ACUTE KIDNEY INJURY... FOCUS ON OBSTETRICS

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AIMS & OBJECTIVES

- * Review the functions of the kidney
- * Identify renal physiological changes in pregnancy
- Define Acute Kidney Injury (AKI)
- Look at general risk factors for AKI
- Raise awareness of additional risk factors for AKI in pregnancy
- * Identify the 3 groups of kidney injury
- * Understand appropriate fluid management

So... what are the kidneys?

- * The kidneys are 2 bean shaped organs, each about the size of a fist
- * They are located just below the rib cage, one on each side of the spine



FUNCTIONS OF THE KIDNEY

- Urine production
- Removal of waste products
- Maintenance of fluid balance
- Maintenance of electrolytes
- Regulation of acid base balance
- Production of erythropoetin
- Production of renin
- Vitamin D3 synthesis

Functional changes in pregnancy..

- Kidneys increase in size by up to 2cm
- * Ureters dilate
- Increased renal blood flow due to increased cardiac output and renal vasodilatation
- * Decrease in systolic pressure due to vasodilatation and reduced vascular resistance
- * Increased renal blood flow leads to increased glomerular filtration rate (GFR), begins shortly after conception, peaks at beginning of 2nd trimester and remains until after delivery

Acute Kidney Injury....

Abrupt reduction in kidney function (within 48hrs) & encompasses a wide spectrum of injury to the kidneys, not just kidney failure.

NICE (2013)



Acute kidney injury

- Seen in 13-18% of all people admitted to hospital
- Older adults particularly at risk
- £434M £630M per year to NHS, Excluding Community
- More than breast, lung and skin cancer altogether
- Suboptimal care contributing factor
- NCEPOD (2009) 50% received good care
- Failures in prevention, recognition, therapy and timely referral to specialist services.
- NICE Guidance (2013), reviewed 2017.

NICE RECOMMENDATIONS

- Assess Risk
- Prevent AKI
- Detect AKI
- Identify Cause
- Manage AKI
- Information and support

Prevention is better than cure....



AKI Risk factors

General

- * > 65 years
- Chronic kidney disease
- History of AKI
- Patients having surgery
- Nephrotoxic drugs
- * Liver failure
- * EWS Deterioration
- * Hypovolaemia

Pregnancy

- Gestational diabetes
- * Sepsis
- * C Section
- * Pre eclampsia
- * Hyperemesis gravidarum
- * Maternal haemorrhage

Clinical Features of AKI

- Asymptomatic
- Oliguria
- Deranged bloods Urea, creatinine,
 Potassium
- Confusion
- Nausea and vomiting
- Loss of appetite

Urine output...

- Normal urine output ranges from 1.5 2litres daily
- Oliguria is defined as the production of between 100
 400mls per 24hours
- * Anuria defined as the production of less than 100mls in 24hrs
- * Absolute anuria reflects no urine output and should be attributed to a urinary tract obstruction until ruled out

So.... What do your kidneys need

- * An adequate blood supply
- * Ability to function/ filter
- * No obstruction between kidneys & urethra or urinary catheter



Causes of AKI....

* Pre – renal Inadequate perfusion / blood flow

* Renal (intrinsic) Have they kidneys suffered an insult or been poisoned?

* Post-renal Obstruction to flow

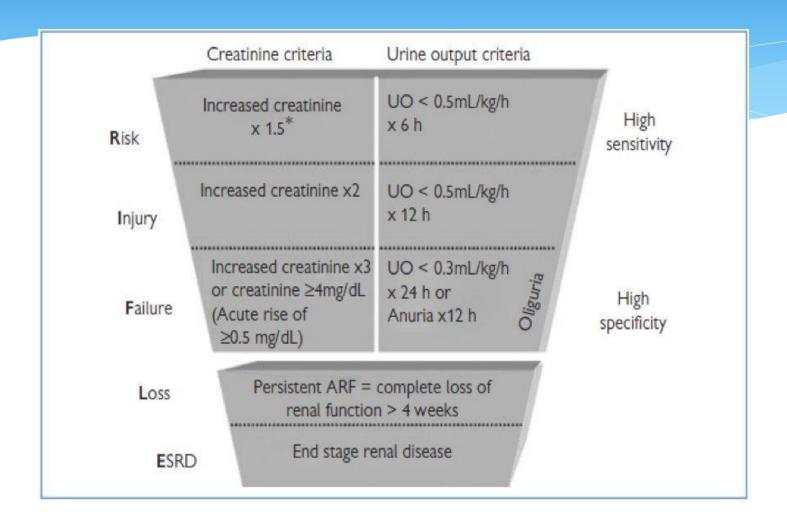
Oliguria in pre eclampsia

- * Pre eclampsia causes a decrease in glomerular filtration and renal blood flow
- Vasospasm and glomerular capillary endothelial oedema – reduced functional glomeruli
- * 2 renal blood flow protein losses from the vascular space & increased sensitivity to angiotensin II
- * Oliguria often detected in immediate postnatal period
- * At this point more at risk of pulmonary oedema
- * Cautious fluid restriction (1ml/kg/hr) / & strict input output monitoring

PREVENT AKI

- Monitor bloods
- Review drugs –e prescribing
- Track and trigger system (NEWS / NEWS 2)
 with accurate fluid balance
- Iodinised contrast fluids (? acetyl cystine)

Detect AKI



IDENTIFY CAUSE

- URINALYSIS ASAP
 document results and
 think early referral to
 Nephrologist if no
 cause found of
 haematuria &
 proteinuria
- USS Not routine if cause identified
- Within 6hrs if pyelonephritis suspected
- Within 24hrs if AKI and no identified cause

Complications from AKI – Increased mortality

- * Hyperkalaemia
- * Acidosis
- * Pulmonary oedema
- * Uraemic encephalopathy
- * Uraemic pericardial effusion
- * Uraemic GI Bleed

MANAGE AKI

- * Relieve obstruction
- * Pharmacological intervention do not routinely offer loop diuretics
- * Refer for Renal Replacement therapy early –No response to medical management complications
- * Referral to nephrologist

CASE STUDY

- * You are called to assess a 39 year old lady on the antenatal ward. She is 38 weeks pregnant with a 4 day history of vomiting. She is complaining of a painful knee and asks if she can have her ibuprofen which is due now.
- * The midwives tell you she hasn't passed urine since admission 4 hours ago....
- * A Patent and able to speak
- * B RR 28, SpO2 94% on room air
- * C HR 110, BP 90/65, CRT 4 secs
- * D AVPU Alert, Pupils normal, Blood Glucose 5.6
- * E Swollen left knee, not hot to touch

CASE STUDY

- * What risk factors can you identify?
- * ABCDE
- * Fluid challenge & Fluid I/O
- * Bladder scan, ? Catheter
- * Review drug history
- * Review notes
- * Investigations..... which?
- * Monitor.... How?

Summary

- The kidneys and adequate function are vital to maintain homeostasis
- * There are 3 categories of renal failure
- Risk assessments can prevent AKI or pick up additional risk factors early preventing long term damage
- Early referral to specialists is essential improved outcomes
- * A multi disciplinary approach is required including pharmacists, medical staff, dieticians and GP's

ANY QUESTIONS?.....

