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PICTORIAL ESSAY

Pearls and pitfalls of early obstetric ultrasound in the acute setting

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Abstract First trimester ultrasound is commonly performed to establish dates or evaluate early pregnancy complications. With improvement in ultrasound technology, visualization of fetal structures has improved. While the emergent evaluation does not typically focus on detailed fetal anatomic evaluation (since this is typically performed at 18–20 weeks), various fetal structural abnormalities can now be visualized, especially during the late first trimester and early second trimester. We present a pictorial review of potential pitfalls encountered in early obstetric ultrasound with an emphasis on fetal structural abnormalities as well as normal fetal anatomy that can be confused with developmental abnormalities.

Keywords Fetal ultrasound · Obstetric ultrasound · Abnormalities · Early detection

Background

First trimester ultrasound is commonly performed to establish dates or evaluate early pregnancy complications in the acute setting. As ultrasound technology has improved, so has the visualization of fetal structural anatomy. While the acute evaluation does not typically focus on detailed fetal structural evaluation, many fetal structural abnormalities can be visualized during the late first trimester and early second trimester. While a detailed structural evaluation is typically performed at a gestational age of 18–20 weeks, not all patients have routine prenatal care. We present a pictorial review of potential pitfalls encountered in early obstetric ultrasound with an emphasis on fetal structural abnormalities as well as normal fetal anatomy that can be confused with developmental abnormalities.

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Central nervous system

Normal brain

Open rhombencephalon

The rhombencephalon, a normal brain structure, forms along with the prosencephalon (which later forms the cerebral hemispheres) and mesencephalon (which forms the midbrain) by the sixth week. As the rhombencephalon segments into the metencephalon and myelencephalon, the communicating and dividing sulcus which later forms the cerebral aqueduct and fourth ventricle becomes a well-delineated normal cystic structure ("open rhombencephalon") within the posterior fossa around 7–9 weeks (Fig. 1) [1]. The open rhombencephalon should not be mistaken for hydrocephalus or posterior fossa malformations.

Appearance after 10 weeks

The calvarium begins to ossify at about 10 weeks, and at 11–14 weeks, ossification is predominantly along the lateral aspects of the frontal and parietal bones and may not be present at the midline. The ventricles should fill almost the entirety of the cerebral hemispheres, and the echogenic choroid plexus fills most of the lateral ventricles [2].

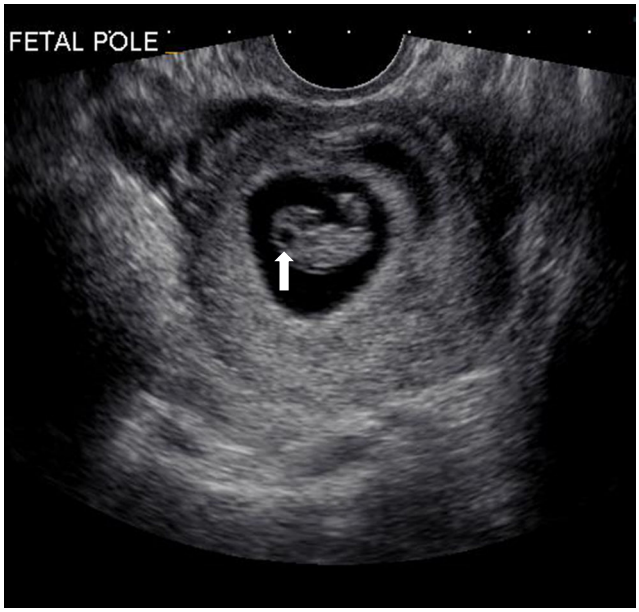

Fig. 1 An eight-week fetus with cyst in the posterior fossa (*white arrow*), which is an open rhombencephalon

Encephalocele

Encephalocele is an open neural tube defect where the meninges or meninges and brain herniate outside the cranial boundaries due to a cranial defect or external disruption (Fig. 2). Most commonly, this occurs posteriorly (75 %) or anteriorly (13 %) along the midline. The appearance of a posterior encephalocele can overlap with a cystic hygroma (a lymphatic malformation arising from the posterior neck). Given sufficient calvarial ossification, an encephalocele should have a calvarial defect. Encephaloceles can be isolated or be associated with genetic syndromes.

Anencephaly, acrania, and exencephaly

Acrania, exencephaly, and anencephaly are thought to represent a spectrum. Cranial ossification should be evaluated specifically since underlying cranial tissue may initially appear normal. Absent cranial ossification is termed acrania. Acrania is complete or partial absence of the cranium with complete (though abnormal) development of brain tissue. However, acrania is most commonly associated with exencephaly or anencephaly (Figs. 3 and 4). Exencephaly is the presence of normal or abnormal brain tissue above the level of the orbits. Most cases of exencephaly progress to anencephaly (Fig. 4), where there is no cranium or underlying brain tissue superior to the orbits [3, 4]. Cranial ossification should be evaluated on both frontal and axial plains, since cranial ossification is greatest along the lateral aspects of the frontal and parietal bones at 11–14 weeks.

Holoprosencephaly

Failure of the prosencephalon to divide into two cerebral hemispheres results in holoprosencephaly. Alobar holoprosencephaly, the most severe form, results in a single large ventricle with brain tissue (the fused thalami) centrally. The falx and interhemispheric fissure should be absent, and no peripheral brain tissue should be seen, differentiating this condition from severe hydrocephalus. Holoprosencephaly is frequently associated with facial abnormalities.

Nuchal translucency

Nuchal translucency is the measurement of fluid collecting posteriorly along the fetal neck. A thin layer of fluid is normally seen. Increased thickness of this fluid, when properly measured, has a high association with aneuploidy (Fig. 5). However, even if the translucency resolves or the karyotype is normal, nuchal translucency remains associated with an increased risk of structural abnormalities such as congenital diaphragmatic hernia, cardiac anomalies, and other genetic syndromes [5, 6].

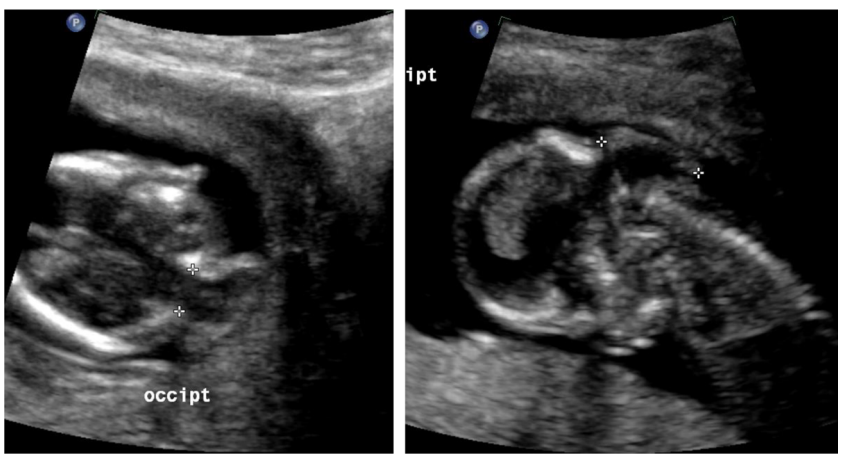

Fig. 2 Encephalocele. A skull defect is seen (*left*), posterior to which is a midline posterior cystic-appearing mass

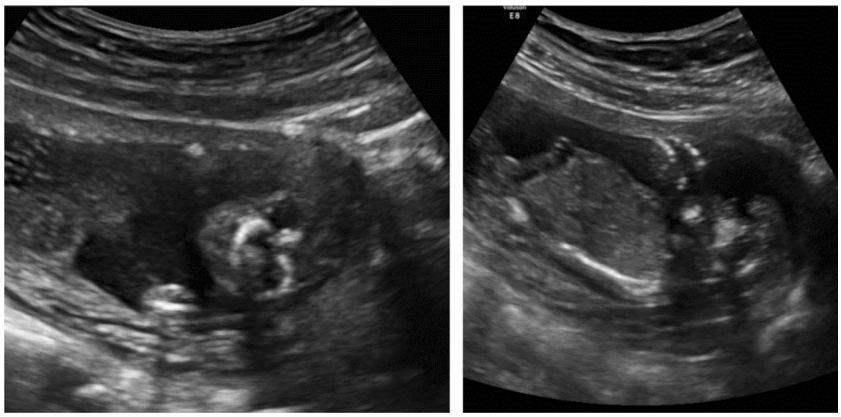

Fig. 3 Acrania/exencephaly. Malformed brain tissue is seen above the level of the orbits, on the coronal view (*left*) and sagittal view (*right*), but there is no cranial ossification

Complex nuchal fluid with septations is associated with a greater risk of associated abnormalities [5, 7]. In the urgent setting, standardized nuchal translucency measurements are not routinely acquired; guidelines stipulate a good sagittal section of the fetus in a neutral position be acquired, among other parameters. A measurement of greater than 3 mm from skin surface to membrane is considered abnormal when proper measurements are obtained. A potential pitfall in nuchal evaluation is mistaking the presence of an unfused amnion as the skin surface. The unfused amnion should be visualized as a structure separate from the nuchal skin surface.

Body wall

Physiologic umbilical herniation

Between 8 and 11 weeks, fetal intestine normally herniates into the umbilical cord base, appearing as an echogenic focus at the base of the umbilical cord insertion on the abdomen (Fig. 6). The intestine returns to the abdominal cavity by 12 weeks as abdominal cavity growth catches up to the more rapid midgut growth. Fluid is typically seen within the fetal stomach by 12–13 weeks [8]. Omphalocele should not be diagnosed before 12 weeks or if the crown-rump length is less than 45 mm [9]. However, if the liver of the stomach protrudes into the sac or the amount of herniation is greater than 7 mm, omphalocele can be diagnosed at any time [10, 11]. Follow-up ultrasound evaluation in 1 to 2 weeks is advisable to re-evaluate the cord insertion if the diagnosis is suspected.

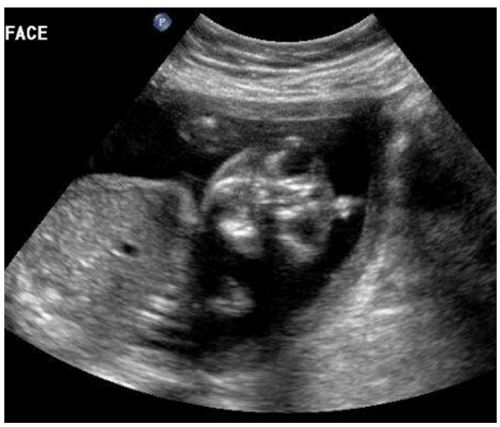

Fig. 4 Anencephaly. Essentially no brain tissue is seen above the orbits on this coronal view

Omphalocele

When intestine or visceral contents within a hernia sac, with the umbilical cord insertion at the apex of the hernia sac, an omphalocele is present (Fig. 7). Omphaloceles are often associated with aneuploidy, most commonly trisomy 18, as well as various developmental syndromes [11].

Gastroschisis

Bowel loops herniate through an abdominal wall defect at the umbilical insertion, without an enveloping membrane, in gastroschisis (Fig. 8). Bowel loops float freely in the surrounding amniotic fluid. Distinguishing gastroschisis from omphalocele is important, as gastroschisis is rarely associated with chromosomal abnormalities.

Lymphangiectasia

Lymphangiectasia is generalized edema around the body of the fetus (Fig. 9). Prognosis is bleak and is associated with impending fetal demise. Lymphangiectasia often occurs as a component of hydrops fetalis, where fluid accumulates and involves at least two fetal components: pleural effusion, pericardial effusion, ascites, nuchal thickening, and cystic hygroma.

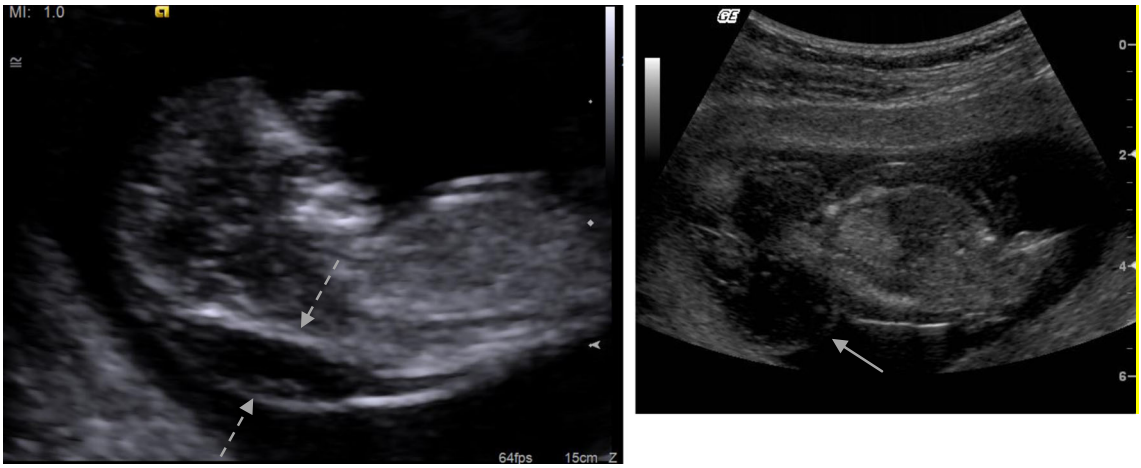

Fig. 5 Nuchal translucency (*left*). Cystic hygroma (*right*)

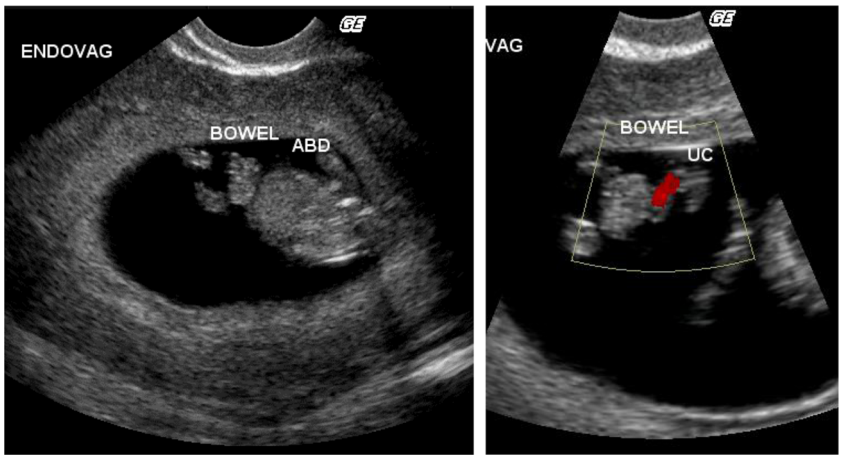

Fig. 6 Physiologic umbilical herniation. The umbilical cord insertion is seen at the apex of bowel loops protruding from the abdomen

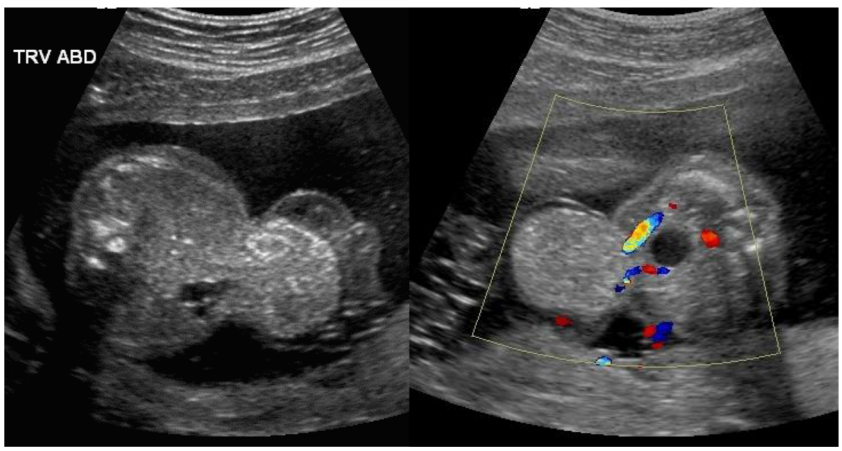

Fig. 7 Omphalocele. Bowel loops herniate through the anterior abdomen into a membrane-enclosed sac

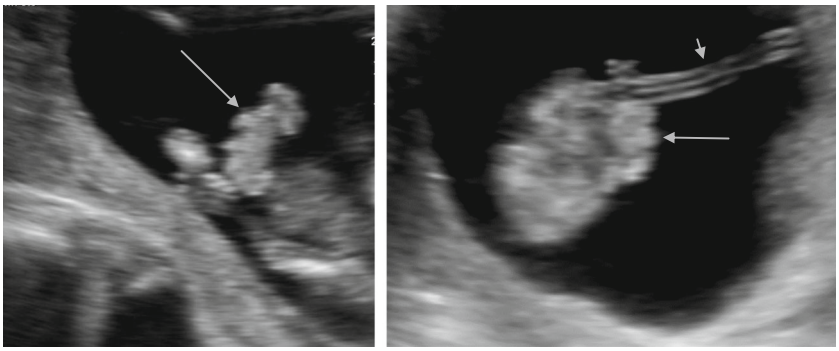

Fig. 8 Gastroschisis. Loops of bowel (*arrow*) float within the amniotic space. *Left* The umbilical cord (*short arrow*) is seen lateral to bowel loops

Triploidy, molar pregnancy, and the cystic placenta

Occurring in about 1 % of all conceptions, the presence of three complete chromosomal sets (triploidy) is the most frequent gestational chromosomal abnormality and typically results in first trimester spontaneous abortion [12]. Less than 1 % of patients with partial molar pregnancies develop persistent gestational trophoblastic disease [13]. A classic complete hydatidiform molar pregnancy is the result of abnormal fertilization (empty ovum) resulting in a diffusely cystic placenta without fetal tissue present. Classically, partial molar pregnancy from triploidy results in a fetus with concomitant molar (cystic) degeneration of the placenta (Fig. 10). Though the survival to term is rare, identification is important for management and genetic counseling regarding future pregnancies. The appearance of a twin pregnancy with a normal fetus and complete molar degeneration of the second fetus is similar to a partial molar pregnancy. However, these entities may be differentiated; identification of a separate normal placenta would be consistent with a twin pregnancy with a normal fetus and complete molar degeneration of the twin fetus.

Placental cysts have numerous etiologies, and a thorough discussion is beyond the scope of this summary. Fetal demise is a well-recognized cause of placental cystic degeneration. A small number of central cysts in the placenta (placental venous lakes) may be seen in up to nearly 20 % of pregnancies in the second trimester and tend to be associated with thicker (>3 cm) placentas [14]. No strong link between placental lakes and adverse pregnancy outcomes exists, though placental cysts within the first trimester are more frequently associated with abnormal pregnancies.

Placental mesenchymal dysplasia (PMD), a recently recognized entity, has an appearance which sonographically overlaps partial molar pregnancy and is likely underdiagnosed [15]. PMD may present with elevated hCG levels, as do molar pregnancies. While PMD fetal karyotypes are normal, PMD is associated with increased preterm deliveries and other adverse pregnancy outcomes including

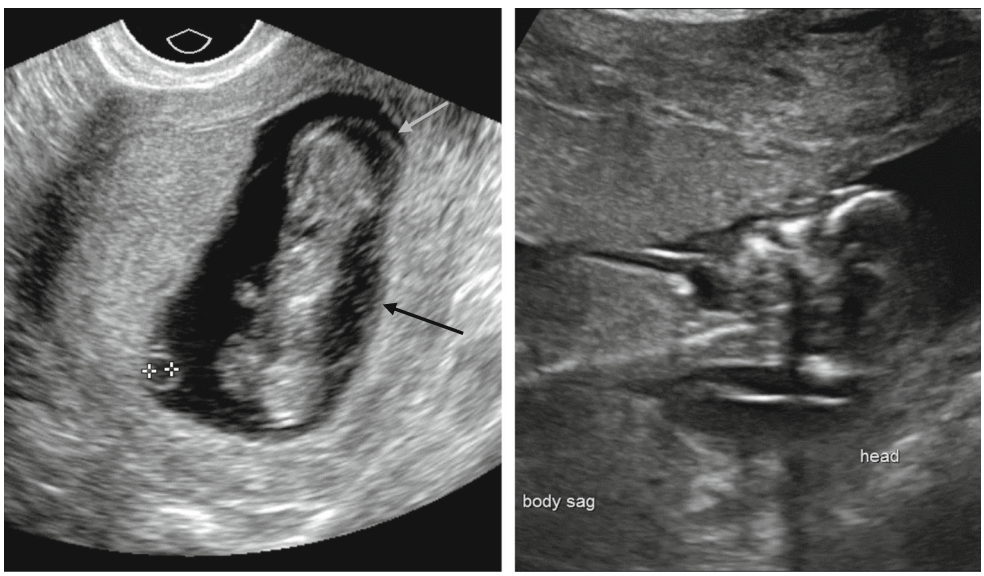

Fig. 9 Lymphangiectasia. *Left* Diffuse edema is seen along the fetus. *Right* Edema is seen posteriorly and anteriorly

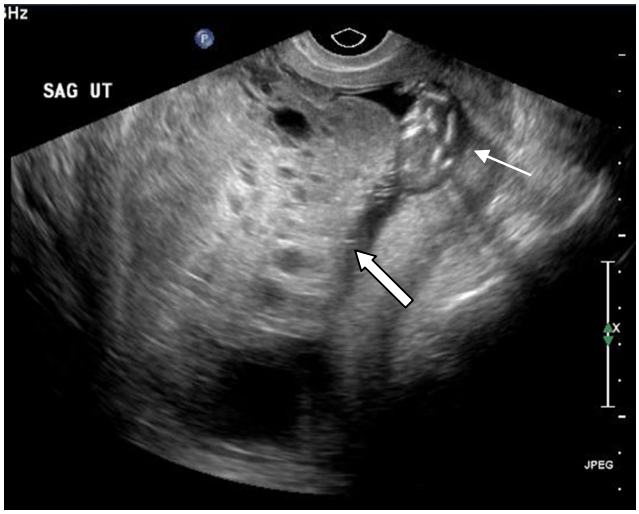

Fig. 10 Partial hydatidiform mole (triploidy). Fetal parts (*thin arrow*) are seen adjacent to an enlarged placenta with cystic degeneration (*thick arrow*)

Beckwith-Wiedemann syndrome. Currently, the diagnosis of PMD can only be confirmed by histologic evaluation of the placenta. Given possible diagnostic uncertainty and the overlap of several distinct entities, follow-up sonography including complete fetal surveys are recommended, depending upon gestational age, and supplemented with laboratory and genetic evaluation as indicated.

Conclusion

Endovaginal sonography plays a dominant role in the evaluation of pregnancy-related complications. Even during routine emergent evaluation, continued improvement in sonographic resolution now permits the visualization of detailed fetal anatomy that was previously much more difficult to visualize and acquire. Awareness of both normal and abnormal findings and fetal anatomy improves the detection of anomalies and avoidance of potential pitfalls.

Conflict of interest The authors declare that they have no conflict of interest.

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