RRT for AKI: when? How much? Which Modality and When to stop

22-25th 2025 – Kuwait - Kuwait

Prof Basset El Essawy

M.B.B.CH, Msc, MD PhD FASN FAST

Maitre es Science Medical (Nephrology, France)

DIU Organ Transplantation (France)

DIU Pediatric Nephrology (France)

AFSA Clinical Nephrology and Hypetension (France)

C1 & C2 Immunology and Immunopathology (France)

Diplome Des Etude Approfonde (DEA) Immunology of Organ transplantation (France)

Prof. of Internal Medicine and Nephrology AlAzhar University & X-Associae Dean Ras ALKhaimah School of Medicine.

Adunject Prof Sharjah, Ras Al-Khaimah & Fakeeh university Hospital

Scientist, TRC- Renal Division; Brigham and women's Hospital.

Professor of Medicine - Harvard Medical School- USA

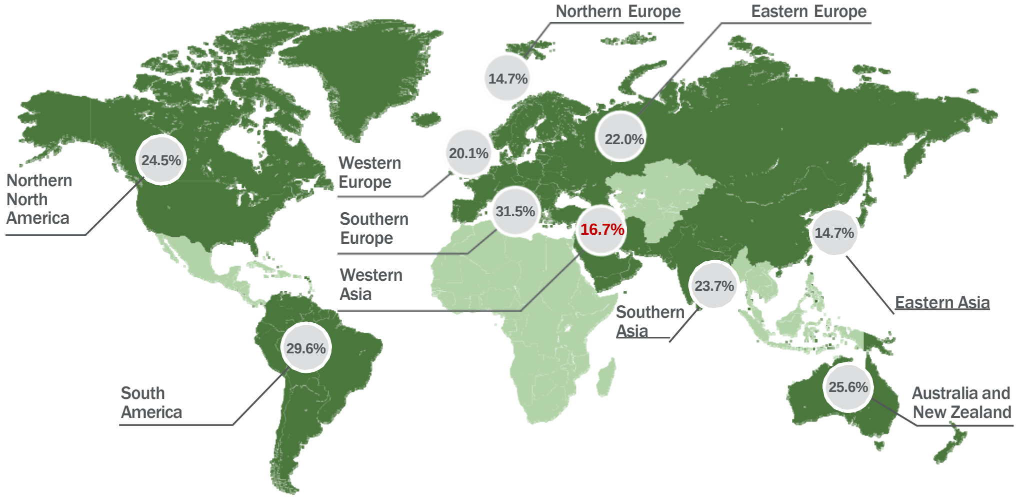

HAYAT حياة
SEHA KIDNEY CARE
UNIVERSITY OF SHARJAH
ISN
EMAN
فقيه
Fakeeh University Hospital
Fakeeh Care Group
WORLD
KIDNEY DAY MEETING
25th
UPDATE IN NEPHROLOGY,
METABOLIC DISORDERS
AND TRANSPLANTATION
12 - 13
APRIL, 2025
MARRIOT AL JADDAF
DUBAI HOTEL
UNDER PROCESS
CME
HOURS
PROF. MONA AL-RUKHAMI
EMAN-T PRESIDENT
DR. MOHAYMEN ABDELGHANY
VICE PRESIDENT FAKEEH CARE AND CEO FUH
PROF. BASSET EL ESSAWY
CONFERENCE PRESIDENT
THC
TOP HOUSE CONFERENCES
FOR MORE INFORMATION
tophconferences
info@tophconferences.com
+971 52 387 3015
Download Program
Register Now

RRT for AKI: when? How much? Which Modality and When to stop

- **Epidemiology, Outcome and Prediction Model of AKI**
- **When to Start RRT (Indications and Timing)**
- ***Which Modality and When to stop***
- ***SGLT2I and ACEi/ARBs***

ACUTE KIDNEY INJURY OVERVIEW

Acute kidney injury is **common** among hospitalized patients globally[1]



AKI AFFECTS AN ESTIMATED 20% OF HOSPITALIZED PATIENTS WORLDWIDE

AKI is a **serious** condition

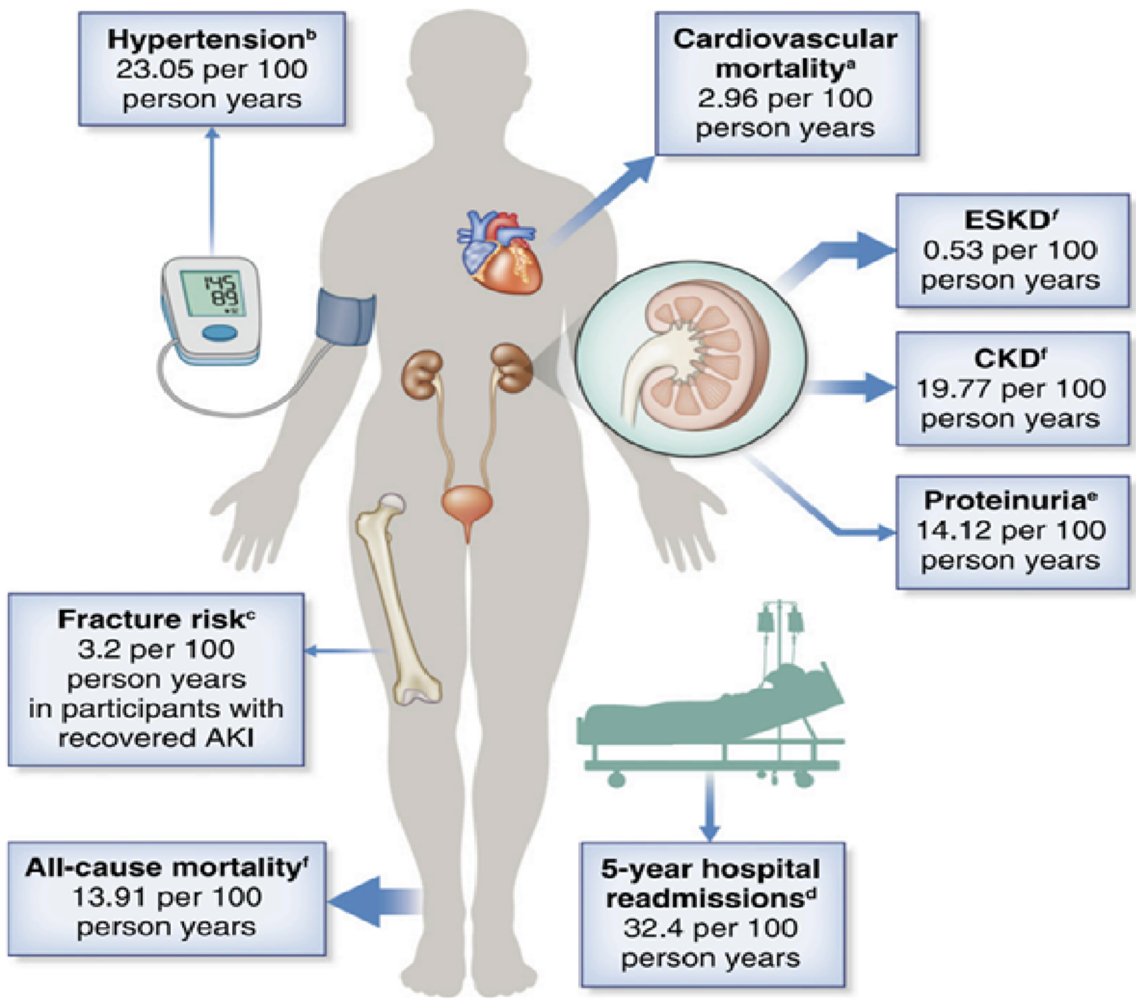
AKI IS ASSOCIATED WITH AN INCREASED RISK OF MORBIDITY and MORTALITY

AKI IS ASSOCIATED WITH AN INCREASED RISK OF CKD, including ESRD

Hsu CY, Ordoñez JD, Chertow GM, et al. The risk of acute renal failure in patients withchronic kidney disease. Kidney Int 2008; 74:101.

*Multicentre meta-analysis of 154 studies (n=3,585,911), primarily in hospital settings, that adopted a KDIGO-equivalent AKI definition between 2004 and 2012. Pooled rates.[1]

CJASN 15: 423–429, 2020. doi: https://doi.org/10.2215/CJN.10410919



. | **Summary of reported event rates for long term outcomes after AKI.** The size of the arrows are a representation of the number of currently available studies reporting this outcome. It should also be noted that, although the rates are reported in the same units (events/100-person-years), data are taken from different sources and are therefore not directly comparable. The superscript letters indicate the sources of the event rates: [a]Odutayo *et al.* (8); [b]Hsu *et al.* (63); [c]Wang *et al.* (64); [d]Brown *et al.* (9); [e]Horne *et al.* (11); and [f]See *et al.* (5).

AKI and Long-Term Risk for Cardiovascular Events and Mortality

Ayodele Odutayo,*† Christopher X. Wong,‡ Michael Farkouh,§ Douglas G. Altman,† Sally Hopewell,† Connor A. Emdin,‖ and Benjamin H. Hunn¶**

*Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada; †Centre for Statistics in Medicine, ‡Nuffield Department of Population Health, ‖St. John's College, and **Department of Physiology, Anatomy and Genetics, University of Oxford, Oxford, United Kingdom; §Peter Munk Cardiac Centre and Heart and Stroke Richard Lewar Centre, University of Toronto, Toronto, Canada; and ¶School of Medicine, University of Tasmania, Hobart, Australia

ABSTRACT

AKI associates with increased long-term risk of mortality, but the prognostic significance of AKI in terms of long-term cardiovascular disease remains unconfirmed. We conducted a systematic review and meta-analysis to assess whether AKI associates with long-term cardiovascular disease. We included cohort studies that examined adults with and without AKI and reported a multivariable-adjusted relative risk (RR) for the association between AKI and cardiovascular mortality, major cardiovascular events, and disease-specific events: congestive heart failure, acute myocardial infarction, and stroke. Twenty-five studies involving 254,408 adults (55,150 with AKI) were included. AKI associated with an 86% and a 38% increased risk of cardiovascular mortality and major cardiovascular events, respectively ([RR 1.86; 95% confidence interval (95% CI), 1.72 to 2.01] and [RR 1.38; 95% CI, 1.23 to 1.55], respectively). For disease-specific events, AKI associated with a 58% increased risk of heart failure (RR 1.58; 95% CI, 1.46 to 1.72) and a 40% increased risk of acute myocardial infarction (RR 1.40; 95% CI, 1.23 to 1.59). The elevated risk of heart failure and acute myocardial infarction persisted in subgroup analyses on the basis of AKI severity and the proportion of adults with baseline ischemic heart disease. Finally, AKI was associated with a 15% increased risk of stroke (RR 1.15; 95% CI, 1.03 to 1.28). In conclusion, AKI associates with an elevated risk of cardiovascular mortality and major cardiovascular events, particularly heart failure and acute myocardial infarction.

J Am Soc Nephrol 28: 377–387, 2017. doi: 10.1681/ASN.2016010105

Original Investigation
FREE
November 14, 2017

Derivation and External Validation of Prediction Models for Advanced Chronic Kidney Disease Following Acute Kidney Injury

Matthew T. James, MD, PhD[1,2,3,4]; Neesh Pannu, MD, SM[5]; Brenda R. Hemmelgarn, MD, PhD[1,2,3,4]; et al
≫ Author Affiliations | Article Information

JAMA. 2017;318(18):1787-1797. doi:10.1001/jama.2017.16326

A Point values for each variable

Age, y	Points
<50	0
50-59	1
60-69	2
70-79	2
80-89	2
≥90	3

Sex	Points
Men	0
Women	3

Baseline SCr, mg/dL	Points
<0.6	0
0.6-<0.7	1
0.7-<0.8	1
0.8-<0.9	2
0.9-<1.0	2
1.0-<1.1	3
1.1-<1.2	3
1.2-<1.3	4
≥1.3	5

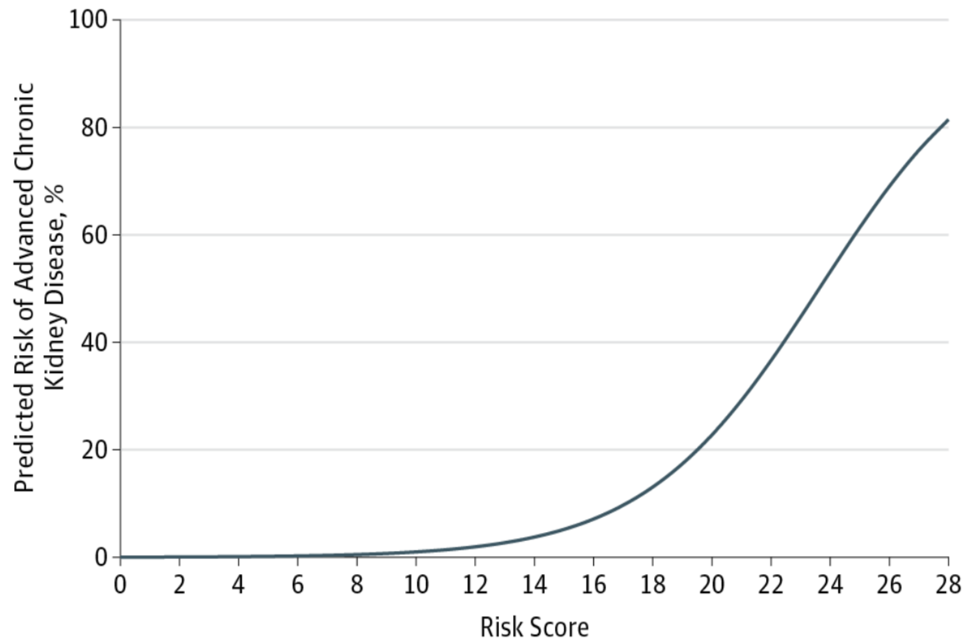
Albuminuria	Points
Normal	0
Mild	1
Heavy	3
Not measured	1

Acute kidney injury stage	Points
1	0
2	1
3	3

Discharge SCr, mg/dL	Points
<1.0	0
1.0-<1.3	3
1.3 -<1.6	6
1.6 -<1.9	7
≥ 1.9	11

Summation of Points:	
Age	+________
Sex	+________
Baseline serum creatinine value	+________
Albuminuria	+________
Acute kidney injury stage	+________
Discharge serum creatinine value	+________
Total Risk Score =	

B Predicted risk of advanced chronic kidney disease



Total Risk Score	Predicted Risk of Advanced Chronic Kidney Disease, %	Percentage of Patients in Risk Category, %	
		Derivation Cohort	External Validation Cohort
1-8	<1	44.0	45.0
9-14	1-<5	43.6	43.8
15-17	5-<10	7.2	7.1
18-19	10-<20	2.5	2.1
≥20	≥20	2.7	2.0

Summary of knowledge gaps in current understanding of the long-term outcomes of AKI

Areas of Uncertainty/Knowledge Gaps in Our Understanding of the Long-Term Outcomes of AKI	Suggestions for Next Steps
To what degree the association between AKI and mortality is causal	Prospective data collection with adequate consideration of confounding variables, intervention studies that demonstrate reduced mortality
Understanding of the mechanism of the link between AKI and CKD in humans	Translation of animal models to humans; clinical studies incorporating methods that improve mechanistic understanding of the AKI to CKD transition in humans (*e.g.*, biopsy, biomarkers, or imaging)
Incorporation of etiologic factors into assessment and definition of AKI	Development of tools that include AKI etiology in assessment; prospective data collection focusing on precise etiology of AKI
Optimal measurement of kidney function following AKI	Consensus definition to allow for harmonization of data going forward
Understanding the incidence of albuminuria post-AKI because this might be a potential target for future intervention	Incorporation of albuminuria into CKD definition post-AKI in line with the KDIGO CKD definition; further study of albuminuria as an independent risk factor for adverse outcomes in a post-AKI setting
Establish the role of existing or novel biomarkers in the recovery phase of AKI to improve risk stratification and/or provide mechanistic insights into the AKI to CKD transition	Collection of biomarkers at time of AKI combined with collection of biomarkers during recovery
Availability of interventions that reduce long-term sequelae following AKI	Studies into optimal or tailored follow-up strategies and research into gaps in current post-AKI care

KDIGO, Kidney Disease Improving Global Outcomes.

Long-Term Outcomes in Patients with Acute Kidney Injury

There are no established therapeutic interventions to reduce post-AKI sequelae or evidence to inform strategies for health care provision.

Why this is important?

Because post-AKI care has been shown to be variable, with many patients not receiving any planned follow-up even when they have received RRT during their acute

Episode.

CJASN 15: 423–429, 2020. doi: https://doi.org/10.2215/CJN.10410919

RRT for AKI: when? How much? Which Modality and When to stop

- **Epidemiology, Outcome and Prediction Model of AKI**

- **When to Start RRT (Indications and Timing)**

- ***Which Modality and When to stop***

- ***SGLT2i and ACEi/ARBs***

RRT for AKI: When? How much? Which Modality and When to stop

- Urgent Indications:-

●*Fluid overload refractory to diuretic therapy*

●*Severe hyperkalemia (plasma potassium concentration >6.5 mEq/L) or rapidly rising potassium levels*

●*Overt manifestations of uremia, such as pericarditis, encephalopathy, or an otherwiseunexplained decline in mental status*

●*Severe metabolic acidosis (pH <7.1) despite medical management, though the benefit of KRT in patients with lactic acidosis is uncertain.*

● *Certain alcohol and drug intoxications amenable to extracorporeal therapy*

ACUTE KIDNEY INJURY OVERVIEW

Fluid overload is one condition that may adversely impact AKI patient prognosis

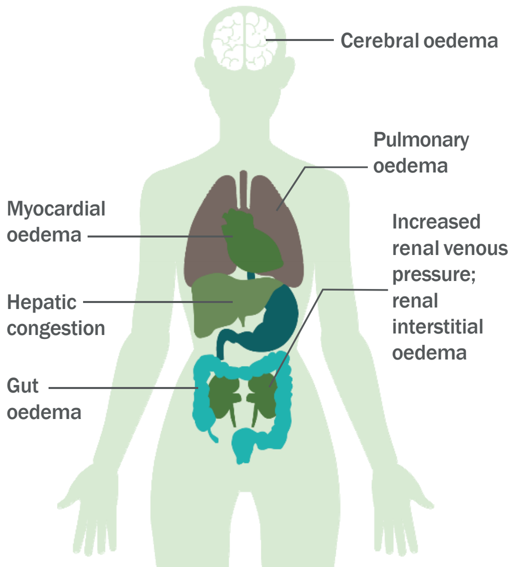
RRT REQUIREMENT[2]

Conditions that can impact AKI patient prognosis

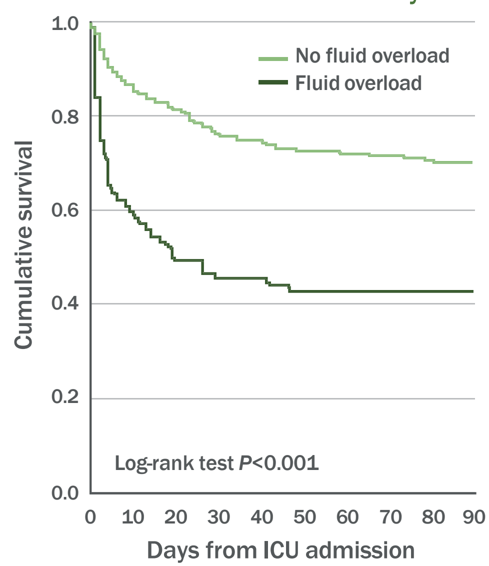
HEMODYNAMIC INSTABILITY[11,12]

FLUID OVERLOAD

Consequences of fluid overload may lead to organ dysfunction



Fluid overload at RRT initiation for AKI has been associated with an increased risk of mortality[11,*]

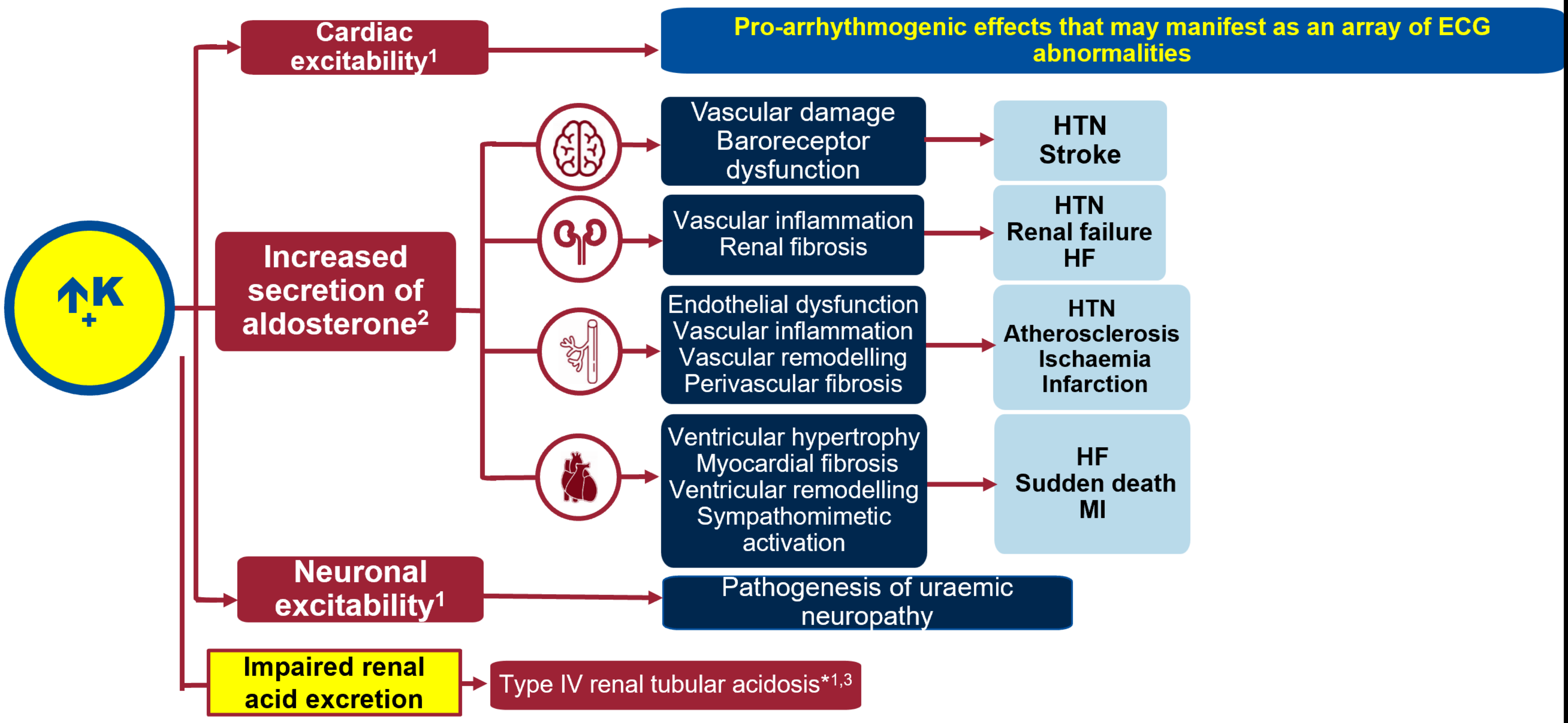


FLUID OVERLOAD IN PATIENTS WITH AKI IS A SERIOUS CONDITION

* Prospective, observational cohort study of 296 adults treated with RRT in 17 Finnish ICUs from Sep 2011–Feb 2012.:.

Zhang L, et al. *J Crit Care*. 2015;30:860.e7-13.
Bouchard J, et al. *Kidney Int*. 2009;76:422-427.

SERIOUS CLINICAL CONSEQUENCES OF CHRONIC HYPERKALAEMIA



1. Hunter RW and Bailey MA. Nephrol Dial Transplant 2019;34:iii2–iii11; 2. Zannad F, et al. The Evolution of Heart Failure Management: The Role of Aldosterone: The Emergence of Aldosterone As a Cardiac Toxin. Presented at Medscape CME & Education; 17th December 2003. Available at: https://www.medscape.org/viewarticle/466090_2 (accessed May 2021);

3. Palmer BF, et al. Adv Ther 2021;38:949–68.

*Exact mechanism unclear.

Timing of Individualized initiation

Individual *initiation of RRT in patients with AKI that is unlikely to resolve quickly and have one or more of the following:*

• S K >6.0 mEq/L that is unresponsive to aggressive medical management, or>5.5 mEq/L if there is ongoing tissue breakdown (eg, Rhabdomyolysis, Crush injury, Tumor Lysis Syndrome).

• Ongoing K absorption (eg, due to severe gastrointestinalbleeding). Elective initiation of RRT in patients before the potassium reaches 6.5 mEq/L can help avoid emergency initiation and potentially life-threatening arrhythmias.

S K can rise rapidly in patients with AKI who have ongoing tissue breakdown or K absorption.

•Severe metabolic acidosis (pH <7.15) without reversible cause (eg, ketoacidosis) and despite optimal medical management (eg, intravenous Nacho3 therapy as volume status permits). Data supporting a precise pH threshold for initiation of RRT int his setting are lacking; some experts would suggest initiation of RRT at higher pH levels(eg, pH <7.2).- STARRT-AKI Investigators, Canadian Critical Care Trials Group, Australian and New ZealandIntensive Care Society Clinical Trials Group, et al. Timing of Initiation of Renal-ReplacementTherapy in Acute Kidney Injury. N Engl J Med 2020; 383:240.

Timing of Individualized initiation

The benefit of RRT in patients with severe metabolic acidosis due to lactic acidosis is controversial, as the rate of clearance that can be provided by RRT is substantially < endogenous generation .

RRT is employed as supportive therapy as a bridge to definitive management of the underlying cause of lactic acidosis (eg, bowel resection for ischemic bowel), there is little evidence of mortality benefit. The exception is the treatment of metformin-associated lactic acidosis, in which RRT reverses the underlying cause.

● Hypervolemic patients who are oliguric or who remain in persistent +ve fluid balance despite high doses of loop diuretics (often used in combination with a thiazide or thiazide-like diuretic), particularly if oxygen requirements are increasing. Elective initiation in such patients can help avoid the need for intubation and mechanical ventilation.

- *There are No specific time threshold (eg, 72 hours of severe AKI) to initiate RRT in the absence of the urgent or Individulized indications as presented above.- - Gaudry S, Hajage D, Benichou N, et al. Delayed versus early initiation of renal replacementtherapy for severe acute kidney injury: a systematic review and individual patient datameta-analysis of randomised clinical trials. Lancet 2020; 395:1506. - Gaudry S, Hajage D, Schortgen F, et al. Initiation Strategies for Renal-Replacement Therapyin the Intensive Care Unit. N Engl J Med 2016; 375:122.*

Timing of initiation of dialysis and its association with mortality

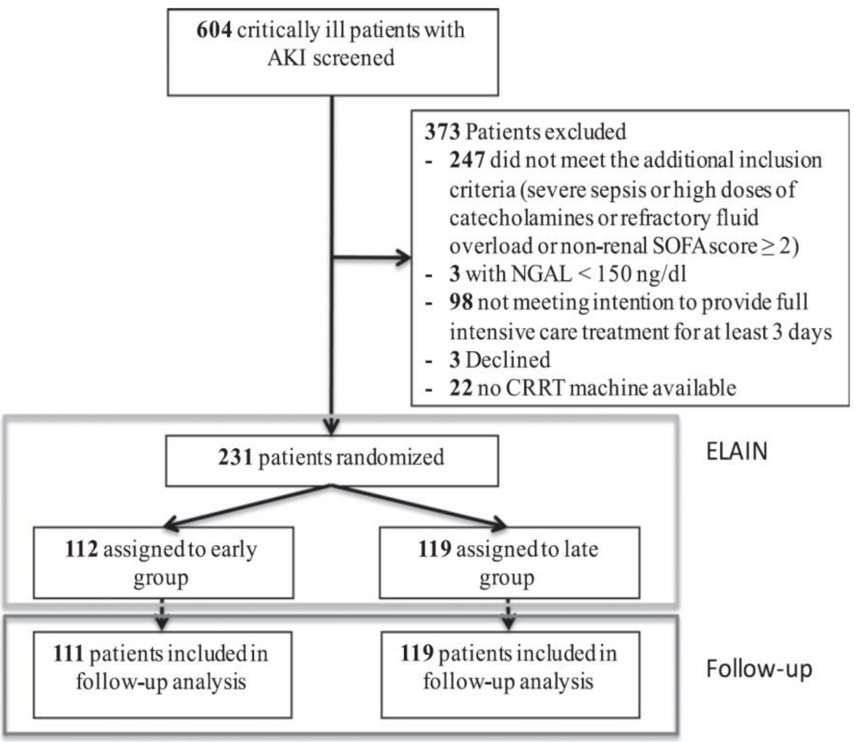
Study	Reference	Year	No. of Patients	Study Design	Predialysis BUN (mg/dl)		Mortality (%)	
					Early	Late	Early	Late
Parsons *et al.*	(6)	1961	33	Cohort with historical control	120 to 150	200	25	88
Fischer *et al.*	(7)	1966	162	Cohort with historical control	152	231	51	77
Kleinknecht *et al.*[a]	(8)	1972	320	Cohort with historical control	93	164	29	42
Conger[a]	(9)	1975	18	Case-control	50	120	20	64
Gettings *et al.*	(10)	1999	100	Retrospective cohort	42.6	94.5	61	80
Bouman *et al.*	(16)	2002	65	Randomized trial	48	105	31	25

[a]Case patients and control subjects differed with respect to both the timing of initiation of dialysis and the dose of dialysis delivered.

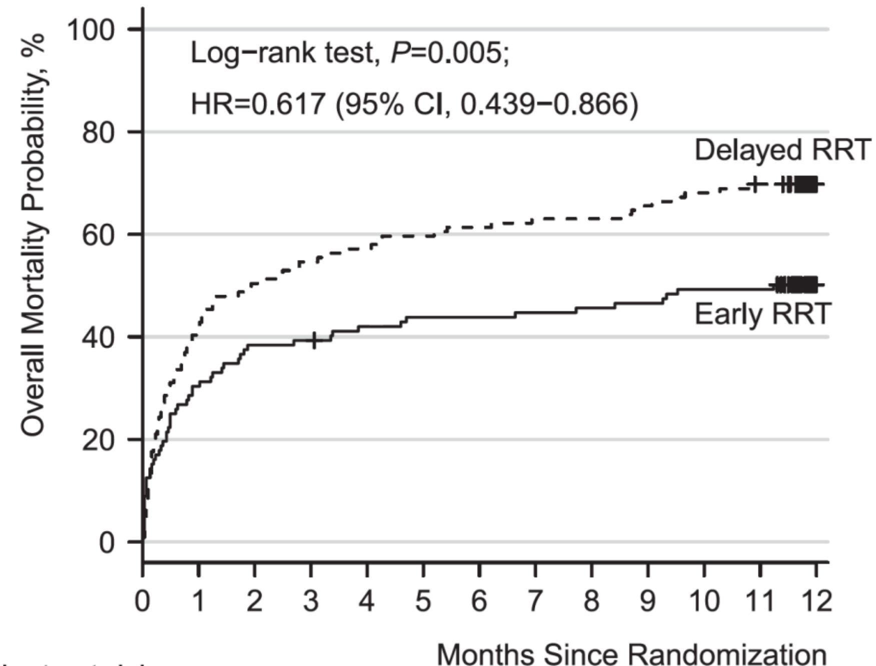
Long-Term Clinical Outcomes after Early Initiation of RRT in Critically Ill Patients with AKI

Melanie Meersch,[1] Mira Küllmar,[1] Christoph Schmidt,[1] Joachim Gerss,[2] Toni Weinhage,[3] Andreas Margraf,[1] Thomas Ermert,[1] John A. Kellum,[4] and Alexander Zarbock[1]

[1]Department of Anaesthesiology, Intensive Care and Pain Medicine and [2]Institute of Biostatistics and Clinical Research, University Hospital of Münster, Muenster, Germany; [3]Department of Pediatric Rheumatology and Immunology, University Hospital Münster, Muenster, Germany; and [4]Center for Critical Care Nephrology, Department of Critical Care Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania



The flowchart of the ELAIN Trial shows 231 patients were included in the initial rial and 1 patient was lost to follow-up for the follow-up analysis. CRRT, continuous RRT; NGAL, neutrophil gelatinase–associated lipocalin.



Patients at risk

Delayed	119	69	59	54	51	48	46	44	44	41	38	35
Early RRT	112	78	69	68	64	62	62	61	60	59	56	56

The Kaplan Meier analysis shows the overall mortality in the early versus the delayed RRT initiation group. Patients with early initiation of RRT showed a significant improved survival as compared with the delayed group (HR, 0.617; 95% CI, 0.439–0.866; *P*=0.005).

Timing of Individualized initiation

Initiating RRT even earlier in the course of AKI (ie, before the patient develops an urgent indication or one of the Individulaized indications) is generally not beneficial.

Early initiation of RRT may be harmful, may delay recovery of kidney function, and results in increased health care utilization.

Multiple trials have compared strategies of early RRT initiation (in the absence of any indications mentioned above) with delayed RRT initiation (once indications have developed).

Wald R, Kirkham B, daCosta BR, et al. Fluid balance and renal replacement therapyinitiation strategy: a secondary analysis of the STARRT-AKI trial. Crit Care 2022; 26:360.

19

Fluid balance and renal replacement therapy initiation strategy: a secondary analysis of the STARRT-AKI trial

Ron Wald[1,2*], Brian Kirkham[3], Bruno R. daCosta[2,3], Ehsan Ghamarian[3], Neill K. J. Adhikari[4], William Beaubien-Souligny[5], Rinaldo Bellomo[6,7,8,9], Martin P. Gallagher[10], Stuart Goldstein[11], Eric A. J. Hoste[12,13], Kathleen D. Liu[14], Javier A. Neyra[15], Marlies Ostermann[16], Paul M. Palevsky[17], Antoine Schneider[18], Suvi T. Vaara[19] and Sean M. Bagshaw[20]

Abstract

Background: Among critically ill patients with acute kidney injury (AKI), earlier initiation of renal replacement therapy (RRT) may mitigate fluid accumulation and confer better outcomes among individuals with greater fluid overload at randomization.

Methods: We conducted a pre-planned post hoc analysis of the STandard versus Accelerated initiation of Renal Replacement Therapy in Acute Kidney Injury (STARRT-AKI) trial. We evaluated the effect of accelerated RRT initiation on cumulative fluid balance over the course of 14 days following randomization using mixed models after censoring for death and ICU discharge. We assessed the modifying effect of baseline fluid balance on the impact of RRT initiation strategy on key clinical outcomes. Patients were categorized in quartiles of baseline fluid balance, and the effect of accelerated versus standard RRT initiation on clinical outcomes was assessed in each quartile using risk ratios (95% CI) for categorical variables and mean differences (95% CI) for continuous variables.

Results: Among 2927 patients in the modified intention-to-treat analysis, 2738 had available data on baseline fluid balance and 2716 (92.8%) had at least one day of fluid balance data following randomization. Over the subsequent 14 days, participants allocated to the accelerated strategy had a lower cumulative fluid balance compared to those in the standard strategy (4509 (−728 to 11,698) versus 5646 (0 to 13,151) mL, $p = 0.03$). Accelerated RRT initiation did not confer greater 90-day survival in any of the baseline fluid balance quartiles (quartile 1: RR 1.11 (95% CI 0.92 to 1.34), quartile 2: RR 1.03 (0.87 to 1.21); quartile 3: RR 1.08 (95% CI 0.91 to 1.27) and quartile 4: RR 0.87 (95% CI 0.73 to 1.03), *p* value for trend 0.08).

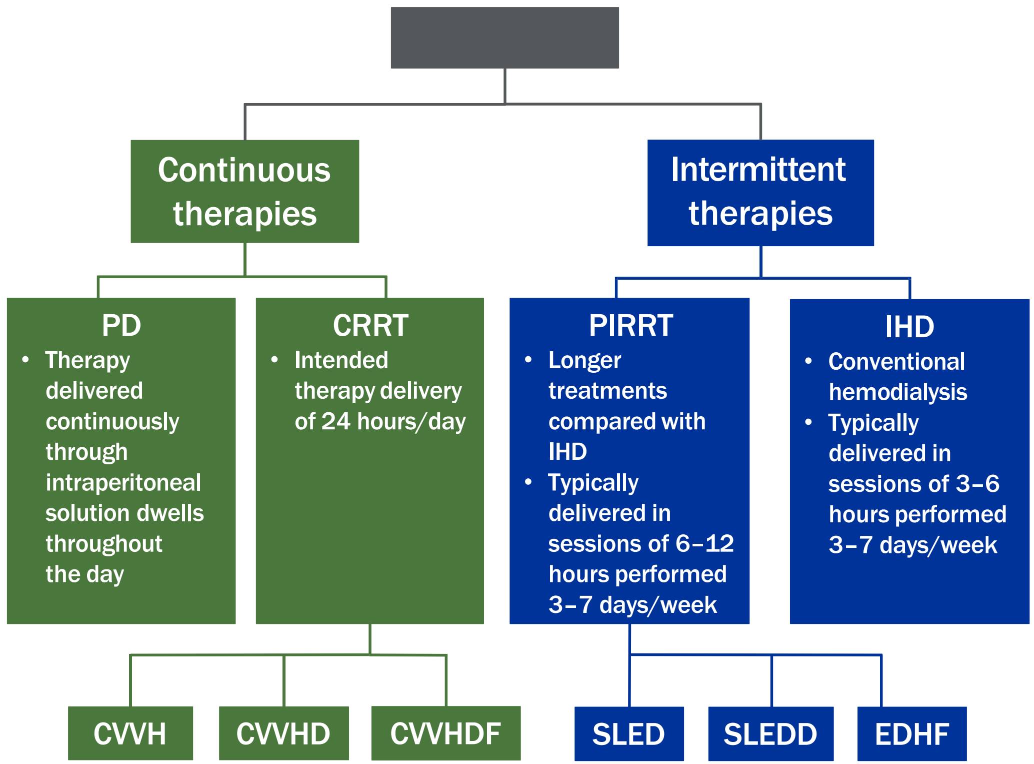
Conclusions: Earlier RRT initiation in critically ill patients with AKI conferred a modest attenuation of cumulative fluid balance. Nonetheless, among patients with greater fluid accumulation at randomization, accelerated RRT initiation did not have an impact on all-cause mortality.

RRT for AKI: when? How much? Which Modality and When to stop

- **Epidemiology, Outcome and Prediction Model of AKI**
- **When to Start RRT (Indications and Timing)**
- ***Which Modality and When to stop***
- ***SGLT2i and ACEi/ARBs***

RRT MODALITIES FOR AKI

Various **renal replacement modalities** are available for the **management of AKI**



ACUTE RRT IS DELIVERED AS EITHER A CONTINUOUS OR INTERMITTENT THERAPY[20]

- Fleming GM. *Organogenesis*. 2011;7:2-12.
- O'Reilly P, Tolwani A. *Crit Care Clin*. 2005;367-378.-
- Pannu N, Gibney RTN. *Ther Clin Risk Manag*. 2005;1:141- 150.
- Sun Z, et al. *Crit Care*. 2014;18:R70.

RRT MODALITIES FOR AKI

Modalities **Differ** in Their Typical Characteristics

Typical RRT modality characteristics and settings for a 70-kg AKI patient[25–27]

	CONTINUOUS THERAPIES			INTERMITTENT THERAPIES	
Parameter	**CVVH**	**CVVHD**	**CVVHDF**	**SLED***	**IHD**
Blood flow (Q_B, mL/min)	150–250	150–250	150–250	100–300	200–300
Predominant solute transport principle			+		
Ultrafiltrate (mL/h)	1500–2000	variable	1000–1500	variable	variable
Dialysate flow (Q_D, mL/h)	0	1500–2000	1000–1500	6000–18,000	18,000–30,000
Replacement fluid for zero balance (mL/h)	1500–2000	0	1000–1500	0	0
Urea clearance (mL/min)	25–33	25–33	25–33	80–90	200–500

Convection Diffusion

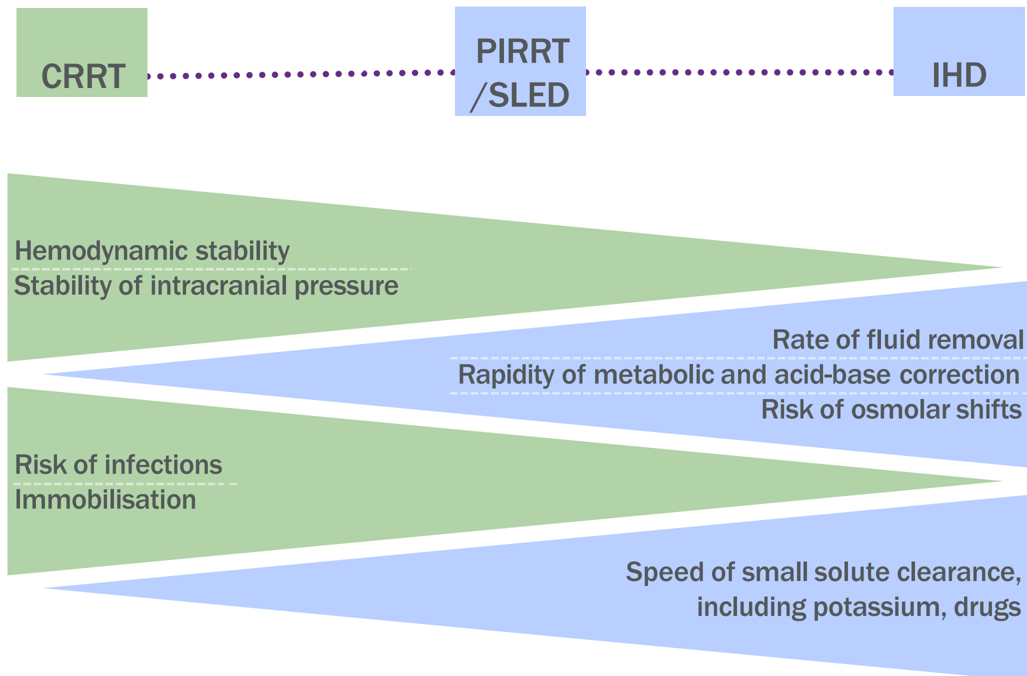
Q_B, Q_D, AND UREA CLEARANCE TEND TO BE LOWER IN CONTINUOUS THERAPIES THAN IN INTERMITTENT THERAPIES

REVACLEAR Dialyzer Technology. 2017. Available from: http://www.baxter.ca/en_CA/assets/downloads/2017/Rev aclear%20Spec%20Sheet%20Brochure%20English.pdf (accessed December 2018).Fresenius Medical Care. Optiflux Dialyzers. 2016. Available from: http://www.fmcna- dialyzers.com/images/pdf/101046-Optiflux- Dialyzer_SpecSheet.pdf (accessed December 2018).

RRT MODALITIES FOR AKI

Individual patient needs can be addressed by considering the characteristics of the various **RRT modalities**[28]

Relative **features**, **risks**, and **burdens** of different RRT modalities[28]



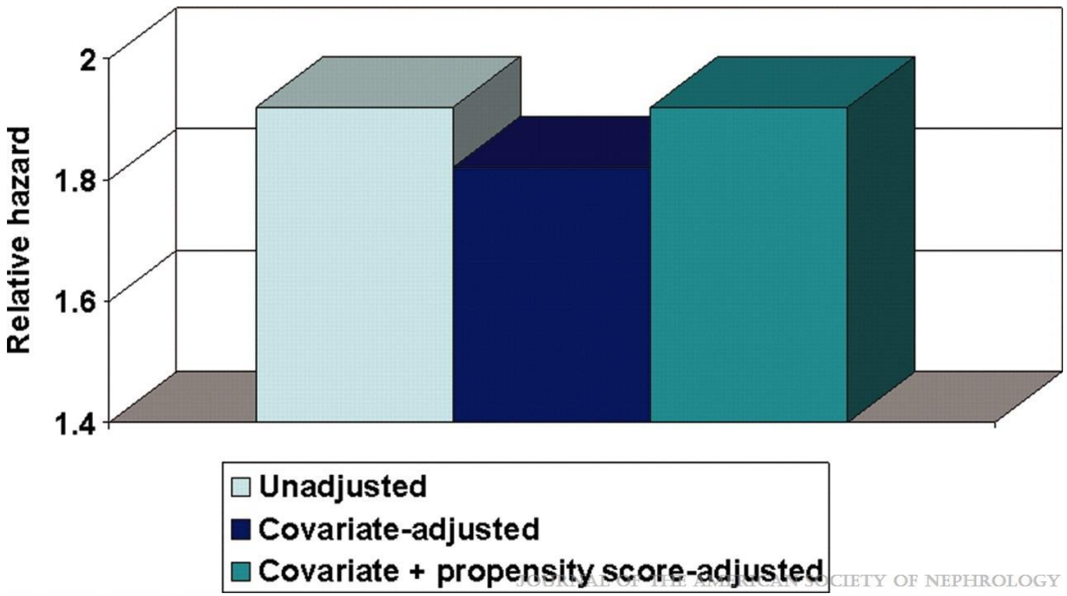
EACH RRT MODALITY HAS POTENTIAL BENEFITS AND LIMITATIONS FOR THE MANAGEMENT OF PATIENTS WITH AKI[28]

11.Ostermann M, et al. *Blood Purif*. 2016;42:224-237.

[Survival by Dialysis Modality in Critically Ill Patients with Acute Kidney Injury]

Cho, Kerry C.; Himmelfarb, Jonathan; Paganini, Emil; Ikizler, T. Alp; Soroko, Sharon H.; Mehta, Ravindra L.; Chertow, Glenn M.

Journal of the American Society of Nephrology17(11):3132-3138, November 2006.
doi: 10.1681/ASN.2006030268



Mortality within 60 d after acute kidney injury requiring dialysis: Continuous renal replacement therapies versus intermittent hemodialysis.

Data do not support the superiority of either CRRT or IHD.

Thus, the selection of modality of RRT should be based upon local expertise and experience in combination with the needs of the individual patient

When to stop = Discontinuations

- *A precise level of kidney function needed to allow discontinuation of RRT has not been established.*

- *However, a creatinine clearance <12 mL/min is probably inadequate to allow discontinuation of therapy.*

- *Inthe VA/NIH ATN study, RRT was discontinued when the measured creatinine clearance exceeded 20 mL/min and was left to the discretion of providers when in the range of 12 to 20mL/min.*

- VA/NIH Acute Renal Failure Trial Network, Palevsky PM, Zhang JH, et al. Intensity of renalsupport in critically ill patients with acute kidney injury. N Engl J Med 2008; 359:7.

RRT for AKI: when? How much? Which Modality and When to stop

- **Epidemiology, Outcome and Prediction Model of AKI**
- **When to Start RRT (Indications and Timing)**
- ***Which Modality and When to stop***
- ***SGLT2I and ACEi/ARBs***

Kidney Outcomes with Sodium–Glucose Cotransporter-2 Inhibitor Initiation after AKI among Veterans with Diabetic Kidney Disease

Daniel P. Murphy,[1] Julian Wolfson,[2] Scott Reule,[1,3] Kirsten L. Johansen,[1,4,5] Areef Ishani,[1,3] and Paul E. Drawz[1]

Key Points

- Post-AKI sodium–glucose cotransporter-2 inhibitor use was associated with a reduced risk for progression of CKD and for recurrent AKI among veterans with diabetic kidney disease even after accounting for recovery from the index AKI.
- A minority of Veterans with diabetic kidney disease received a sodium–glucose cotransporter-2 inhibitor after having had AKI during the study period.

Abstract

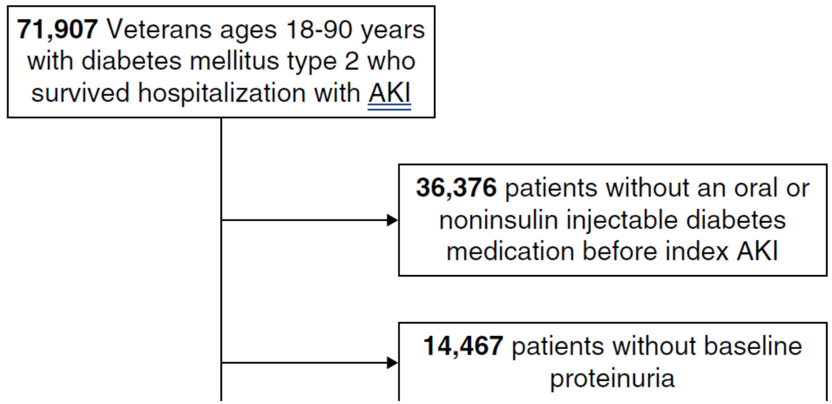
Background The effect of sodium–glucose cotransporter-2 inhibitor (SGLT2i) on kidney function after AKI is unknown.

Methods The study population was drawn from a retrospective cohort of Veterans with diabetes mellitus type 2 (DM2) and proteinuria. The study exposure was time-varying use of SGLT2i after an index AKI hospitalization. The two study outcomes were time to (*1*) a sustained decrease in eGFR over at least 3 months to <60 ml/min per 1.73 m^2 and ≥30% below a post-AKI–updated eGFR and (2) recurrent hospitalization with AKI. AKI was defined as a rise in serum creatinine concentration to ≥50% above a moving outpatient creatinine baseline. DM2 was defined by ≥2 billing codes related to DM2 before the index AKI; proteinuria was defined by the most recent albuminuria, proteinuria, or urinalysis test. Veterans were required to have a baseline eGFR and an eGFR 3–12 months after the index AKI hospitalization ≥30 ml/min per 1.73 m^2.

Results Ten thousand thirty-six Veterans met study inclusion criteria. Two thousand seven hundred and ninety-four (28%) received a SGLT2i. Seven hundred and seventy-five (8%) had CKD progression, and 1816 (18%) had recurrent AKI over a median follow-up of 1.8 and 1.7 years, respectively, which began 1 year after the index AKI hospitalization. SGLT2i use was associated with lower risk for CKD progression (adjusted hazard ratio 0.72 [95% confidence interval, 0.57 to 0.91]) and for recurrent AKI (adjusted hazard ratio 0.75 [95% confidence interval, 0.65 to 0.88]).

Conclusions SGLT2i use was associated with a lower risk for CKD progression and for recurrent AKI among those with diabetic kidney disease and recent AKI.

Kidney360 5: 335–343, 2024. doi: https://doi.org/10.34067/KID.0000000000000375



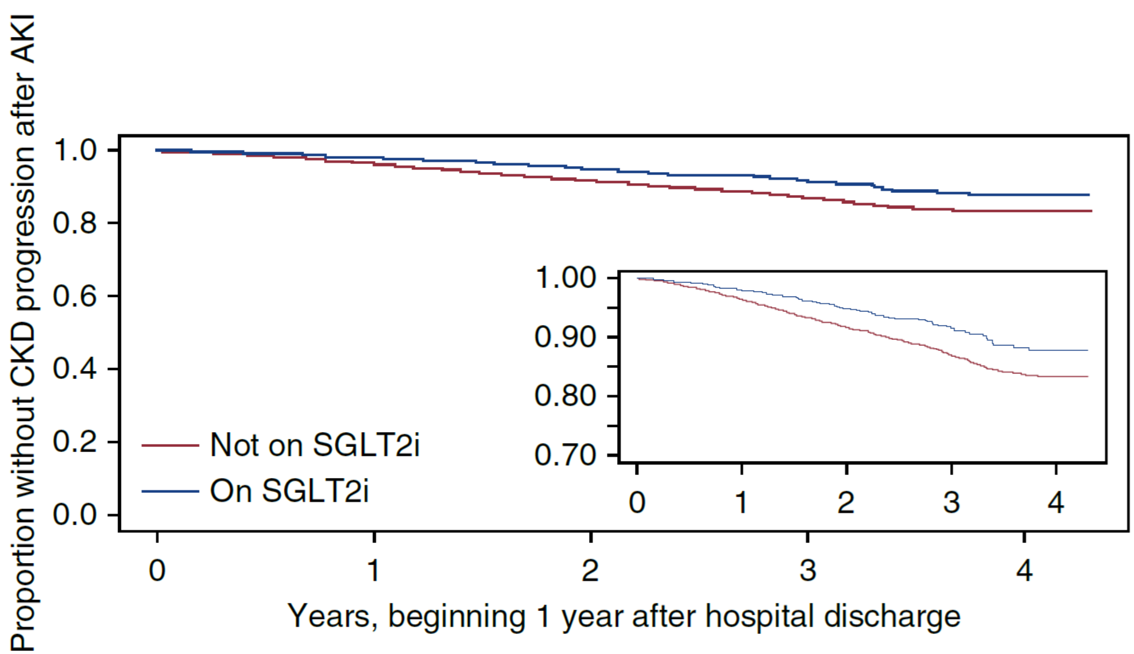
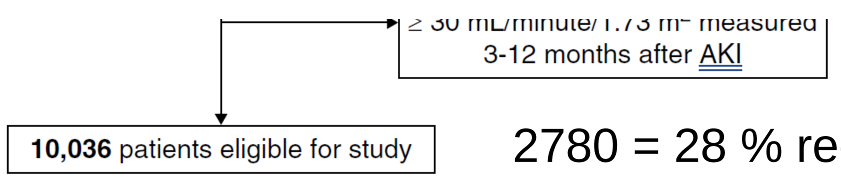


Figure 3. Kaplan–Meier curve for CKD progression after AKI by post-AKI SGLT2i use (N=10,036, P < 0.001).



2780 = 28 % received SGLT2i after AKI

Renin–Angiotensin–Aldosterone System Blockade after AKI with or without Recovery among US Veterans with Diabetic Kidney Disease

Daniel P. Murphy,[1] Julian Wolfson,[2] Scott Reule,[1,3] Kirsten L. Johansen,[1,4,5] Areef Ishani,[1,3] and Paul E. Drawz[1]

[1] Department of Medicine, Medical School, University of Minnesota, Minneapolis, Minnesota
[2] Division of Biostatistics, School of Public Health, University of Minnesota, Minneapolis, Minnesota
[3] Section of Nephrology, Minneapolis Veterans Affairs Health Care System, Minneapolis, Minnesota
[4] Division of Nephrology, Hennepin Healthcare, Minneapolis, Minnesota
[5] Chronic Disease Research Group, Hennepin Healthcare Research Institute, Minneapolis, Minnesota

ABSTRACT

Background Optimal use of angiotensin-converting enzyme inhibitors (ACEis) or angiotensin II receptor blockers (ARBs) after AKI is uncertain.

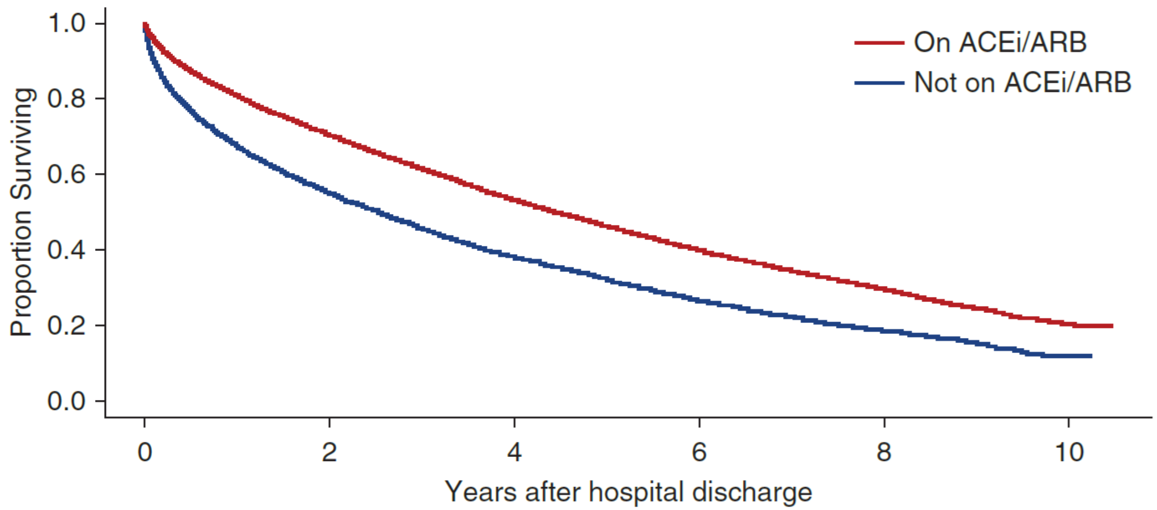
Methods Using data derived from electronic medical records, we sought to estimate the association between ACEi/ARB use after AKI and mortality in US military Veterans with indications for such treatment (diabetes and proteinuria) while accounting for AKI recovery. We used ACEi/ARB treatment after hospitalization with AKI (defined as serum creatinine ≥50% above baseline concentration) as a time-varying exposure in Cox models. The outcome was all-cause mortality. Recovery was defined as return to ≤110% of baseline creatinine. A secondary analysis focused on ACEi/ARB use relative to AKI recovery (before versus after).

Results Among 54,735 Veterans with AKI, 31,146 deaths occurred over a median follow-up period of 2.3 years. Approximately 57% received an ACEi/ARB <3 months after hospitalization. In multivariate analysis with time-varying recovery, post-AKI ACEi/ARB use was associated with lower risk of mortality (adjusted hazard ratio [aHR], 0.74; 95% confidence interval [CI], 0.72 to 0.77). The association between ACEi/ARB use and mortality varied over time, with lower mortality risk associated with earlier initiation (*P* for interaction with time <0.001). In secondary analysis, compared with those with neither recovery nor ACEi/ARB use, risk of mortality was lower in those with recovery without ACEi/ARB use (aHR, 0.90; 95% CI, 0.87 to 0.94), those without recovery with ACEi/ARB use (aHR, 0.69; 95% CI, 0.66 to 0.72), and those with ACEi/ARB use after recovery (aHR, 0.70; 95% CI, 0.67 to 0.73).

Conclusions This study demonstrated lower mortality associated with ACEi/ARB use in Veterans with diabetes, proteinuria, and AKI, regardless of recovery. Results favored earlier ACEi/ARB initiation.

JASN 34: 1721–1732, 2023. doi: https://doi.org/10.1681/ASN.0000000000000196

140,734 patients age 18-90 years with diabetes mellitus who survived AKI up to hospital discharge



Kaplan–Meier survival curves by ACEi/ARB use after hospitalization with AKI among Veterans with diabetes mellitus and proteinuria (*N*=54,735; *P* < 0.001). ACEi/ARB, angiotensin-converting enzyme inhibitor and angiotensin-II receptor blocker.

57 % received ACEi /ARBS within 3 months after discharge

Dr Basset El Essawy

aelessawy@bwh.harvard.edu
belessawy@azhar.edu.eg
Abdel.essawy@rakmhsu.ac.ae
abelessawy@fakeeh.care
belessawy@gmail.com

THANK YOU

RRT MODALITY CONSIDERATIONS

Selection of RRT modality requires careful consideration of many patient- and ICU-specific factors[25,28]

Overview of modality considerations

- CLINICAL CONSIDERATIONS: FLUID OVERLOAD AND HEMODYNAMIC INSTABILITY
- CLINICAL CONSIDERATIONS: LONG-TERM OUTCOMES
- MACHINE AND PRESCRIPTION CONSIDERATIONS
- SOLUTION CONSIDERATIONS
- LONG-TERM COST CONSIDERATIONS
- EQUIPMENT FOOTPRINT AND MOBILITY CONSIDERATIONS

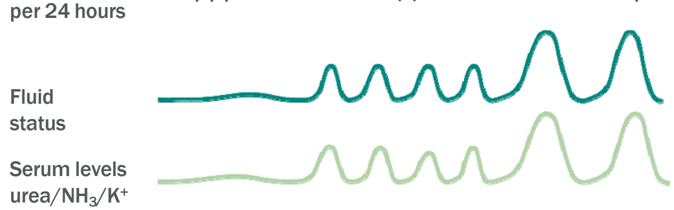
Clinical considerations: fluid overload and hemodynamic instability

Fluid overload in AKI patients can be treated by fluid removal during RRT, but rapid fluid removal that does not allow time for plasma refill may lead to hemodynamic instability[25,29]

Avoiding rapid fluid removal to prevent hypovolaemia may **improve** AKI patient outcomes[25,29]

Modality comparisons[30]

	CRRT	PIRRT /SLED	IHD
	Continuous		Intermittent
Clearance per hour	+	++	+++
Clearance per 24 hours	+++	++	+



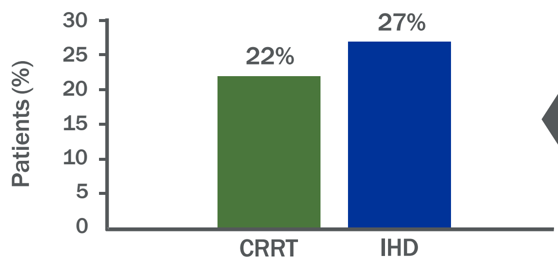
CRRT MAY OFFER MORE PRECISE FLUID MANAGEMENT VS IHD OR PIRRT/SLED[25,30]

CRRT IS A PREFERRED RRT BY MANY CLINICIANS FOR AKI PATIENTS WHO ARE HEMODYNAMICALLY UNSTABLE[25,29]

Clinical considerations: long-term outcomes

AKI is associated with an increased risk of long-term dialysis dependence;[8] acute RRT modality type may impact this risk[31–34]

Patients on chronic dialysis at day 90 by initial RRT modality[31,*]



CHRONIC DIALYSIS HAZARD RATIO (95% CI) FOR CRRT vs IHD WAS 0.75 (0.65–0.87), P<0.0001[31]

Modality comparisons[31–34]

CONTINUOUS	INTERMITTENT	
CRRT	PIRRT	IHD
Patients are less likely to require chronic dialysis following initial AKI episode compared with patients treated with IHD	? Insufficient evidence	It has been reported that patients are more likely to require chronic dialysis following initial AKI episode compared with patients treated with CRRT

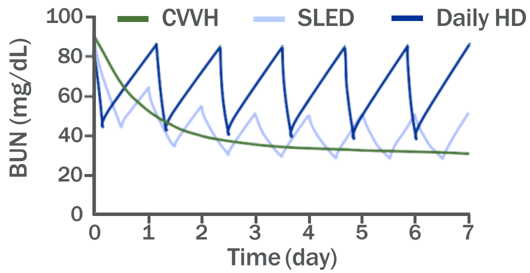
USE OF CRRT FOR AKI MANAGEMENT HAS BEEN ASSOCIATED WITH A LOWER RISK of CHRONIC DIALYSIS COMPARED WITH IHD[31–34]

*Retrospective multicentre cohort study of critically ill adults with AKI between 1996 and 2009. 2004 patients originally treated with CRRT and 2004 patients originally treated with IHD were propensity matched and rates of dialysis dependence were compared.[31]

Machine and prescription considerations

RRT machines deliver different dose intensities over different durations of therapy[21,22,25]

Kinetic modeling of urea clearance by different RRT modalities[35]



A SAWTOOTH PATTERN WAS OBSERVED WHEN USING INTERMITTENT THERAPIES TO REMOVE UREA, WHILE CONTINUOUS THERAPY MAINTAINED A CONSISTENT BUN LEVEL OVER TIME[35]

Modality comparisons[21,22,35]

CONTINUOUS	INTERMITTENT	
CVVH	SLED	IHD
Intended to run 24 h/day • Slow but continuous urea clearance helps avoid spikes in BUN levels	Typically run in 6–12 h sessions delivered 3–7 times/week • Intermittent nature does not allow for continuous urea clearance, which could result in variable BUN levels	Typically run in 3–6 h sessions delivered 3–7 times/week • Intermittent nature does not allow for continuous urea clearance, which could result in variable BUN levels

UNLIKE IHD OR PIRRT, CRRT IS RUN ON MACHINES THAT DELIVER CONTINUOUS SOLUTE REMOVAL[22,35]

RRT MODALITY CONSIDERATIONS

Solution considerations

Typically, CRRT solutions are **commercially** prepared, while IHD and PIRRT use **local water sources** to prepare dialysate[29,36,37]

Preparing solutions on-line from local water sources **necessitates** water **treatment** and routine water **quality monitoring** to assure clean water standards are met[36–38]

Modality comparisons[29,36–40]

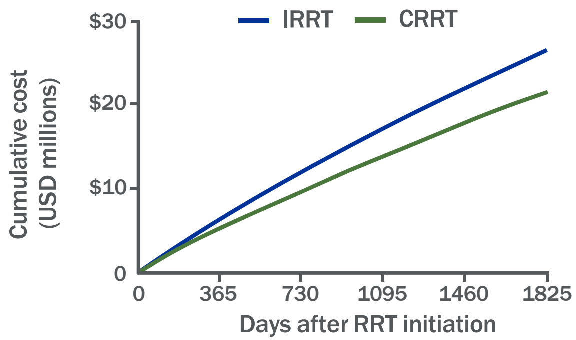
CONTINUOUS	INTERMITTENT	
CRRT	PIRRT	IHD
Because no on-line solutions are typically used, no water treatment systems are required • Monitoring water quality is not applicable	If a centralized water treatment system is unavailable in the ICU, individual water quality monitoring is necessary • If a centralized water treatment system is not used, staff need to monitor dialysate quality for individual patients • Disinfection requirements may limit treatment duration to <12 hours[41]	If a centralized water treatment system is unavailable in the ICU, individual water quality monitoring is necessary • If a centralized water treatment system is not used, staff need to monitor dialysate quality for individual patients

WATER TREATMENT AND QUALITY TESTING MAY CONTRIBUTE TO INCREASED MONITORING WHEN USING SOLUTIONS PREPARED ON-LINE FOR IHD and PIRRT[39,42]

Long-term cost considerations

Because initial RRT modality may impact the risk of chronic dialysis,[31] long-term costs of AKI may also be influenced by initial treatment modality[43]

Cumulative costs of dialysis dependence by initial AKI treatment modality[43,*]



MEAN 5-YEAR TOTAL COST/PATIENT OF AKI-D† WAS $37,780 FOR CRRT AS THE INITIAL MODALITY COMPARED WITH $39,448 FOR IRRT[43]

†Including cost of dialysis dependence. Cost in 2013 USD.

Modality comparisons[43]

CONTINUOUS	INTERMITTENT	
CRRT	PIRRT	IHD
Total costs may be lower due, in part, to a lower risk of chronic dialysis	Insufficient evidence to compare to CRRT or IHD	Total costs may be higher due, in part, to a higher risk of chronic dialysis

THE LONG-TERM COST OF AKI MAY BE LOWER FOR PATIENTS INITIALLY TREATED WITH CRRT COMPARED TO THOSE TREATED WITH IHD[43]

*Health outcomes and healthcare costs were simulated and averaged for a cohort of 1000 patients initiated on CRRT and a cohort of 1000 patients initiated on IRRT. All costs were inflated to 2013 USD.[43]

Equipment footprint and mobility considerations

Water treatment systems required for IHD and PIRRT add to physical **space** requirements and water lines may limit RRT mobility in ICUs without central water treatment systems[37,40]

In ICUs without central water treatment systems, portable **water treatment devices** may be necessary,[40] which can occupy as much as 0.13–0.16 m^2 of floor space[44,45]

Modality comparisons[36,37,40,41,46–49]

CONTINUOUS	INTERMITTENT	
CRRT	PIRRT	IHD
Because the CRRT machine is the only component that contributes to the therapy's physical footprint, treatment mobility may be increased • No space considerations for water treatment systems are necessary	Both the IHD machine and water treatment systems contribute to the therapy's physical footprint, which may impact treatment mobility in ICUs without central water treatment systems • In situations where a central water treatment system is not utilised, the greater physical footprint of the machine + water treatment system may impact ICU spacing	

WATER TREATMENT EQUIPMENT MAY ADD TO THE FOOTPRINT OF IHD AND PIRRT SYSTEMS, POTENTIALLY DECREASING TREATMENT MOBILITY AND IMPACTING SPACING CONSIDERATIONS[40,47–49]

SUMMARY

AKI is a **common** and **costly** condition among ICU patients,[1,17–19] and is associated with increased risks of **morbidity and mortality**[2–9]

Acute RRT is delivered as **either** a **continuous or** an **intermittent** therapy, each of which have unique characteristics, settings, and limitations[20,25–28]

Selection of RRT modality requires careful consideration of many patient- and ICU-specific factors[25,28]

- FLUID OVERLOAD AND HEMODYNAMIC INSTABILITY
- LONG-TERM CLINICAL OUTCOMES
- MACHINE AND PRESCRIPTION
- SOLUTIONS
- LONG-TERM COSTS
- EQUIPMENT FOOTPRINT AND MOBILITY

CRRT IS A PREFERRED RENAL REPLACEMENT THERAPY BY MANY CLINICIANS FOR PATIENTS WITH AKI WHO ARE HEMODYNAMICALLY UNSTABLE[25,28]

ACRONYMS/ABBREVIATIONS/REFERENCES

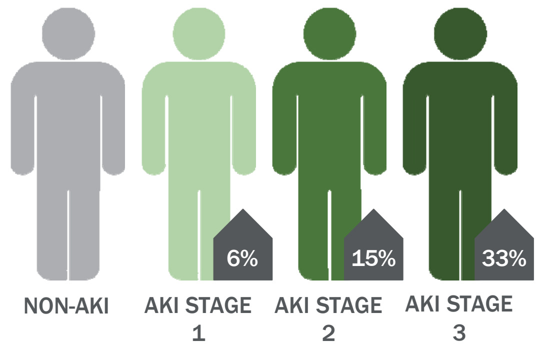
AKI, acute kidney injury; AKI-D, dialysis-requiring AKI; BUN, blood urea nitrogen; CI, confidence interval; CKD, chronic kidney disease; CRRT, continuous renal replacement therapy; CVVH, continuous veno-venous hemofiltration; CVVHD, continuous veno-venous hemodialysis; CVVHDF, continuous veno-venous hemodiafiltration; dL, decilitre; EDHF, extended daily hemofiltration; ESRD, end-stage renal disease; Feb, February; GI, gastrointestinal; h, hour; HD, hemodialysis; ICU, intensive care unit; IHD, intermittent hemodialysis; IRRT, intermittent renal replacement therapy; K^+, potassium ion; KDIGO, Kidney Disease Improving Global Outcomes; kg, kilogram; m^2, square meters; mg, milligram; MI, myocardial infarction; min, minute; mL, millilitre; PD, peritoneal dialysis; PIRRT, prolonged intermittent renal replacement therapy; NH_3, ammonia; Q_B, blood flow rate; Q_D, dialysis flow rate; RRT, renal replacement therapy; Sep, September; SLED, sustained or slow low-efficiency dialysis; SLEDD, sustained or slow low-efficiency daily dialysis; US, United States; USD, United States dollar; vs, versus; VTE, venous thromboembolism

1. Susantitaphong P, et al. *Clin J Am Soc Nephrol*. 2013;8:1482-1493.
2. Coca SG, et al. *Am J Kidney Dis*. 2009;53:961-973.
3. Ricci Z, et al. *Kidney Int*. 2008;73:538-546.
4. Chawla LS, et al. *Clin J Am Soc Nephrol*. 2014;9:448-456.
5. Brown JR, et al. *Ann Thorac Surg*. 2016;102:1482-1489.
6. Wu VC, et al. *J Am Soc Nephrol*. 2014;25:595-605.
7. Ishani A, et al. *J Am Soc Nephrol*. 2009;20:223-228.
8. Coca SG, et al. *Kidney Int*. 2012;81:442-448.
9. Wald R, et al. *JAMA*. 2009;302:1179-1185.
10. Bellomo R, et al. *Crit Care Med*. 2012;40:1753-1760.
11. Vaara ST, et al. *Crit Care*. 2012;16:R197.
12. Kaddourah A, et al. *N Engl J Med*. 2017;376:11-20.
13. Prowle JR, et al. *Nat Rev Nephrol*. 2010:6;107-115.
14. Zhang L, et al. *J Crit Care*. 2015;30:860.e7-13.
15. Bouchard J, et al. *Kidney Int*. 2009;76:422-427.
16. Heung M, et al. *Nephrol Dial Transplant*. 2012;27:956-961.
17. National Clinical Guideline Centre (UK). Acute Kidney Injury: Prevention, Detection and Management Up to the Point of Renal Replacement Therapy. 2013; NICE Clinical Guidelines, No. 169. Introduction.
18. Xu X, et al. *Clin J Am Soc Nephrol*. 2015;10:1510-1518.
19. Silver SA, et al. *J Hosp Med*. 2017;12:70-76.
20. Fleming GM. *Organogenesis*. 2011;7:2-12.
21. O'Reilly P, Tolwani A. *Crit Care Clin*. 2005;367-378.
22. Pannu N, Gibney RTN. *Ther Clin Risk Manag*. 2005;1:141-150.
23. Sun Z, et al. *Crit Care*. 2014;18:R70.
24. Kitchlu A, et al. *BMC Nephrol*. 2015;16:127.
25. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. *Kidney Int Suppl*. 2012;2:1-138.
26. Baxter Corporation. REVACLEAR Dialyzer Technology. 2017. Available from: http://www.baxter.ca/en_CA/assets/downloads/2017/Revaclear%20Spec%20Sheet%20Brochure%20English.pdf (accessed December 2018).
27. Fresenius Medical Care. Optiflux Dialyzers. 2016. Available from: http://www.fmcna-dialyzers.com/images/pdf/101046-Optiflux-Dialyzer_SpecSheet.pdf (accessed December 2018).
28. Ostermann M, et al. *Blood Purif*. 2016;42:224-237.
29. Murugan R, et al. *Blood Purif*. 2016;42:266-278.
30. Ostermann M. In: Bellomo R, et al (eds). 40 Years of Continuous Renal Replacement Therapy. *Contrib Nephrol*. 2018;194:51-59.
31. Wald R, et al. *Crit Care Med*. 2014;42:868-877.
32. Bell M, et al. *Intensive Care Med*. 2007;33:773-780.
33. Cartin-Ceba R, et al. *Intensive Care Med*. 2009;35:2087-2095.
34. Lin YF, et al. *Am J Surg*. 2009;198:325-332.
35. Liao Z, et al. *Artif Organs*. 2003;27:802-807.
36. Coulliette AD, Arduino MJ. *Semin Dial*. 2013;26:427-438.
37. Glorieux G, et al. *Nephrol Dial Transplant*. 2012;27:4010-4021.
38. Azar AT, Ahmad S. Hemodialysis Water Treatment System. In: Azar A (eds). Modelling and Control of Dialysis Systems. Studies in Computational Intelligence. 2013;404:347-378.
39. BC Renal Agency. Clinical Practice Standards and Procedures for Dialysis Water Quality: 2b: Endotoxin Testing of Dialysis Water. 2011. Available from:. http://www.bcrenalagency.ca/resource-gallery/Documents/2bEndotoxin-Testing-of-Dialysis-Water-Final_2012.pdf (accessed December 2018).
40. Bellomo R, et al. *Crit Care Resusc*. 2002;4:281-290.
41. Fresenius Medical Care. 2008K[2] Hemodialysis Machine Operator's Manual. 2016. Available from: https://fmcna.com/wp-content/uploads/documents/490136_Rev_J.pdf (accessed December 2018).
42. Food and Drug Administration. Quality Assurance Guidelines for Hemodialysis Devices. 1991. Available from: https://www.fda.gov/downloads/medicaldevices.deviceregulationandguidance/guidancedocuments/ucm073435.pdf (accessed December 2018).
43. Ethgen O, et al. *Nephrol Dial Transplant*. 2015;90:54-61.
44. Mar Cor Purification. 700 Series Portable. 2014. Available from: http://www.mcpur.com/main/library/12_brochures/1238642_(700).pdf (accessed December 2018).
45. Mar Cor Purification. Millenium HX. 2015. Available from: http://www.mcpur.com/main/library/12_brochures/3027573_(MHX).pdf (accessed December 2018).
46. Baxter Healthcare Corporation. The PRISMAFLEX System. 2016. Available from: https://www.baxter.com/sites/g/files/ebysai746/files/2017-11/Prismaflex-07.11-Brochure-New_Accts.pdf (accessed December 2018).
47. Ledebo I, Blankestijn PJ. *NDT Plus*. 2010;3:8-16.
48. Kasparek T, Rodriguez OE. *Clin J Am Soc Nephrol*. 2015;10:1061-1071.
49. Poeppel K, et al. *Vet Clin Small Anim*. 2011;177-191.

AKI is associated with substantial financial burden[17–19,*]

AKI status impacts daily costs[18]

PATIENTS WITH AKI HAVE SIGNIFICANTLY HIGHER DAILY COSTS COMPARED WITH PATIENTS WITHOUT AKI[18,†]



AKI is expensive even relative to other acute medical conditions[19]

Acute medical condition	Adjusted mean cost difference, in 2012 USD (95% CI)[a]
AKI-D[b]	11,016 (10,468, 11,564)
Sepsis	4822 (4696, 5068)
VTE	3782 (3611, 3953)
Acute pancreatitis	1802 (1676, 1929)
AKI[c]	1795 (1692, 1899)
Pneumonia	1705 (1584, 1825)
Stroke	1427 (1281, 1573)
MI	14 (–91, 119)
GI bleed	–860 (–961, –759)

[a]Compared with reference group without the condition of interest.
[b]Compared with patients without AKI. [c]Includes patients with dialysis-requiring AKI (AKI-D).

THE INCREMENTAL COST OF AKI-D OR AKI IS HIGHER THAN FOR MANY OTHER CONDITIONS FOUND IN HOSPITALIZED PATIENTS[19,‡]

WHILE EXPENDITURES MAY VARY BY COUNTRY,
AKI is a COSTLY CONDITION[17–19]

*Costs for hospitalisation due to AKI may vary from country to country.
†Multicentre, retrospective cohort study of 659,945 adult hospital admissions across central China in 2013.[18]
‡2012 multicentre, retrospective study of 29,763,649 adult US hospitalisations without ESRD.[19]

RRT for AKI: When? How much? Which Modality and When to stop

22-25th 2025 – Kuwait - Kuwait

Prof Basset El Essawy

M.B.B.CH, Msc, MD PhD FASN FAST

Maitre es Science Medical (Nephrology, France)

DIU Organ Transplantation (France)

DIU Pediatric Nephrology (France)

AFSA Clinical Nephrology and Hypetension (France)

C1 & C2 Immunology and Immunopathology (France)

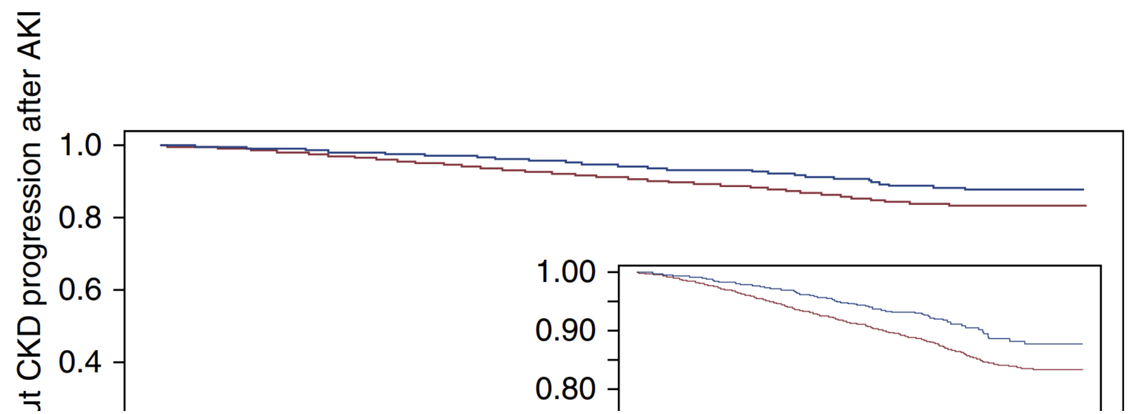
Diplome Des Etude Approfonde (DEA) Immunology of Organ transplantation (France)

Prof. of Internal Medicine and Nephrology AlAzhar University & X-Associae Dean Ras ALKhaimah School of Medicine.

Adunject Prof Sharjah, Ras Al-Khaimah & Fakeeh university Hospital

Scientist, TRC- Renal Division; Brigham and women's Hospital.

Professor of Medicine - Harvard Medical School- USA



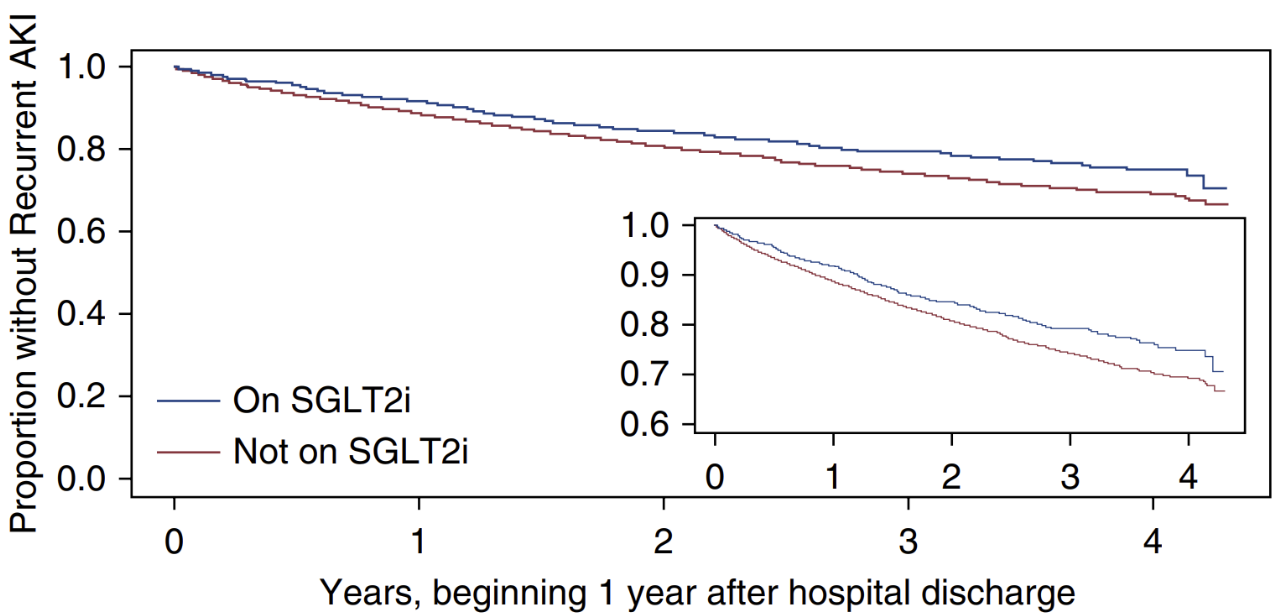
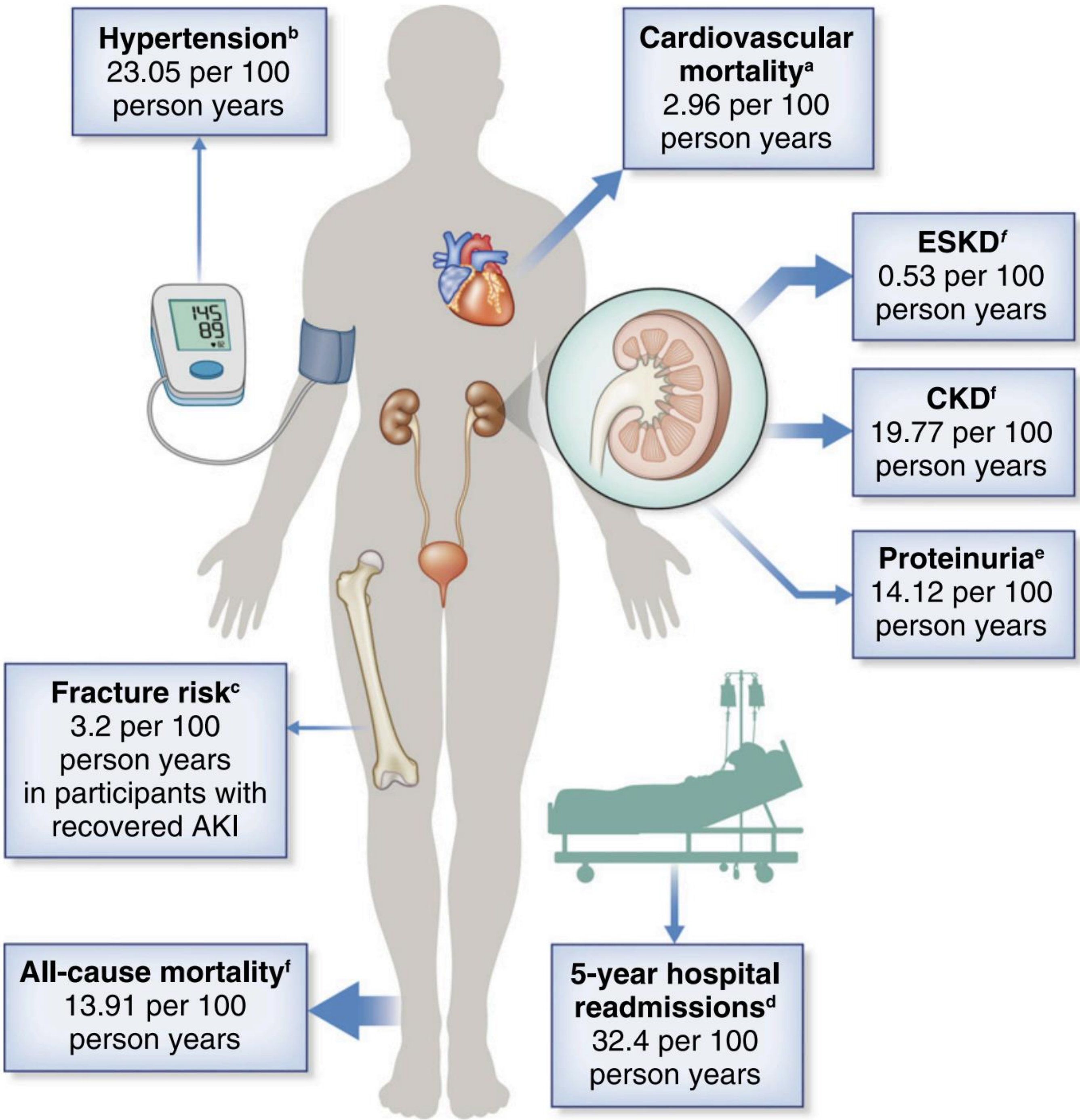


Figure 4. Kaplan–Meier curve for recurrent AKI progression after AKI by post-AKI SGLT2i use (N=10,036, P < 0.001).


Hypertension[b]
23.05 per 100
person years
Cardiovascular
mortality[a]
2.96 per 100
person years
145
89
ESKD[f]
0.53 per 100
person years
CKD[f]
19.77 per 100
person years
Proteinuria[e]
14.12 per 100
person years
Fracture risk[c]
3.2 per 100
person years
in participants with
recovered AKI
All-cause mortality[f]
13.91 per 100
person years
5-year hospital
readmissions[d]
32.4 per 100
person years

Downloaded from http://journals.lww.com/jasn by RcBvNY1uM1lVgyO1SxzNZJyCc4tvCEAoxU4D9d5uzkzbt9LK4CRg Piqb49ghXR+RX+IS539OrJG62ueOMpD5T5pskVNjpfF6lwgk9NNuHrk/oVzN+t9L1VYYprC84I9+lewYsW87axMf/iiBTZgU3ZnJ HVamR/Cj on 01/24/2025

Table 3. Subgroup Analysis for the Association Between Acute Kidney Injury and Acute Myocardial Infarction

Stratification	Number of Studies	Setting[a]	Relative Risk	95% CI	I^2, %	P Value
AMI – setting						
CIN	5	-	1.43	1.18 to 1.75	48	0.93
Peri-operative	3	-	1.39	1.08 to 1.77	47	
Other	2	-	1.31	0.82 to 2.09	55	
AMI – recovery						
Complete recovery	2	CIN (1) Peri-op (1) Other (0)	1.14	0.87 to 1.48	0	0.12
Partial or no recovery	2	CIN (1) Peri-op (1) Other (0)	1.55	1.16 to 2.07	0	
AMI – severity						
Mild AKI	4	CIN (2) Peri-op (2) Other (0)	1.45	1.28 to 1.65	18	0.64
Moderate to severe AKI	4	CIN (2) Peri-op (2) Other (0)	1.53	1.27 to 1.84	0	
AMI – median proportion baseline CHF (min–max)						
0.04 (0–0.06)	4	CIN (0) Peri-op (2) Other (2)	1.42	1.19 to 1.69	38	0.91
0.15 (0.10–0.17)	3	CIN (2) Peri-op (1) Other (0)	1.40	1.17 to 1.67	0	
AMI – median proportion baseline IHD (min–max)						
0.12 (0.10–0.72)	3	CIN (1) Peri-op (2) Other (0)	1.32	1.10 to 1.57	2	0.44
1 (1–1)	7	CIN (4) Peri-op (1) Other (2)	1.45	1.23 to 1.71	51	
AMI – median proportion baseline stroke (min–max)						
0.04 (0–0.06)	4	CIN (1) Peri-op (2) Other (1)	1.37	1.14 to 1.65	62	0.49
0.10 (0.07–0.95)	4	CIN (3) Peri-op (1) Other (0)	1.59	1.08 to 2.34	47	
AMI – median proportion baseline CKD (min–max)						
8.8 (0–0.18)	5	CIN (3) Peri-op (2) Other (0)	1.32	1.09 to 1.61	43	0.45
0.28 (0.20–1)	4	CIN (2) Peri-op (1) Other (1)	1.47	1.23 to 1.75	37	

AMI, acute myocardial infarction; CIN, contrast induced nephropathy; peri-op, peri-operative or intensive care unit.
[a]Number of studies is provided in brackets.

Table 2. Timing of initiation of dialysis and its association with mortality

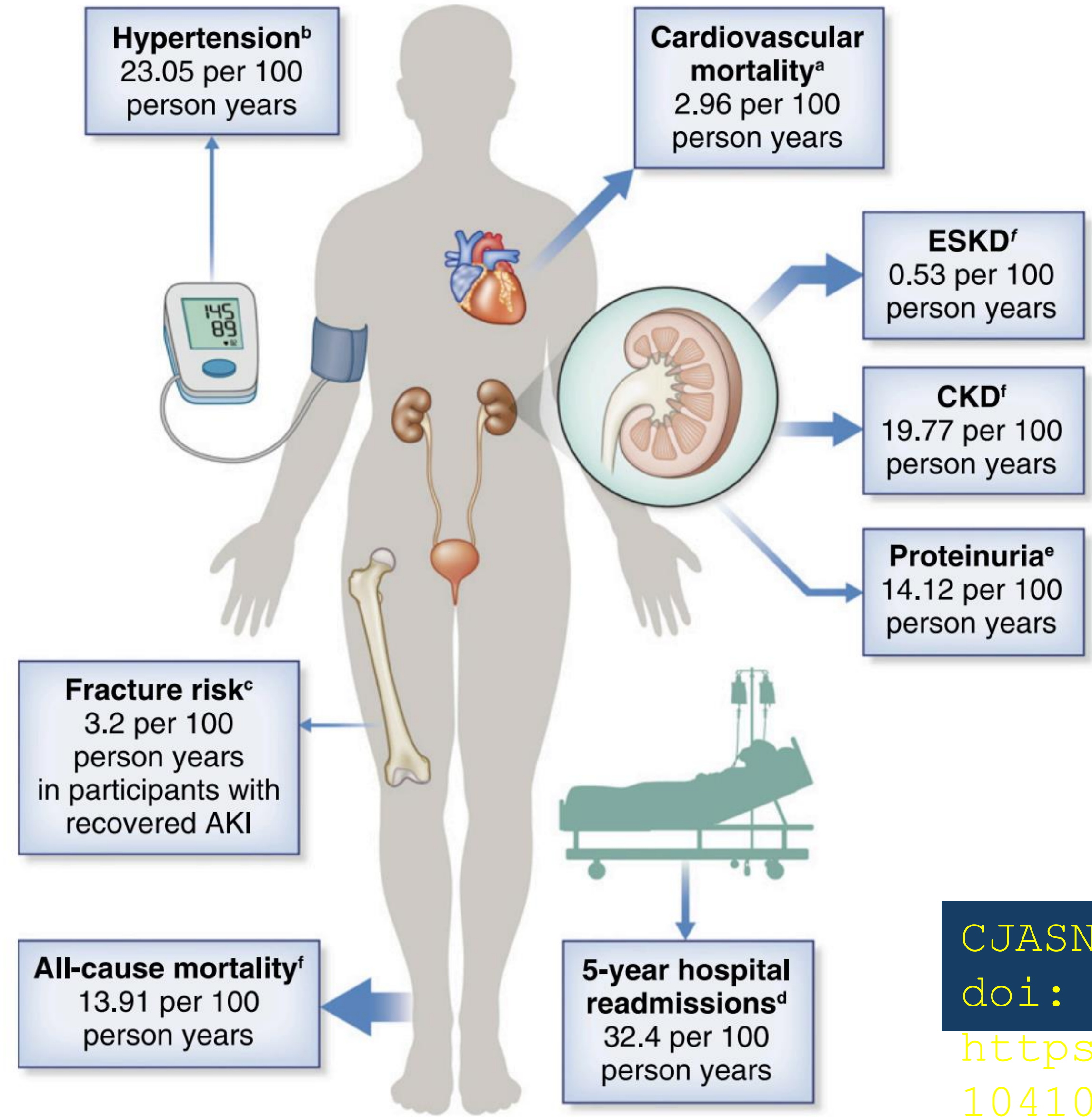
Study	Reference	Year	No. of Patients	Study Design	Predialysis BUN (mg/dl)		Mortality (%)	
					Early	Late	Early	Late
Parsons *et al.*	(6)	1961	33	Cohort with historical control	120 to 150	200	25	88
Fischer *et al.*	(7)	1966	162	Cohort with historical control	152	231	51	77
Kleinknecht *et al.*[a]	(8)	1972	320	Cohort with historical control	93	164	29	42
Conger[a]	(9)	1975	18	Case-control	50	120	20	64
Gettings *et al.*	(10)	1999	100	Retrospective cohort	42.6	94.5	61	80
Bouman *et al.*	(16)	2002	65	Randomized trial	48	105	31	25

[a] Case patients and control subjects differed with respect to both the timing of initiation of dialysis and the dose of dialysis delivered.

Dr Basset El Essawy

aelessawy@bwh.harvard.edu
belessawy@azhar.edu.eg
Abdel.essawy@rakmhsu.ac.ae
belessawy@gmail.com

THANK YOU



CJASN 15: 423-429,
doi:
https://doi.org/10.
10410919

Figure 1. | **Summary of reported event rates for long term outcomes after AKI.** The size of the arrows are a representation of the number of currently available studies reporting this outcome. It should also be noted that, although the rates are reported in the same units (events/100-person-years), data are taken from different sources and are therefore not directly comparable. The superscript letters indicate the sources of the event rates: [a]Odutayo *et al.* (8); [b]Hsu *et al.* (63); [c]Wang *et al.* (64); [d]Brown *et al.* (9); [e]Horne *et al.* (11); and [f]See *et al.* (5).