Supplementary Data

Table 1: Primary cause of AKI

Primary cause of AKI	Total patients (%) N=176
Sepsis syndrome	75 (43%)
Infection / sepsis	67
Pancreatitis	8
Extracellular fluid depletion	28 (16%)
Dehydration	10
Abdominal Aorta Aneurysm (AAA) rupture	4
ALD / hepatorenal syndrome	8
Bleed other than AAA	6
Heart failure	21 (12%)
Heart failure	8
Myocardial infarct	7
Cardiac arrest	6
Intrinsic renal	12 (7%)
CKD progression	3
Rhabdomyolysis	3
Vasculitis	2
Acute Interstitial Nephritis	2
Minimal Change Disease	1
Henoch Schönlein Purpura	1
Obstructive uropathy	5 (3%)
Nephrotoxins as single primary cause	4 (2%)
Oral nephrotoxic medication	3
Intravenous contrast	1
Multiple organ failure / other	31 (17%)
Post-op	21
Malignancy	4
Renal artery stenosis	2
Status epilepticus	1
Road Traffic Accident	1
Ischaemic stroke	1
Compartment syndrome	1

Table 2: Cause of death

Cause of death	Total patients (%) N=94
Sepsis syndrome	28 (30%)
Cardiovascular	23 (24%)
Cardiac arrest	11
Heart failure	5
Bleed	3
Sudden death / MI	1
Ischaemic stroke	1
Haemorrhagic stroke	1
Limb ischaemia	1
Malignancy	16 (17%)
Liver disease / hepatorenal syndrome	10 (11%)
Other	9 (10%)
Multiple Organ Failure	3
Respiratory failure	2
Frailty	2
Status epilepticus	1
Bowel perforation	1
Unknown	8 (8%)

Table 3: Pathophysiology of medication interacting with the renovascular system.

Agent	Causal path of AKI	Number of patients affected N=37
ACEi/ARB/Antihypertensives	Reduction of auto regulation of GFR in context of reduced effective plasma volume (e.g. in sepsis)	22
Trimethoprim	Exacerbation of hyperkalaemia and reduced	2
Metformin	Exacerbation of acidaemia	8
Opiods	Reduction of effective plasma volume due to vasodilation	2
NSAIDs	In context of reduced effective plasma volume inappropriate prostaglandin inhibition causes vasodilation.This causes reduced renal perfusion and reduced RAS-activity. NSAID induced interstitial nephritis is less common.	2
Contrast	Vasoconstriction causing reduced medullary perfusion and direct tubular toxicity	11

In thirty-seven patients interacting drugs were identified of which seventeen patients received more than one type of interacting drugs.

ACEi: Angiotensin Converting Enzyme inhibitor, ARB: Angiotensin-Receptor Blocker, GFR; Glomerular Filtration Rate, NSAIDs: Non-Steroidal Anti-Inflammatory Drugs, RAS: Renin-Angiotensin System.

Figure 1: Medication use as primary cause or contributing factor to the development of AKI in our population