

Screening of Fetal Thorax and Abdomen

Kazuo Maeda

Professor (Emeritus), Department of Obstetrics and Gynecology, Tottori University Medical School, Yonago, Japan

Correspondence: Kazuo Maeda, 3-125, Nadamachi, Yonago, Tottoriken 683-0835, Japan, Phone and Fax: 81-859-22-6856
e-mail: maedak@mocha.ocn.ne.jp

ABSTRACT

Ultrasonic screening was studied in the congenital and acquired disorders of fetal thorax cavity, diaphragm, lung and heart; those of fetal peritoneal cavity, liver, alimentary tract, abdominal wall; those of fetal kidneys, ureter and urinary bladder; and those of fetal genital organs.

Keywords: Ultrasound, Screening, Fetus, Thorax, Abdomen, Hydrothorax, Lung, Diaphragm, CHD, Arrhythmia, Intestine, Liver, Kidney, Ureter, Bladder, Genitalia.

FETAL THORAX

Fetal thorax abnormalities are diagnosed by 2D, 3D, 4D ultrasound imaging, color Doppler flow mapping and pulsed Doppler flow velocity waveform. Abnormal number of fetal ribs with associated anomalies were reported.^{1,2}

Deformity of Fetal Thorax

Chondroectodermal dysplasia, thanatophoric dwarfism, various chondrodystrophy or dwarfism develop fetal thorax deformity, including bell-shape thorax with hypoplastic lung.

Hypoplastic Fetal Lung

Fetal lung volume is determined by using volume measuring function of 3D ultrasound.^{3,4}

Immature Fetal Lung

Fetal lung immaturity diagnosis is significant in preterm labor to predict neonatal respiratory distress syndrome. Although fetal lung maturity has been estimated by the chemical or physical properties of amniotic fluid obtained by amniocentesis, it is noninvasively estimated with corrected gestational weeks with crown-rump length (CRL), ultrasonically estimated fetal weight, mature placental image, normal fetal arterial Doppler flow velocity wave form, fetal nasal color Doppler respiratory wave or ultrasonic tissue characterization, e.g. B-mode image texture, special mean gray level or the gray level histogram width (GLHW), where the product of lung/liver GLHW ratio and corrected gestational weeks detected 96% of immature fetal lungs.⁵

Hydrothorax, Pleural Effusion

Fetal pleural effusion in the thorax and ascites of abdomen of fetus are the sign of fetal immunological and nonimmunological hydrops. Ultrasonic image shows dark echoless space surrounding the fetal lung (Fig. 1).

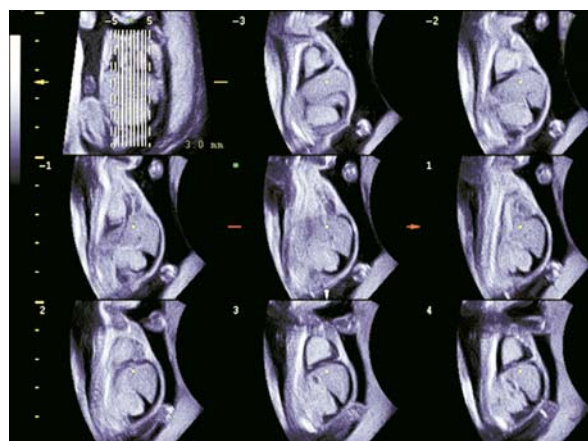


Fig. 1: Pleural effusion and ascites at 19 weeks of gestation. Tomographic ultrasound imaging of fetal thorax and abdomen. Lung and liver float due to pleural effusion and ascites in the thoracic and abdominal spaces respectively. This was 45X/46XY mosaicism case and both pleural effusion and ascites repeatedly appeared and resolved spontaneously during pregnancy (Courtesy: Dr. Pooh, CRIFM PMC)

Chylothorax

B-mode change of the chylothorax is similar to pleural effusion while usually it does not accompany abdominal ascites because it is caused by local abnormality of lymphatic system, where the lymph is confirmed by the puncture and aspiration of accumulated fluid in fetal thorax.

Congenital Cystic Adenomatoid Malformation (CCAM)

The abnormality is the most frequent fetal lung disease and caused by the proliferation of terminal bronchioli. The development is usually in unilateral fetal lung. CCAM is classified into three types according to cyst size. Type I is the large cyst formation (Fig. 2), Type II is accompanied by small cysts (< 2 cm), and Type III formed by too small cysts to recognize by ultrasound (Fig. 3) or MRI (Fig. 4) as a bright

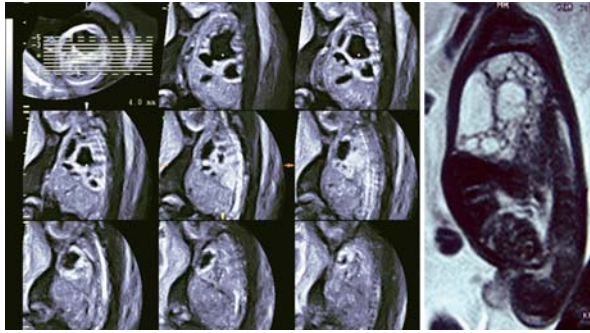


Fig. 2: CCAM (congenital cystic adenomatoid malformation) type I at 24 weeks of gestation. Left; Tomographic ultrasound imaging of fetal thorax. Multiple cysts with various size are seen. Right; Magnetic resonance imaging. This case had favorable pre-/postnatal prognosis after of simultaneous cyst-amniotic and thoracic-amniotic shunting procedures (Courtesy: Dr Pooh, CRIFM PMC)

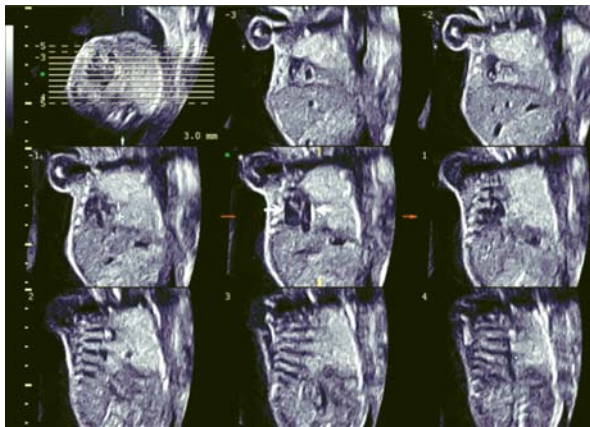


Fig. 3: CCAM (congenital cystic adenomatoid malformation) type III at 21 weeks of gestation. Tomographic ultrasound imaging of fetal thorax and abdomen. The heart is replaced to fetal right side (arrows) due to left-sided CCAM mass. Note the liver-lung border and stomach bubble location are intact (Courtesy: Dr Pooh, CRIFM PMC)

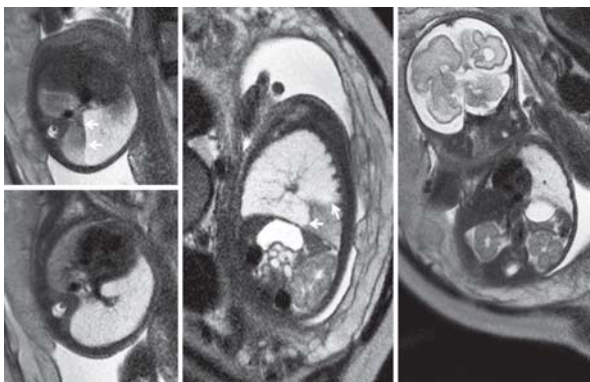


Fig. 4: CCAM (congenital cystic adenomatoid malformation) type III at 28 weeks of gestation. Left; axial sections. Middle; Sagittal section, Right; Coronal section. CCAM is demonstrated as a hyperchogenic mass. MRI demonstrates clear borderline (arrows) and obvious contrast difference between normal lung tissue and CCAM (Courtesy: Dr Pooh, CRIFM PMC)

structure caused mediastinal dislocation. Diagnostic marker is cystic pulmonary mass complicated with fetal hydrops. CCAM was used to be thought as lethal congenital disease. However, CCAM masses often shrink and resolve spontaneously during pregnancy, regardless of types. As fetuses with other complications, such as hydrops due to hypercardiac output, may have poor prognosis and outcome, careful serial observation during pregnancy is required.

Sequestration

Bronchopulmonary sequestration is the separated pulmonary tissue or cystic mass which contains independent bronchus. It is classified into two types according to the relation between the tumor and fetal lung. Ultrasonically, it is a solid mass in the thorax. Its outcome is generally ominous, particularly if it is accompanied by the other congenital anomalies.

Congenital Diaphragmatic Hernia (CDH)

The CDH is the congenital anomaly related to both fetal thorax and abdomen, it is caused by the defect of fetal diaphragm through which fetal abdominal organs, including the stomach, intestine and sometimes liver, enter into fetal thorax and compress fetal lung (Fig. 5), and therefore it causes fetal lung dysplasia and frequently neonatal death in spite of the neonatal surgery to close the diaphragmatic defect after birth.

Ultrasound studies disclose three characteristic changes including hydramnios, mediastinum dislocation and the presence of abdominal organs including fetal stomach, intestine and, in severe cases, fetal liver in the thorax. Fetal heart is dislocated to the opposite side. The ultrasonic diagnosis will be the CDH if the abdominal organ is located at the same height as four chamber view image of the fetal heart.

The prenatal surgery of fetal CDH after opening pregnant uterus was tried under monitoring fetal ECG, pulse oxymetry

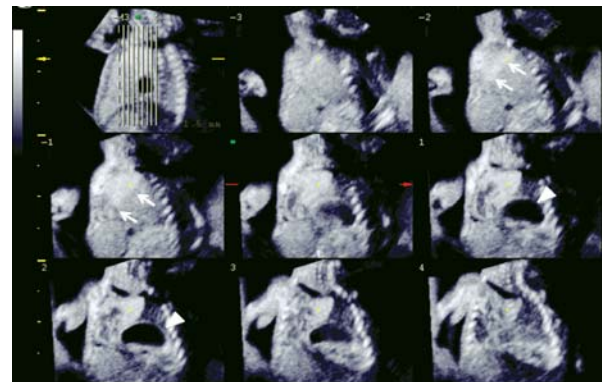


Fig. 5: Congenital diaphragmatic hernia at 16 weeks of gestation. Tomographic ultrasound imaging, coronal sections of fetal thorax. Lung-liver border (arrows) indicates left lobe of the liver is up in the thorax. The stomach (arrowheads) is located on the diaphragm (Courtesy: Dr Pooh, CRIFM PMC)

and ultrasound to open fetal thorax, getting back the abdominal organs to the abdomen through the hernia aperture of diaphragm and closing the hernia with plastic patch then close fetal thorax, uterus and maternal abdomen. However, Harrison⁶ tried the occlusion of fetal trachea by a plug or clamp with the purpose to increase fetal lung volume by the liquid accumulation in the lung and to improve the pulmonary hypoplasia, which develops after the birth threatening neonatal life in spite of hernia repairing surgery. The antenatal tracheal occlusion is now widely tried in antenatal CDH treatment.

CONGENITAL HEART DISEASES (CHD)

Since the congenital fetal heart diseases (CHD) are discussed in the chapter of screening of fetal heart for the congenital heart disease in this issue, including hypoplastic left heart syndrome (HLHS), atresia or stenosis of aortic valve/pulmonary artery, tricuspid valve atresia, Ebstein anomaly, common atrio-ventricular canal, ASD and VSD, Fallot's tetralogy, translocation of large vessels, endocardial cushion defect (ECCD) and endocardial fibroelastosis (EFE), only fetal heart arrhythmias are discussed in this Article.

Fetal Cardiac Arrhythmia

Although fetal bradycardia is diagnosed by the fetal heart rate traced by a fetal monitor, the tachycardia higher than 210 bpm is hardly recorded correctly by the fetal heart rate (FHR) monitor while sometimes half of true heart rate is traced on the monitoring chart. However, since fetal heart beat monitoring sound of fetal monitor may suggest the presence of fetal tachycardia, suspected fetal tachycardia may be studied precisely by the fetal pulsed Doppler arterial flow velocity waveform, which will correctly report the tachycardic fetal heart rate.

Fetal Tachycardia

Supraventricular Tachyarrhythmia: The supraventricular origin is common in fetal tachyarrhythmia higher than 200 bpm, which is shown by the pulsed Doppler method. Maternal transplacental pharmaceutical therapy with digoxin, etc is common in its treatment.

Bradycardia

Atrioventricular block: Ultrasonic M-mode disclose the dissociation of atrial and ventricular motion where atrial movement shows normal fetal heart rate, but the ventricular motion shows bradycardia.

Sick sinus: The heart rate tracing of sinus bradycardia transiently increases synchronizing with fetal movement burst on the ultrasonic Doppler actocardiogram, where neonatal condition was favorable.⁷

Hypoxic bradycardia: The transient bradycardia delayed from every uterine contraction repeatedly appearing in the late deceleration, of which typical ominous case accompanies no FHR acceleration and the loss of baseline variability.

Sudden decrease of fetal heart rate below 60 to 70 bpm and its continuous persistence may suggest ominous hypoxia preceding fetal damage.

Vagal reflex bradycardia: So-called variable deceleration of FHR tracing is a U-shaped, sudden FHR decrease and quick recovery to normal baseline FHR within one minute, which may be caused by the compression of umbilical cord vessels, where fetal blood pressure rise is prevented by elevated vagal tone which suppresses fetal heart action to maintain normal blood pressure by reducing the heart rate. The release of cord compression makes blood pressure and FHR to normal level. However, prolonged deceleration longer than 1 to 2 minutes will show the association of fetal hypoxia.

Premature Heart Beats—Extrasystole

The premature beat occurs after short diastolic interval followed by compensatory longer diastole than normal beat-to-beat interval. The QRS of direct FECG or pulsed Doppler fetal arterial blood flow velocity wave reveals the phenomena. Also, instantaneous FHR tracing records transient rise followed by transient fall. The ultrasonic Doppler fetal heart beat detector sound reveals the premature heart beat. According to the locus to develop the premature beat, the premature beats are classified into atrial or ventricular premature beats. The premature beats are usually harmless and disappear after birth.

Esophageal Obstruction

The esophageal obstruction is classified into five types according to the presence of tracheoesophageal fistula. Antenatally, its obstruction is suspected but hardly diagnosed in cases of polyhydramnios and the loss of fetal stomach vesicle which are caused when the fetus is unable to swallow the amniotic fluid.

FETAL ABDOMEN

Fetal Liver

Ultrasonic image of fetal liver is the large organ in the upper abdomen, it is separated from fetal thorax by the diaphragm, it is generally highly echogenic, and its transection with umbilical vein is the representative image of fetal abdominal axial plane. The gall bladder is visualized at the right side of umbilical vein, and the stomach is a cyst located at left upper abdomen. The alimentary tract, including the stomach and intestine is filled with liquid in ultrasonic image.

Obstruction of the Alimentary Tract

Duodenal and ileum obstructions are common in the congenital gut atresia.

Polyhydramnios is frequently associated with duodenal atresia, which is ultrasonically diagnosed by the double-bubble sign (Fig. 6), and its treatment is postnatal surgery. The intestinal atresia can be multiple, and it is frequently accompanied with congenital anomaly including 21 trisomy and others.

Antenatal anal atresia is present in some fetuses, but its antenatal diagnosis is difficult.

Megacolon (Hirschsprung's disease) is ultrasonically diagnosed by the dilated fetal colon in fetal abdomen.

Meconium peritonitis is characterized by ultrasonically high brightness of intestinal wall.



Fig. 6: Double-bubble sign in a case of duodenal atresia at 25 weeks of gestation (Courtesy: Dr Pooh, CRIFM PMC)

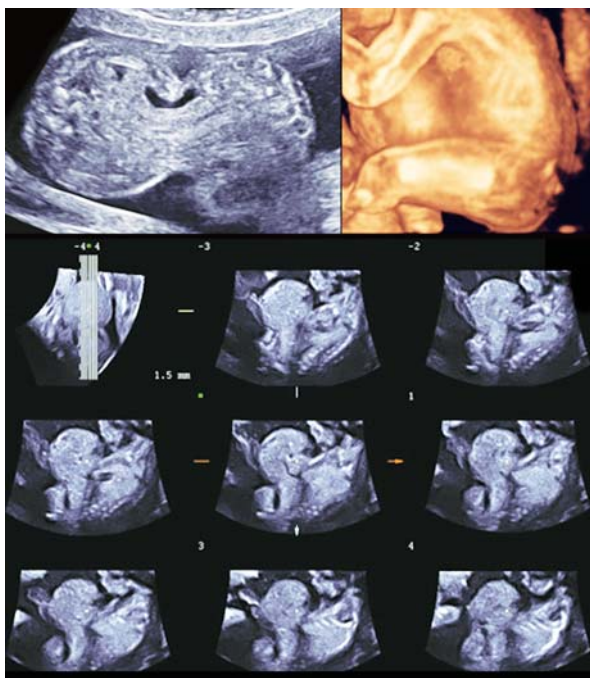


Fig. 7: Umbilical hernia at 20 weeks of gestation. Left upper; 2D axial ultrasound image of fetal abdomen. Large umbilical hernia is seen. Right upper; 3D reconstruction image. Lower; Tomographic ultrasound imaging of umbilical hernia (Courtesy: Dr Pooh, CRIFM PMC)

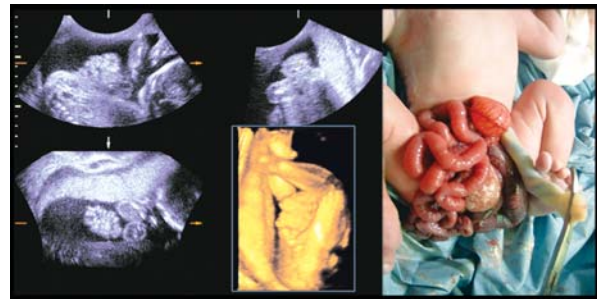


Fig. 8: Gastroschisis at 20 weeks of gestation and macroscopic picture after birth. Left; 3D orthogonal view and reconstructed image. Right; Macroscopic picture. Prognosis after surgery was favorable in this case (Courtesy: Dr Pooh, CRIFM PMC)

Umbilical hernia (Fig. 7) is shown as enlarged umbilical cord which attached fetal abdominal wall incorporating fetal abdominal organs, including intestine, liver and others in the hernia sac. Postnatal surgery is its treatment.

Physiological abdominal hernia is ultrasonically observed at around 13 weeks of pregnancy which disappears after the period of pregnancy.

Gastroschisis (Fig. 8) is the perforated fetal abdominal wall showing multiple intestines floating in the amniotic fluid.

Ascites is the fluid accumulation in peritoneal cavity which is usually the partial symptom of fetal hydrops where the treatment is proper therapy for the fetal hydrops. Conservative treatment of massive ascites will be abdominal puncture and drainage.

Prune-belly syndrome is the disease of fetal abdominal wall composed of enlarged fetal abdomen, defect of abdominal wall muscles, enlarged asthenic urinary bladder and enlarged ureter.

Sacral Masses

Teratoma, hemangioma or the other mass develops at fetal gluteal region, which is ultrasonically diagnosed and treated by postnatal surgery.

UROGENITAL SYSTEM

Normal External Genitalia

Male or female genitalia is detected by ultrasound with the medical diagnostic purpose on the fetus.

Normal Kidneys

Fetal kidneys are detected in the screening of fetus at the upper part of fetal abdomen located at both sides of spinal column. Fetal adrenal glands are detected above the kidneys as triangular organs.

Renal Agenesis

No bilateral kidneys are ultrasonically detected with the absence of amniotic fluid (severe oligohydramnios). No content of urinary bladder is also visualized.

Postnatal outcome is fatal because of no urine production.

Polycystic Kidney (PCK)

The tissues of bilateral kidneys are replaced by small cystic tissues, which has no urine producing function. Ultrasound image reveals neither kidney image nor cystic tissue but only tumor like enlargement of bilateral kidneys or atrophic kidneys (Fig. 9). The outcome is ominous.

Multicystic Dysplastic Kidney (MCDK)

Multiple cysts are recognized by ultrasound B-mode examination in bilateral or unilateral kidney(s). Urine formation function is normal in case of unilateral existence of multiple cysts. The outcome is unfavorable if the bilateral kidneys are affected by the cystic changes (Fig. 10), though unilateral multicystic kidney is usually harmless.

Hydronephrosis

The kidney pelvis is dilated (Figs 11 and 12) by the urinary accumulation due to stenosis or obstruction of ureter or other causes. The outcome may be unfavorable when the urinary tract obstruction is bilateral, and it is recommended to insert the drainage catheter connecting the enlarged kidney pelvis to amniotic cavity under the ultrasonic guidance in the antenatal treatment.

Megacystis

The urinary bladder is enlarged (Fig. 13) due to urethral occlusion, posterior urethral valve or the prune-belly syndrome. Severe oligohydramnios develops due to no fetal urine output. The urine accumulated in the bladder is drained into the amniotic cavity by the insertion of catheter into the bladder in the antenatal treatment.



Fig. 9: Bilateral polycystic atrophic kidney at 16 weeks of gestation. 3D volume contrast image shows the atrophic hyperechogenic kidneys (arrowheads) (Courtesy: Dr Pooh, CRIFM PMC)

Ovarian Masses

There was the axial torsion of ovarian cyst in the abdomen of a female fetus, and the outcome was intrauterine fetal death.⁸ Cases of antenatal ovarian tumor were reported.⁹

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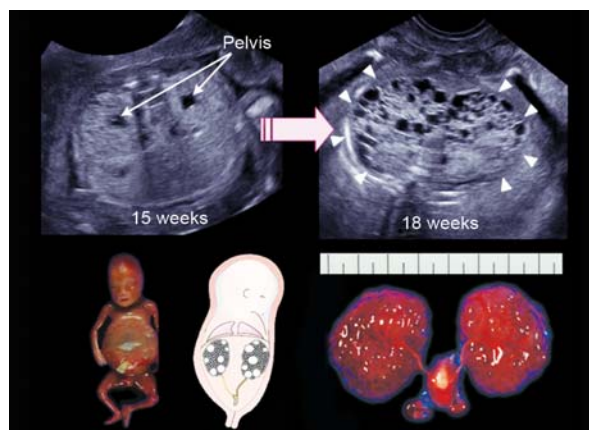


Fig. 10: Bilateral multicystic dysplastic kidney (MCDK), changing renal appearance during the early second trimester. Left upper; Horizontal view of kidneys. Although kidneys are demonstrated as hyperechogenic and enlarged structure bilaterally, both pelvis (arrows) indicate existence of urination function. Right upper; At 18 weeks, renal appearance changed into bilateral multicystic structure with numerous cysts. Lower left; Picture of aborted fetus. Abdominal space was occupied by large renal masses. Lower middle; Schematic picture of bilateral MCDK. Lower right; Macroscopic picture of MCDKs, ureters, bladder. Bilateral MCDK is the lethal condition (Courtesy: Dr Pooh, CRIFM PMC)

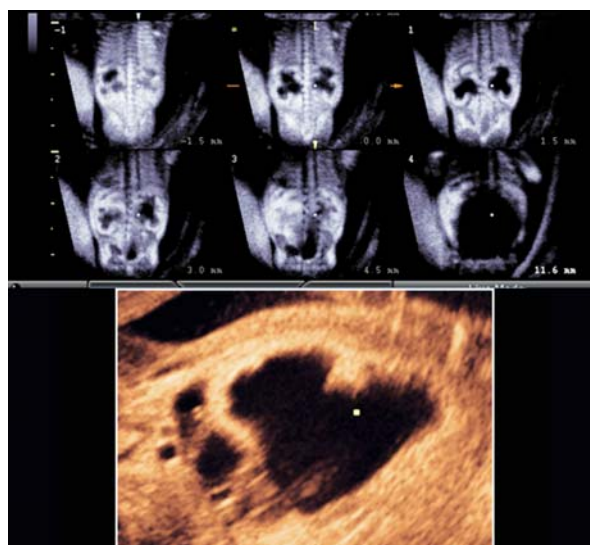


Fig. 11: Hydronephrosis in the second trimester. Upper; Tomographic ultrasound imaging of hydronephrosis at 15 weeks of gestation. Bilateral dilated pelvises are clearly demonstrated. Lower; Volume contrast imaging of hydronephrosis at 18 weeks. Thin parenchyma is obvious with pelvic dilatation (Courtesy: Dr Pooh, CRIFM PMC)

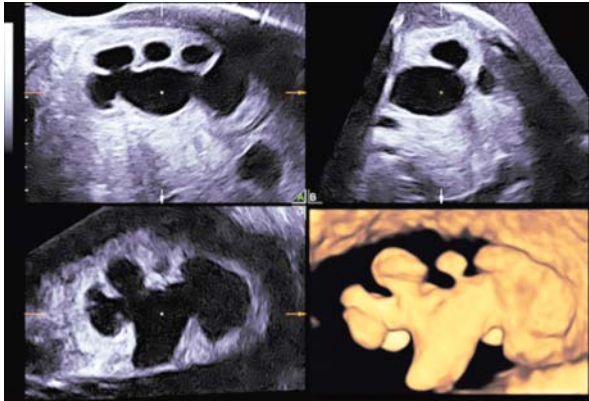


Fig. 12: Moderate hydronephrosis in the third trimester. 3D orthogonal view with 3D inversion reconstructed image shown in right lower figure (Courtesy: Dr Pooh, CRIFM PMC)



Fig. 13: Megacystis at 16 weeks of gestation. 2D ultrasound imaging, Midsagittal sections of fetus. Megacystis is obvious. After vesicoamniotic shunting was performed at 17 weeks, the pregnant course, delivery and postnatal course were favorable with good renal function (Courtesy: Dr Pooh, CRIFM PMC)

REFERENCES

1. Achiron R, Gindes L, Zalel Y, Lipitz S, Weisz B. Three- and four-dimensional ultrasound: New methods for evaluating fetal thoracic anomalies. *Ultrasound Obstet Gynecol* 2008;32:36-43.
2. Gindes L, Benoit B, Pretorius DH, Achiron R. Abnormal number of fetal ribs on 3-dimensional ultrasonography: Associated anomalies and outcomes in 75 fetuses. *J Ultrasound Med* 2008;27:1263-71.
3. Hata T, Kuno A, Dai SY, Inubasiri E, Hanaoka U, Kanenishi K, et al. Three-dimensional sonographic volume measurement of fetal lung. *J Obstet Gynecol Res* 2007;33:793-98.
4. Gerards FA, Enges MA, Twisk JW, van Vugt JM. Normal fetal lung volume measured with three-dimensional ultrasound. *Ultrasound Obstet Gynecol* 2006;27:134-44.
5. Serizawa M, Maeda K. Noninvasive fetal lung maturity prediction based on ultrasonic gray level histogram width. *Ultrasound Med Biol* 2010;16:1998-2003.
6. Harrison MR, Adzick NS, Flake AW, VanderWall KJ, Bealer JF, Howell LJ, et al. Correction of congenital diaphragmatic hernia in utero VIII: Response of the hypoplastic lung to tracheal occlusion. *J Pediatr Surg* 1996;31:1339-48.
7. Minagawa Y, Akaiwa A, Hidaka T, Tsuzaki T, Tatsumura M, Ito T, Maeda K. Severe fetal supraventricular bradyarrhythmia without fetal hypoxia. *Obstet Gynecol* 1987;70:454-56.
8. Makio A, Nozue K, Ohtani Y, Utsu M, Torii Y, Narita S, et al. Intrauterine fetal death due to axial torsion of fetal ovarian cyst. *Current Obstetrics and Gynecology* 1999;2:175-76.
9. Sbragia L, Paek BW, Fekdstein VA, Farrell JA, Harrison MR, Albanese CT, Farmer DL. Outcome of prenatally diagnosed solid fetal tumors. *J Pediatr surg* 2001;36:1244-47.