Evaluation of congenital anomalies of the kidney and urinary tract (CAKUT)

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INTRODUCTION

Congenital anomalies of the kidney and urinary tract (CAKUT) constitute approximately 20 to 30 percent of all anomalies identified in the prenatal period [1]. Routine antenatal ultrasonography during pregnancy detects the majority of CAKUT.

Antenatal screening for CAKUT and the postnatal evaluation of infants diagnosed prenatally with CAKUT are discussed here.

ANTENATAL SCREENING

The majority of kidney malformations are detected antenatally because of the widespread use and sensitivity of fetal ultrasonography. In 2002, a prenatal ultrasound was performed in approximately two-thirds of all live births in the United States. The frequency of congenital anomalies of the kidney and urinary tract (CAKUT) as detected sonographically in unselected populations has been reported to be between 0.1 to 0.7 percent [2-4].

In general, the optimal timing recommended for a screening antenatal ultrasound is between 16 to 20 weeks of gestation because of the following factors at this gestational age:

- There is good visualization of anatomy with a high sensitivity in detecting anomalies.

- It is early enough in the pregnancy to allow completion of prenatal diagnostic procedures (eg, fetal karyotype, additional imaging studies) while legal termination of pregnancy is possible, if desired.

Fetal kidney — Between the 12th and 15th week of gestation, the fetal kidney can be detected by transabdominal ultrasonography [1]. On transverse ultrasound images, normal fetal kidneys are hypoechoic ovoid masses located in the renal fossa on either side of the spine corresponding to the level of the second lumbar vertebrae. The kidney cortex and medulla are distinctly demonstrated by ultrasound by the 20th to 25th week of gestation. Fetal kidney length based upon gestational age is a marker of kidney growth and is illustrated in the table (table 1) [5].

Normally, the fetal ureters are not seen on ultrasonography. However, if they are visualized, it may be indicative of ureteric or bladder obstruction, or vesicoureteral reflux (VUR).

The urine-filled bladder is normally identified at 13 to 15 weeks gestation [6]. Urine in the bladder suggests at least one functioning kidney. The bladder wall is normally thin. If the bladder wall is thick, urethral obstruction such as posterior urethral valves in a male fetus may be present. If the bladder is not seen, consider the diagnosis of bladder exstrophy.

The sensitivity of detecting kidney malformations by antenatal ultrasonography depends upon the gestational age and the skill of the ultrasonographer. In one study, the sensitivity of antenatal screening for kidney malformations was reported as 82 percent at a mean gestational age of 23 weeks [7].

Amniotic fluid — Assessment of amniotic fluid volume and analysis of biochemical markers are used to evaluate fetal kidney function.

Volume — Although fetal urine production begins at nine weeks of gestation, its contribution to amniotic fluid volume becomes significant at the start of the second trimester. By 20 weeks gestation, fetal urine accounts for more than 90 percent of the amniotic fluid volume [7]. Thus, a decrease in amniotic fluid volume (oligohydramnios) at or beyond the 20th week of gestation is an excellent predictor of abnormal fetal kidney function and CAKUT [8].

Severe oligohydramnios due to CAKUT either involves both kidneys or occurs in a solitary kidney in the fetus. Bilateral kidney agenesis (RA) or severe dysgenesis, bilateral ureteric obstruction, or obstruction of the bladder outlet or urethra can result in severe oligohydramnios as early as 18 weeks gestation. Because an adequate amniotic fluid volume is critical for lung development, severe oligohydramnios due to abnormal fetal kidney function in the second trimester can result in lung hypoplasia, a potentially fatal disorder [9,10]. In its most severe form, this

sequence of events results in Potter's syndrome, which consists of a typical facial appearance characterized by pseudoepicanthus, recessed chin, posteriorly rotated, flattened ears and flattened nose, decreased fetal movement, musculoskeletal features including clubfoot and clubhand, hip dislocation and joint contractures, and pulmonary hypoplasia (picture 1).

The assessment of amniotic fluid volume is discussed in greater detail separately. (See "Assessment of amniotic fluid volume".)

Analysis — Although oligohydramnios is the most reliable predictor of abnormal fetal kidney function, its absence does not assure normal fetal kidney function. Because amniotic fluid is predominantly composed of fetal urine, measurement of biochemical markers contained in amniotic fluid (fetal urine) can be used to assess fetal kidney function.

With increasing gestational age, renal tubular resorptive function increases. As a result, the urinary levels of sodium and beta-2-microglobulin decrease with increasing gestational age, while urine osmolality increases [11,12]. Impaired resorption is seen in fetuses with bilateral renal dysplasia or severe bilateral obstructive uropathy resulting in abnormal urinary levels of electrolytes, beta-2-microglobulin, and osmolality [13,14].

In general, high urinary electrolyte excretion, sodium and chloride concentration greater than 90 mEq/L (90 mmol/L), and urinary osmolality less than 210 mosmol/kg H2O (210 mmol/kg H2O) in the amniotic fluid are indicative of fetal renal tubular impairment and poor kidney prognosis [15]. When analyzing the results, it is important to use gestation specific cut-offs because with increasing gestational age, renal tubular resorptive function increases [16]. However, in a systematic review of fetal urine analysis, none of the urinary tests provide significant clinical accuracy to correctly predict poor postnatal kidney function [16].

Tests to assess fetal glomerular function include fetal serum measurement of cystatin C and beta-2-microglobin [17]. However, these tests are not used in clinical practice because of technical difficulties in obtaining fetal blood.

In addition, amniocentesis can be used to detect chromosomal abnormalities often associated with kidney defects such as trisomy 18 [18]. (See "Diagnostic amniocentesis".)

Management — Counseling of families with fetuses with CAKUT should be universally available. If the fetal prognosis is poor, as determined by severe bilateral disease, bilateral RA, oligohydramnios, or unfavorable amniotic fluid analysis, legal termination, if possible, can be offered.

In all other cases, continued counseling throughout the pregnancy including discussion of postnatal management is required. In particular, discussion with parents regarding their wishes on the level of support given to offspring with severe oligohydramnios, who are at risk for lung hypoplasia that may be incompatible with life, is helpful in establishing guidelines for initial postnatal care.

In utero intervention — Intervening in pregnancy to attempt definitive or temporary correction of fetal kidney anomalies would be reasonable if one could prevent the development of renal dysplasia, kidney scarring, chronic kidney failure, or the occurrence of pulmonary hypoplasia [19-22]. Although there have been case series of antenatal surgery in fetuses with severe hydronephrosis and oligohydramnios, this intervention has not been shown to improve kidney outcome. These procedures may increase the amount of amniotic fluid, thus potentially improving lung development and survival rate. In these rare cases, the procedure should only be performed in select centers with expertise and in infants with severe bilateral hydronephrosis, absent of severe renal parenchymal or cystic disease, favorable urinary electrolyte levels and osmolality, and normal karyotype.

The prenatal care of the fetus with severe oligo- or anhydramnios or those with fetal hydronephrosis are discussed separately. (See "Oligohydramnios: Etiology, diagnosis, and management in singleton gestations", section on 'Postdiagnostic evaluation' and "Fetal hydronephrosis: Etiology and prenatal management" and "Oligohydramnios: Etiology, diagnosis, and management in singleton gestations".)

POSTNATAL EVALUATION

History and physical examination — After delivery, a detailed maternal and pregnancy history, and careful physical examination should be performed in all infants with an antenatally detected kidney malformation.

- Pulmonary evaluation especially in fetuses with severe oligohydramnios who are at risk for lung hypoplasia. In these severely affected neonates, decisions on the use of intensive supportive care are often made in the delivery room. If at all possible, prior discussion with the family regarding management decisions is helpful in establishing guidelines for initial postnatal care.

- Examination of the abdomen to detect the presence of a mass that could represent an enlarged kidney due to obstructive uropathy or multicystic dysplastic kidney (MCDK).

- A palpable bladder in a male infant, especially after voiding, may suggest bladder outlet obstruction such as the presence of posterior urethral valves.

- A male infant with prune belly syndrome (also known as Eagle-Barrett syndrome) will have deficient abdominal wall musculature and undescended nonpalpable testes. The presence of associated anomalies should be investigated. (See "Assessment of the newborn infant" and "Prune-belly syndrome".)

- The presence of outer ear abnormalities is associated with an increased risk of congenital anomalies of the kidney and urinary tract (CAKUT). (See "Congenital anomalies of the ear", section on 'Association with kidney anomalies'.)

- A single umbilical artery is associated with an increased risk of CAKUT, particularly vesicoureteral reflux (VUR). (See "Assessment of the newborn infant", section on 'Umbilical cord'.)

- Genital examination for female infants because müllerian defects (eg, uterine didelphys and/or vaginal duplication) are common since the Wolffian and müllerian ducts are contiguous [23]. However, detection of müllerian defects by physical examination is limited. (See "Congenital uterine anomalies: Clinical manifestations and diagnosis", section on 'Associated anomalies in other organ systems'.)

Timing of postnatal renal studies — Postnatal evaluation by ultrasonography is performed within the first 24 hours of life for neonates with bilateral involvement, a solitary kidney, and/or a history of oligohydramnios because they are at increased risk for a serious kidney anomaly that may be amenable to intervention. As an example, a distended bladder with thickened bladder wall and bilateral hydronephrosis may be caused by posterior urethral valves (PUV) that require surgical intervention.

Kidney growth can be assessed by measuring the kidney length with the infant in the prone position.

In general, conditions, which have unilateral involvement, do not need immediate attention. Kidney ultrasonography is recommended after the infant returns to birth weight (after 48 hours of age and within the first week of life) to ensure volume repletion and increased urine output as renal plasma flow and glomerular filtration rate (GFR) rise in the first 48 hours of life [24]. Thus, in an infant with hydronephrosis, the level of severity might be underestimated if ultrasonography is performed before 48 hours of life. In addition, hydronephrosis may not be present on kidney ultrasonography in an infant with obstructive uropathy that has intrinsic anuric kidney failure.

Imaging should also be performed for other relevant organ systems to detect associated anomalies (eg, müllerian defects in female infants).

Other diagnostic tests — Diagnostic testing includes measurement of serum creatinine to assess kidney function and other radiologic tests that can be useful in determining underlying kidney pathology, kidney function, and the presence of other urological anomalies.

Serum creatinine — Estimation of the kidney function by measurement of the serum creatinine concentration is used clinically to assess the presence and extent of kidney impairment and to follow the infant's kidney function. Measurement of creatinine should be considered when there is bilateral kidney disease or an affected solitary kidney.

The serum creatinine concentration at birth is similar to that in the mother (usually ≤1.0 mg/dL [88 micromol/L]). It declines to normal values (serum creatinine 0.3 to 0.5 mg/dL [27 to 44 micromol/L]) in approximately one week in term infants and two to three weeks in preterm infants. Serum creatinine should be measured after the first 24 hours to avoid overestimation of creatinine that may be high and reflective of maternal creatinine values. (See "Neonatal acute kidney injury: Pathogenesis, etiology, clinical presentation, and diagnosis", section on 'Serum creatinine'.)

Voiding cystourethrography — Voiding cystourethrography (VCUG) is the definitive method for assessment of the lower urinary tract. It requires urethral catheterization and injection of a contrast agent. Indications include any suspicion of a thick-walled bladder, ureteric dilatation, hydronephrosis, and in male infants, any urethral pathology (eg, posterior urethral valves) detected by ultrasound.

VCUG is the definitive study to demonstrate VUR, which often accompanies other CAKUT (eg, multicystic dysplastic, hypoplastic, or ectopic kidney). VUR is detected in as many as 15 percent of infants with antenatal hydronephrosis. Infants with postnatal hydronephrosis (without evidence of another cause of the hydronephrosis) are candidates to undergo VCUG. The decision to perform a VCUG depends on the severity of the hydronephrosis and how a finding of VUR will determine clinical management. Infants who are at high risk for VUR should be given prophylactic antibiotics. Infants with significant VUR demonstrated by VCUG are typically continued on prophylactic antibiotics. (See "Management of vesicoureteral reflux".)

Dynamic renal scan — Dynamic radionuclide scans assess kidney excretory function and utilize 99mTc-mercaptotriglycylglycine (MAG-3 or MAG3) as a radiotracer. MAG-3 is injected intravenously, absorbed from the blood by the proximal tubules, and secreted into the tubular lumen and then into the bladder.

Dynamic renal scans are used to differentiate between obstructive versus nonobstructive causes of hydronephrosis. (See "Fetal hydronephrosis: Postnatal management", section on 'Diuretic renography'.)

Static renal scan — Static radionuclide scan is most useful for detection of focal renal parenchymal abnormalities and the differential assessment of kidney function between the two kidneys. Technetium Tc-99m succimer (dimercaptosuccinic acid; DMSA) is used as the radiotracer. Following intravenous (IV) injection, DMSA is taken up by proximal tubular cells with only a minimal amount excreted in the urine, so the tracer accumulates over several hours within the tubule, providing a static image of functioning nephrons. There is no minimum age at which a DMSA scan can be performed. However, the quality of both dynamic and static radionuclide scans improves with kidney maturity.

DMSA scan is used to assess whether a suspected kidney lesion contains normal-functioning nephrons, the differential function of the two kidneys and/or detection of kidney scarring. If possible, the DMSA renal scan should be performed four to six weeks after birth in a full-term infant as poor function measured by DMSA renal scan does not necessarily imply irreversible damage in a neonate, but may be a reflection of immature kidney function. A repeat scan should be undertaken over three months of age or following therapeutic intervention because there may be recovery of kidney function after surgical intervention.

Serial ultrasound — Serial ultrasounds are used to assess compensatory kidney growth of unaffected kidneys in patients with unilateral CAKUT. In our practice, growth is monitored by ultrasound scans every six months in the first year of life, and then yearly or every second year until puberty is completed. In addition, serial ultrasounds are used to monitor for progressive hydronephrosis in patients with mild/moderate obstructive uropathy or changes in the affected kidneys (eg, size of multicystic dysplastic kidney).

No role for routine genetic testing — The yield for routine genetic testing is low. For example, in a retrospective study, genetic mutation was detected in only 14 of 66 patients (approximately 20 percent) [25]. No mutations were detected in the six patients with urinary tract obstruction, whereas patients with bilateral kidney lesions were more likely to have an underlying genetic mutation. However, diagnosing a genetic condition did not alter long-term kidney prognosis. As a result, genetic testing is not recommended for routine evaluation and should be reserved for research purposes.

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Prenatal hydronephrosis (The Basics)")

SUMMARY

Congenital anomalies of the kidney and urinary tract (CAKUT) constitute approximately 20 to 30 percent of all anomalies identified in the prenatal period.

- Routine antenatal ultrasonography during pregnancy detects CAKUT in most affected patients.
- The fetal kidney can be detected by the 12th to 15th week of gestation, however, the kidney cortex and medulla are not distinctly differentiated from one another until the 20th to 25th week of gestation. Fetal kidney length based upon gestational age is a marker of kidney growth (table 1). (See 'Fetal kidney' above.)
- By 20 weeks gestation, fetal urine accounts for more than 90 percent of the amniotic volume. A decrease in amniotic volume (oligohydramnios) at or beyond 20 weeks gestation is an excellent predictor of abnormal fetal kidney function and indicates bilateral fetal kidney dysfunction or, rarely, a poorly functioning solitary kidney. (See 'Volume' above.)
- Biochemical analysis of amniotic fluid is useful in assessing fetal kidney function. Sodium and chloride concentration >90 mEq/L (90 mmol/L), and urinary osmolality less than 210 mosmol/kg H2O (210 mmol/kg H2O) in the amniotic fluid is associated with a poor fetal kidney prognosis. Amniotic beta-2-microglobulin concentration ≥6 mg/L is also associated with severe kidney damage. (See 'Analysis' above.)

- Counseling should be available to all families with fetuses who have a CAKUT. If the fetal prognosis is poor, legal termination, if appropriate and available, can be offered. In all other cases, continued counseling including discussion of postnatal management is required. (See 'Management' above.)

- Initial postnatal evaluation by ultrasonography is performed within the first 24 hours of life for infants with bilateral involvement, a solitary affected kidney, and/or a history of oligohydramnios. Infants with unilateral involvement should be evaluated after 48 hours of life. (See 'Timing of postnatal renal studies' above.)

- Further postnatal diagnostic testing includes measurement of serum creatinine to assess kidney function in cases of bilateral kidney disease or in an affected solitary kidney, and the following radiological studies:

  - Voiding cystourethrography (VCUG) is indicated in male infants suspected to have urethral pathology (eg, posterior urethral valves). It detects vesicoureteral reflux and abnormalities of the male urethra. VCUG may be indicated in infants with hydronephrosis and without any other evidence of kidney-urinary tract abnormality depending on the severity of the hydronephrosis. (See "Fetal hydronephrosis: Postnatal management", section on 'Persistent moderate to severe hydronephrosis'.)

  - 99mTc-mercaptotriglycylglycine (MAG-3) renal scan can be used to differentiate between obstructive versus nonobstructive causes of hydronephrosis. (See 'Dynamic renal scan' above.)

  - Technetium Tc-99m succimer (dimercaptosuccinic acid; DMSA) is used to detect ectopic kidney tissue and/or to determine differential function between the two kidneys, or detect renal dysplasia or scarring. (See 'Static renal scan' above.)

  - Serial ultrasounds are used to monitor compensatory kidney growth of unaffected kidneys in patients with unilateral CAKUT, progressive hydronephrosis in patients with mild/moderate obstructive uropathy, or changes in the affected kidneys. (See 'Other diagnostic tests' above.)

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REFERENCES

1. Dugoff L. Ultrasound diagnosis of structural abnormalities in the first trimester. Prenat Diagn 2002; 22:316.
2. Madarikan BA, Hayward C, Roberts GM, Lari J. Clinical outcome of fetal uropathy. Arch Dis Child 1988; 63:961.
3. Livera LN, Brookfield DS, Egginton JA, Hawnaur JM. Antenatal ultrasonography to detect fetal renal abnormalities: a prospective screening programme. BMJ 1989; 298:1421.
4. Sanghvi KP, Merchant RH, Gondhalekar A, et al. Antenatal diagnosis of congenital renal malformations using ultrasound. J Trop Pediatr 1998; 44:235.
5. Cohen HL, Cooper J, Eisenberg P, et al. Normal length of fetal kidneys: sonographic study in 397 obstetric patients. AJR Am J Roentgenol 1991; 157:545.
6. Cohen HL, Kravets F, Zucconi W, et al. Congenital abnormalities of the genitourinary system. Semin Roentgenol 2004; 39:282.
7. Vanderheyden T, Kumar S, Fisk NM. Fetal renal impairment. Semin Neonatol 2003; 8:279.
8. Leibovitch L, Kuint J, Rosenfeld E, et al. Short-term outcome among term singleton infants with intrapartum oligohydramnios. Acta Paediatr 2012; 101:727.
9. POTTER EL. Bilateral renal agenesis. J Pediatr 1946; 29:68.
10. Mehler K, Beck BB, Kaul I, et al. Respiratory and general outcome in neonates with renal oligohydramnios--a single-centre experience. Nephrol Dial Transplant 2011; 26:3514.
11. Nicolini U, Fisk NM, Rodeck CH, Beacham J. Fetal urine biochemistry: an index of renal maturation and dysfunction. Br J Obstet Gynaecol 1992; 99:46.
12. Muller F, Dommergues M, Bussières L, et al. Development of human renal function: reference intervals for 10 biochemical markers in fetal urine. Clin Chem 1996; 42:1855.
13. Muller F, Dommergues M, Mandelbrot L, et al. Fetal urinary biochemistry predicts postnatal renal function in children with bilateral obstructive uropathies. Obstet Gynecol 1993; 82:813.
14. Abdennadher W, Chalouhi G, Dreux S, et al. Fetal urine biochemistry at 13-23 weeks of gestation in lower urinary tract obstruction: criteria for in-utero treatment. Ultrasound Obstet Gynecol 2015; 46:306.
15. Glick PL, Harrison MR, Golbus MS, et al. Management of the fetus with congenital hydronephrosis II: Prognostic criteria and selection for treatment. J Pediatr Surg 1985;

20:376.

16. Morris RK, Quinlan-Jones E, Kilby MD, Khan KS. Systematic review of accuracy of fetal urine analysis to predict poor postnatal renal function in cases of congenital urinary tract obstruction. Prenat Diagn 2007; 27:900.
17. Bökenkamp A, Dieterich C, Dressler F, et al. Fetal serum concentrations of cystatin C and beta2-microglobulin as predictors of postnatal kidney function. Am J Obstet Gynecol 2001; 185:468.
18. Limwongse C, Cassidy SB. Syndromes and malformations of the urinary tract. In: Pediatric Nephrology, 5th ed, Avner ED, Harmon WE, Niaudet P (Eds), Williams & Wilkins, Philadelphi a 2004. p.93.
19. Mandell J, Blyth BR, Peters CA, et al. Structural genitourinary defects detected in utero. Radiology 1991; 178:193.
20. Josephson S. Suspected pyelo-ureteral junction obstruction in the fetus: when to do what? II. Experimental viewpoints. Eur Urol 1991; 19:132.
21. Elder JS, Duckett JW Jr, Snyder HM. Intervention for fetal obstructive uropathy: has it been effective? Lancet 1987; 2:1007.
22. Freedman AL, Johnson MP, Smith CA, et al. Long-term outcome in children after antenatal intervention for obstructive uropathies. Lancet 1999; 354:374.
23. O'Flynn O'Brien KL, Bhatia V, Homafar M, et al. The Prevalence of Müllerian Anomalies in Women with a Diagnosed Renal Anomaly. J Pediatr Adolesc Gynecol 2021; 34:154.
24. Fawer CL, Torrado A, Guignard JP. Maturation of renal function in full-term and premature neonates. Helv Paediatr Acta 1979; 34:11.
25. Ishiwa S, Sato M, Morisada N, et al. Association between the clinical presentation of congenital anomalies of the kidney and urinary tract (CAKUT) and gene mutations: an analysis of 66 patients at a single institution. Pediatr Nephrol 2019; 34:1457.

Topic 6106 Version 25.0

GRAPHICS

Normal fetal renal lengths

Gestational age, weeks	Mean kidney length, cm	95% CI, cm
18	2.2	1.6 - 2.8
19	2.3	1.5 - 3.1
20	2.6	1.8 - 3.4
21	2.7	2.1 - 3.2
22	2.7	2.0 - 3.4
23	3.0	2.2 - 3.7
24	3.1	1.9 - 4.4
25	3.3	2.5 - 4.2
26	3.4	2.4 - 4.4
27	3.5	2.7 - 4.4
28	3.4	2.6 - 4.2
29	3.6	2.3 - 4.8
30	3.8	2.9 - 4.6
31	3.7	2.8 - 4.6
32	4.1	3.1 - 5.1
33	4.0	3.3 - 4.7
34	4.2	3.3 - 5.0
35	4.2	3.2 - 5.2
36	4.2	3.3 - 5.0
37	4.2	3.3 - 5.1
38	4.4	3.2 - 5.6
39	4.2	3.5 - 4.8
40	4.3	3.2 - 5.3
41	4.5	3.9 - 5.1

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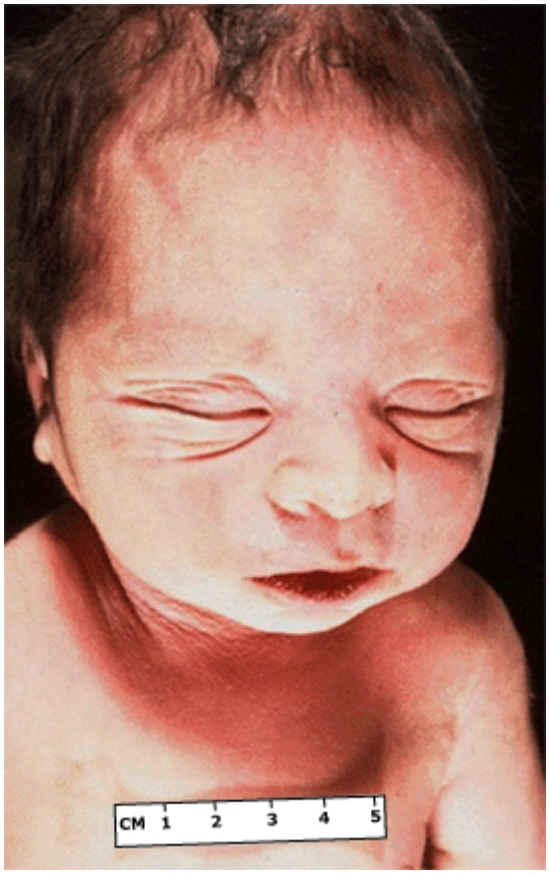
Graphic 74636 Version 11.0

Facial appearance of a patient with Potter sequence



Typical facial appearance observed in Potter sequence. Characteristic abnormalities include pseudoepicanthus, recessed chin, posteriorly rotated, flattened ears, and flattened nose.

Graphic 72847 Version 3.0

Contributor Disclosures

Norman D Rosenblum, MD, FRCP No relevant financial relationship(s) with ineligible companies to disclose. **Tej K Mattoo, MD, DCH, FRCP** Consultant/Advisory Boards: Alnylam Pharmaceuticals [Primary hyperoxaluria]. All of the relevant financial relationships listed have been mitigated. **Laurence S Baskin, MD, FAAP** No relevant financial relationship(s) with ineligible companies to disclose. **Laurie Wilkie, MD, MS** No relevant financial relationship(s) with ineligible companies to disclose.

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