Twitter Thread by Buku Renal



Buku Renal
@BukuRenal



“Should I give them more fluid?”

Extremely common question during AKI referrals from junior docs.

■■ Fluid management is tricky, often misunderstood, & can cause huge iatrogenic harm ■■

We try to convey important principles in THREAD ■

#tipsfornewdocs #medtwitter #tweetorial

Currently too often patients with high creatinine are given too much IV fluid, driven by an AKI ➡■ fluid reflex

We’ve seen 7 litres be given in one shift.

Medics & surgeons, juniors & seniors, it seems everyone loves to reach for extra IV fluid (especially if urine output poor)

Why does huge volume IV fluid ‘resuscitation’ consistently occur?

Lots of reasons, including FALSE beliefs:
■ IV fluid always “treats AKI”
■ Hypotension = hypovolaemia
■ Lactate >2 means fluid deficiency
■ Septic patients are very hypovolaemic

The rule with IV fluids and AKI is to remember this 5 word phrase:

■ ■ Aim for euvolaemia, then stop■ ■

Fluid status is like Goldilocks - hypovolaemia is bad for the kidneys BUT excess volume is also harmful for renal function and will cause other complications too.

It is easy to say “target euvolaemia” - but we agree it is more difficult to do!

Fluid status assessment is certainly made harder by a belief that we must only rely on a snapshot assessment of examination features which are NOT specific / sensitive / reliable to elicit like….

■ Cool peripheries
■ Bibasal crackles - highly nonspecific in older patients!
■ Dry skin
■Tachycardia
■ x and y descent of the JVP wave…..

Even postural BP drop, one of the better performing signs for hypovolaemia, still has a wide differential diagnosis.

Serial physical examinations by one clinician improves assessment, but this is challenging given shift patterns.

And unfortunately the blood tests can be misleading too - for example check out our previous post about other causes of raised urea ■ https://t.co/CSiRRnMi1a

> Quick learning: think isolated high urea = \u201cdehydration\u201d?
>
> Some other differentials:
> \u2757\ufe0fGI bleeding
> \u2757\ufe0f\u2019apparent hypovolaemia\u2019 in CCF (when the kidney sees low perfusion pressure)
> \u2705 corticosteroids
> \u2705 high protein intake
>
> Not all \u2b06\ufe0furea needs IV fluid!#medtwitter #tipsfornewdocs
>
> — Buku Renal (@BukuRenal) September 17, 2020

TIP - things get better when the adage of ‘history trumps examination’ is observed:

1■■ Has there been volume loss?
2■■ Has there been decreased intake?

If the answer is “not really” to both of these, then think carefully about what you think more fluid will accomplish.

So….

When phoned as renal reg on-call on day 2-3 we’re usually not thinking “easy hypovolaemic AKI” but are concerned something is being missed.

Three common things:

1. AKI is part of sepsis / hypotension / MOF badness: patient needs ICU & vasopressors, not more IV fluid

2. AKI is intrinsic or post-renal: patient needs a diagnosis (think urinalysis, ultrasound, discussion), not more IV fluid

3. AKI is an additional signal that palliative care may be appropriate (remember stage 3 AKI has terrible 42% mortality at 30 days in patients >75 years)

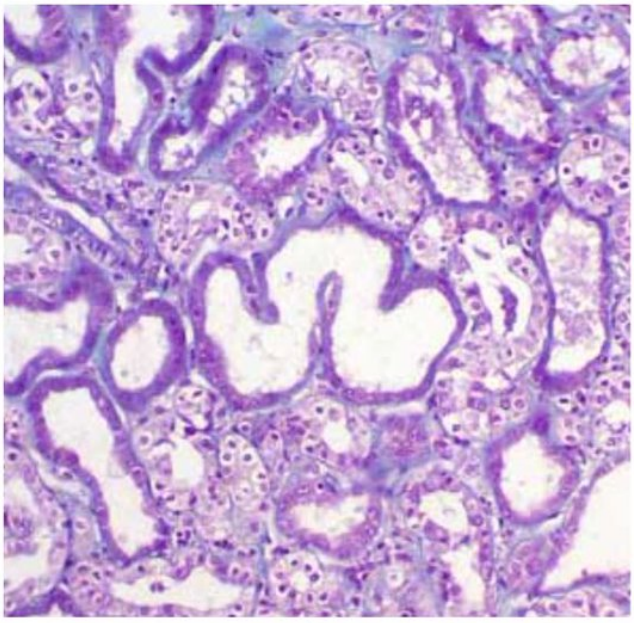
Table 2.2 30 day mortality by peak stage of AKI and demographics for patients with an AKI episode in 2018

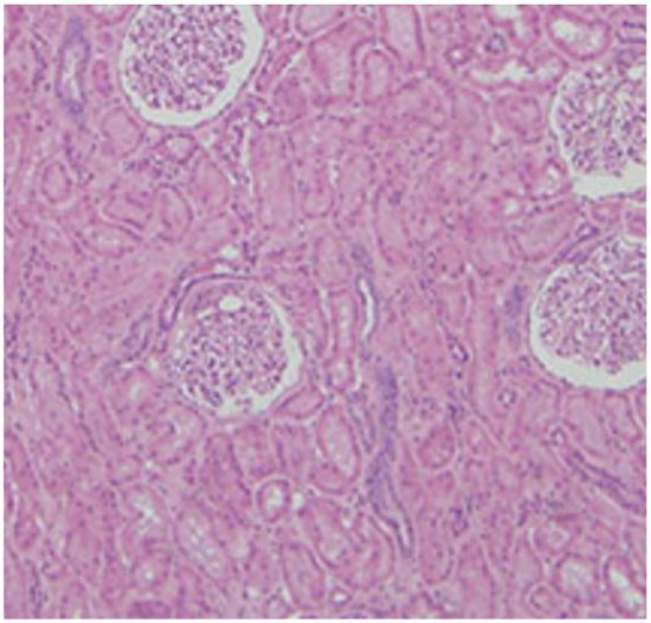
	All AKI episodes		Peak stage of AKI					
			1		2		3	
Variable	N	Unadj. mortality (%)	N	Unadj. mortality (%)	N	Unadj. mortality (%)	N	Unadj. mortality (%)
All	488,856	18.5	342,382	13.4	82,769	28.6	63,705	32.7
Age group (years)								
<18	11,204	3.0	8,072	1.8	2,001	4.7	1,131	9.1
18-39	42,374	2.7	34,143	1.4	4,970	5.7	3,261	11.4
40-64	106,491	10.9	73,208	6.7	17,395	17.8	15,888	23.1
65-74	97,928	16.9	65,924	11.7	17,644	25.8	14,360	29.9
≥75	230,859	26.2	161,035	20.2	40,759	38.3	29,065	42.6
Sex								
Male	233,457	20.0	157,562	15.0	38,995	29.0	36,900	31.8
Female	255,399	17.0	184,820	11.9	43,774	28.2	26,805	34.1
Deprivation quintile								
1 - least deprived	82,698	19.2	58,546	14.1	13,904	29.6	10,248	33.9
2	93,807	19.2	66,098	13.8	15,850	30.2	11,859	34.3
3	99,469	18.6	69,778	13.6	16,908	28.5	12,783	32.9
4	102,600	17.9	71,762	12.9	17,169	27.3	13,669	31.8
5 - most deprived	109,598	17.5	75,779	12.4	18,782	27.3	15,037	31.1
Month of AKI alert								
Jan-Mar	128,252	22.6	87,753	16.7	22,751	33.4	17,748	38.0
Apr-Jun	118,485	17.6	83,565	12.7	19,897	27.5	15,023	31.7
Jul-Sep	116,762	16.3	82,371	11.7	19,396	26.1	14,995	29.4
Oct-Dec	125,357	17.0	88,693	12.2	20,725	26.6	15,939	31.0

Pre-renal AKI purely due to hypovolaemia generally improves fast.

If the patient has a great story for hypovolaemia but their creatinine is rising/stuck, then they often have a degree of acute tubular necrosis (ATN).

Let us show you something (it's not as boring as it appears!)





On left is ATN with gaping spaces in tubules, & on right is healthy kidney with nice plump tubules.

Appreciate that there is actual histological change - this needs TIME to recover

✳■ Just as injured cells in stroke or MI don't magically heal with IV saline, neither does ATN ✳■

■ Sometimes you'll get a patient with ATN to euvolaemia & they still won't pee. That's OK - not an indication for more fluids

■ Remember low urine output is a warning you have less margin for error before causing hypervolaemia - again, shake off the reflex just to ■■fluid rate

"Are there exceptions?"

Not really - there's different risk:benefit in different scenarios
■ in tumour lysis, myeloma or rhabdo for example we do try to target higher urine outputs

BUT when patient is well-filled & not peeing much, we're re-thinking rather than ploughing on.

"But does volume overload even matter?"

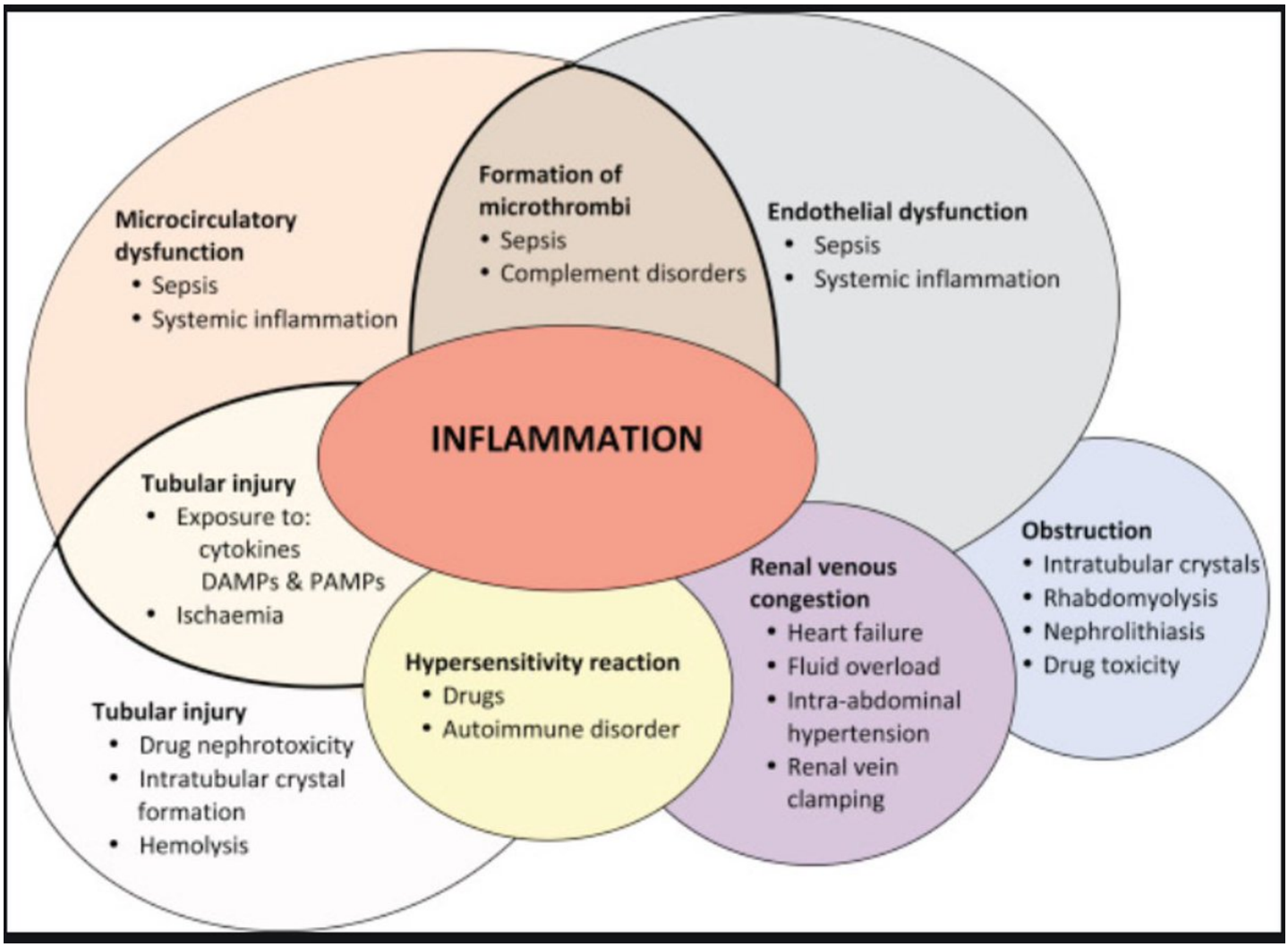
YES - and it's not just the obvious pulmonary oedema badness.

Excess fluid ➡■ high venous pressure ➡■ decreased perfusion pressure for heart, brain, liver (& kidneys!!)

Patients with excess volume have:
■ longer AKI
■ higher mortality

In hypovolaemic AKI aim to stop fluids before complications develop - you do not need to “swell to get well”.

Avoidable dialysis for ■■volume continues because of myth that the hugely complex pathophysiology of AKI is easily remedied by IV salty water (pic from Ostermann et al)



So apologies for the rant. But we hope we have made you think about why:

■ “I can’t give fluid for their AKI because they are already overloaded” makes no sense ■

And why:

■ “I’ll keep giving IV fluids until their oxygen sats drop” is just bonkers ■

Take home points:

■ Prescribe IV fluid with the care you would warfarin
■ Achieve euvolaemia, then sit on your hands
■ If urine output or BP stays low think “Is a different approach needed here?” rather than autoreaching for more IV fluid
■ Break your AKI ➡■ fluid reflex

As ever all comments/disagreements very welcome - it’s a tricky topic to teach & not done really at med school, so we at @BukuRenal (@jamiekwillows this time) appreciate any feedback.

Would love to hear views on how you approach teaching ward junior docs @NephroGuy @icmteaching