



ACUTE KIDNEY INJURY FOLLOW UP

David Broodbank

EMEESY Network Meeting 10/10/14



Definition

- *Creatinine rise $>26\text{mmol/L}$ within 48 hours*
- *$>50\%$ rise in creatinine over proceeding 7 days*
- *Urine output $<0.5\text{ml/Kg/hr}$ over 8 hours*
- *$>25\%$ fall in eGFR over proceeding 7 days*



Table 6. Pediatric-modified RIFLE (pRIFLE) criteria

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	Estimated CCI	Urine output
Risk	eCCI decrease by 25%	<0.5 ml/kg/h for 8 h
Injury	eCCI decrease by 50%	<0.5 ml/kg/h for 16 h
Failure	eCCI decrease by 75% or eCCI <35 ml/min/1.73 m ²	<0.3 ml/kg/h for 24 h or anuric for 12 h
Loss	Persistent failure >4 weeks	
End stage	End-stage renal disease (persistent failure >3 months)	

eCCI, estimated creatinine clearance; pRIFLE, pediatric risk, injury, failure, loss and end-stage renal disease.

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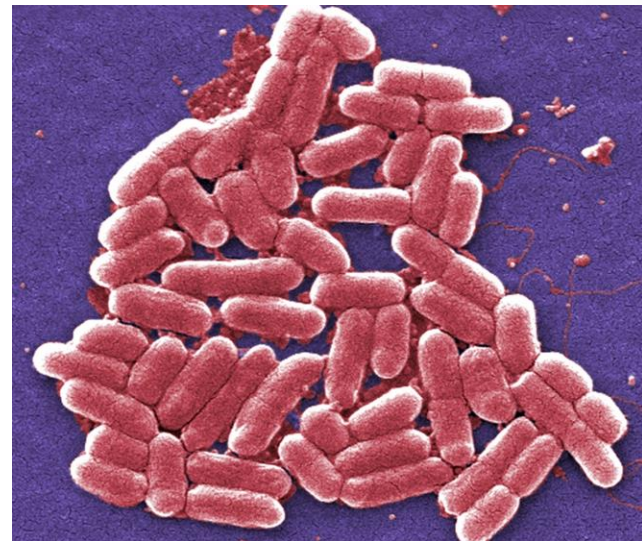


Some NICE Guidance

- 1.5.16 Monitor^[Z] serum creatinine after an episode of acute kidney injury. Consider referral to a nephrologist or paediatric nephrologist when eGFR is 30 ml/min/1.73 m² or less in adults, children and young people who have recovered from an acute kidney injury
- 1.5.17 Consider referral to a paediatric nephrologist for children and young people who have recovered from an episode of acute kidney injury but have hypertension, impaired renal function or 1+ or greater proteinuria on dipstick testing of an early morning urine sample
- ^[Z] The frequency of monitoring should be based on the stability and degree of renal function at the time of discharge.

So Why Follow Up?

- AKI does progress to CKD
 - Special cases, HUS.
- JAMA. 2003 Sep 10;290(10):1360-70.
- *Long-term renal prognosis of diarrhea-associated hemolytic uremic syndrome: a systematic review, meta-analysis, and meta-regression.*
- Garg AX¹, Suri RS, Barrowman N, Rehman F, Matsell D, Rosas-Arellano MP, Salvadori M, Haynes RB, Clark WF.
 - 25% of survivors demonstrate long-term renal sequelae (>3400 pts in study)

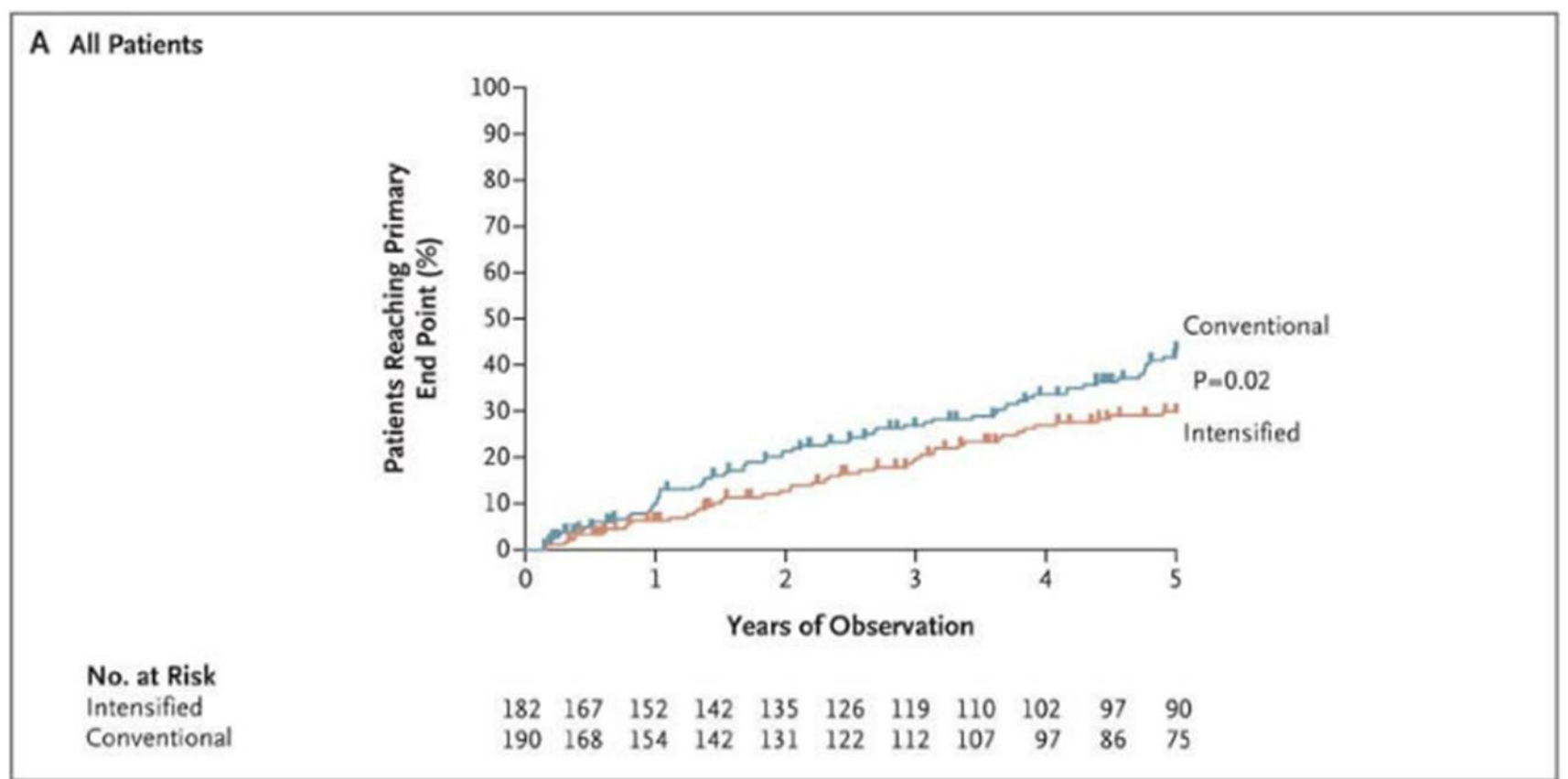


So Why Follow Up

- And the rest?
 - Less clear
 - Epidemiology may give a clue (more secondary causes of AKI and thus CKD)
 - New Biomarkers may help tracking AKI to CKD e.g. Neutrophil gelatinase-associated lipocalin (NGAL), Kidney Injury Molecule 1 (KIM 1), Liver Type Fatty acid binding protein (L-FABP)
 - Viaud et al:- 2/3 of those with severe AKI had evidence of renal injury (all cause) at 12 years (received RRT) (13 pts)
 - Mammen et al:- 10% of all AKI admitted to PICU had CKD at 3 years (126 pts)
 - Preterm infants may be even more significantly affected

Why to follow (2)

- CKD is a progressive but modifiable disease



50% reduction in GFR, GFR <10 ml/min/1.73 or RRT

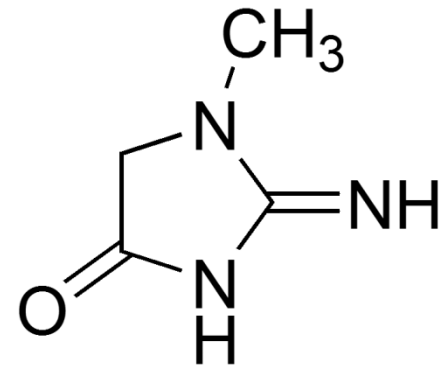
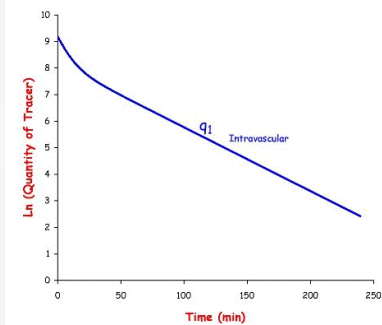
Who to follow?

- All patients with significant AKI
 - Can probably exclude those with rapidly resolving pre-renal causes
- Certainly all who received RRT

	Serum Creatinine	Urine Output
Stage 1	eGFR < 75 ml/min/1.73m ² Or decrease in eGFR by 25% Or increase creatinine 1.5 – 2 x baseline	<0.5 ml/kg/hr for 6 hours
Stage 2	eGFR < 50 ml/min/1.73m ² Or decrease in eGFR by 50% Or increase creatinine 2 – 3 x baseline	<0.5 ml/kg/hr for 12 hours
Stage 3	eGFR <35 ml/min/1.73m ² Or decrease in eGFR by 75% Or increase in creatinine > 3 x baseline Or requirement for renal replacement therapy	<0.3 ml/kg/hr for 24 hours Or anuria for 12 hours



How to follow?

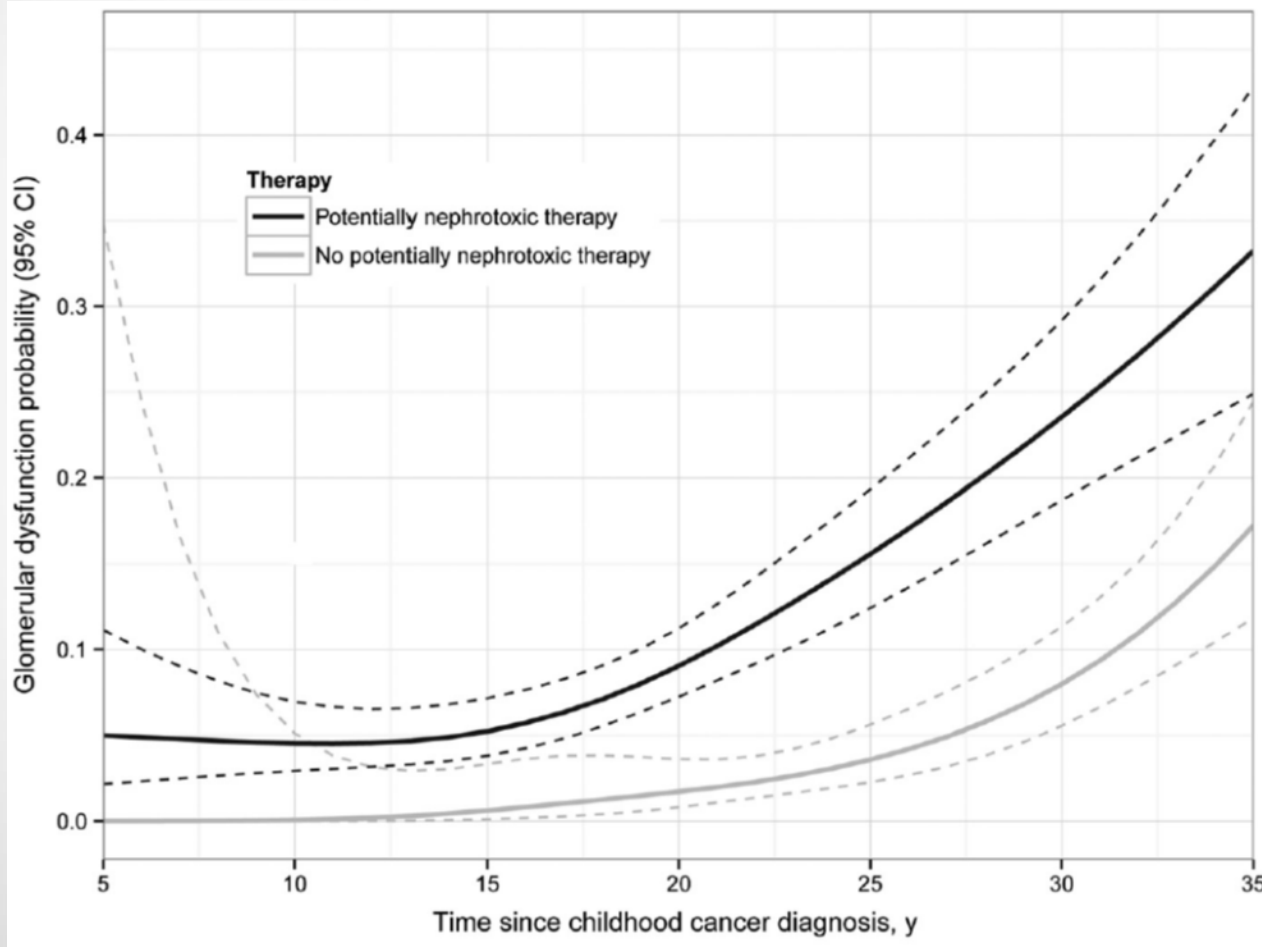


For the less visual learners...

- Creatinine check year 1 and year 5
- Annual BP check
- Annual urine dip or protein : creatinine ratio
- Formal GFR (for those who received RRT and those with HUS) at 1 yr, 5 yrs and post puberty.

Special Cases -Oncology

- Ifosphamide and platinum chemotherapy
 - Both effect glomerular function (AKI and CKD)
 - Both effect tubular function
- Some effects may resolve
 - E.g. tubular effects of ifosphamide
- Others persist
 - E.g. tubular effects of cisplatin or glomerular dysfunction
- Follow up therefore involves
 - Urine dip / PCR
 - Assessment of tubular function, phosphate, bicarbonate etc
 - Creatinine
 - Blood pressure
- Risk factors alone (high cumulative dose, single kidney) will not predict all cases of ongoing kidney disease



Likelihood of
GFR <90 at 35
years

- 26.4% if
potentially
nephrotoxic
treatment
received
- 6.6% if no
nephrotoxic
treatment
($p < 0.001$)

Special cases -Neonates

- 20 Neonates <1000g with AKI (rise in creat >48 hrs or U/O <0.5ml/kg/hr) 18 year review
- Mean age 7.5 yrs
- 9 GFR <75ml/min/1.73m².
- Predictors for CKD
 - P:CR at 1 year
 - Serum creatinine at 1 year
- How to follow?
 - Urine dip
 - Creatinine
 - Blood pressure

Carolyn L. Abitbol • Charles R. Bauer
Brenda Montané • Jayanthi Chandar
Shahnaz Duara • Gastón Zilleruelo

**Long-term follow-up of extremely low birth weight infants
with neonatal renal failure**

References

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- NICE (2013) *Acute kidney injury: prevention, detection and management of acute kidney injury up to the point of renal replacement therapy*.
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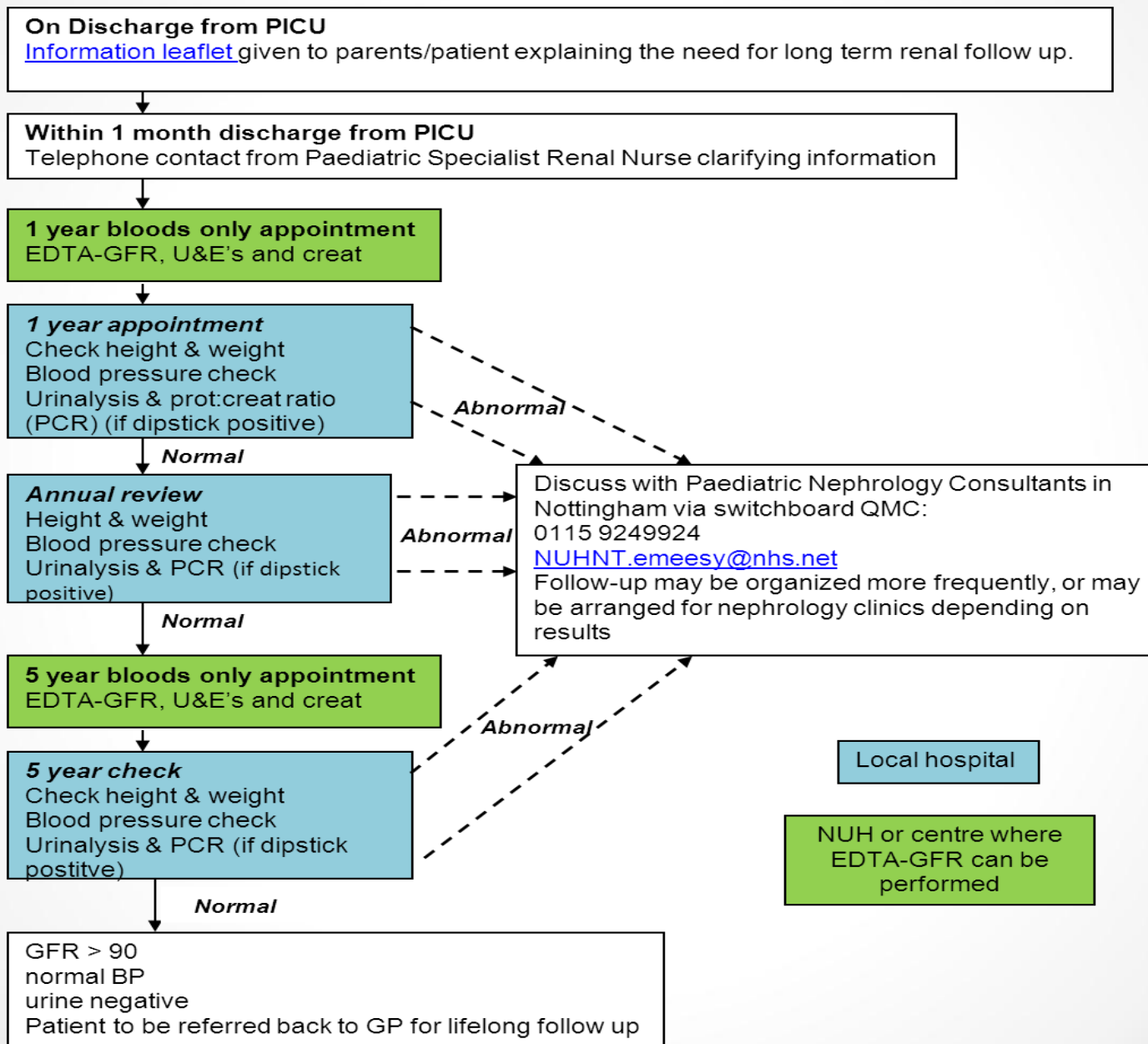
Network Follow up

Molly McLaughlin
Paediatric Renal Critical Care Nurse

Background

- Historically: no defined pathway for follow up
- January 2014: AKI follow up introduced for all patients having CRRT within PICU
- Initially 5 year follow up, now extended to lifelong
- Plan to widen this to all AKI(level 3, persistent, all receiving RRT)

Protocol for Follow-up of patients discharged home following Renal Replacement Therapy for AKI (CVVH/PD)



Referrals to date

- 16 patients identified since January for follow up within EMEESY PICUs
 - Sheffield 4
 - 2 SCH
 - 1 Doncaster
 - 1 Rotherham (currently being seen at SCH)
 - Nottingham 5
 - 3 NUH
 - 1 Kettering
 - 1 Cambridge
 - Leicester (LRI/GH) 7
 - 3 NUH
 - 2 UHL
 - 1 Kettering
 - 1 Boston

Potential challenges

- Identifying patients & ensuring initial referral proving problematic
- Referral pathway
 - Do all patients come under a Nephrologist at NUH?
 - Bloods only appointment in other centre
- Follow up of all AKI