THE FETAL GENITOURINARY TRACT

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DISCLOSURES

Beverly G. Coleman, MD
No Relevant Financial Relationships

LEARNING OBJECTIVES

After completing this presentation, the learner will be able to discuss:

1. The normal sonographic appearance of all of the organs and structures that constitute the fetal GU tract
2. A systematic approach to analyzing fetal scans referred for suspected GU tract anomalies
3. Tips for recognizing renal developmental variants
4. Distinguishing features of urinary tract obstruction compared to cystic renal disease

OUTLINE

- The Normal Urinary Tract
- Renal Developmental Variants
- Urinary Tract Obstruction
- Cystic Renal Disease

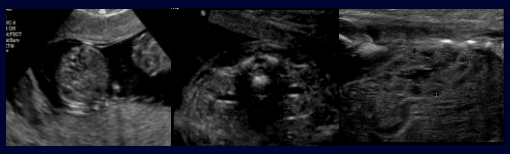
THE NORMAL URINARY TRACT

- Viz at 11-13 wks (TV) and 14-16 wks (TA)
- Corticomedullary differentiation at 16-18 wks
- Exponential ↑ in size with GA; RC/AC ratio = 0.27- 0.30; Nomograms for renal length 14-42 wks & renal volume 15-42 wks
- Increased conspicuity by 3rd trimester with thicker cortex, renal sinus/perinephric fat, best corticomedullary differentiation
- Color Doppler ⟶ renal arteries & veins

THE NORMAL URINARY TRACT

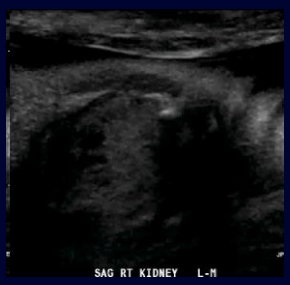
Normal Fetal Kidneys

14 weeks	***20 weeks***	***30 weeks***
8 Mhz TV	***9Mhz***	***12 Mhz***



THE NORMAL URINARY TRACT

Normal CM Differentiation at 27 weeks



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THE NORMAL URINARY TRACT

The Normal Fetal Bladder

- Anechoic, midline, anterior, pear shaped with a thin wall
- First structure to be visualized at 9-10 wks on both TA & TV scans
- Fills and empties every 25-30 minutes
- Max volume ↑ from mean of 1 mL at 20 wks to 36 mL at 41 wks
- Color Doppler ⟶ umbilical arteries

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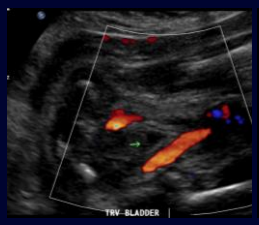
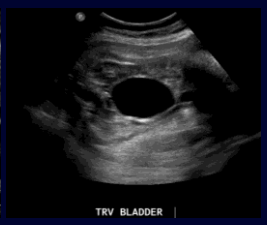
THE NORMAL URINARY TRACT

The Normal Fetal Bladder

- ***Collapsed*** ***Distended***





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THE NORMAL URINARY TRACT

- ***Normal Fetal Adrenals***
  - ➢Disc shaped, "ice cream sandwich"
  - ➢Viz at 20-30 wks, ↑ length with GA
  - ➢Nomograms – length, volume, weight, etc.
- ***Normal Fetal Gender***
  - ➢Male, female identical to 11wks; phallus direction "angle of the dangle"
  - ➢Testicles descend after 25 wks; both >97% after 32 wks; Small hydroceles common in 15% of normal males
  - ➢Uterus can be viz late; ovaries rarely ever viz

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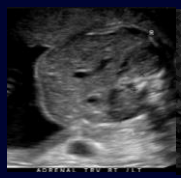
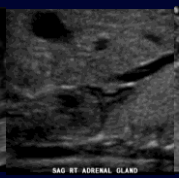
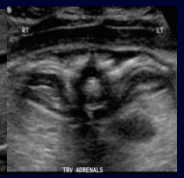
THE NORMAL URINARY TRACT

Normal Fetal Adrenals

- ***2003*** ***2007*** ***2012***







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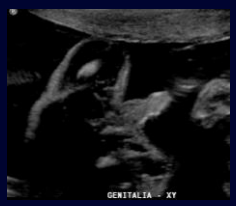
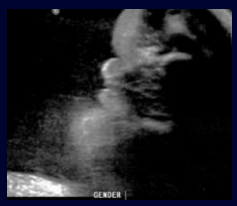
THE NORMAL URINARY TRACT

The Normal Genitalia

- ***XY 23 wks*** ***XX 28 wks***





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Importance of Fetal Gender Assessment

- Confirmation for specific structural anomalies
- Family history of X-linked disorders
- Familial syndromes with genital anomalies
- Gender assignment in multiple gestations
- Exclude maternal cell contamination on amnio

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Fetal Urine Production

- Begins at the 9th week of embryonic life
- Varies with gestational age: 2-5 ml/hr at 20 wks ⟶10 ml/hr at 30 wks ⟶28 ml/hr at 40 wks
- After 14 wks, 2/3's of the amniotic fluid is derived from fetal urination and 1/3 from pulmonary fluid

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THE NORMAL URINARY TRACT

Amniotic Fluid What Do we Know?

- Before 10 wks, AF = ultrafiltrate maternal plasma
- 10-20 wks resembles fetal plasma; vol c/w weight
- AFV ave 500ml at 20 wks ⟶1200ml at 32 wks⟶ 800ml at term
- Amniotic Fluid Evaluation
  - Subjective assessment studies indicate this is very reliable by experienced observers
  - Semi-quantitative Criteria-
    - AFI Nomograms for 4 Quadrants at 16-42 wks (1990)
    - DVP Method=Vertical Depth of largest single pocket (exclude the umbilical cord & fetal parts)

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THE NORMAL URINARY TRACT

Fluid Production	*Fluid Removal*
• Kidneys	GI Tract
• Lung	Lung
• Membranes & Cord	Membranes & Cord
• Skin	

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Approach to GU Tract Analysis

- Kidneys normal – number, size, location, echogenicity?
- If not, is there unilateral versus bilateral disease?
- If obstruction is present, how severe & at what point?
- Are there renal cysts, where and how significant?
- Are the ureters and bladder visible?
- Are the adrenals and genitalia normal?
- Is amniotic fluid volume normal, increased or decreased?

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Renal Developmental Variants

Absent/ectopic tissue ***Abnormal fusion/ascent***

- Renal Agenesis
- Pelvic Kidney
- Horseshoe Kidney
- Crossed Fused Renal Ectopia

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Renal Agenesis

Unilateral	***Bilateral***
Excellent Prognosis!	LETHAL
AFI in normal range	Severe oligo/anhydramnios
One "lying down" adrenal	Two "lying down" adrenals
+/-Compensatory hypertrophy	May be isolated; Numerous
Asso with VACTERL; cardiac, genital, MSK, GI,	Asso anomalies=15% cardiac 40% non-cardiac
Recurrence risk 1% (nl parents)	Recurrence risk 4%

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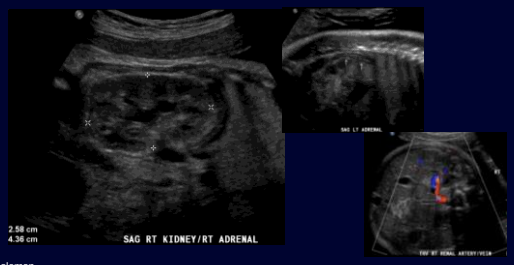
Renal Developmental Variants

Unilateral Renal Agenesis

- ***Solitary RK normal in size & echotexture***



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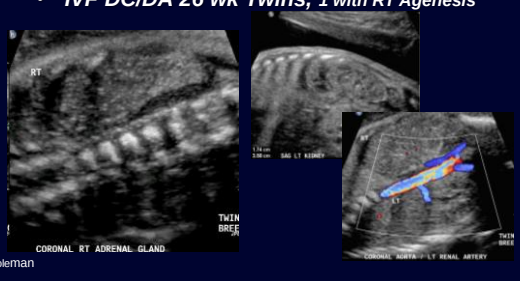
Renal Developmental Variants

Unilateral Renal Agenesis

- ***IVF DC/DA 26 wk Twins, 1 with RT Agenesis***



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Bilateral Renal Agenesis

- Incidence 1:4000 with 2.5:1 male/female ratio
- Imaging Features = anhydramnios, "absent bladder"
- Adrenals may = size of early kidneys; diff shape
- Pulmonary hypoplasia- MUST measure C/T ratio and compare TC to gestational age nomograms
- ↓ Fetal movement → talipes, other joint contractures
- Potter's facies = flat nose, low set ears, micrognathia

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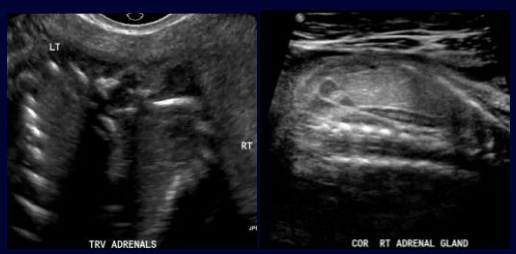
Bilateral Renal Agenesis

"Lying Down" Adrenal Glands



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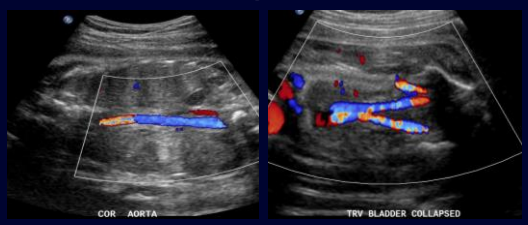
Bilateral Renal Agenesis

Color Doppler for Absent RA's & Collapsed Bladder



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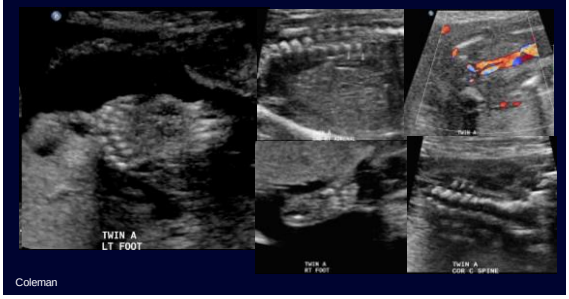
Bilateral Renal Agenesis

- ***VACTERL in Di-Di Twins***



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RENAL ECTOPIA

- No kidney observed in the flank, usually unilateral RA (54%)
- Most common forms of renal ectopia:
  1. Pelvic kidney (37%)
  2. Horseshoe kidneys (5%)
  3. Crossed fused (4%)
- +/-Mild to moderate dilatation 2° obst/reflux
- Abnormal morphology 2° malrotation, dysplasia

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PELVIC KIDNEY

- Empty renal fossa with flattened “lying down” adrenal gland
- Kidney can be missed as often similar to bowel in texture, superior to bladder, ± malrotation
- Normal size or ↑ contralateral kidney
- Color Doppler ⟶ follow RA to pelvis

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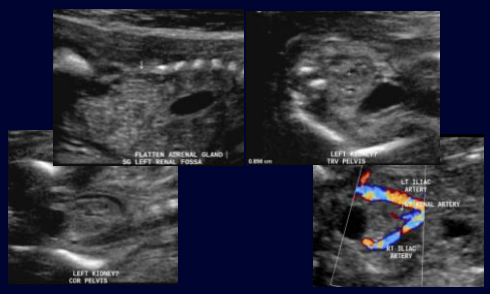
PELVIC KIDNEY

- ***Is One Kidney Truly Missing?***



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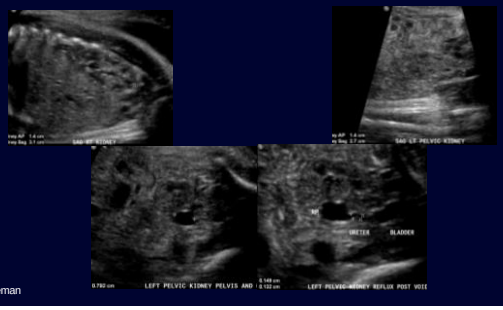
PELVIC KIDNEY

- ***Left Kidney with Vesicoureteral Reflux***



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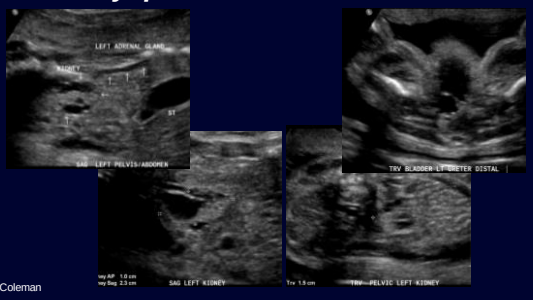
PELVIC KIDNEY

- ***Dysplastic LK with Ureterocele***



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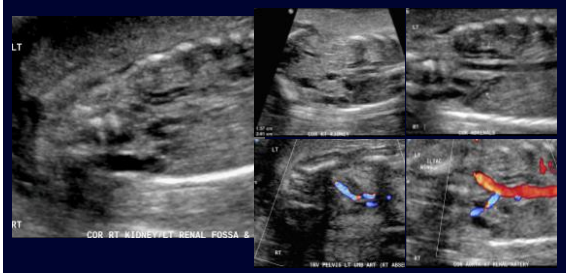
PELVIC KIDNEY

- ***Dysplastic Right Pelvic Kidney & LRA***



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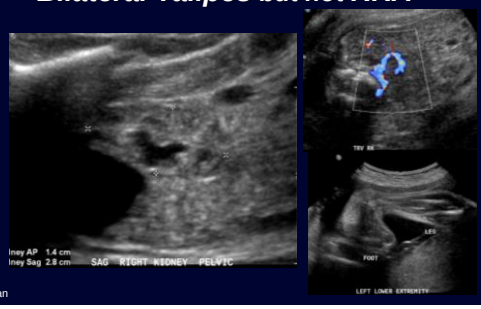
PELVIC KIDNEY

- ***Bilateral Talipes but not RRA***



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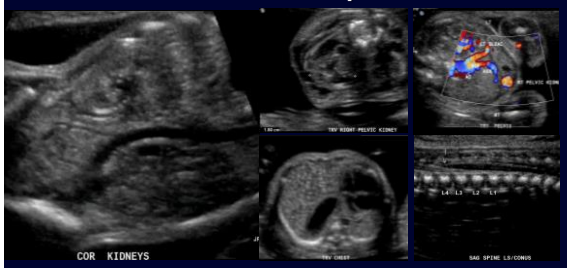
PELVIC KIDNEY

- ***Association with Multiple Anomalies***



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HORSESHOE KIDNEY

- Lower poles connected by isthmus, parenchymal or fibrous; rare in upper pole
- Kidneys more inferior in location 2° ↓ ascent
- Malrotation is common, clue=anterior pelves
- Incidence=1:400 in the general population
- Associations – T_{18}, XO, VACTERL, various syndromes, etc

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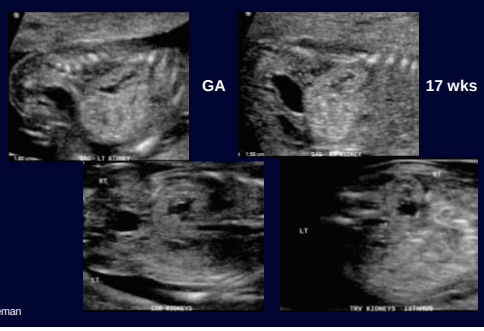
HORSESHOE KIDNEY



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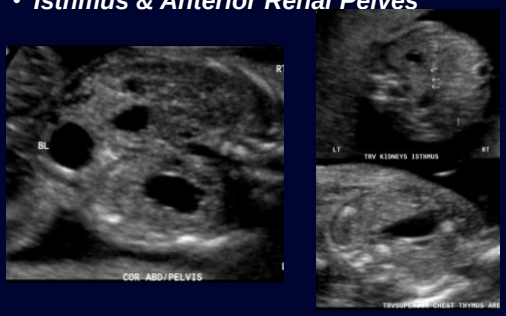
HS KIDNEY in DiGeorge Syndrome

- ***Isthmus & Anterior Renal Pelves***



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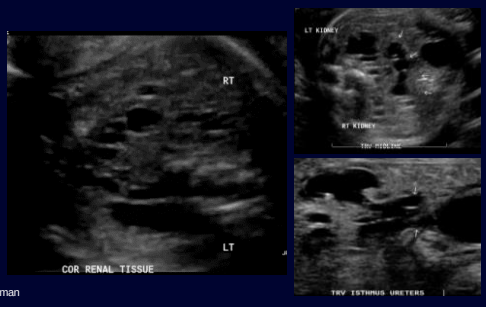
HS KIDNEY in Cloacal Malformation

- ***UTD A2-3 in mono-di Twin A***



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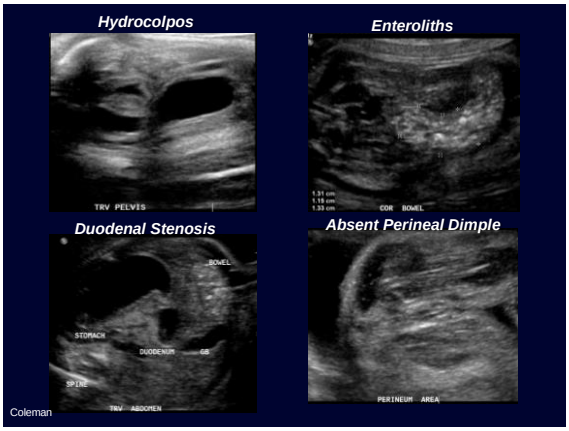

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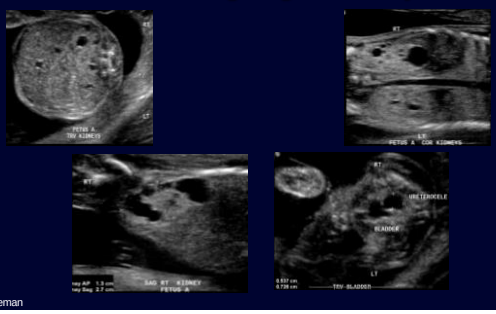
Twins with HS Kidney

- ***MCDK involving Right Moiety***



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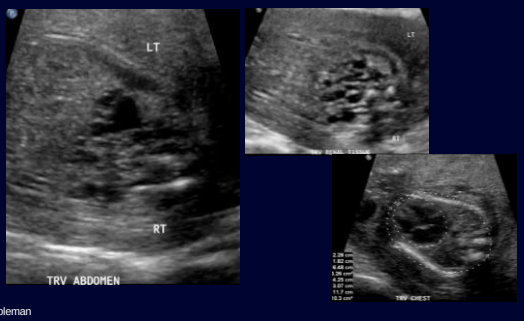
Dysplastic HS KIDNEY



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CROSSED RENAL ECTOPIA

- Ectopic kidney with both on the same side; unilateral empty renal fossa
- Most often L⟶R, 95% fused; freq malrotated
- Kidney appears large and bilobed; ureter crosses the midline to insert at bladder
- Renal arteries, one usu more inferior
- Associations – vertebral segmentation anomalies, spina bifida, sacral agenesis, etc

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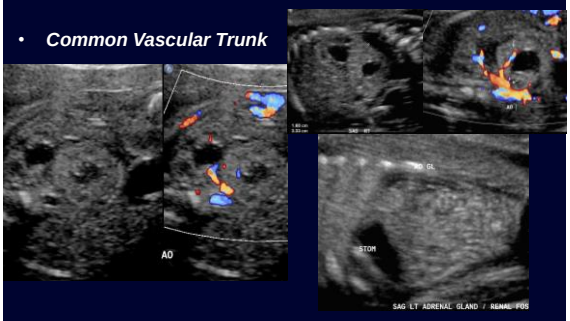
CROSSED RENAL ECTOPIA

- ***Common Vascular Trunk***



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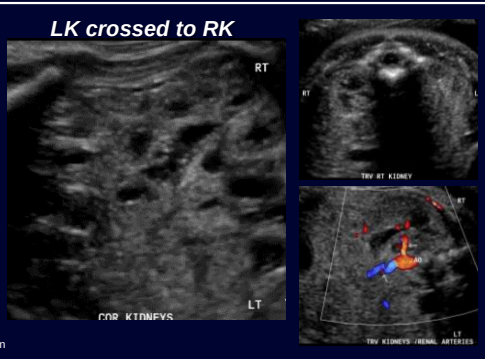
CROSSED RENAL ECTOPIA

LK crossed to RK



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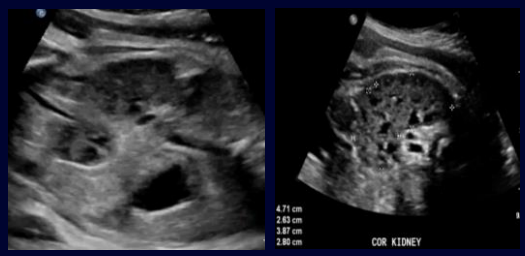
CROSSED RENAL ECTOPIA

***CFRE** asso with MCA*



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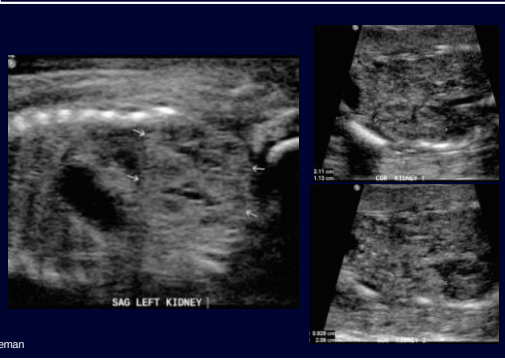
"PANCAKE" KIDNEY



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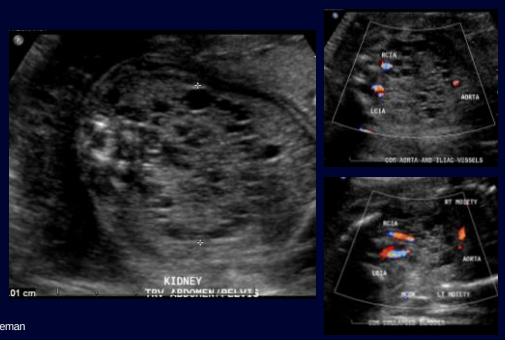
DYSPLASTIC "PANCAKE" KIDNEY



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URINARY TRACT OBSTRUCTION

- Ureteropelvic Junction Obstruction
- Ureterovesical Pathology
- Lower Urinary Tract Obstruction

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TERMINOLOGY

***AVOID** nonspecific terms like:*

- **Hydronephrosis**
- **Pyelectasis**
- **Pelviectasis**
- **Uronephrosis**
- **Urinary tract or pelvic fullness/prominence**

Multidisciplinary consensus on the classification of prenatal and postnatal urinary tract dilation (UTD classification system)

Hiep T. Nguyen [A,C,*], Carol B. Benson [B,*], Bryann Bromley [B], Jeffrey B. Campbell [C,*], Jeanne Chow [E], Beverly Coleman [A,D], Christopher Cooper [B,*], Jude Crino [E], Kassa Darge [B], C.D. Anthony Herndon [B,*], Anthony O. Odibo [F], Michael J.G. Somers [I], Deborah R. Stein [J]

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[E] Society for Maternal-Fetal Medicine (SMFM), Washington, D.C., USA
[F] Society for Pediatric Urology (SPU), Beverly, MA, USA
[G] Society for Pediatric Radiology (SPR), Reston, VA, USA
[H] Society of Radiologists in Ultrasounds (SRU), Reston, VA, USA

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TERMINOLOGY

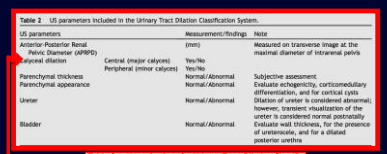

Table 2 US parameters included in the Urinary Tract Dilation Classification System.

US parameters		Measurement/findings	Note
Anterior-Posterior Renal Pelvic Diameter (APRPD)		(mm)	Measured on transverse image at the maximal diameter of intrarenal pelvis
Calyceal dilation	Central (major calyces)	Yes/No	
	Peripheral (minor calyces)	Yes/No	
Parenchymal thickness		Normal/Abnormal	Subjective assessment
Parenchymal appearance		Normal/Abnormal	Evaluate echogenicity, corticomedullary differentiation, and for cortical cysts
Ureter		Normal/Abnormal	Dilation of ureter is considered abnormal; however, transient visualization of the ureter is considered normal postnatally
Bladder		Normal/Abnormal	Evaluate wall thickness, for the presence of ureterocele, and for a dilated posterior urethra

Table 3 Normal values for Urinary Tract Dilation Classification System.

Ultrasound findings	Time at presentation		
	16–27 weeks	≥28 weeks	Postnatal (>48 h)
Anterior-Posterior Renal Pelvis Diameter (APRPD)	<4 mm	<7 mm	<10 mm

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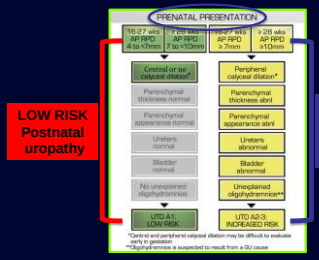
ANTENATAL CLASSIFICATION SYSTEM



LOW RISK Postnatal uropathy

INCREASED RISK Postnatal uropathy

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UPJ OBSTRUCTION

- Most common site for prenatal obstruction, 1:2,000 live births
- Tip: ↑ RP ± calyces with no dilated ureter or bladder
- Several causes – ischemia, stricture, valves, anomalous vessels or crossing bands, high ureteral insertion, muscle abnormality
- Contralateral renal anomaly, 25%; Extra-renal anomalies occur in 10%
- AFV usually normal; "paradoxical" polyhydramnios occurs in 33%
- Bilateral UPJ, incidence ~10%

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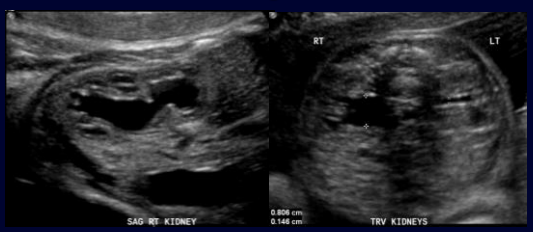
UNILATERAL UPJ

A2-3 High Risk



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UNILATERAL UPJ

A2-3 High Risk, Echogenic Parenchyma

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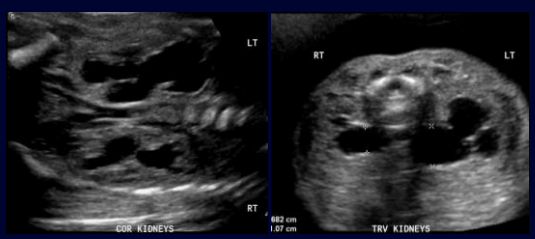
BILATERAL UPJ

A2-3 High Risk, Renal Pelves 7 & 11 mm



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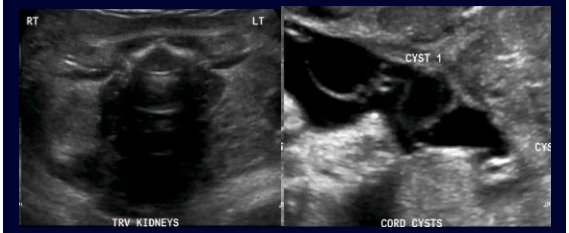
BILATERAL UPJ

A2-3 High Risk, Renal Pelves 9 & 10 mm



Coleman

URETEROVESICLE PATHOLOGY

- Complete ureteral duplication with ectopic ureterocele
- Ectopic ureter with single collecting system
- Ureterovesicle junction stenosis
- Congenital megaureter

Coleman

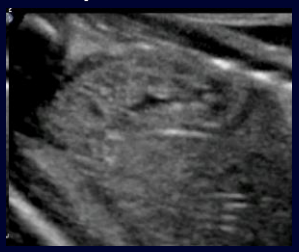
DUPLICATED COLLECTING SYSTEM

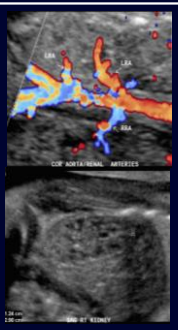
- Imaging features-intervening band of renal tissue separates upper and lower pole pelves; bilateral in 10-20% of cases
- Asym renal size in unilateral cases; length >95% on the affected side
- Upper pole ⟶ ectopic ureter obstructs
- Lower pole ⟶ normotopic ureter refluxes
- Scan bladder several times; check external genitalia

Coleman

DUPLICATED COLLECTING SYSTEM???

- ***Duplicated LRA***

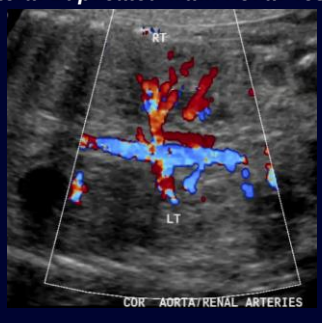




Coleman

DUPLICATED COLLECTING SYSTEM???

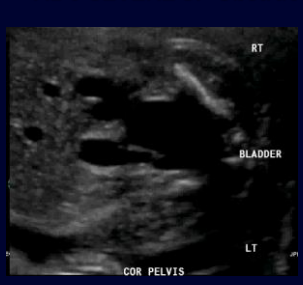
- ***Bilateral Duplicated Main Renal Vessels***

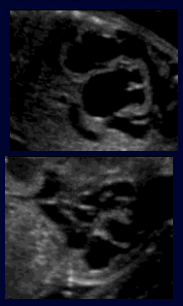


Coleman

DUPLICATED COLLECTING SYSTEM

- ***A2-3 SEVERE UP Obstruction & LP Reflux***

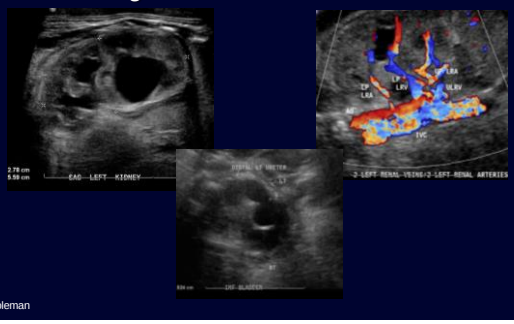




Coleman

UNILATERAL DUPLICATION

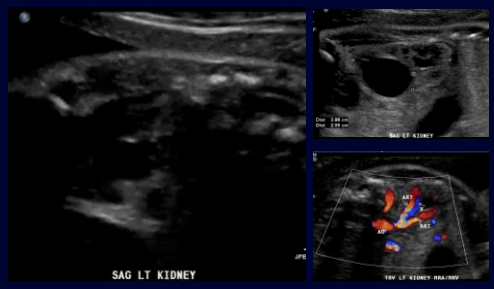
- *A2-3 High Risk LK Obstruction >> Reflux*



Coleman

UNILATERAL DUPLICATION

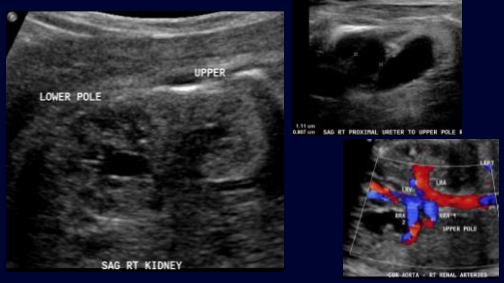
- *A2-3 Severe RK Reflux >>> Obstruction*



Coleman

UNILATERAL DUPLICATION

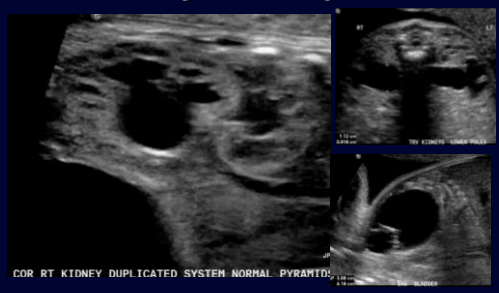
- *A2-3 High Risk with UP Renal Dysplasia*



Coleman

BILATERAL DUPLICATIONS

- *A2-3 with Large Obstructing Ureterocele*



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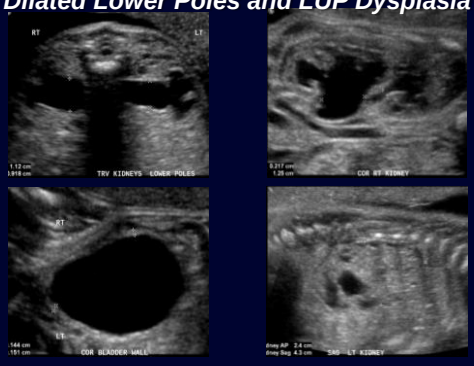
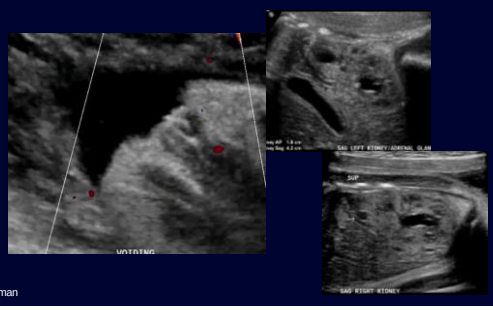
- *Dilated Lower Poles and LUP Dysplasia*



Coleman

BILATERAL DUPLICATIONS

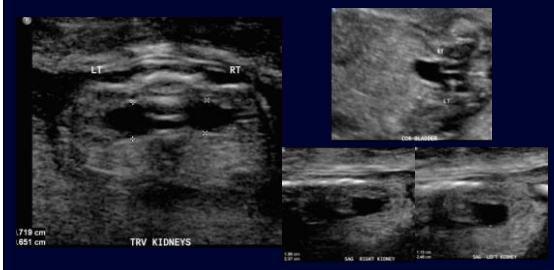
- *A2-3 with Focal OCD in Polar Regions*



Coleman

Bilateral Ectopic Ureters

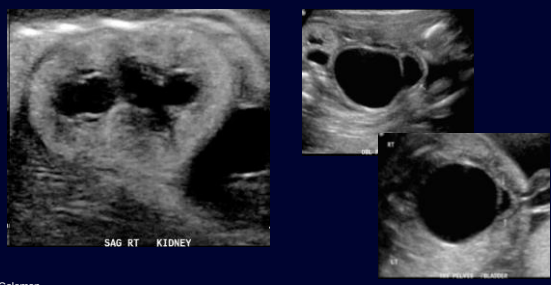
- *Ectopic Ureters without Duplicated Systems*



Coleman

CONGENITAL MEGAURETER

- *A2-3 UTD with URETER >>>>> BLADDER*



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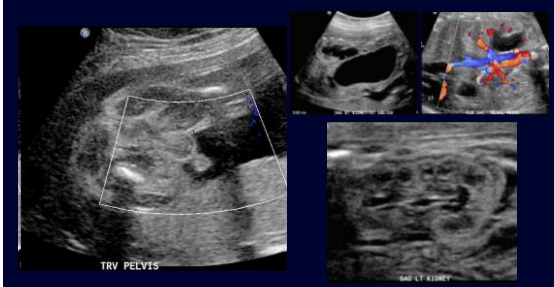
- *In Utero Voiding*



Coleman

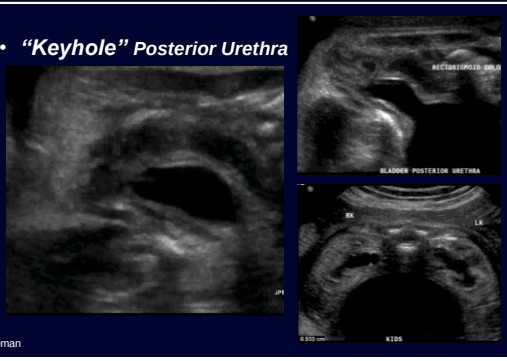
Lower Urinary Tract Obstruction

- Imaging features-=dilated bladder, renal pelvis, calyces and ureters with oligohydramnios; ± dysplasia with cysts; ± urinary ascites
- Degree of collecting system dilatation does not = degree of renal dysplasia
- Causes =posterior/anterior valves, urethral atresia, meatal stenosis, cloacal malformation, etc
- 43% cases have associated anomalies; aneuploidy

Coleman

Lower Urinary Tract Obstruction

- *"Keyhole" Posterior Urethra*



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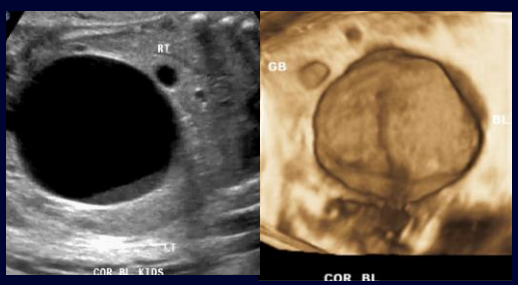
- *Bladder Volume of 80 ml*



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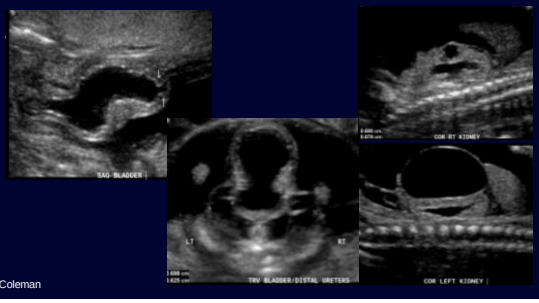
Lower Urinary Tract Obstruction

- ***Patent Urachus with Urinary Ascites***



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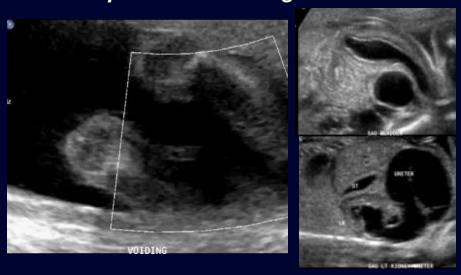
Lower Urinary Tract Obstruction

- ***Incomplete with Voiding & Normal AFI***



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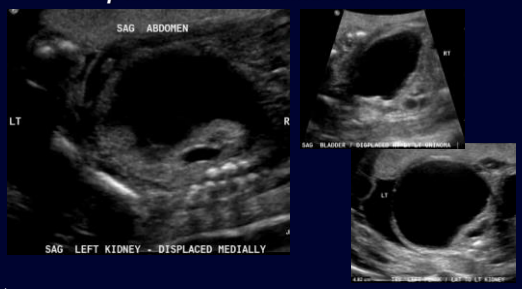
Lower Urinary Tract Obstruction

- ***Ruptured with Urinoma & Normal DVP***



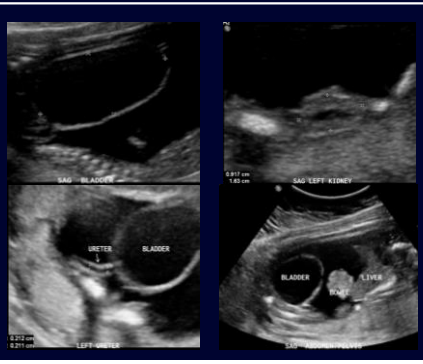
Coleman

CAUSES of MEGACYSTIS

- Posterior urethral valves
- Urethral atresia/stricture
- Prune belly syndrome
- Megalourethra
- Cloacal malformation
- Megacystis-microcolon-intestinal hypoperistalsis syndrome

Coleman

PRUNE BELLY SYNDROME



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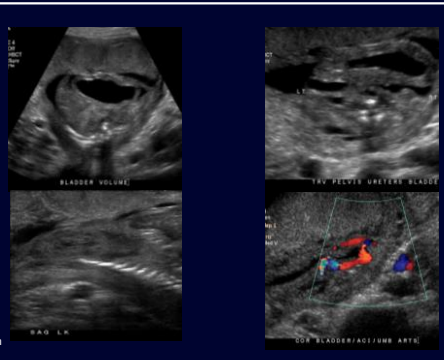
PRUNE BELLY SYNDROME



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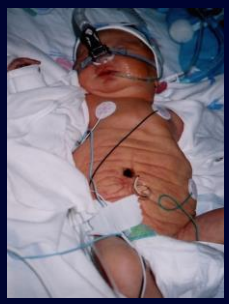

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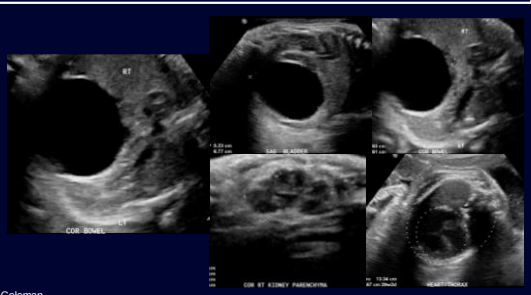
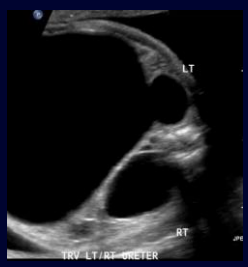
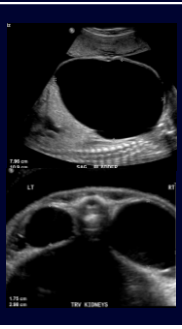
Megacystis Microcolon Intestinal Hypoperistalsis Syndrome



Coleman

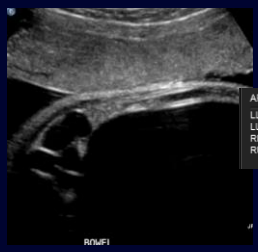
Megacystis Microcolon Intestinal Hypoperistalsis Syndrome

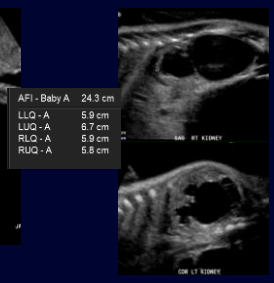




Coleman

Megacystis Microcolon Intestinal Hypoperistalsis Syndrome





Coleman

CYSTIC RENAL DISORDERS

- Obstructive Cystic Dysplasia
- Multicystic Dysplastic Kidney
- Autosomal Dominant Polycystic Renal Disease
- Autosomal Recessive Polycystic Renal Disease

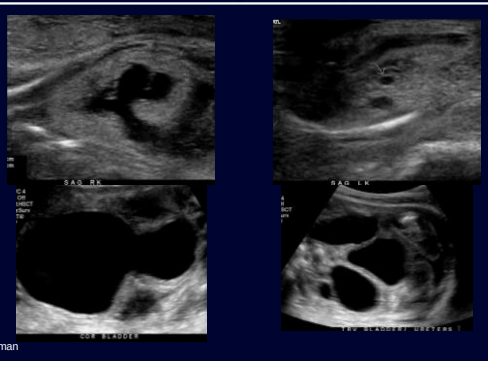
Coleman

Obstructive Cystic Dysplasia

- GU tract obstruction ⟶ renal macrocysts and/or microcysts; most common cause is urethral obstruction; ureteropelvic or ureterovesical junction obstruction less common
- Unilateral, bilateral, rarely segmental
- Renal size may be ↑, ↓ or normal
- Worse prognosis is obstruction ⟶ OCD before 20 wks

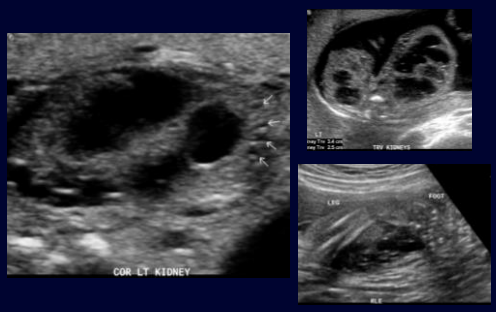
Coleman

Obstructive Cystic Dysplasia



Coleman

Obstructive Cystic Dysplasia



Coleman

Antenatal Predictors of Poor Postnatal Renal Function

Ultrasound	***Fetal Urine***
Severe Oligohydramnios esp if early onset	⇧ Sodium Level (Na+)
	⇧ Calcium Level (Ca^{2+})
Increased renal echogenicity	⇧ B_2-Microglobulin
Renal Cortical Cysts	⇧ Osmolality
Slow Bladder Filling s/p vesicocentesis	***Fetal Blood***
	⇧ B2-Microglobulin

Coleman

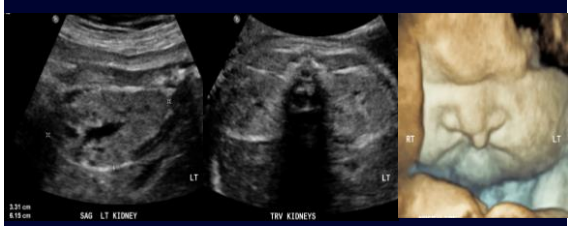
ECHOGENIC KIDNEYS

- Obstructive Cystic Dysplasia
- Hereditary Cystic Diseases
- Nonhereditary Fetal Diseases
  - *Infection, Renal Vein Thrombosis, Toxic Agents*
- Syndromes
  - *T13, Meckel Gruber, Beckwith Wiedemann*
- Chromosomal Anomalies
- Congenital Nephrotic Syndromes
- Maternal & Metabolic Diseases
- Nephroblastomatosis
- Normal Variant

Coleman

TRISOMY 13

- ***Echogenic, Enlarged Kidneys with MCA***



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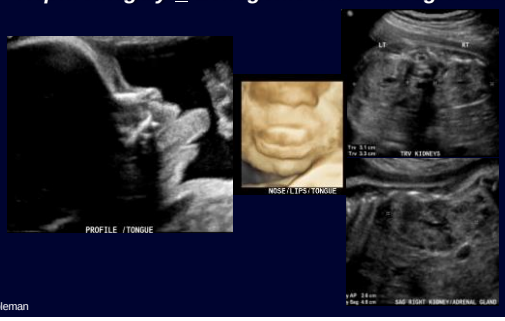
Beckwith Wiedeman Syndrome

- ***Nephromegaly ± Echogenic with Macroglossia***



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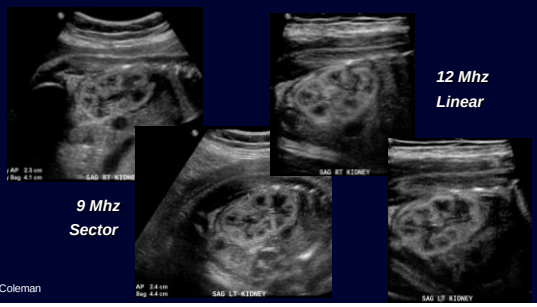
NORMAL VARIANT

- *Echogenic Kidneys with Normal AFI & Renal Size*



Coleman

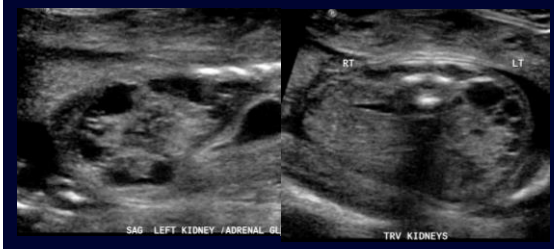
MULTICYSTIC DYSPLASTIC KIDNEY

- Non functioning kidney in 90%; may be late sequelae of renal obstruction
- Variants=pelvic, horseshoe or duplicated kidney
- MCDK usu involutes; rarely complicated by infection, hypertension, Wilm's tumor,
- F/U scans to check fluid, contralateral kidney
- Neonatal work up required in EVERY case!

Coleman

Unilateral MCDK

- *80% of cases, L>R renal involvement*



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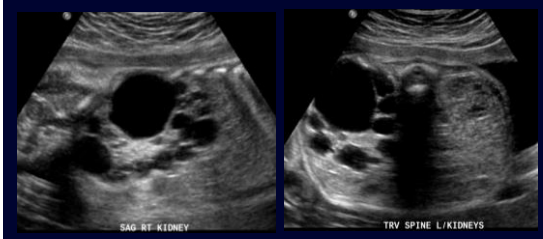
Unilateral MCDK

- *Cysts vary in size & may enlarge dramatically*



Coleman

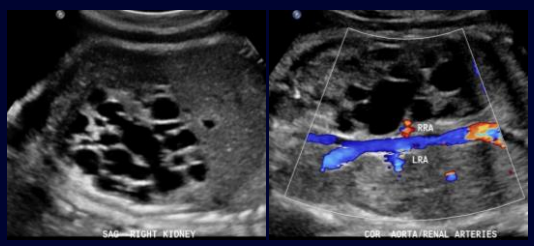
MULTICYSTIC DYSPLASTIC KIDNEY

FEATURE	MCDK	HYDRONEPHROSIS
Cysts	Random distribution	Calyces in a row
Cyst Size	Quite variable	Uniform unless compound
Communication	None	Calyces → infundibulum
Parenchyma	Islands between cysts	Intact, peripheral to calyces
Renal Shape	Absent reniform contour	Contour preserved

Coleman

Unilateral MCDK

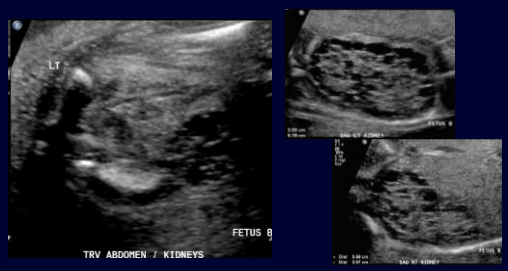
- *Random Cyst Distribution & Patent MRA*



Coleman

Bilateral MCDK

- *Di-di twins with replaced renal parenchyma*



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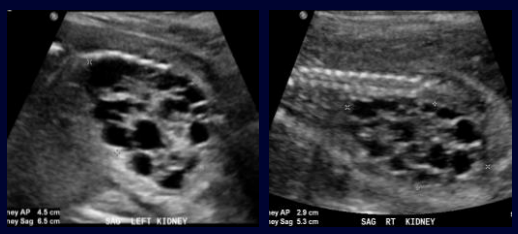
Bilateral MCDK

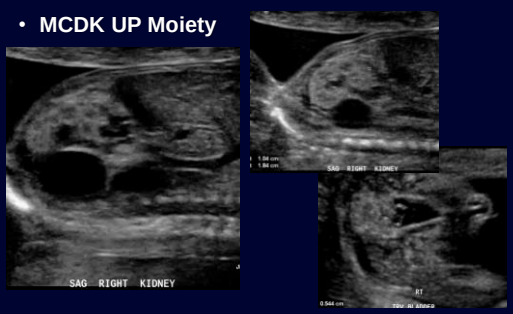
- Completely replaced renal parenchyma=LETHAL



Coleman

Segmental MCDK

- MCDK UP Moiety



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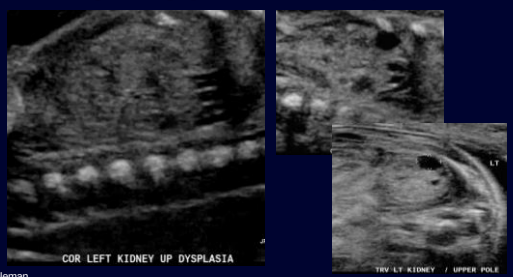
Segmental MCDK

- UP Cystic Dysplasia without duplication



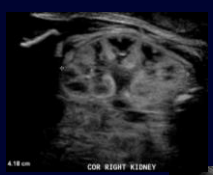
Coleman

Polycystic Kidney Disease

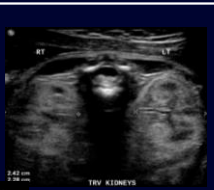
	Autosomal Dominant	Autosomal Recessive
Size	Often Enlarged	Markedly Enlarged
Cysts	Cysts +/- viz, usu late	Most below limits of resolution for US
Parenchyma	Preserved medullary region	Echogenic medullary region
AFI	Normal	Profound oligo, absent fluid
Prognosis	Normal renal fn at birth	Dismal

Coleman

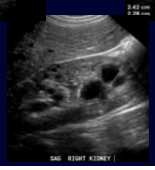
Autosomal Dominant Polycystic Kidney Disease



CMD
No cysts



Maternal

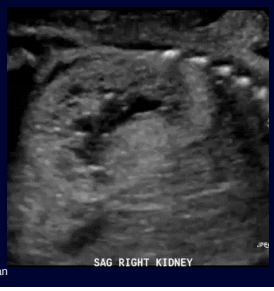


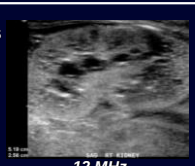
Kidney

Coleman

Autosomal Dominant Polycystic Kidney Disease

- Scattered Parenchymal Cysts





12 MHz

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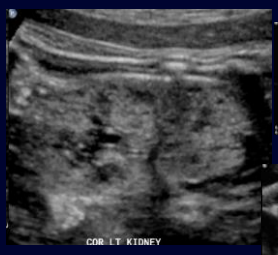
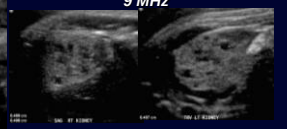
Autosomal Dominant Polycystic Kidney Disease



9 MHz



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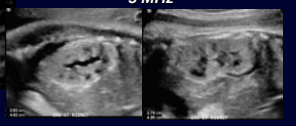

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Autosomal Recessive Polycystic Kidney Disease

- Imaging Features = ↑ hyperechoic kidneys, ± 1-2 mm cysts and echogenic parenchyma; kidneys may look normal up to 20 wks
- Kidney Size > 2 SD above the mean for the expected gestational age
- Oligohydramnios/anhydramnios with non-visualized bladder
- Check for signs of pulmonary hypoplasia such as ↓TC and ↑ C/T ratio without cardiomegaly

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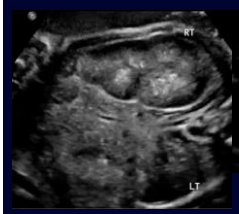
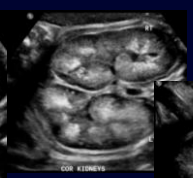
Autosomal Recessive Polycystic Kidney Disease

- *Parents are Known Carriers*





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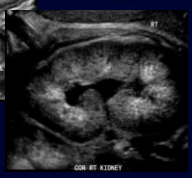
12 MHz



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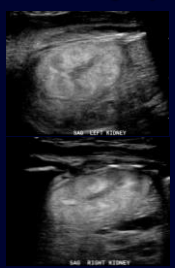
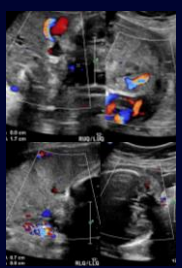
Autosomal Recessive Polycystic Kidney Disease

- *Severe Oligohydramnios*





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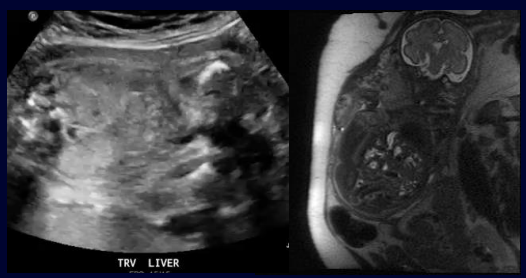
- *Severe Biliary Ectasia*



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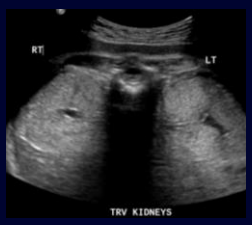
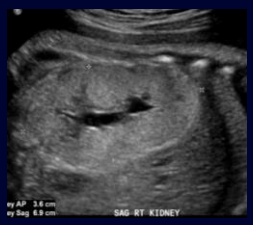
Autosomal Recessive Polycystic Kidney Disease

- **"MIRACLE CHILD"** with normal AFI, micromelia & polydactyly





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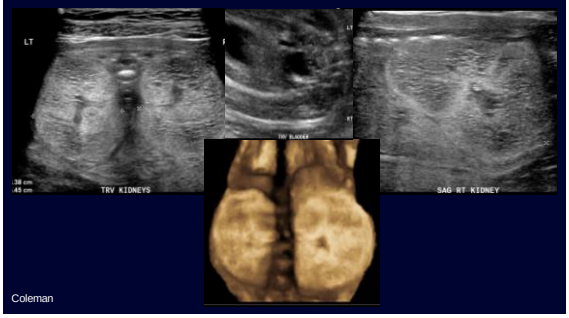
Metabolic Polycystic Kidney Disease



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CONCLUSIONS

- It is important to examine the entire GU tract using a systematic approach with the aim to make the most specific diagnosis possible and/or to narrow the differential as much as possible.
- A thorough knowledge of what is normal or is a normal GU variant is critical in order to minimize patient anxiety.
- Any associated anomalies involving other organs and structures should be carefully imaged and analyzed as GU tract anomalies are common in cases of aneuploidy and various syndromes.

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CONCLUSIONS

- When GU tract obstruction is expected, the most important aspect of the fetal evaluation is to determine the site of the obstruction in addition to assessing the severity of the degree of obstruction.
- A clear understanding of the numerous sonographic features that help distinguish renal cystic disease from obstruction is crucial in making the correct diagnosis in many cases.
- As much information as possible is critical for parents to make an informed decision regarding pregnancy management with all options fully discussed.

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KEY REFERENCES

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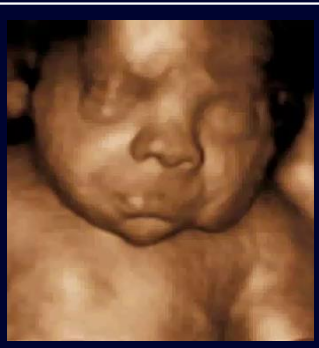
SAD with My Diagnosis



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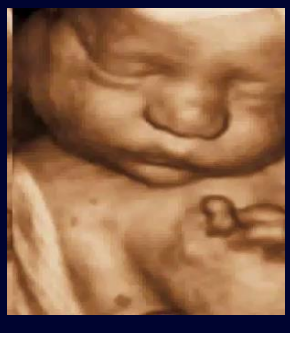
THRILLED with My Diagnosis



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