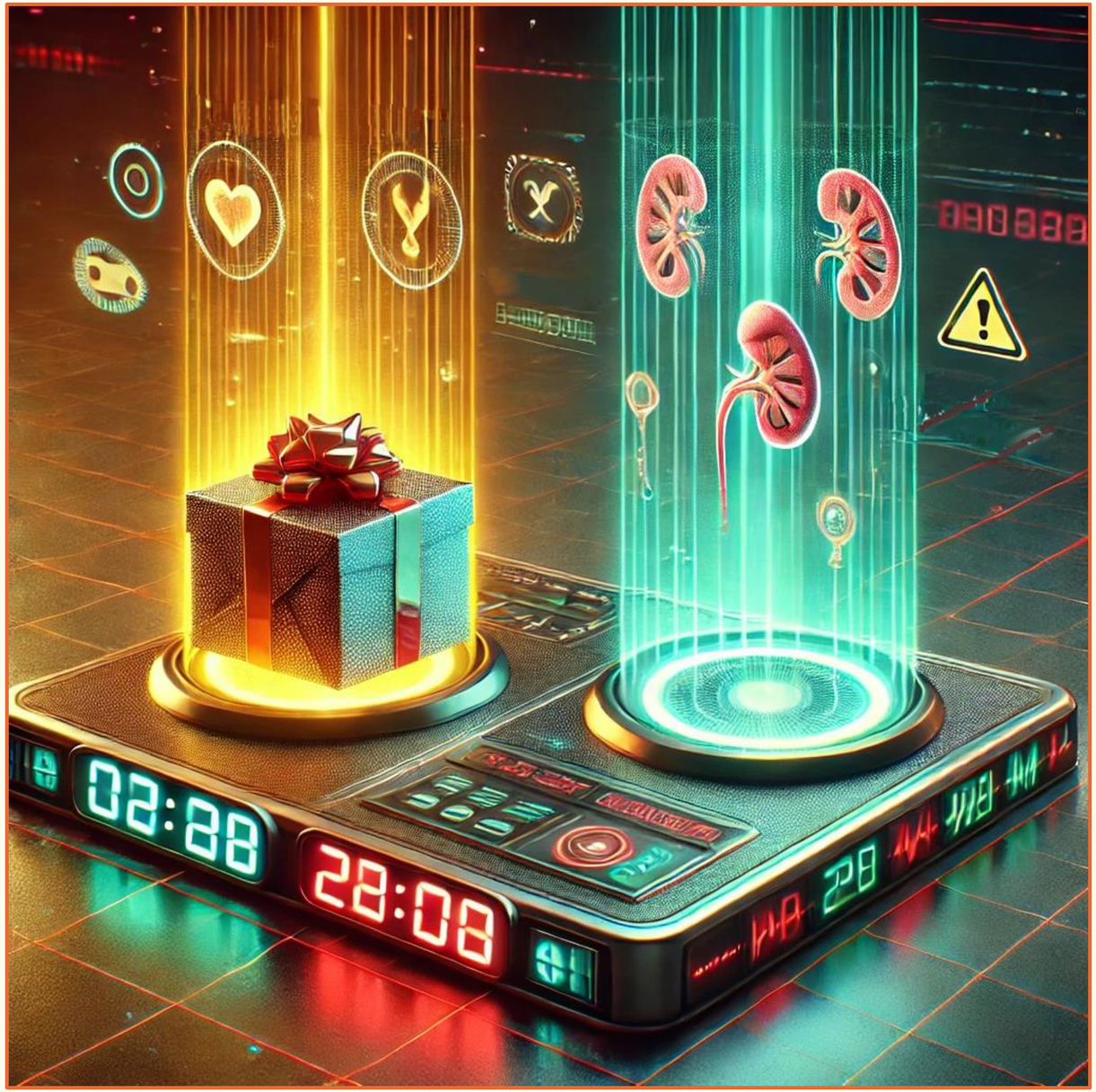

Obesity in Kidney Transplant

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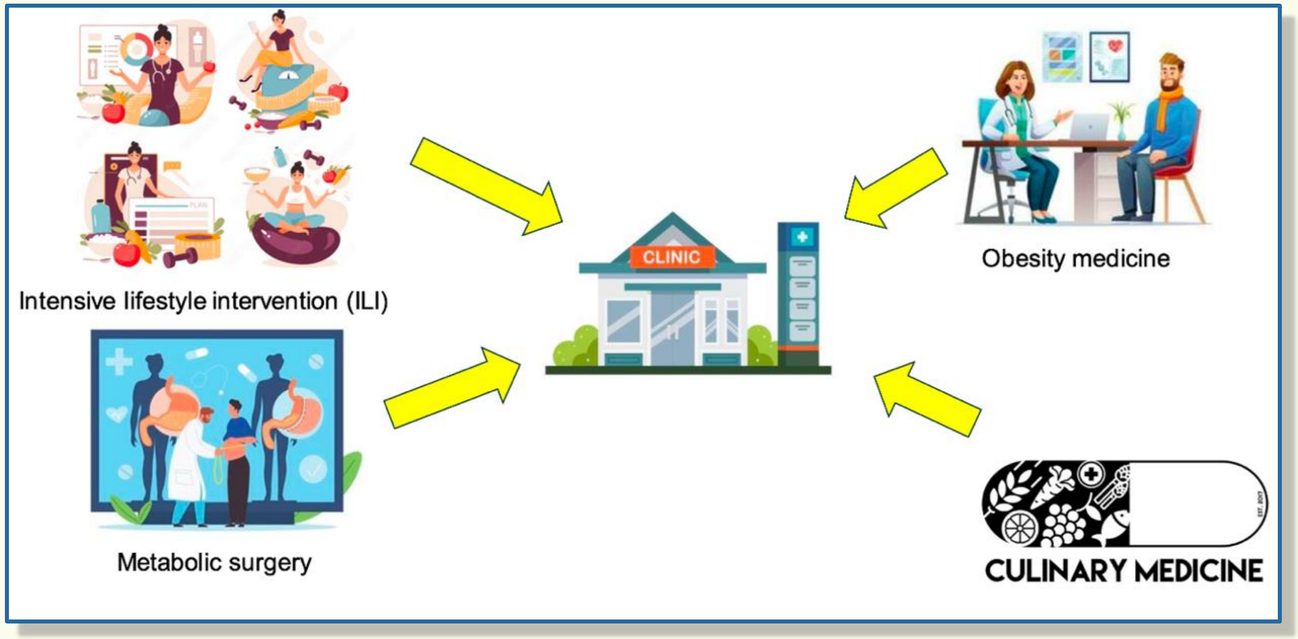
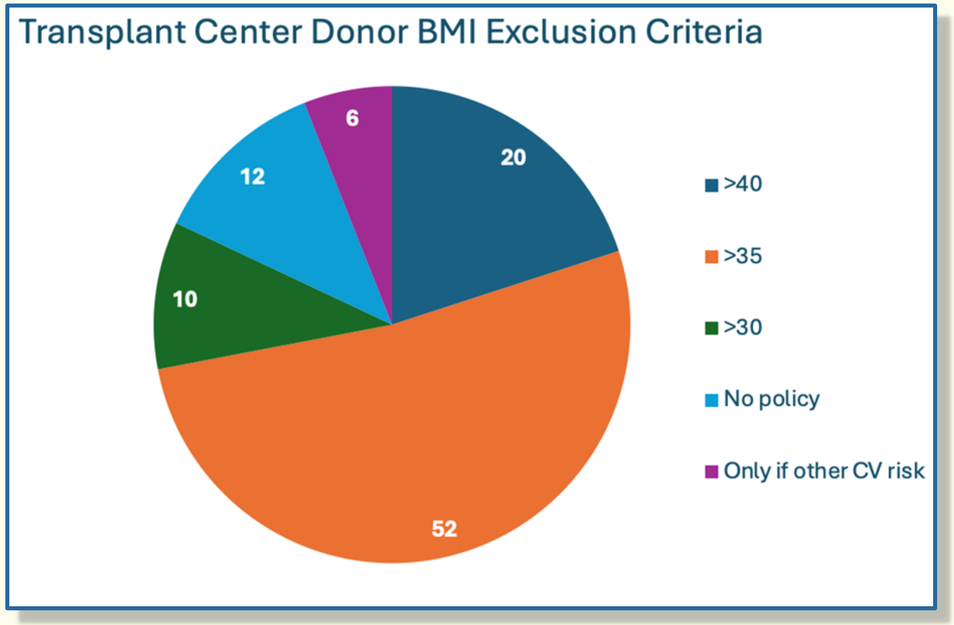
Obesity in Kidney Transplant Donors

Obesity in Kidney Transplant Donors

- >90,000 individuals on the kidney transplant waiting list in the U.S.
  - We need to find ways to expand donor pool!
- No standard BMI threshold for ability to donate
- About ⅔ of donors in the U.S. have a BMI > 30 kg/m^2
- Obesity in kidney transplant donors increases the risk of development of ESRD 1.9 fold above non-obese donors
- Treatment of obesity should be multi-modal
  - Past data shows potential donors rarely are able to lose the weight
  - Donors frequently (re)gain weight post donation
- Therapies to promote health are essential both pre and post donation!





What is the Impact of Obesity as a Barrier to Living Kidney Donation?

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RETROSPECTIVE

Single center

Potential Living Kidney Donors
n=104

2008-2012

18% BMI <25 NORMAL

37% BMI 25-29.9 OVERWEIGHT

23% BMI 30-34.9 CLASS I OBESITY

16% BMI 35-39.9 CLASS II OBESITY

6% BMI >40 CLASS III OBESITY

85%

OVERWEIGHT to MORBIDLY OBESE

22%

BMI >35 MODERATELY and MORBIDLY OBESE

Only 13% succeeded at losing weight and donating

Conclusion: Obesity may be a frequent barrier to living kidney donation, directly leading to exclusion as a potential kidney donor in about one in five instances. Successful weight loss leading to donation appears to be infrequent, suggesting need to address obesity in the donor population.

Reference: Sachdeva M, et al, Obesity as a barrier to living kidney donation: a center-based analysis, Clin Transplant, 2013

VA by @edgarvlermamd

Obesity in Kidney Transplant Recipients

Post-Transplant Obesity

- ~30% of patients are obese at the time of kidney transplant
- Weight gain is common post kidney transplant
- Obesity increases short and long term risks post kidney transplant
  - Delayed graft function
  - Poor wound healing
  - Cardiovascular disease
  - Posttransplant diabetes mellitus
- Fear of movement and low self-efficacy are common in transplant recipients
- There is limited data of pharmaceutical agents in this population
  - Limited data of GLP-1s, DPP4 inhibitors, and metformin suggest safety in carefully selected patients
- Bariatric surgery has been shown to be effective in kidney transplant recipients.
  - Higher doses of tacrolimus needed due to decreased absorption
- Even with increased risks of kidney transplant in obese patients, obese kidney transplant patients still perform better than obese ESKD patients who remain on the waiting list

Category	N	Death rate/100 patient years at risk
Obese		
Waiting list	7443	6.6
Deceased donor	1719	3.3
Living donor	552	1.9
Non-obese		
Waiting list	23,219	6.3
Deceased donor	4795	2.8
Living donor	1528	1.3

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Does extreme obesity impact kidney transplant success?

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Nationwide Cohort (2001-2016, OPTN/UNOS)

44,560 first-time deceased-donor kidney recipients

Mate-Kidney Model

Followup: 3.9 yrs

BMI Categories

- 18-25 kg/m² (normal)
- >25-30 kg/m² (overweight)
- >30-35 kg/m² (obese)
- >35 kg/m² (extreme obesity)

Delayed Graft Function

Risk decreases as BMI decreases

BMI 18-25 kg/m² → 58% lower (OR 0.42)
BMI >25-30 kg/m² → 45% lower (OR 0.55)
BMI >30-35 kg/m² → 27% lower (OR 0.73)

Death-censored graft failure

Lower risk in Lower BMI

BMI 18-25 kg/m² → 34% lower risk (HR 0.66)
BMI >25-30 kg/m² → 21% lower risk (HR 0.79)
BMI >30-35 kg/m² → ✕ No significant difference (HR 0.91)

Hospital stay & patient survival

No significant BMI-related differences

Conclusion: Despite an increased risk of DGF likely unrelated to donor organ quality, long-term transplant outcomes among recipients with a BMI >35 kg/m2 are similar to those among recipients with a BMI >30-35 kg/m2, supporting a flexible approach to kidney transplantation candidacy in candidates with extreme obesity

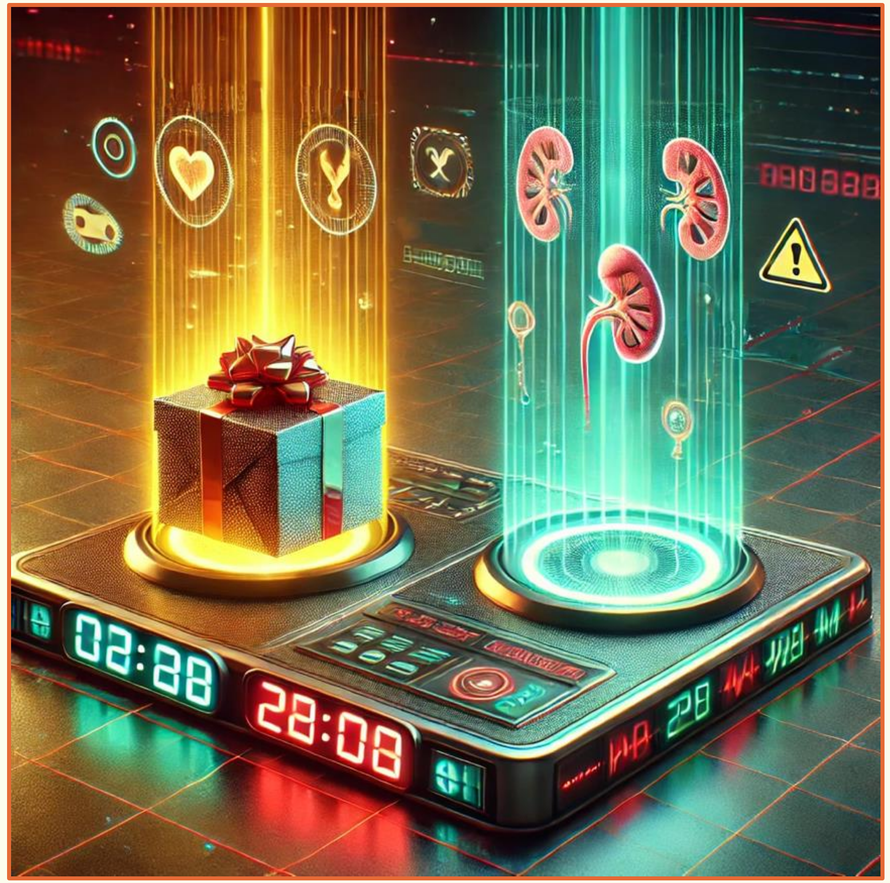
Reference:

Kalathil K Sureshkumar et al, Recipient Obesity and Kidney Transplant Outcomes: A Mate-Kidney Analysis, Am J Kidney Dis, 2021

VA by Dr PS Vali MD DM 𝕏 @DrPSVali

EFFLUENT EIGHT ROUND

Pick Your Champion for the Obesity in Kidney Transplant Region



Obesity in Donors

VS

Obesity in Recipients

NEPH MADNESS 2025

Minimal Change Disease

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Expert:
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Region Execs:
Ana Catalina Alvarez-Elías
Matthew Sparks

NEPH MADNESS 2025

Minimal Change Disease Diagnosis & Pathogenesis

Minimal Change Disease Pathophysiology

Minimal Change Disease (MCD) is a histopathological diagnosis characterized by foot effacement of the podocytes which are part of the glomerular filtration barrier. The clinical presentation in adult and pediatric population is massive proteinuria, hypoalbuminemia and edema (nephrotic syndrome)

ADULTS → Diagnosis through a kidney biopsy

Children
1-12 years
(could be older) → Diagnosis through clinical response to corticosteroid therapy. Traditionally assumed in Steroid Sensitive Nephrotic Syndrome (SSNS)

Physiopathology remains UNKNOWN!

PREVIOUS THEORIES

- T cell mediated
- Permeable circulating factor
- Genetics

NEW THEORIES

- ✓B cells mediated
- ✓Anti-Nephrin antibodies
- ✓Atypical B cells
- ✓Genetics

Childhood nephrotic syndrome (NS) according to steroid response (KDIGO Guidelines)

- Steroid sensitive NS (SSNS): Complete remission after 4 weeks of treatment with recommended dose of steroids.
- Frequent relapsing NS (FRNS): More than 2 relapses within 6 months or more than 4 relapses within 12 months
- Steroid dependent NS (SDNS): Two relapses while in steroid therapy or within 14 days of discontinuing steroid therapy
- Steroid resistant NS (SRNS): Fail to achieve remission after 4 weeks of recommended steroid treatment

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What are pathophysiological mechanisms involved in childhood idiopathic nephrotic syndrome?

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Triggers

- Infections
- Allergies
- Gut dysbiosis (such as less abundant butyric acid-producing bacteria)

B cells

Autoantibody:
- antinephrin
- anti-UCHL1
- anti-CD40

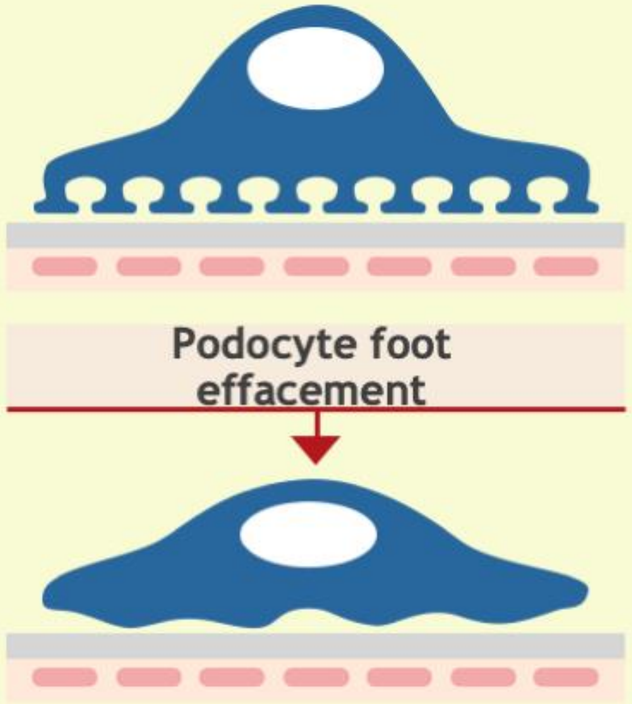
Crosstalk

T cells

- ↑ T-helper-17 activity
- ↓ T regulatory cell response

Permeability factors

Hemopexin, CLCF-1, suPAR



Genetic mutations

NPHS1
NPHS2
WT1
LAMB2
PLCE1
INF2
TRPC6
COQ10

Conclusion: The pathophysiology of childhood idiopathic nephrotic syndrome is not clearly understood. Several immune disorders have been linked with increased permeability of the filtation barrier. Steroid-resistant variants are more frequently caused by mutations involving podocyte-related genes.

Reference: Marina Vivarelli et al. *Childhood nephrotic syndrome*. Lancet, 2023.

VA by @CTeodosiu

Minimal Change Disease Relapse

Minimal Change Disease Relapse

Initial treatment

Prednisone or prednisolone:
- ✓ 60 mg/m^2/day or 2 mg/kg/day for 4-6 weeks (with a maximum dose of 60 mg/day) and
- ✓ 40 mg/m^2/ every other day or 1.5 mg/kg/ every other day for 4-6 weeks, for a total duration of 8-12 weeks for initial treatment [*KDIGO Guidelines*]

Relapse treatment

Prednisone or prednisolone:
- ✓ 60 mg/m^2/day or 2 mg/kg/day (maximum 60 mg/day) until the child remits completely for more than ≥3 days.
- ✓ After achieving complete remission, reduce dose to 40 mg/m^2 or 1.5 mg/kg (maximum 50 mg) on alternate days for ≥ 4 weeks. [*KDIGO Guidelines]*

The steroid-sparing drug of use depends on center practices, patient's preference, availability and cost. No strong evidence about a specific drug being more effective.

Steroid-sparing drugs for childhood nephrotic syndrome

- **Calcineurin Inhibitors:** Tacrolimus or cyclosporine
- **Antiproliferative:** Mycophenolate Mofetil or Mycophenolic Acid
- **Alkylating agents:** Cyclophosphamide
- **Antiparasitic:** Levamisole (not available in USA or Canada)
- **Anti-CD20:** Rituximab, Ofatumumab and Obinutuzumab

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Prevalence of minimal change disease in patients with nephrotic syndrome by age

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Conclusion: Minimal change disease is the most common cause of nephrotic syndrome in children >1 year of age.

Reference: Marina Vivarelli et al, *Childhood nephrotic syndrome*, The Lancet, 2023

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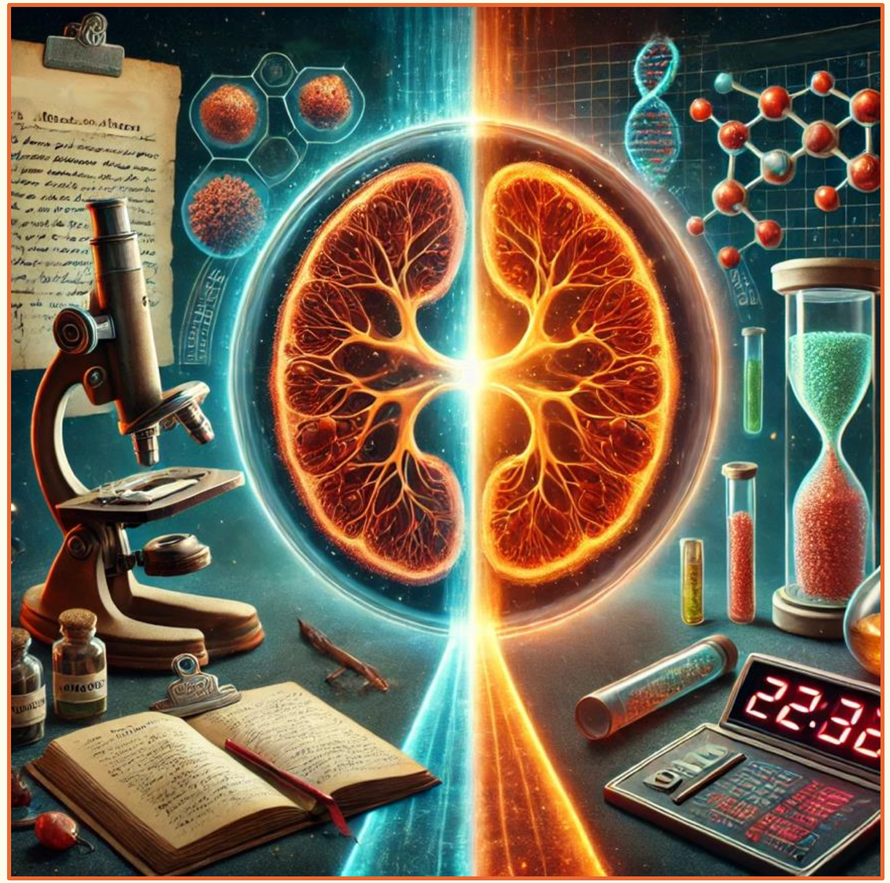
EFFLUENT EIGHT ROUND

Pick Your Champion for the Minimal Change Disease Region



MCD Dx & Pathogenesis

VS

MCD Relapse

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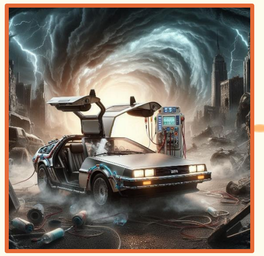
From your Effluent 8, pick your Filtered 4



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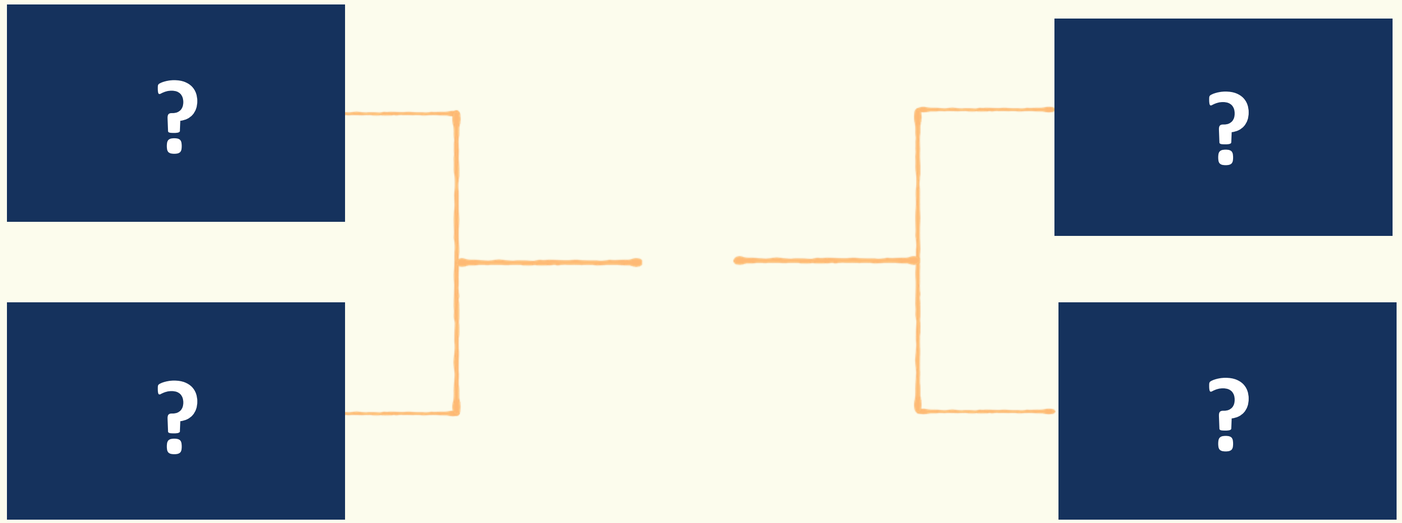
From your Filtered 4, pick your Left and Right Kidneys



Crown your NephMadness 2024 CHAMPION:

Thanks for playing and good luck!

- **Submit brackets by March 31, 2025 on Tourneytopia**
- **Claim CME and MOC credit through NKF PERC**
- **Discuss on social media using #NephMadness**

Important Dates:
March 1, Saturday (7:00 am Eastern): Bracket entry opens
March 31, Monday (11:59 pm Eastern): Deadline for entering contest
April 2, Wednesday: Effluent 8 results
April 4, Friday: Filtered 4 results
April 7, Monday: Left & Right Kidney results
April 8, Tuesday: NephMadness Champion crowned

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