KDIGO Guidelines on AKI

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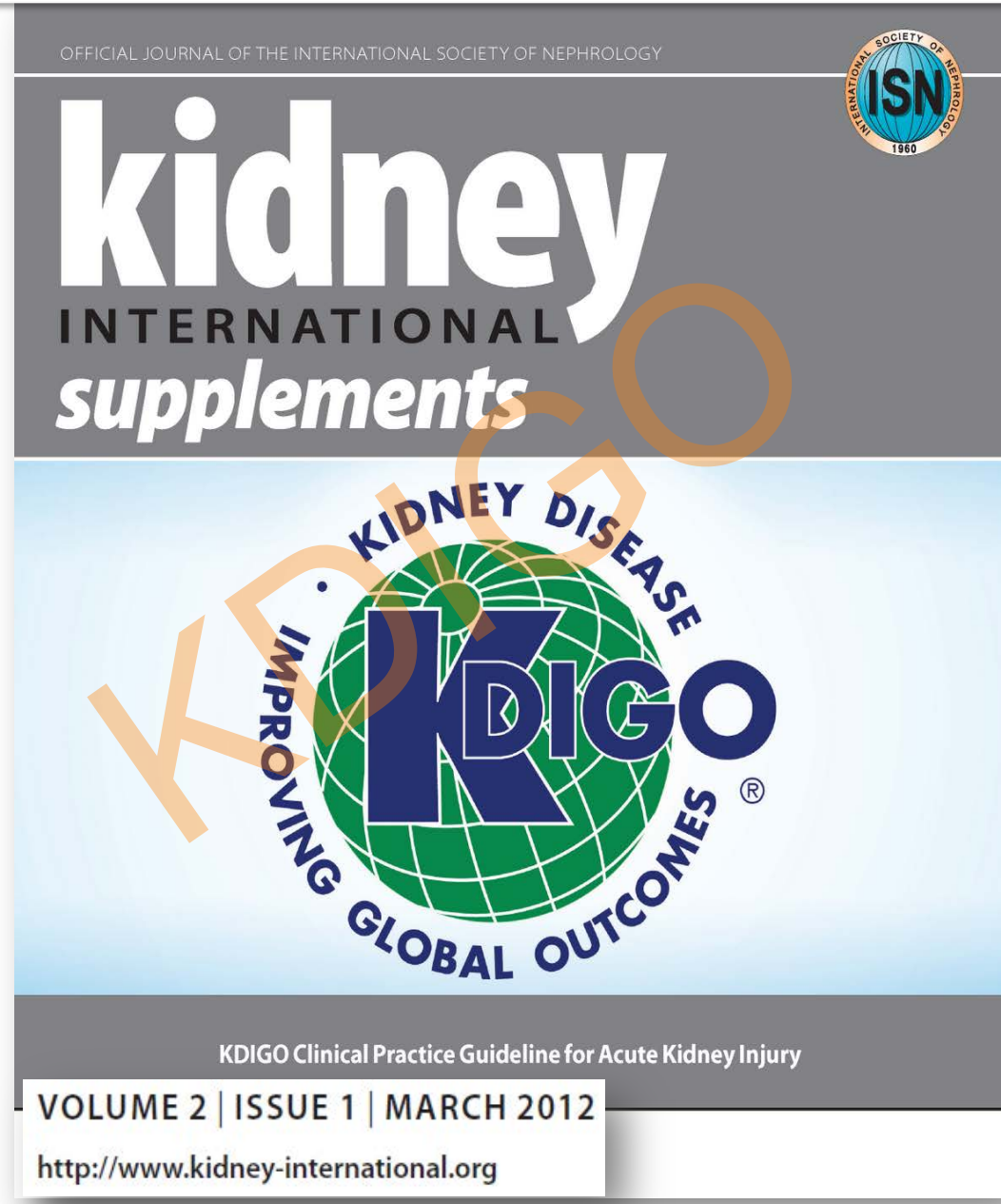
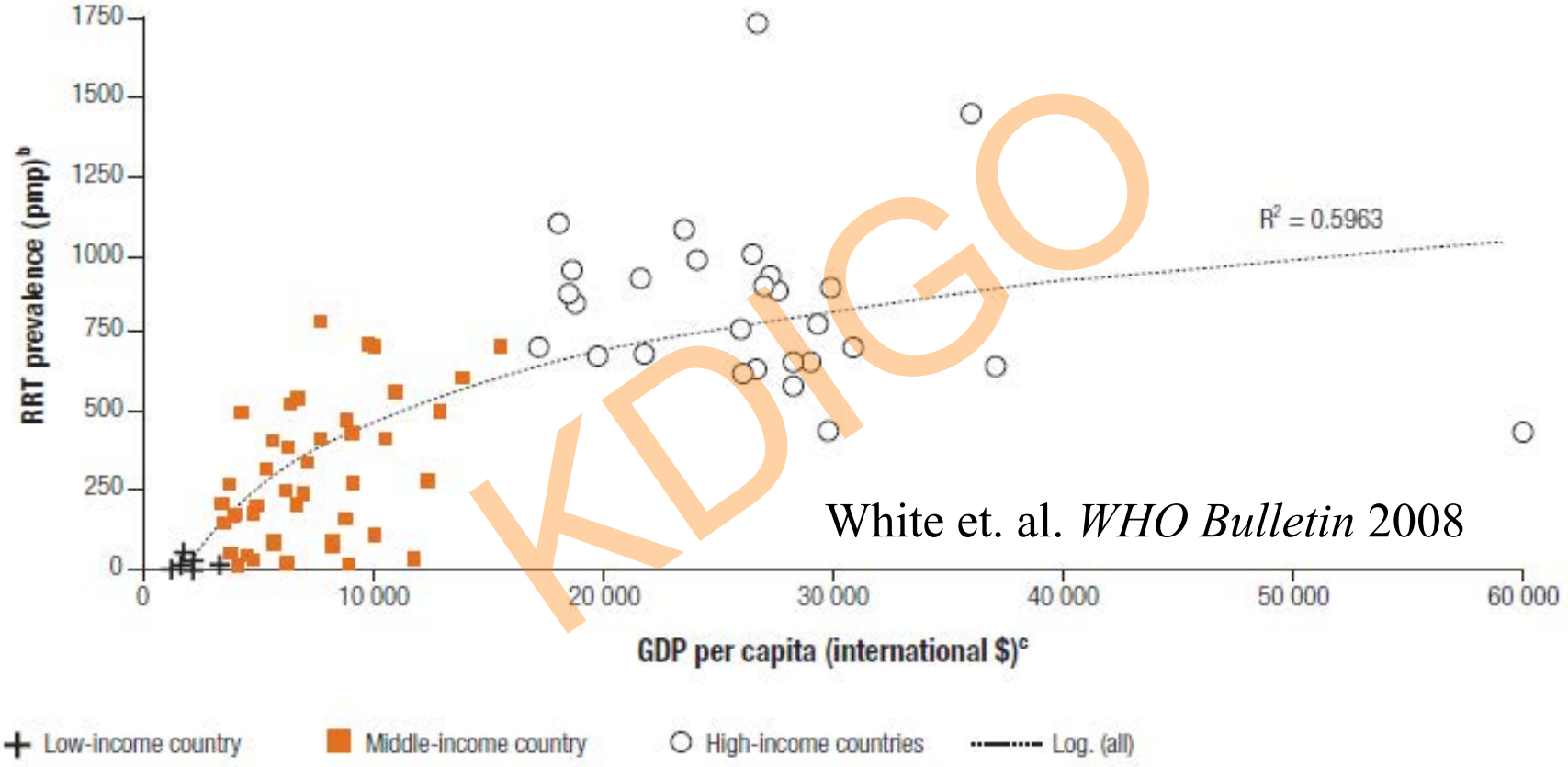
www.kdigo.org/home/guidelines/acute-kidney-injury



KDIGO – AKI Guidelines

Global context



White et. al. *WHO Bulletin* 2008

Estimated ¼ to ½ of people who might benefit from RRT currently have access to it

No reliable estimate treatment gap for AKI

Introduction

- KDIGO Co-Chairs appointed 2 Co-Chairs of the Work Group
- Assembled experts in nephrology, critical care medicine, internal medicine, pediatrics, cardiology, radiology, infectious diseases and epidemiology
- Every effort made to avoid actual or reasonably perceived conflicts of interest of a member of the Work Group
- The Work Group was supported by
  - Evidence Review Team at Tufts Medical Center, Boston
  - KDIGO staff (infrastructure)

KDIGO – AKI Guidelines

Introduction

- Work Group worked for two years
- Divided in sub-groups
- Over 18,000 citations were screened
- Four face to face meetings and teleconferences
- Finalized by the Co-Chairs of the Work Group
- Work-Group agreement on the final document
- Publication in KI in March 2012

The final Clinical Practice Guideline document was based upon the best information available as of February 2011

KDIGO – AKI Guidelines

Rating the Guideline Recommendations

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Strength

Grade*	Implications: Clinicians
Level 1 "We recommend"	Most patients should receive the recommended course of action.
Level 2 "We suggest"	Different choices will be appropriate for different patients. Each patient needs help to arrive at a management decision consistent with her or his values and preferences.

Quality

Grade	Quality of evidence
A	High
B	Moderate
C	Low
D	Very low

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KDIGO – AKI Guidelines

Rating the Guideline Recommendations

- 61 graded and 26 ungraded statements
- 11 (18%) recommendations graded A
- 20 (32.8%) were B
- 23 (37.7%) were C
- 7 (11.5%) were D
- 22 (36.1%) recommendations graded 1
- 39 (63.9%) graded 2
- Only 9 (14.8%) recommendations graded 1A
- 10 (16.4%) were 1B
- 3 (4.9%) were 1C and 0 (0%) were 1D

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KDIGO – AKI Guidelines

Document structure

- Summary of recommendation statements
- Section 1 – Introduction and methodology
- Section 2 – AKI definition
- Section 3 – Prevention and treatment of AKI
- Section 4 – Contrast-induced AKI
- Section 5 – Dialysis interventions for treatment of AKI

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KDIGO – AKI Definition

Section 2.1 - Definition

2.1.1: AKI is defined as any of the following:

- Increase in SCr by ≥ 0.3 mg/dl (≥ 26.5 µmol/l) within 48 hours; or
- Increase in SCr to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- Urine volume <0.5 ml/kg/h for 6 hours

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KDIGO – AKI Definition

Section 2.1 - Definition

2.1.2: AKI is staged for severity according to the following criteria (Table 2)

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline OR ⩾0.3 mg/dl (⩾26.5 μmol/l) increase	<0.5 ml/kg/h for 6–12 hours
2	2.0–2.9 times baseline	<0.5 ml/kg/h for ⩾12 hours
3	3.0 times baseline OR Increase in serum creatinine to ⩾4.0 mg/dl (⩾353.6 μmol/l) OR Initiation of renal replacement therapy OR, In patients <18 years, decrease in eGFR to <35 ml/min per 1.73 m^2	<0.3 ml/kg/h for ⩾24 hours OR Anuria for ⩾12 hours

KDIGO – AKI Definition

RIFLE

Increase in SCr X 1.5 or GFR decrease > 25%

AKIN

Increase in SCr ≥0.3 mg/dl or increase ≥150% to 200% (1.5- to 2-fold) from baseline within 48 h

KDIGO

Increase in SCr by ≥0.3 mg/dl within 48 h or to ≥1.5 times baseline, known or presumed to have occurred within the prior 7 d

KDIGO – AKI Definition Validation

- Prospective study 1,050 people in first 7 days after hospitalisation with AMI

- AKI defined by RIFLE in 14.8% and KDIGO 36.6%

- Both RIFLE and KDIGO associated with increased 30-day and one-year death rate

People diagnosed as not having AKI by RIFLE, but AKI by KDIGO had increased risk of death

Rodrigues et. al. *PLOS One* 2013

KDIGO – AKI Definition

Section 2.1 - Definition

2.1.3: The cause of AKI should be determined whenever possible (Not Graded)

Table 5 | Causes of AKI and diagnostic tests

Selected causes of AKI requiring immediate diagnosis and specific therapies	Recommended diagnostic tests
Decreased kidney perfusion	Volume status and urinary diagnostic indices
Acute glomerulonephritis, vasculitis, interstitial nephritis, thrombotic microangiopathy	Urine sediment examination, serologic testing and hematologic testing
Urinary tract obstruction	Kidney ultrasound

Identification of nephrotoxic drugs

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KDIGO AKI – Risk Assessment

Section 2.2 - Risk assessment

2.2.1: We recommend that patients be stratified for risk of AKI according to their susceptibilities and exposures (1B)

2.2.2: Manage patients according to their susceptibilities and exposures to reduce the risk of AKI) (Not Graded)

2.2.3: Test patients at increased risk for AKI with measurements of SCr and urine output to detect AKI. (Not Graded) Individualize frequency and duration of monitoring based on patient risk and clinical course. (Not Graded)

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KDIGO AKI – Risk Assessment

Section 2.2 - Risk assessment

Table 6 | Causes of AKI: exposures and susceptibilities for non-specific AKI

Exposures	Susceptibilities
Sepsis	Dehydration or volume depletion
Critical illness	Advanced age
Circulatory shock	Female gender
Burns	Black race
Trauma	CKD
Cardiac surgery (especially with CPB)	Chronic diseases (heart, lung, liver)
Major noncardiac surgery	Diabetes mellitus
Nephrotoxic drugs	Cancer
Radiocontrast agents	Anemia
Poisonous plants and animals	

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KDIGO AKI – Evaluation and General Management

Section 2.3 - Evaluation and general management of pts with and at risk for AKI

2.3.1: Evaluate patients with AKI promptly to determine the cause, with special attention to reversible causes. (Not Graded)

2.3.2: Monitor patients with AKI with measurements of SCr and urine output to stage the severity, according to Recommendation 2.1.2. (Not Graded)

2.3.3: Manage patients with AKI according to the stage (see Figure 4) and cause. (Not Graded)

2.3.4: Evaluate patients 3 months after AKI for resolution, new onset, or worsening of pre-existing CKD. (Not Graded)

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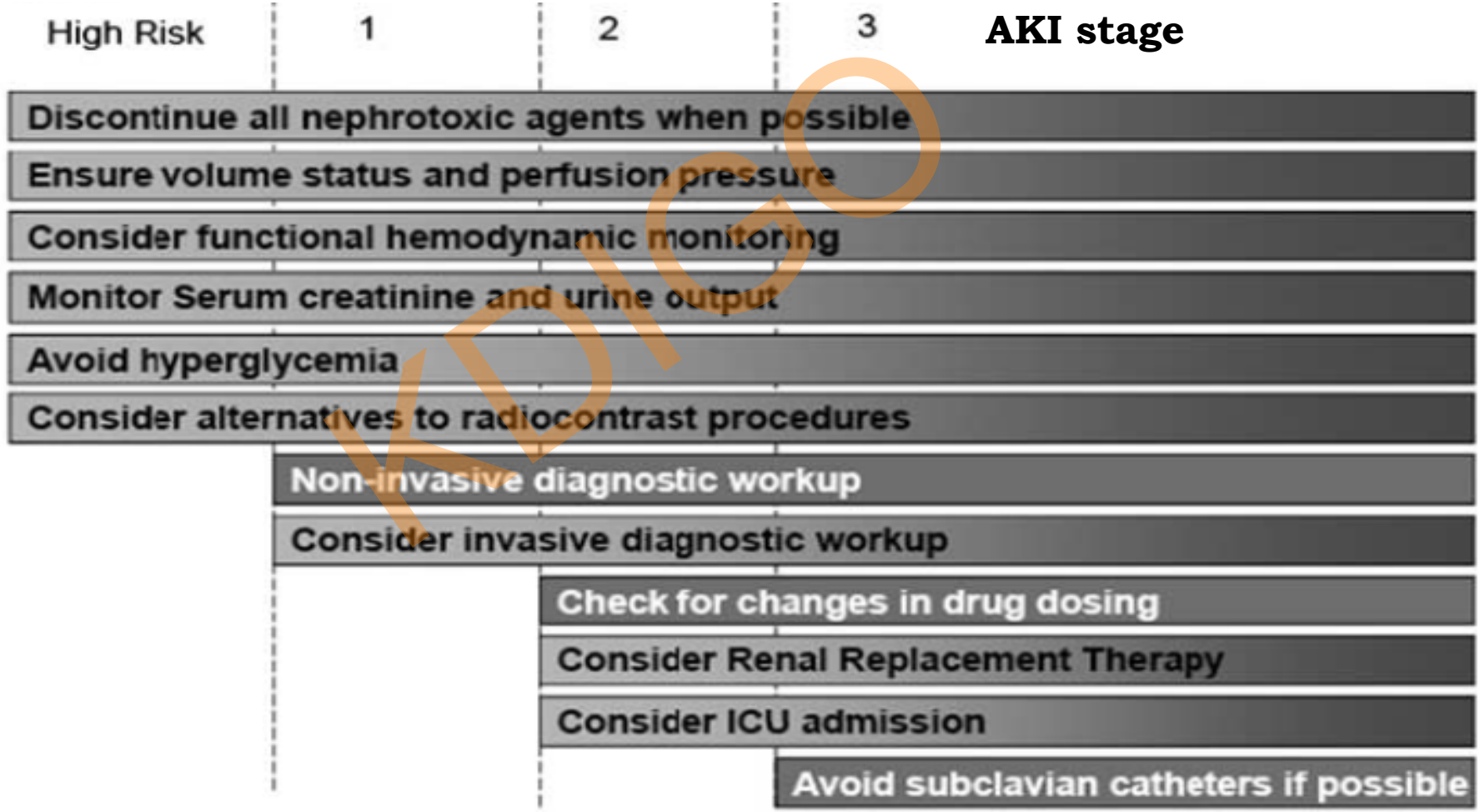
KDIGO AKI – Evaluation and General Management

Section 2.3 - Evaluation and general management of pats with and at risk for AKI



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Chapter 3.1: Fluids

3.1.1: In the absence of hemorrhagic shock, we suggest using isotonic crystalloids rather than colloids (albumin or starches) as initial management for expansion of intravascular volume in patients at risk for AKI or with AKI. (2B)

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Prevention and Treatment of AKI – Fluids

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Hydroxyethyl Starch 130/0.42 versus Ringer's Acetate in Severe Sepsis

HES 130/0.42: 398 pts

Ringer acetate: 400 pts

Perner et al, NEJM 2012

Prevention and Treatment of AKI - Fluids

HES vs. Ringer Acetate

Renal Replacement Therapy

Ringer acetate: 65 pts (16%)

HES 130/0.42: 87 pts (22%)

RR 1.35; 95% CI 1.01 to 1.80; p = 0.04

Perner et al, NEJM 2012

Prevention and Treatment of AKI – Fluids

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Hydroxyethyl Starch or Saline for Fluid Resuscitation in Intensive Care

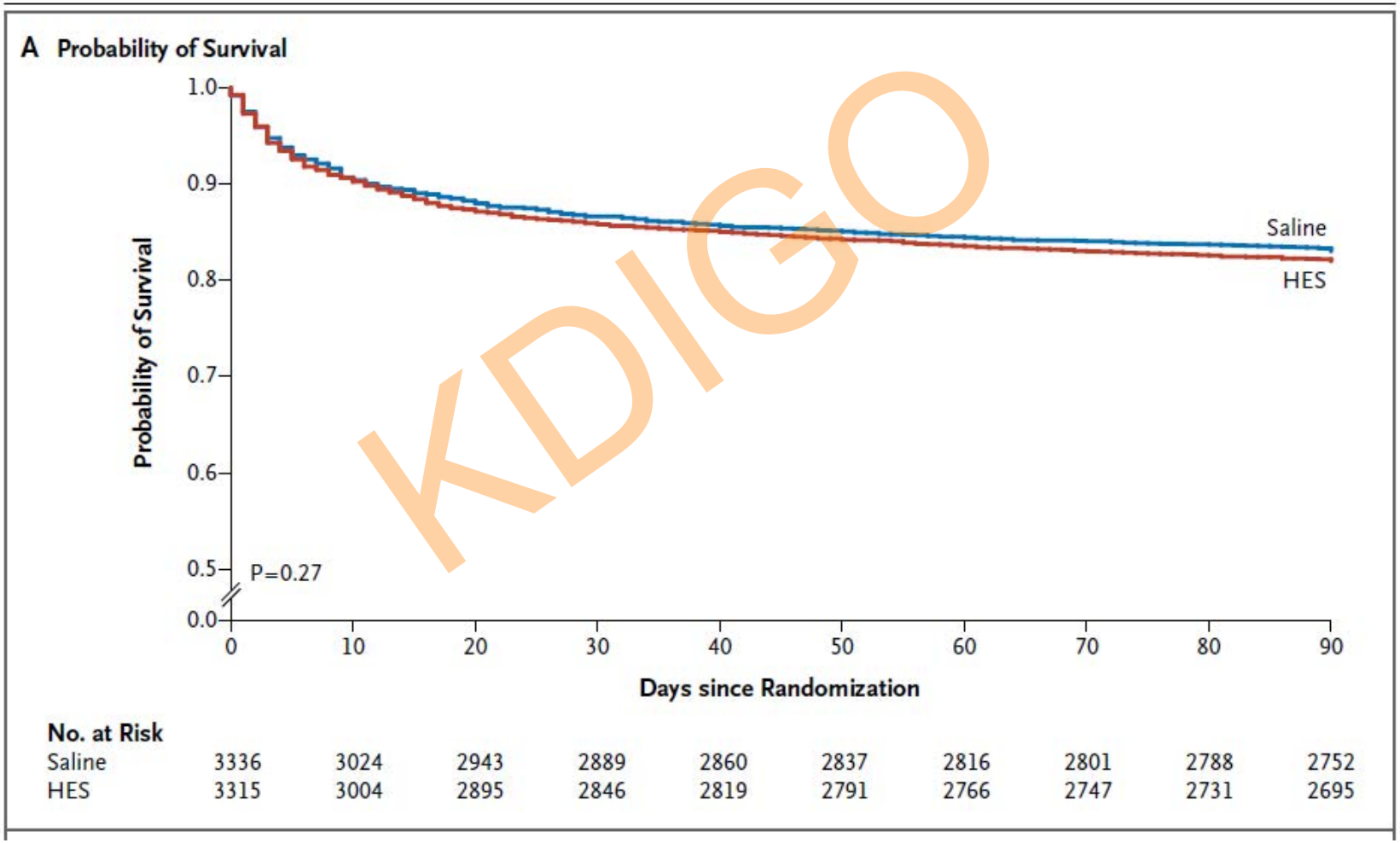
HES 130/0.4: 3,315 pts

Saline: 3,336 pts

Myburgh et al, NEJM 2012

Prevention and Treatment of AKI – Fluids

HES vs. Saline in ICU pts

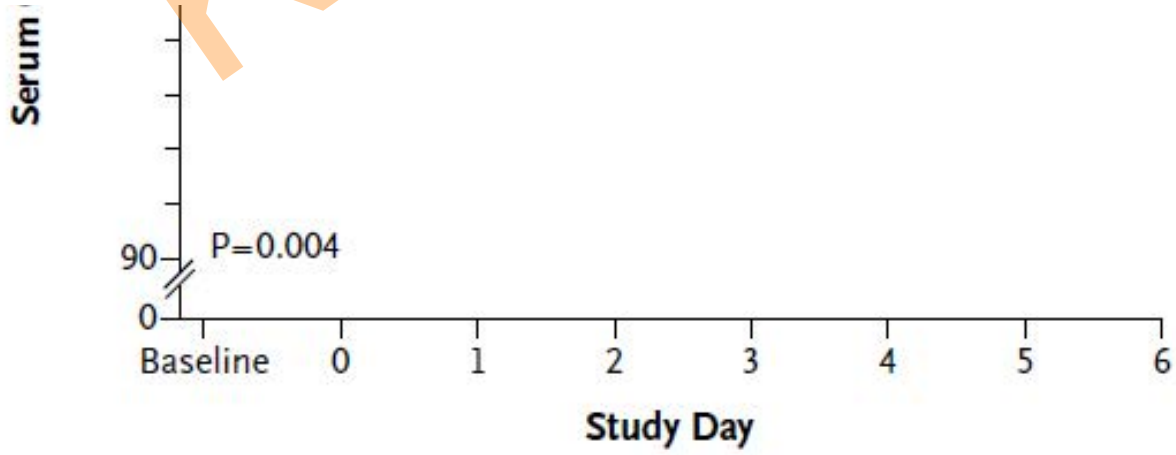


Myburgh et al, NEJM 2012

Prevention and Treatment of AKI – Fluids

HES vs. Saline in ICU pts

RRT 7.0% in the HES group and 5.8% in the saline group (RR, 1.21; 95% CI, 1.00 to 1.45; P = 0.04)



Myburgh et al, NEJM 2012

Vassopressors

3.1.2: We recommend the use of vasopressors in conjunction with fluids in patients with vasomotor shock with, or at risk for, AKI (1C)

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Prevention and Treatment of AKI - Vasopressors

Does perioperative hemodynamic optimization protect renal function in surgical patients? A meta-analytic study

Nicola Brienza, MD, PhD; Maria Teresa Giglio, MD; Massimo Marucci, MD; Tommaso Fiore, MD

Data Sources, Study Selection, Data Extraction: **A systematic literature review, using MEDLINE, EMBASE, and The Cochrane Library databases through January 2008 was conducted and 20 studies met the inclusion criteria (4220 participants). Data synthesis was obtained by using odds ratio (OR) with 95% confidence interval (CI) by random-effects model.**

Crit Care Med 2009 Vol. 37, No. 6

Prevention and Treatment of AKI - Vasopressors

fluids

Study or Subgroup	Treatment Events	Total	Control Events	Total	Odds Ratio M-H, Random, 95% CI
Chytra [33]	0	80	1	82	0.34 [0.01, 8.41]
Gan [25]	2	50	4	50	0.48 [0.08, 2.74]
McKendry [26]	1	89	3	85	0.31 [0.03, 3.05]
Noblett [29]	0	51	2	52	0.20 [0.01, 4.19]
Wakeling [44]	3	64	2	64	1.52 [0.25, 9.45]
Subtotal (95% CI)		**334**		**333**	**0.55 [0.20, 1.47]**
Total events	6		12		

Heterogeneity: Tau² = 0.00; Chi² = 2.00, df = 4 (P = 0.74); I² = 0%

Test for overall effect: Z = 1.20 (P = 0.23)

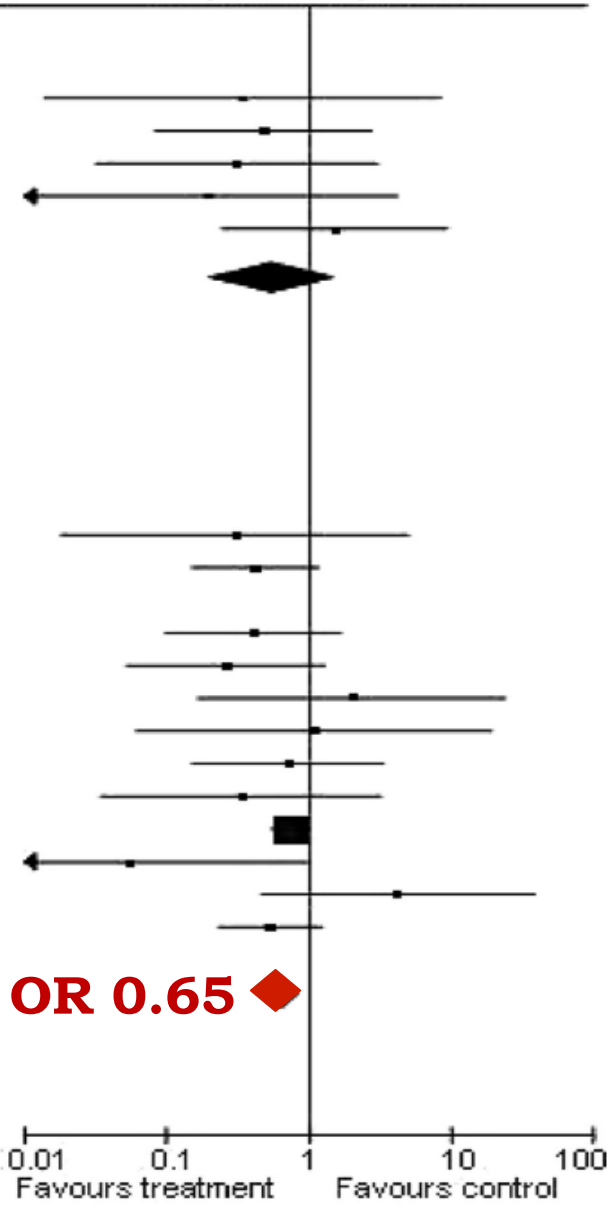
fluids & inotropes

Bender [28]	0	51	0	53	Not estimable
Berlauk [30]	1	68	1	21	0.30 [0.02, 4.99]
Bishop [32]	6	50	16	65	0.42 [0.15, 1.16]
Bonazzi [31]	0	50	0	50	Not estimable
Boyd [34]	3	53	7	54	0.40 [0.10, 1.65]
Donati [36]	2	68	7	67	0.26 [0.05, 1.30]
Lobo [38]	2	19	1	18	2.00 [0.17, 24.19]
Malhotra [37]	1	13	1	14	1.08 [0.06, 19.31]
Pearse [40]	3	62	4	60	0.71 [0.15, 3.32]
Polonen [41]	1	196	3	197	0.33 [0.03, 3.22]
Sandham [42]	70	941	95	965	0.74 [0.53, 1.02]
Shoemaker [39]	0	28	14	60	0.06 [0.00, 0.98]
Valentine [43]	4	60	1	60	4.21 [0.46, 38.86]
Wilson [45]	16	92	13	46	0.53 [0.23, 1.24]
Ziegler [46]	0	32	0	40	Not estimable
Subtotal (95% CI)		**1783**		**1770**	**0.65 [0.50, 0.85]**
Total events	109		163		

Heterogeneity: Tau² = 0.00; Chi² = 10.33, df = 11 (P = 0.50); I² = 0%

Test for overall effect: Z = 3.17 (P = 0.002) **P=0.002**

OR 0.65



Brienza et al, Crit Care Med 2009

Prevention and Treatment of AKI - Vasopressors

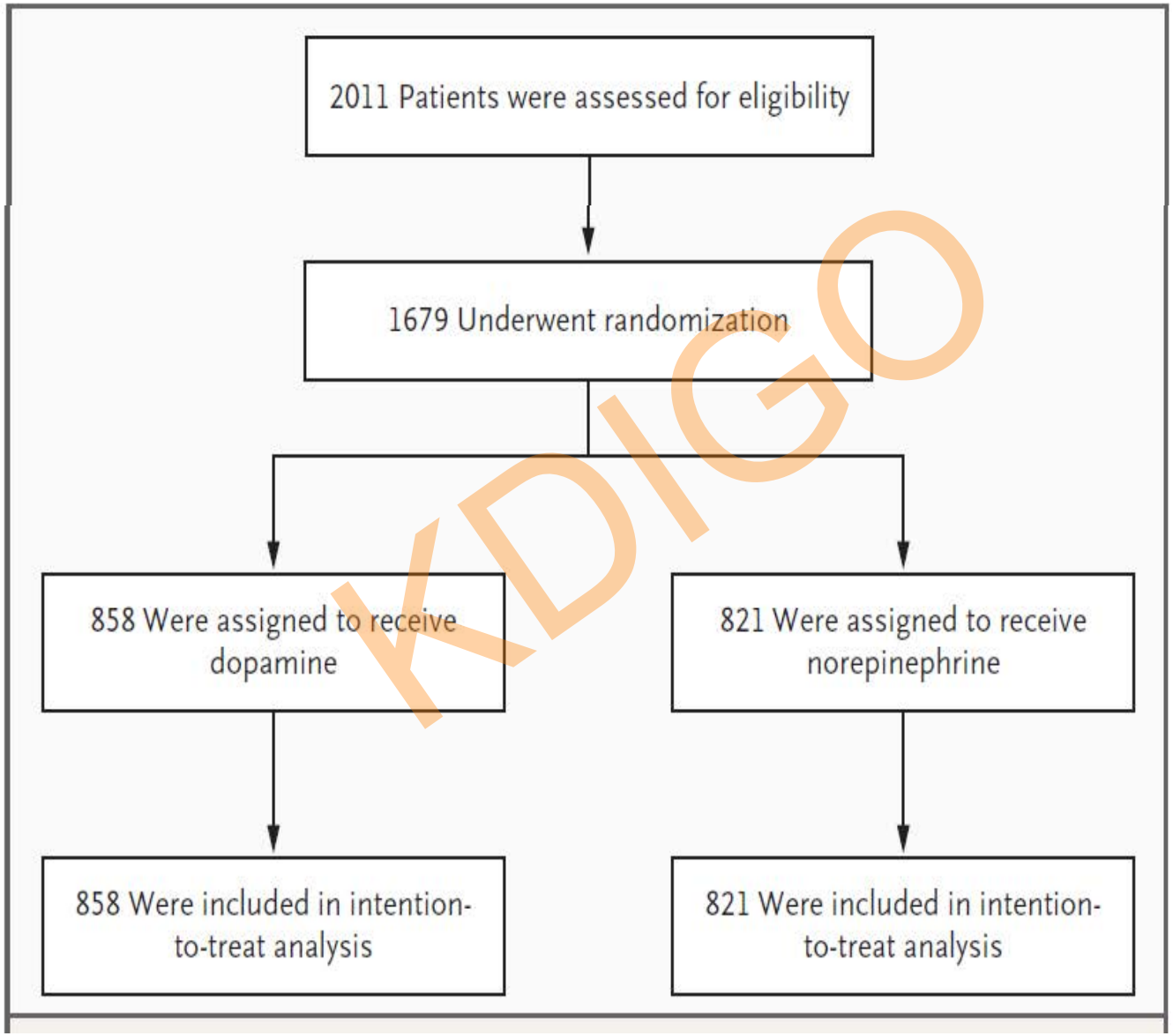
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 4, 2010 VOL. 362 NO. 9

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., and Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators*

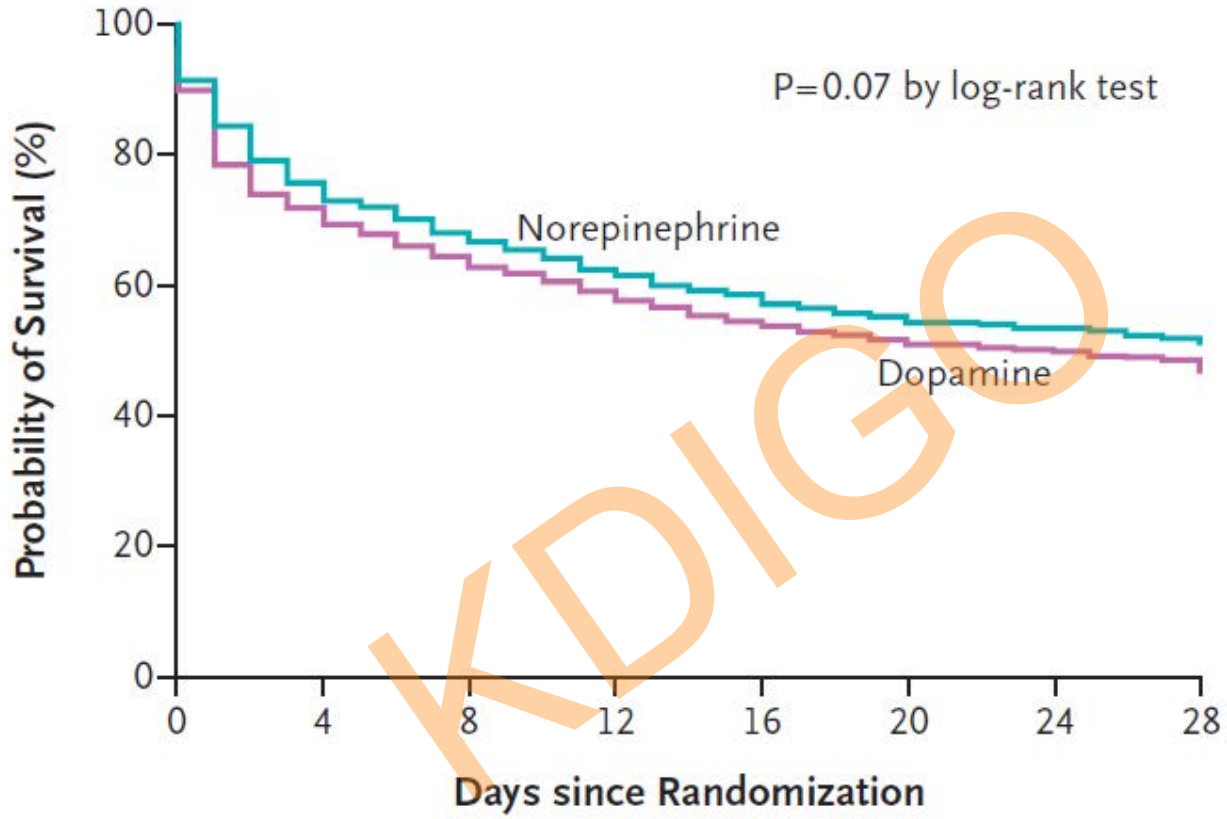
Prevention and Treatment of AKI - Vasopressors



NEJM

de Backer et al, NEJM 2010

Prevention and Treatment of AKI - Vasopressors



No. at Risk								
Norepinephrine	821	617	553	504	467	432	412	394
Dopamine	858	611	546	494	452	426	407	386

Figure 2. Kaplan–Meier Curves for 28-Day Survival in the Intention-to-Treat Population.

de Backer et al, NEJM 2010

Prevention and Treatment of AKI - Vasopressors

Nora: Tendency to Less Need for Renal Support

Table 3. Secondary Outcomes and Adverse Events.*

Variable	Dopamine (N=858)	Norepinephrine (N=821)	P Value
Support-free days through day 28			
Vasopressors not needed			
Trial drug	11.0±12.1	12.5±12.1	0.01
Open-label vasopressors	12.6±12.5	14.2±12.3	0.007
Mechanical ventilation not needed	8.5±11.2	9.5±11.4	0.13
Renal support not needed	12.8±12.4	14.0±12.3	0.07
Intensive care not needed	8.1±10.3	8.5±10.3	0.43

NEJM

de Backer et al, NEJM 2010

KDIGO - Prevention and Treatment of AKI

Protocolized Management

3.1.3: We suggest using protocol-based management of hemodynamic and oxygenation parameters to prevent development or worsening of AKI in high-risk patients in the perioperative setting (2C) or in patients with septic shock (2C)

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Prevention and Treatment of AKI – Protocolized Therapy

EARLY GOAL-DIRECTED THERAPY IN THE TREATMENT OF SEVERE SEPSIS AND SEPTIC SHOCK

EMANUEL RIVERS, M.D., M.P.H., BRYANT NGUYEN, M.D., SUZANNE HAVSTAD, M.A., JULIE RESSLER, B.S., ALEXANDRIA MUZZIN, B.S., BERNHARD KNOBLICH, M.D., EDWARD PETERSON, PH.D., AND MICHAEL TOMLANOVICH, M.D., FOR THE EARLY GOAL-DIRECTED THERAPY COLLABORATIVE GROUP*

NEJM 2001

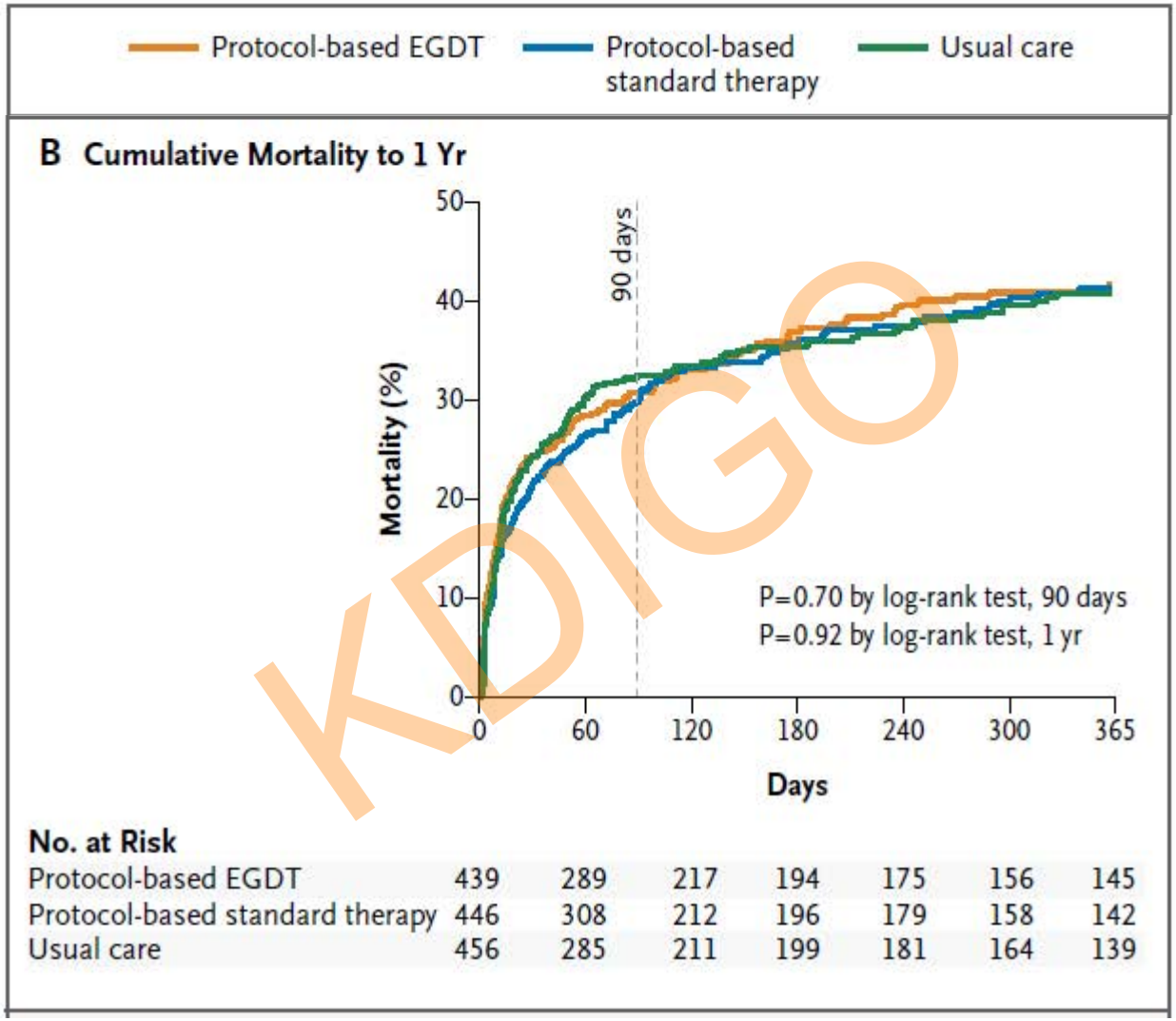
- EGDT (a resuscitation strategy based upon achieving specific physiologic end-points within 6 hours of hospital admission) vs standard
- EGDT
  - In-hospital mortality 30.5% vs 46.5%
  - Lower APACHE II scores
- Did not look at AKI outcomes
- Criticisms – open-label, small, single-centre study

Prevention and Treatment of AKI – Protocolized Therapy

- Guidelines note 3 large multi-centre trials underway in US (ProCESS), UK (PRoMISe) and Australia (ARISE)
- ProCESS study reported online *NEJM* last month
  - Study in 31 EDs, enrolled 1341 patients
  - Randomised to protocol-based EGDT, protocol-based standard therapy, or usual care

No significant differences in 90-day mortality, 1-year mortality, or the need for organ support

Prevention and Treatment of AKI – Protocolized Therapy



KDIGO - Supportive management of AKI

Glycemic control

3.3.1: In critically ill pts, we suggest insulin therapy targeting plasma glucose 110 – 149 mg/dl (6.1 – 8.3mmol/L) (2C)

- Tight glycemic control
  - Greater incidence hypoglycaemia
  - No clear benefit in terms of mortality or RRT

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KDIGO - Supportive management of AKI

Nutritional Support

3.3.2: We suggest achieving a total energy intake of 20–30 kcal/kg/d in patients with any stage of AKI. (2C)

3.3.3: We suggest to avoid restriction of protein intake with the aim of preventing or delaying initiation of RRT. (2D)

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KDIGO - Supportive management of AKI

Nutritional Support

3.3.4: We suggest administering 0.8–1.0 g/kg/d of protein in non catabolic AKI patients without need for dialysis (2D), 1.0–1.5 g/kg/d in patients with AKI on RRT (2D), and up to a maximum of 1.7 g/kg/d in patients on CRRT and in hypercatabolic patients. (2D)

3.3.5: We suggest providing nutrition preferentially via the enteral route in patients with AKI. (2C)

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KDIGO - Supportive management of AKI

Use of diuretics in AKI

3.4.1: We recommend not using diuretics to prevent AKI. (1B)

3.4.2: We suggest not using diuretics to treat AKI, except in the management of volume overload. (2C)

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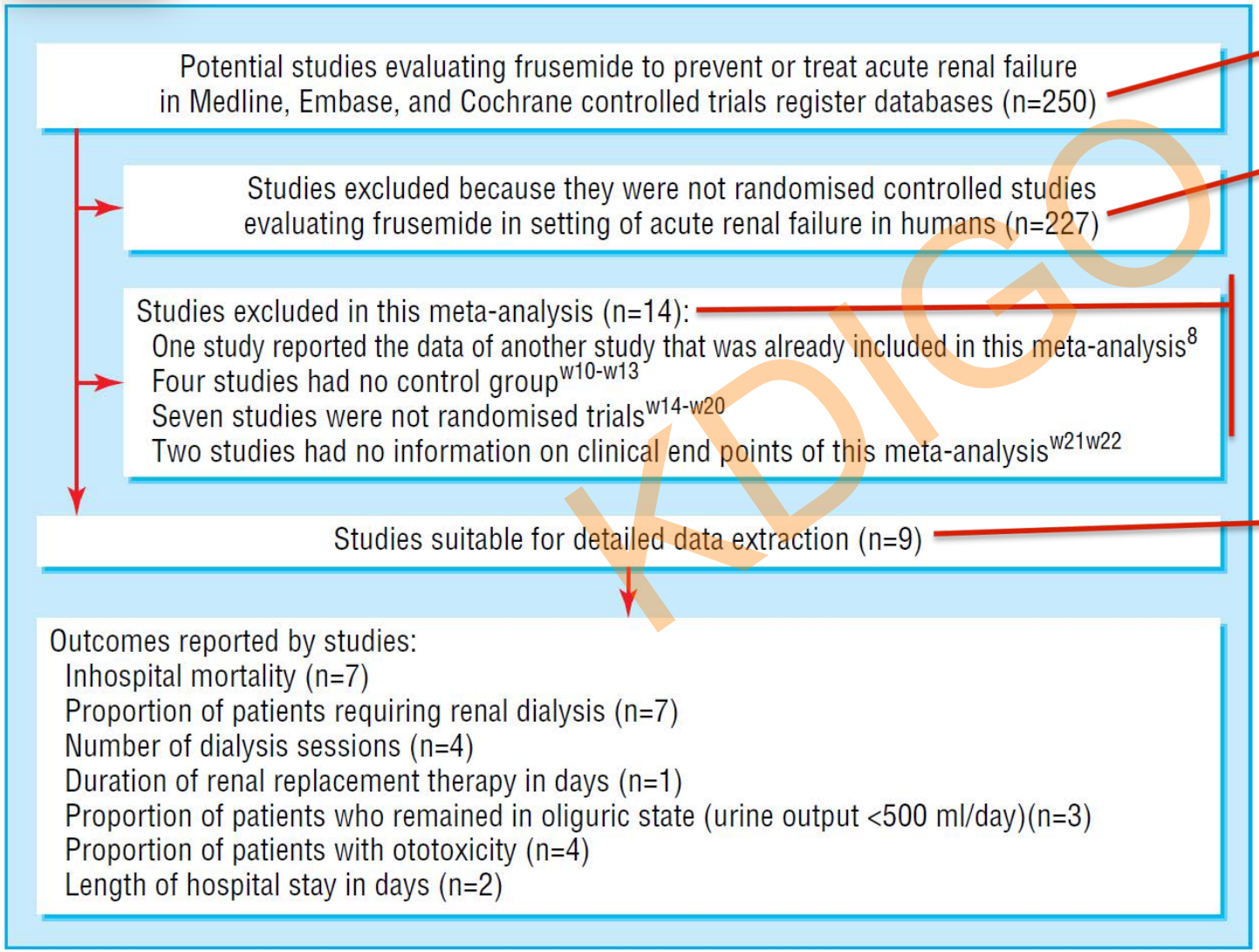
Prevention and Treatment of AKI – Diuretics

BMJ

Meta-analysis - Furosemide to prevent or treat AKI



250 studies

227 excluded

14 excluded in meta-analysis

9 included

849 pts

Ho and Sheridan, BMJ 2006

Prevention and Treatment of AKI – Diuretics

BMJ

Meta-analysis - Furosemide to prevent or treat AKI

What is already known in this topic

Frusemide, a potent loop diuretic, can induce diuresis in some patients with acute renal impairment

What this study adds

Frusemide is not associated with any clinical benefits when used to prevent and treat acute renal failure in adults

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High doses of frusemide may be associated with an increased risk of ototoxicity

Ho and Sheridan, BMJ 2006

KDIGO - Supportive management of AKI

Vasodilator therapy: dopamine, fenoldopam, and natriuretic peptides

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3.5.1: We recommend not using low-dose dopamine to prevent or treat AKI. (1A)

3.5.2: We suggest not using fenoldopam to prevent or treat AKI. (2C)

3.5.3: We suggest not using ANP to prevent (2C) or treat (2B) AKI.

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Prevention and Treatment of AKI – Dopamine

Meta-Analysis: Low-Dose Dopamine Increases Urine Output but Does Not Prevent Renal Dysfunction or Death

Jan O. Friedrich, MD, DPhil; Neill Adhikari, MD, CM; Margaret S. Herridge, MD, MPH; and Joseph Beyene, PhD

- 61 trials – 3,359 pts
- no effect of low-dose dopamine
  - Mortality: RR 0.96 [95% CI, 0.78 - 1.19]
  - Need for RRT: RR 0.93 [CI, 0.76 - 1.15]
  - Adverse events: RR 1.13 [CI, 0.90 - 1.41]

Annals of Internal Medicine
Established in 1927 by the American College of Physicians

Ann Intern Med. 2005;142:510-524.

KDIGO – Contrast-induced AKI

Contrast-induced AKI

4.2.1: Assess the risk for CI-AKI and, in particular, screen for pre-existing impaired kidney function in patients who are considered for a procedure that requires intravascular administration of iodinated contrast medium. (Not Graded)

4.2.2: Consider alternative imaging methods in patients at increased risk for CI-AKI. (Not Graded)

4.3.1: Use the lowest possible dose of contrast medium in patients at risk for CI-AKI. (Not Graded)

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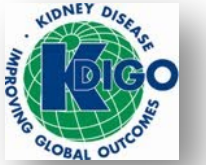
KDIGO – Contrast-induced AKI



Contrast-induced AKI

4.3.2: We recommend using either iso-osmolar or low-osmolar iodinated contrast media, rather than high-osmolar iodinated contrast media in patients at increased risk of CI-AKI. (1B)

4.4.1: We recommend i.v. volume expansion with either isotonic NaCl or NaBiC solutions, rather than no i.v. volume expansion, in patients at increased risk for CI-AKI. (1A)

4.4.2: We recommend not using oral fluids alone in patients at increased risk of CI-AKI. (1C)

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KDIGO – Contrast-induced AKI

Contrast-induced AKI

4.4.3: We suggest using oral NAC with i.v. isotonic crystalloids, in patients at increased risk of CI-AKI. (2D)

4.4.4: We suggest not using theophylline and fenoldopam to prevent CI-AKI. (2C)

4.4.5: We recommend not using fenoldopam to prevent CI-AKI. (1B)

4.5.1: We suggest not using prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for contrast-media removal in patients at increased risk for CI-AKI. (2C)

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KDIGO - Dialysis for Treatment of AKI

Timing of RRT in AKI

5.1.1: Initiate RRT emergently when life-threatening changes in fluid, electrolyte and acid-base balance exist. (Not Graded)

5.1.2: Consider the broader clinical context, the presence of conditions that can be modified with RRT, and trends of laboratory tests, rather than single BUN and creatinine thresholds alone, when making the decision to start RRT. (Not Graded)

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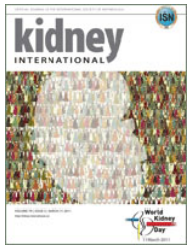
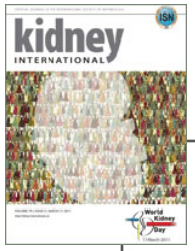


Timing of RRT in AKI

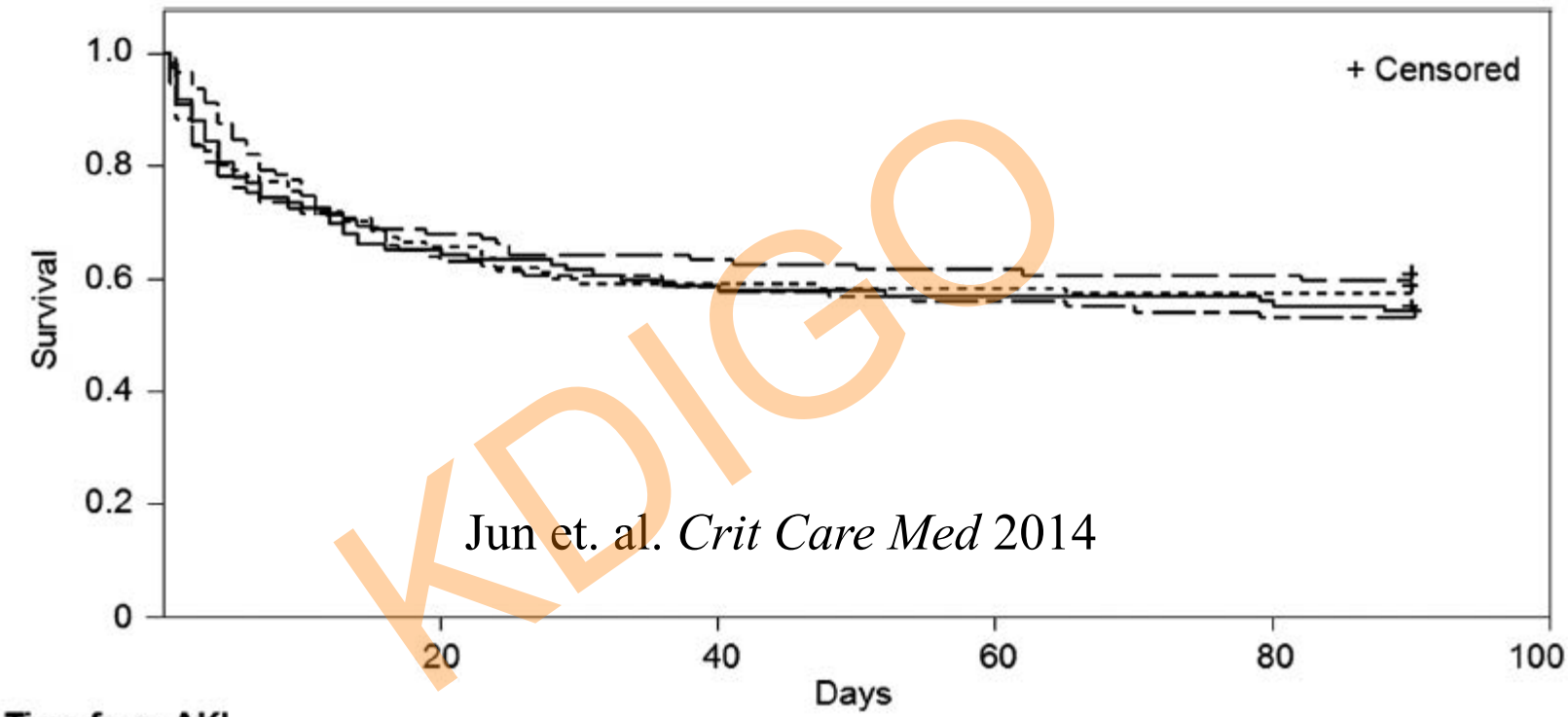
Whether or not to provide RRT, and when to start, are two of the fundamental questions facing nephrologists and intensive-care practitioners in most cases of severe AKI.

The timing of initiation of RRT has not been included as a factor in any of the large RCTs in this area. The optimal timing of dialysis for AKI is not defined.

Dialysis for Treatment of AKI



Timing of RRT in AKI



Jun et. al. *Crit Care Med* 2014

Time from AKI (RIFLE-I) to randomization — — — <7.1 hrs ····· ≥7.1 to <17.6 hrs ——— ≥17.6 to <46.0 hrs — - — - ≥46.0 hrs

At risk (n)

<7.1 hrs	109	74	69	67	66	0
≥7.1 to <17.6 hrs	110	72	65	64	63	0
≥17.6 to <46.0 hrs	109	71	64	62	61	0
≥46.0 hrs	111	71	65	62	59	0

Timing of RRT in AKI

Table 17 | Potential applications for RRT

Applications	Comments
Renal replacement	This is the traditional, prevailing approach based on utilization of RRT when there is little or no residual kidney function.
Life-threatening indications	No trials to validate these criteria.
Hyperkalemia	Dialysis for hyperkalemia is effective in removing potassium; however, it requires frequent monitoring of potassium levels and adjustment of concurrent medical management to prevent relapses.
Acidemia	Metabolic acidosis due to AKI is often aggravated by the underlying condition. Correction of metabolic acidosis with RRT in these conditions depends on the underlying disease process.
Pulmonary edema	RRT is often utilized to prevent the need for ventilatory support; however, it is equally important to manage pulmonary edema in ventilated patients.
Uremic complications (pericarditis, bleeding, etc.)	In contemporary practice it is rare to wait to initiate RRT in AKI patients until there are uremic complications.
Nonemergent indications	
Solute control	BUN reflects factors not directly associated with kidney function, such as catabolic rate and volume status. SCr is influenced by age, race, muscle mass, and catabolic rate, and by changes in its volume of distribution due to fluid administration or withdrawal.
Fluid removal	Fluid overload is an important determinant of the timing of RRT initiation.
Correction of acid-base abnormalities	No standard criteria for initiating dialysis exist.

Timing of RRT in AKI

Table 17 | Potential applications for RRT

Applications	Comments
Renal support	This approach is based on the utilization of RRT techniques as an adjunct to enhance kidney function, modify fluid balance, and control solute levels.
Volume control	Fluid overload is emerging as an important factor associated with, and possibly contributing to, adverse outcomes in AKI. Recent studies have shown potential benefits from extracorporeal fluid removal in CHF. Intraoperative fluid removal using modified ultrafiltration has been shown to improve outcomes in pediatric cardiac surgery patients.
Nutrition	Restricting volume administration in the setting of oliguric AKI may result in limited nutritional support and RRT allows better nutritional supplementation.
Drug delivery	RRT support can enhances the ability to administer drugs without concerns about concurrent fluid accumulation.
Regulation of acid-base and electrolyte status	Permissive hypercapnic acidosis in patients with lung injury can be corrected with RRT, without inducing fluid overload and hypernatremia.
Solute modulation	Changes in solute burden should be anticipated (e.g., tumor lysis syndrome). Although current evidence is unclear, studies are ongoing to assess the efficacy of RRT for cytokine manipulation in sepsis.

KDIGO - Dialysis for Treatment of AKI

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5.2.1: Dis no longer required, idney function that it is adequa or because t with the goals

"The doctors never gave up."

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KDIGO - Dialysis for Treatment of AKI

Vascular access for RRT in AKI

5.4.1: We suggest initiating RRT in patients with AKI via an uncuffed non tunneled dialysis catheter, rather than a tunneled catheter. (2D)

5.4.2: When choosing a vein for insertion of a dialysis catheter in patients with AKI, consider these preferences (Not Graded):

- First choice: right jugular vein;
- Second choice: femoral vein;
- Third choice: left jugular vein;
- Last choice: subclavian vein with preference for the dominant side.

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Vascular access for RRT in AKI

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5.4.3: We recommend using ultrasound guidance for dialysis catheter insertion. (1A)

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KDIGO - Dialysis for Treatment of AKI

Modality of RRT in AKI

5.6.1: Use continuous and intermittent RRT as complementary therapies in AKI patients. (Not Graded)

5.6.2: We suggest using CRRT, rather than standard intermittent RRT, for hemodynamically unstable patients. (2B)

5.6.3: We suggest using CRRT, rather than intermittent RRT, for AKI patients with acute brain injury or other causes of increased intracranial pressure or generalized brain edema. (2B)

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KDIGO – Modality of RRT in AKI

Table 22 Theoretical advantages/ disadvantages of dialysis modalities

Modality	Potential setting in AKI	Advantages	Disadvantages
PD	Hemodynamically unstable Coagulopathy Difficult access Patients at risk of increased intracranial pressure Under-resourced region	Technically simple Hemodynamic stability No anticoagulation No need for vascular access Lower cost Gradual removal of toxins	Poor clearance in hypercatabolic patients Protein loss No control of rate of fluid removal Risk of peritonitis Hyperglycemia Requires intact peritoneal cavity Impairs diaphragmatic movement, potential for respiratory problems
IHD	Hemodynamically stable	Rapid removal of toxins and low-molecular-weight substances Allows for "down time" for diagnostic and therapeutic procedures Reduced exposure to anticoagulation Lower costs than CRRT	Hypotension with rapid fluid removal Dialysis disequilibrium with risk of cerebral edema Technically more complex and demanding
CRRT	Hemodynamically unstable Patients at risk of increased intracranial pressure	Continuous removal of toxins Hemodynamic stability Easy control of fluid balance No treatment-induced increase of intracranial pressure User-friendly machines	Slower clearance of toxins Need for prolonged anticoagulation Patient immobilization Hypothermia Increased costs

Dialysis for AKI – Peritoneal

Discuss in presentation on Saturday

KDIGO - Dialysis for Treatment of AKI

Buffer for RRT in AKI

5.7.1: We suggest using bicarbonate, rather than lactate, as a buffer in dialysate and replacement fluid for RRT in patients with AKI. (2C)

5.7.2: We recommend using bicarbonate, rather than lactate, as a buffer in dialysate and replacement fluid for RRT in patients with AKI and circulatory shock. (1B)

5.7.3: We suggest using bicarbonate, rather than lactate, as a buffer in dialysate and replacement fluid for RRT in patients with AKI and liver failure and/or lactic acidemia. (2B)

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KDIGO - Dialysis for Treatment of AKI

Dose of RRT in AKI

5.8.1: The dose of RRT to be delivered should be prescribed before starting each session of RRT.
(Not Graded) We recommend frequent assessment of the actual delivered dose in order to adjust the prescription. (1B)
5.8.2: Provide RRT to achieve the goals of electrolyte, acid-base, solute, and fluid balance that will meet the patient's needs. (Not Graded)

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KDIGO - Dialysis for Treatment of AKI

Dose of RRT in AKI

5.8.3: We recommend delivering a Kt/V of 3.9 per week when using intermittent or extended RRT in AKI. (1A)

5.8.4: We recommend delivering an effluent volume of 20–25 ml/kg/h for CRRT in AKI (1A). This will usually require a higher prescription of effluent volume. (Not Graded)

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Dialysis for AKI – Dose

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Intensity of Renal Support in Critically Ill Patients with Acute Kidney Injury

The VA/NIH Acute Renal Failure Trial Network*

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Intensity of Continuous Renal-Replacement Therapy in Critically Ill Patients

The RENAL Replacement Therapy Study Investigators*

Dialysis for AKI – My Summary Recommendation