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Preventive Renal Medicine

“I am tomorrow” or some future day, what I establish today. I am today what I established yesterday or some previous day”. (James Joyce). Many of the renal insults leading to CKD in later life begin in the paediatric age group.

CKD: World wide incidence of ESRD – 75 – 350/million. In Asia alone, approx. 350, 000 patients were on dialysis by end 2004. Paediatric incidence of ESRD – 3 – 15 / million. 1,00,000 patients develop CKD / year in India. Majority may never see a nephrologist. Denotes advanced Renal damage. Important to recognise conditions which progress to CKD.

CKD (Chronic Kidney Disease) – Either of the following.

1) Renal damage for 3 months or more, defined by structural or functional abnormalities of the kidneys with or without decreased GFR, manifested by 1 or more of the following:

- a) Abnormalities in blood or urine composition.
- b) Abnormalities in Imaging Tests.
- c) Abnormalities of Kidney Biopsy.

2)GFR less than 60ml/min/1.73m², for 3 months or more with or without the above mentioned signs of kidney damages.

Classification of stages of CKD

Stage	GFR (ml/min/1.73m ²)	Type
1.	90	K. damage with N/ GFR
2.	60-89	K. damage with mild GFR
3.	30-59	Moderate GFR
4.	15-29	Severe GFR
5.	< 15	Renal failure

GFR – *Glomerular Filtration Rate*

$$\text{GFR} - (\text{ml/min/1.73m}^2) = \frac{0.55 \times \text{Ht(cm)}}{\text{Sr. creatinine (mgm/dl)}}$$

Principles of management of children with CKD

A) Screening, early detection, early intervention of Kidney disease - “SEEK” . B) Prevention of progression of CKD. C) Prevention and early treatment of Complication of CKD.

- **CKD – Cause** **Glomerular Nephritis** - Idiopathic (MPGN, FSGS), RPGN / Crescentic GN, SLE **Familial** - CNS, Alports Syndrome, Juvenile Nephronophthisis, **Amyloidosis, HUS, Congenital** - Dysplasia, Hypoplasia, PCKD, **VUR, Obstructive** – Obstructions, PUV, Calculi, **Miscellaneous** - Wilms tumor, Renal cortical necrosis, **Acute on chronic insult** – severe shock, AGE etc., **IN INDIA** – CGN, OBSTRUCTIVE UROPATHY: MORE COMMON IN CHILDREN.

Risk factors for CKD - VUR (severe), Recurrent UTi, Renal scarring, HT, Obstructive uropathy, H/o nephritis, NS, DM, SLE, HSP, Family h/o ESRD, PKD, Perinatal / Neonatal insults , Genetic / Chromosomal defects, Dysplasia / Hypoplasia , IUGR/ EPT/ PT/SGA

Progression of renal damage

Nephron loss leading to raised Arteriolar pressure - Hyperfiltration Injury to matrix – Proteinuria - TGF β , GF, endothelins, IL - Focal sclerosis/tubular & glomerular damage Nephron loss

Introduction to antenatal fetal renal medicine - Era of revolutionized fetal renal medicine, 1% of all new borns births, Significant abnormalities - 2/1000 live births, Renal anomalies are the commonest (20%), 80% of these NNB are asymptomatic at birth, Congenital nephropathies - diverse group of disorders – leading to 30% of Pediatric ESRD

Indications of fetal U/S include the following - Past +ve h/o fetal renal abnormalities, Positive family history, Oligo / Polyhydramnios, Persistent breech presentation, Elevated MSAFP in 2nd Trimester, Marked IUGR, Fetal urine production - 10th week GA, Fetal kidney abn. - 16th week of GA.

Hydronephrosis - Commonest entities diagnosed antenatally, 1.4% of all fetuses have hydronephrosis, Management - evolved rapidly, HN is not synonymous with obstruction, Majority of these NNB are normal O/E with normal blood and urine evaluation, Low grade of HN resolve postnatally.

Approach to antenatal hydronephrosis - USG within 7 days, followed by an X-Ray & MCU



Vesicoureteric Reflux - Abnormal situated / distal ureteral orifice, Embryologically muscular tone of trigone, AD, genetic, Sibs need screening, Graded from I to V, 15-30% of ESRDs had UTI with VUR.

THE VULNERABILITY OF THE PERINATAL KIDNEY Prematurity. Incidence of premature births in India in a general population - 14% (data of 2004), 3 to 4 million preterms are born annually.

GFR in preterm – is practically 1/5th of an adult so a small insult can cause permanent damage - New Borns and infants can lose > 50% of renal function with no significant change in serum creatinine.

Fetal complications associated with angiotensin – converting enzyme inhibitors & Non-steroidal anti inflammatory drug : fetal nephrotoxicity - Reduced fetal urine production, Oligohydramnios, Abnormal tubular and glomerular growth and differentiation, Impaired nephrogenesis., Interstitial renal fibrosis.

UTI : Introduction - 150 million people per year become infected, **5%** of general practitioner visits are for UTIs, **Prevalence -** In girls <1 year is 6.5%, boys is 3.3, In girls >1 year is 8.1%, boys is 1.9%, **7%** of children <2 yrs. who have fever without a source, **Renal scarring** - twice as common in this age, group.

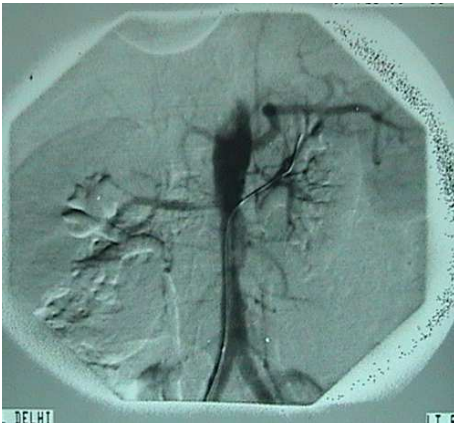
Clinical - Signal underlying **genitourinary tract abnormality**, **Difficult to diagnose** because symptoms are non-specific in this age group and testing is often invasive, **New borns and Infants** – poor feeding, vomiting, poor weight gain, irritability, fever, hypothermia, prolonged jaundice, sepsis, apnea, etc. , **Toddlers** – fever, vomiting, dysuria, incontinence, haematuria, frequency, enuresis, **Older Children** – any of above, abdominal pain etc., **Detailed and careful history taking** – previous fevers, missed or partially treated or partially investigated UTIs, UTI is a silent killer, 25-30% of recurrent UTI with severe VUR lead to CKD, 1/3 of cases of UTI in infancy have association with VUR, Hypertension – Proteinuria – scarring – CKD, UTI is a common cause of fever without a source, Symptoms are non-specific, high level of suspicion, Urine C/S – mandatory, mode of collection, Children < 5 yrs require 2 imaging studies after initial UTI, School Health Screening Programmes are crucial.

Hypertension : Incidence -20% in adult population, 5% Task force definition, 1% req. antiHT Rx, General incidence 1.2% to 13%. (varying studies), Standard normograms mandatory (BP : age /sex), Increases morbidity & mortality (CAD / CVA), 1/3rd of adult US population has HT, HT in adults has its antecedents in childhood, Complex & multifactorial. Early diagnosis and treatment prevents long term end organ damage.

Symptoms of HT - Hidden, varied, high index of suspicions, **Significant HT -** Chase vigorously, NB – RD, irritability, Lethargy, FTT, CCF, Vomiting, Fits, Sweating, Pallor, Cynosis, Sepsis picture, Apnea, Older – Fatigue, Headache, Vision, Epistaxis, Facial N. Palsy, Polydipsia, Polyuria.

HT : Examination - BP & Pulses in all four limbs should be checked with a detailed systemic examination including skin & eyes.

Lt. RAS



Management - Most CKDs continue to progressed due to hyper filtration which is a functional adaptation following a critical loss of nephron units, **CAKUT –** Congenital Abnormalities of Kidney & Urinary Tract, Height, Weight, BP, Failure to Thrive, urinary infections, hypertension, rickets, nephrotic picture, dehydration – electrolytes, Regular screening of children in at “risk category”, Nation wide school screening programmes – detection of proteinuria, hematuria, hypertension, anaemia, Education & Public Awareness.

