Pictorial Essay

Pattern of prenatal ultrasound diagnosed anterior abdominal wall defects at the University College Hospital, Ibadan, Nigeria: A pictorial essay

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Abstract

Anterior abdominal wall defects form a wide spectrum of congenital abnormalities that allow the abdominal contents to protrude through an unusual opening on the abdominal wall. These defects could be physiological or pathological depending on the time of diagnosis. They include physiological gut herniation, congenital umbilical cord hernia, omphalocele, gastroschisis, ectopia cordis, bladder exstrophy, body-stalk anomaly, Prune-Belly Syndrome, and pentalogy of Cantrell. Correct prenatal diagnosis of these anomalies with ultrasound (US) is extremely important for patient management. Evaluation of the defect relative to the umbilical cord insertion site is fundamentally important in differentiating among the various malformations. We present a pictorial essay of the spectrum of anterior abdominal wall defects diagnosed prenatally with US seen over a 5-year period at the University College Hospital, Ibadan.

Keywords: Bladder exstrophy, body-stalk anomaly, gastroschisis, omphalocele, prenatal ultrasound

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INTRODUCTION

Anterior abdominal wall defects form a wide spectrum of congenital abnormalities that allow the abdominal contents to protrude through an unusual opening on the abdominal wall. There is an overall prevalence of six cases per 10,000 births.[1] These defects could be physiological or pathological depending on the time of diagnosis.[1,2] They include physiological gut herniation, congenital umbilical cord hernia, omphalocele, gastroschisis, ectopia cordis, bladder exstrophy, body-stalk anomaly, Prune-Belly Syndrome (PBS), and pentalogy of Cantrell.[2,3]

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The prenatal diagnosis of these anomalies is very important because they all differ greatly regarding associated structural anomalies and perinatal as well as neonatal morbidity and mortality. Furthermore, it helps the parents in their decision whether to continue with the pregnancy or opt for elective termination of the pregnancy especially in lethal cases or those associated with multiple severe anomalies.[3]

Prenatal ultrasound (US) screening is the key imaging modality available at present time for the diagnosis of anterior abdominal wall defects. The American Institute of Ultrasound in Medicine[4] and the International Society

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of Ultrasound in Obstetrics and Gynecology[5] have guidelines for the performance of the second trimester US examination, which mandates demonstration of umbilical cord insertion on the fetal abdomen [Figure 1]. This view is essential to exclude an anterior abdominal defect, and when a defect is present, the relationship of the defect to the cord insertion site may be used to reach a specific diagnosis, amidst the spectrum of anomalies.

Prenatal US screening for fetal anomalies has only been introduced in our institution in the last 5 years. We present a pictorial essay of the anterior abdominal wall defects that were diagnosed prenatally and some of the postnatal outcomes.

PHYSIOLOGIC GUT HERNIATION

Physiological gut herniation is a natural phenomenon that occurs in early pregnancy. It usually occurs from around the 6th week until the 12th week *in utero*. During this period, there is a normal herniation of midgut, which will form the small intestine, into the proximal portion of the umbilical cord.[6] This is due to a number of factors, including the rapid growth of the cranial end of the midgut faster than the abdominal cavity and the large size of the developing liver and kidneys, while the midgut is within the umbilical cord, it rotates 90° counter-clockwise along the axis of the superior mesenteric artery. At approximately 12 weeks' gestation, the abdomen enlarges, and the midgut returns to its normal position in the abdominal cavity, undergoing an additional 180° rotation along the axis of the superior mesenteric artery.[6,7] Physiologic gut herniation is usually diagnosed on US with the presence of fetal bowel outside the anterior abdominal wall, at the base of the umbilical cord [Figure 2]. The extent of herniation is comparatively small (often <7 mm). It should not be seen after 12–13 weeks' gestational age and the "hernia" should not contain other organs such as the liver.[7]

CONGENITAL UMBILICAL HERNIA

A congenital umbilical hernia is one of the most common abnormalities seen in the first few months of life. It consists of a protrusion of bowel and sometimes omentum through a defect in the linea alba, which may be the result of incomplete closure or weakness at the umbilical ring. There is usually normal insertion of the cord into the umbilical ring with intact skin covering the ring in an umbilical hernia.[8]

The defect is significantly more common among the black population, with one study reporting a prevalence of 25% in black children compared with 3% in white neonates.[9] The condition is also found more frequently in premature infants, occurring in 75% of those weighing <1500 g.[10] The prenatal US usually shows a bulge on the anterior abdominal wall, which is covered by the skin, and the umbilical cord inserts normally into the fetal abdominal wall [Figure 3]. The abdominal organs are usually normal with no associated tumor or organomegaly.

OMPHALOCELE

Omphalocele is one of the most common anterior abdominal wall defects with a prevalence of about 2–3 in 10,000 live births.[2,11] It is a sporadic abnormality that results from herniation of abdominal viscera through an enlarged umbilical ring to the base of the umbilical cord. The etiology of omphalocele is not known; however, various theories have been postulated, which include failure of the bowel to return into the abdomen by 10–12 weeks, failure of lateral mesodermal body folds to

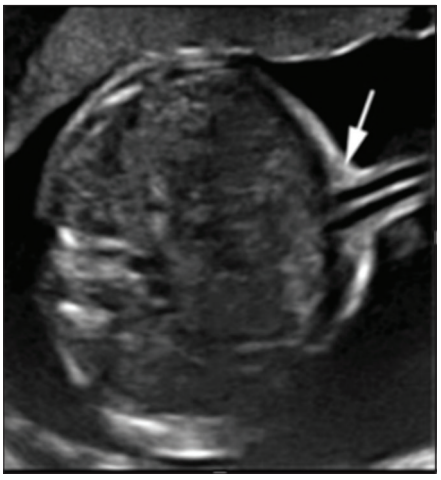

Figure 1: Transverse scan of the fetal abdomen at 20 weeks showing normal insertion of the umbilical cord (arrow)

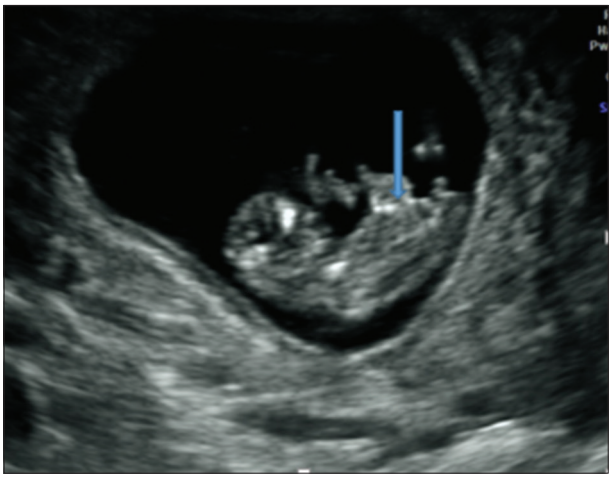

Figure 2: Ultrasound of a fetus at 10 weeks' gestation showing physiological herniation of midgut (arrow)

migrate centrally, and persistence of the body stalk beyond 12 weeks of gestation.[2,12]

Prenatal diagnosis of an omphalocele with two-dimensional US is based on the demonstration of the midline abdominal wall defect and the herniated sac with its visceral contents [Figures 4-6]. This mass has a smooth surface represented by peritoneal-amniotic membrane, and the umbilical cord inserts into a sac covering the herniated viscera.[1,2] This sac is composed of peritoneum, amnion, and Wharton's jelly [Figures 4]. Visualization of the covering sac confirms the diagnosis of omphalocele and virtually excludes gastroschisis.

Three-dimensional US, if available, helps in confirming the diagnosis of omphalocele, and it may also help the parents to understand the condition better, which will help in their decision-making.[13] The size of the omphalocele can vary from a small abdominal defect with extrusion of the bowels [Figure 6] to a large abdominal wall defect with extrusion of all abdominal viscera. [Figures 4, 7 and 8].

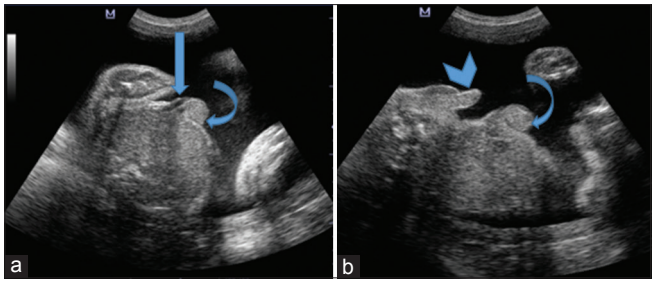

Figure 3: (a and b) Prenatal ultrasound scan of a fetus with congenital umbilical hernia: sagittal views through the fetal abdomen shows a small bulge (curved arrow) in the anterior abdominal wall covered by the skin. The umbilical cord (straight arrow) inserts normally into the fetal abdomen below the bulging area. The fetus here is a boy (arrowhead)

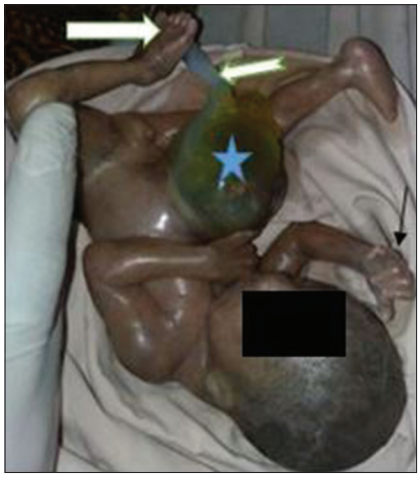

Figure 5: Picture of the fetus in Figure 4 with omphalocele after termination of pregnancy showing the extruded abdominal content covered with a jelly-like membrane (star). The umbilical cord is seen to arise from the middle of the covering membrane (notched white arrow) also noted is the webbed foot (thick white arrow) and the fused middle three fingers (black arrow)

Up to 75% of cases of omphalocele have associated chromosomal and nonchromosomal congenital abnormalities. The most frequently associated anomalies are cardiac, gastrointestinal, genitourinary, and neural tube defects. One of the cases diagnosed had associated ascites and bilateral hydrocalycosis [Figure 4] on US. The pregnancy was terminated, and in addition to the aforementioned anomalies, webbed feet and syndactyly of the fingers were also detected [Figure 5].

GASTROSCHISIS

Gastroschisis is a congenital paraumbilical abdominal wall defect through which abdominal content herniates and is directly exposed to the amniotic fluid without a covering membrane for the greater period of gestation. The defect is usually small, <4 cm in diameter, and it is located on the right side of the umbilicus in 95% of cases.[2,14] It has an estimated prevalence of 1 in 4000 live births.[1] Gastroschisis

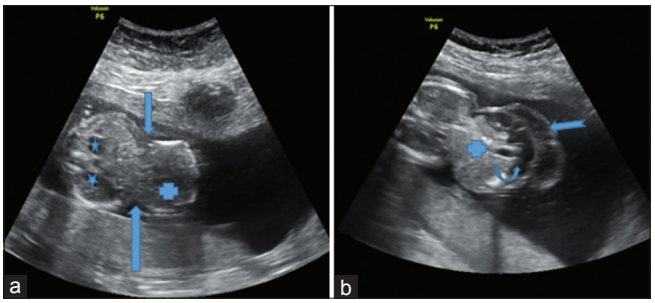

Figure 4: (a and b) Prenatal ultrasound scan of a 22-week-old fetus with major omphalocele: Transverse views of the abdomen of the fetus show a large anterior abdominal wall defect (thick arrows) with extrusion of the abdominal contents (cross), which are covered by a thick membrane (notched arrow). There is also associated bilateral calyceal dilatation (stars) as well as ascites (curved arrow)

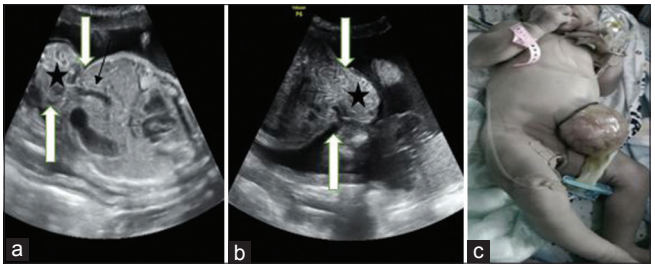

Figure 6: (a and b) Prenatal ultrasound scan of a 20-week-old fetus with minor omphalocele: Transverse views through the abdomen show a small anterior abdominal wall defect with extrusion of abdominal contents (star) that are covered by a membrane. The umbilical vein (long arrow) is seen entering the extruded content. (c) Picture of the baby after delivery showing extruded abdominal contents covered by a transparent membrane consistent with omphalocele. The umbilical cord is seen arising from the omphalocele

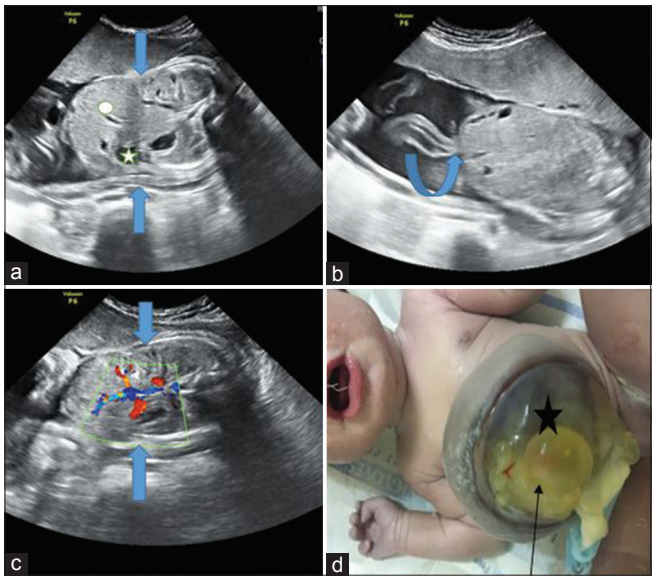

Figure 7: (a-c) Prenatal ultrasound scan of a 21-week-old fetus with major omphalocele: Transverse views through the abdomen show a large anterior abdominal wall defect (block arrows) with extrusion of the liver (circle) and the stomach (star). The umbilical cord (curved arrow) is seen inserting into the sac that covers the herniated viscera. The umbilical vein (color) is seen entering the extruded content. (d) Picture of the baby postdelivery confirming the omphalocele evidenced by the large anterior abdominal wall defect with extrusion of the abdominal contents (star) which are covered by a jelly-like membrane (thin arrow)

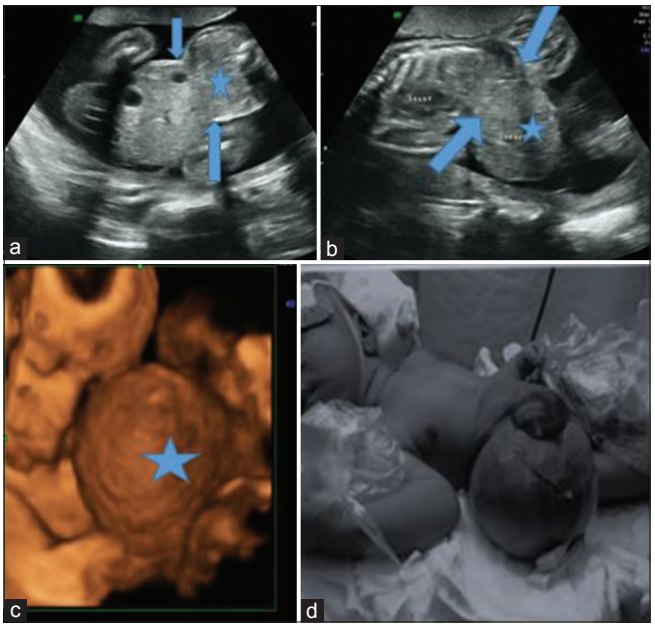

Figure 8: (a and b) Prenatal ultrasound scan of a 24-week-old fetus with major omphalocele: Sagittal and transverse ultrasound scan through the abdomen show a large anterior abdominal defect (block arrows) with extrusion of echogenic solid content that suggests the liver. (c) Three-dimensional ultrasound image of the fetus confirming the omphalocele as a mass (star) in front of the fetus. (d) Picture of the baby postdelivery showing the extruded abdominal contents (star) covered by a membrane

can produce intrauterine and neonatal complications, which may include postnatal bowel dysfunction, bowel atresia, bowel necrosis, and subsequent short-bowel syndrome. The etiology of gastroschisis is unknown, although several hypotheses have been proposed, which include the herniation of the intestine as a result of the rupture of the amniotic membrane through the umbilical ring, anterior abdominal wall defect resulting from vascular disruption in the early embryo, and abnormal body wall folding leading to the anterior abdominal wall defect and subsequent gut herniation.[1,14] Gastroschisis usually occurs as an isolated anomaly; however, it is important to look for other malformations.[15] This is because Barisic *et al*.[15] found additional anomalies in 14% of fetuses with gastroschisis among which central nervous system and cardiac anomalies were the most common. Intrauterine demise is uncommon, and when it happens, it is most often related to other major anomalies.[16]

Prenatal evaluation by ultrasonography plays an important role in classification of the abnormality, determination of the presence of any associated abnormality, prognostication, and guiding management decision. The most common abdominal structure to herniate is the small bowel followed by the large bowel and sometimes the stomach. It is, however, rare for the solid visceral organ such as the liver to eviscerate [Figure 9c].

One major US feature in the diagnosis of gastroschisis is the normal insertion of the umbilical cord vessels into the fetal abdomen, with the paraumbilical defect usually occurring to the right of the umbilical cord. Another feature is the visualization of freely floating loops of bowel within the amniotic fluid with the absence of a covering membrane [Figure 9a and b].[16] Common sonographic predictors associated with poor prognosis with gastroschisis include the presence of intra-abdominal bowel dilatation, bowel wall thickness, intrauterine growth restriction, liver herniation [Figure 9c], and urinary bladder herniation.[15-17]

PRUNE-BELLY SYNDROME

PBS, also known as the Eagle-Barrett syndrome or triad syndrome, is a rare congenital anomaly characterized by a triad of distinctive features including anterior abdominal wall underdevelopment, cryptorchidism, and urinary tract anomalies.[18] Urinary tract abnormalities include massive dilatation of the ureters and upper tracts and a very large bladder as well as undescended testes. Anterior and posterior urethra may be dilated resulting in megalourethra. The incidence has been reported to range between 1/29,000 and 1/40,000 in different studies, and it is an almost exclusively male disorder with about 95% occurring in males.[18]

Its etiology and pathogenesis are uncertain and may result from primary obstructive urinary anomalies or defective mesodermal development.[19]

The most common mode of diagnosis in the developed world is by obstetric US scan usually in the 2nd–3rd trimester, although diagnosis in the 1st trimester between 12 and 14 weeks has been reported.[18,19] The hallmark of the prenatal diagnosis of PBS is the elongated, tortuous, and dilated appearance of the ureters [Figure 10a].[20] Other features, include hydronephrosis and megacystis with oligohydramnios, which eventually result in pulmonary hypoplasia [Figure 10a].[5,8] Postnatally, an infant with PBS may show marked distention of the abdomen with laxity and wrinkling of the abdominal wall as well as bulging flanks [Figure 10b]. Radiographs of the chest and abdomen confirm the hypoplasia of the lungs as well as the absent abdominal wall vasculature [Figure 10c] while the abdominal US shows the dilated ureters with hydrocalycosis [Figure 10d]. Termination of pregnancy can be offered before viability. The prognosis depends on the degree of renal function compromise. Early urinary obstruction leads to renal failure, pulmonary hypoplasia, and death in the neonatal period.[21]

BODY-STALK ANOMALY

Body-stalk anomaly, also called limb-body wall complex, is a sporadic, lethal abnormality with a reported incidence of 0.12 cases per 10,000 births (including both live and stillbirths).[22] The pathogenesis is uncertain; however, three pathogenetic mechanisms have been proposed which are as follows: abnormal folding of the trilaminar embryo during the first 4 weeks of development, early amnion rupture with amniotic band syndrome, and early generalized compromise of embryonic blood flow.[23] The anomaly can be classified into two types based on the malformation phenotypes: type I is based on the craniofacial defect, while the type II is recognized based on the ventral wall defects.[24,25]

The ultrasonographic features are usually a major abdominal wall defect, myelomeningocele and/or caudal regression, a short or absent or rudimentary umbilical cord, and limb abnormalities [Figures 11a-d].[23,25] The placenta may be attached to the fetus. These typical features of the body-stalk anomaly can be detected using US by the end of the first trimester.[25] As the body-stalk anomaly is incompatible with life, it is important to diagnose the lesions prenatally and to differentiate them from other anterior abdominal wall defects. It should be differentiated from common abdominal wall defects such as gastroschisis, omphalocele, and uncommon entities such as ectopia cordis, amniotic band syndrome, cloacal dystrophy, and urachal cyst.

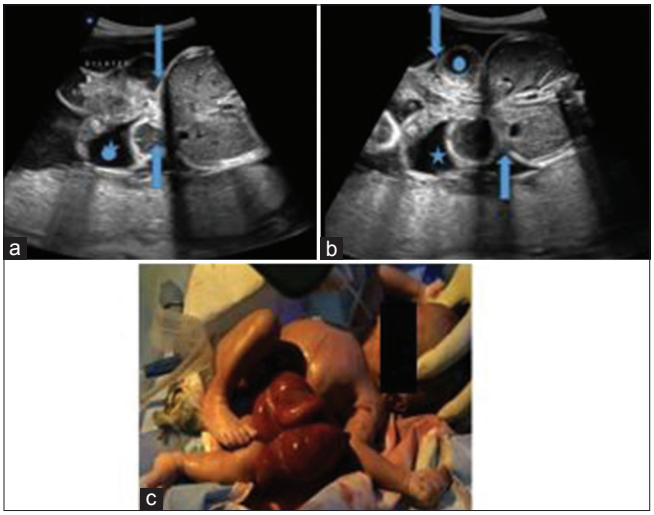

Figure 9: (a and b) Prenatal ultrasound scan of a fetus with gastroschisis: transverse sonographic views through the fetal abdomen show an anterior abdominal wall defect with herniated dilated bowel loops (arrows) and stomach (star) floating within the amniotic fluid (circles). These bowel loops lack a covering membrane. (c) Photograph of the baby with gastroschisis post cesarean section confirming the herniated bowel loops and liver through the anterior abdominal wall. The baby had the first stage of surgery within 4-h postdelivery but died 24-h postsurgery

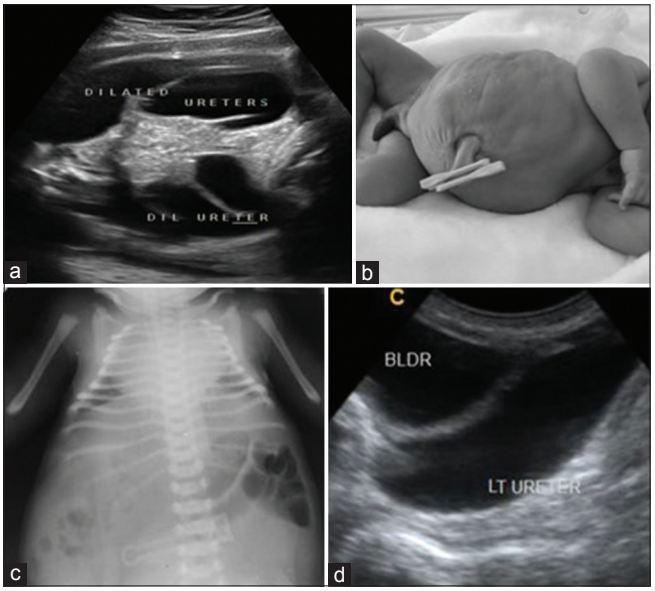

Figure 10: (a) Prenatal ultrasound scan of a fetus with Prune-Belly syndrome: Coronal view of the fetal abdomen shows markedly dilated, elongated, and tortuous ureters as well as oligohydramnios (b) Picture of the baby showing the wrinkled and lax anterior abdominal wall. There is a hypoplastic scrotum with bilateral undescended testes. The penile shaft appears normal for age. (c) Chest radiograph showing a bell-shaped small thoracic cavity with hypoplastic lungs bilaterally and an apparent cardiomegaly. There is also abdominal distension with bulging of the flanks. (d) Abdominal ultrasound of the baby postdelivery showing dilated ureter with normal-sized urinary bladder

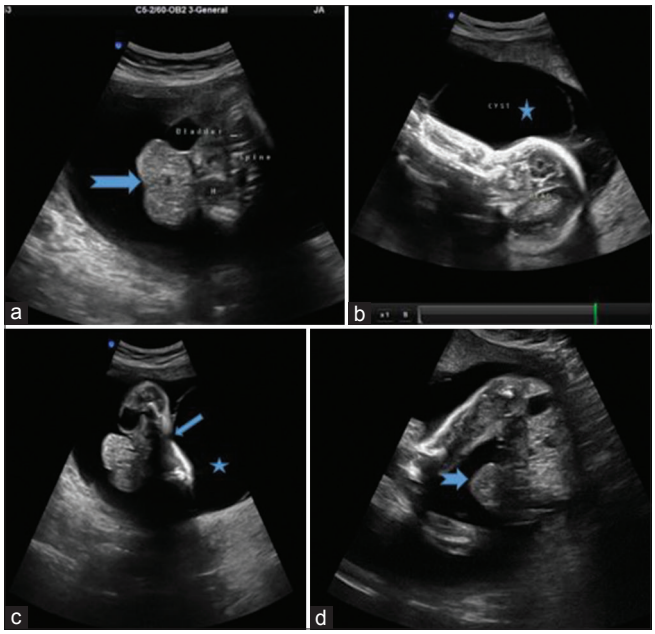

Figure 11: (a) Prenatal ultrasound scan of a 20-week fetus with a body-stalk anomaly: sagittal view of the fetus shows a very large abdominal wall defect with extrusion of the liver (notched arrow) and the urinary bladder into the amniotic fluid without a covering membrane. The heart (H) is seen within the thorax. There is a distortion of the anatomy of the spine. (b and c) A huge cystic mass (star) consistent with a meningocele is seen arising from a defect (arrow) in the spine. (d) Image of the fetus also showing the anterior abdominal wall mass (notched arrow) with nonvisualization of the umbilical cord, which is likely absent

BLADDER EXSTROPHY

Exstrophy of the bladder, also known as ectopia vesicae, is a rare congenital abnormality with an incidence of 1 per 30,000–50, 000 live births, and male-to-female ratio ranging from 1.5:1 to 5:1.[26] The condition is thought to be caused by incomplete development of the infraumbilical part of the anterior abdominal wall, associated with incomplete development of the anterior wall of the bladder, which results in eversion and exteriorization of the pelvic viscera on the abdominal surface, inferiorly displaced umbilicus, divergent pubic rami, and abnormal exterior genitalia.[13,27]

Another school of thought hypothesized that the natural history of this congenital anomaly begins with a failure of the closure of the lower abdominal wall with an abnormal distension of the urinary bladder (megacystis) that protrudes through the abdominal wall defect, which is usually apparent at around 16 weeks of gestation. Subsequently, the megacystis ruptures and its walls evert. The mucosal surface of the urinary bladder is then exposed and opens anteriorly into the amniotic cavity, which is seen as a complex mass on the anterior abdominal wall below the umbilical cord insertion.[28]

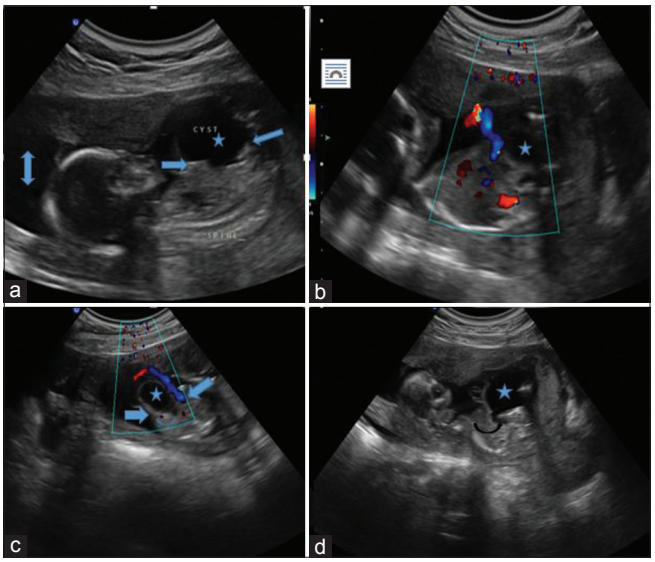

Figure 12: (a-d) Prenatal ultrasound scan of a 17-week fetus with bladder exstrophy: sagittal view of the fetus shows a wide anterior abdominal wall defect (block arrows) below the umbilical cord insertion (blue-colored vessel). A large cystic mass (star) is seen protruding through the defect consistent with a distended urinary bladder (megacystis), and this is seen to continue as a blind-ending smaller tubular structure in the lower abdomen in keeping with the urethra (curved arrows). No other pelvic cystic structure that suggests a normally placed urinary bladder was seen. The amniotic fluid was adequate (up-down arrow)

Prenatal US findings are usually those of a cystic or complex lower anterior abdominal wall mass (depending on when the diagnosis is made) seen below the umbilical cord insertion. The fetal bladder is not visualized in its normal position within the pelvis; however, the kidneys are located normally with normal amniotic fluid volume [Figures 12a-d]. Other findings include a small penis, epispadias, and splayed iliac bones.[13,29]

CONCLUSION

The use of prenatal US allows the diagnosis of the majority of abdominal wall defects with subsequent opportunities for parental counseling, fetal intervention, and optimal perinatal management. Cases diagnosed with abdominal wall defects should be considered for *in utero* transport to perinatal center to benefit from an optimal management with a multidisciplinary team. In cases with associated lethal or multiple severe abnormalities, parents may opt for elective termination of the pregnancy.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will

not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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