Fetal and Placental MR

Optimizing Perinatal Management

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http://hvra.com/our_services/maternal_fetal_imaging-for_physicians

Placental Magnetic Resonance Imaging (MR)

- adherent placentation – increta, percreta
- "extreme" placental pathologies and early onset of unexplained IUGR.
  - massive villous fibrin deposition (maternal floor infarction)
  - fetal-maternal thrombotic (hemoragic) vasculopathy
  - maternal arterial malperfusion
  - villitis of unknown etiology

ADHERENT PLACENTATION – PLACENTA ACCRETA, INCRETA AND PECRETA

- The goal of imaging is to identify "at risk" population into low risk and high risk categories as they relate to increta/percreta and risk for clinically significant hemorrage and need for hystercectomy.
- Imaging is not helpful in identifying mild placenta accreta.
- Adherent placentation occurs when there is a defect in the decidua basalis allowing the anchoring vill to adhere to the myometrium.

- Frequency of adherent placentation has increased ten-fold over the last twenty years and is now observed in approximately 9% of women with placenta previa or 1 per 500 deliveries.
- Cesarean delivery is the most common cause of decidual defects.
- In patients with prior cesarean deliveries who have placenta previa or low lying anterior placenta the risk of adherent placentation increases from 24% for one prior C-section to 67% for four prior C-sections.
- Incidence of placenta previa with at least one C-section is 10%

OTHER RISK FACTORS FOR CLINICALLY SIGNIFICANT ADHERENT PLACENTATION

- Subserosal uterine myomas
- Prior myomectomy
- Asherman's syndrome
- Maternal age over 35 years of age
- Smoking
- Elevated alpha fetoprotein levels
  PAPPA > 3.0 MOM

Imaging assessment of adherent placentation – 2D US, Color Doppler US, MR

- Identification of clinical risk factors: AMA, prior CS, placenta previa, prior uterine surgery, multiple D&C.
- Placenta lacunae – number, morphology, doppler flow patterns.
- Placenta/myometrial interface – 2D and MR
- Bizarre vascularity – color flow mapping
- MR - posterior placenta; extent of percreta – vascular encasement , abdominal wall, intestinal involvement.

HVRA census increta/percreta. 2008-2014: 124 cases

- gravid patients at risk for clinically significant adherent placentation.
- referred by MFMs who desired second opinion after their own ultrasound studies.
- all patients had MR followed by transabdominal, transvaginal color Doppler ultrasound studies.
- all patients had MD performed US scanning and MR interpretation by one radiologist (DJC).
- delivery, operative and pathology reports reviewed for all patients

HVRA's increta/percreta study - 124 cases

Interpretive endpoint

- "High-risk" categorization – patient at high-risk for increta/percreta or patterns of accreta that might result in hysterectomy.
- "Low-risk" – no signs of percreta/increta. Accreta of a type that might result in hysterectomy unlikely, but cannot be entirely excluded.

HVRA's Imaging Census for Placenta Increta/Percreta from January 2008 through September 13, 2014. 140 patients studied.

	No. of pts
True positive:	41
True negative:	84
False positive:	9
False negative:	6
Sensitivity	87%
Specificity	90%
Positive Predictive Value	82%
Negative Predictive Value	90%
False Positive Rate	10%
False Negative Rate	13%
Accuracy	89%

HVRA review of four false negative cases

- Complete previa with one C-section. Imaged at 29 weeks. Less than 5 lacunae with no focally unusual vascularity and intact sub placental myometrium.

  Bleeding with delivery 10 days later. Pathology – increta.

- Uterine rupture with hysterectomy at 24 weeks. (At site of previous posterior fundal hysterotomy.) Ultrasound and MR imaging at 22 weeks identified focal loss of myometrium and focal placental bulge– no lacunae and no abnormal vascularity. Interpreted as uterine window, but no increta. Path – increta with microscopic percreta.

Review of HVRA's false negative cases for increta/percreta

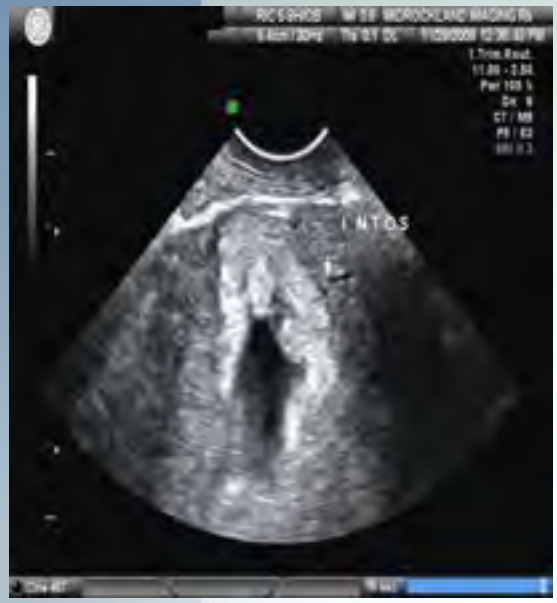
- Patient imaged at 32 weeks with placenta previa and two prior C-sections. Path report describes two sites of fundal percreta. Case review – placenta extended from fundus to cervix. Fundus was not imaged on MR and ultrasound.

- Patient imaged at 28 weeks demonstrating a focal lower uterine segment placental bulge with myometrial thinning, but no lacunae and no focally unusual vascularity. Case interpretation as no percreta and low-risk for increta. Bleeding at 32 weeks with follow up outside imaging demonstrating increasing lacunae. Delivered at 33 weeks with pathology demonstrating increta.

Review of HVRA's false positive cases, "high-risk" for increta/percreta – 8 cases

Six out of eight false positive cases all demonstrated low-lying placenta or previa with greater than five lacunae, focal placental bulging and myometrial thinning and focally abnormal vascularity.

First trimester diagnosis of placenta increta in CS scar



OBSTETRIC MR CLINICAL VIGNETTE

Adherent placentation – true negative for increta/percreta.

Complete previa.

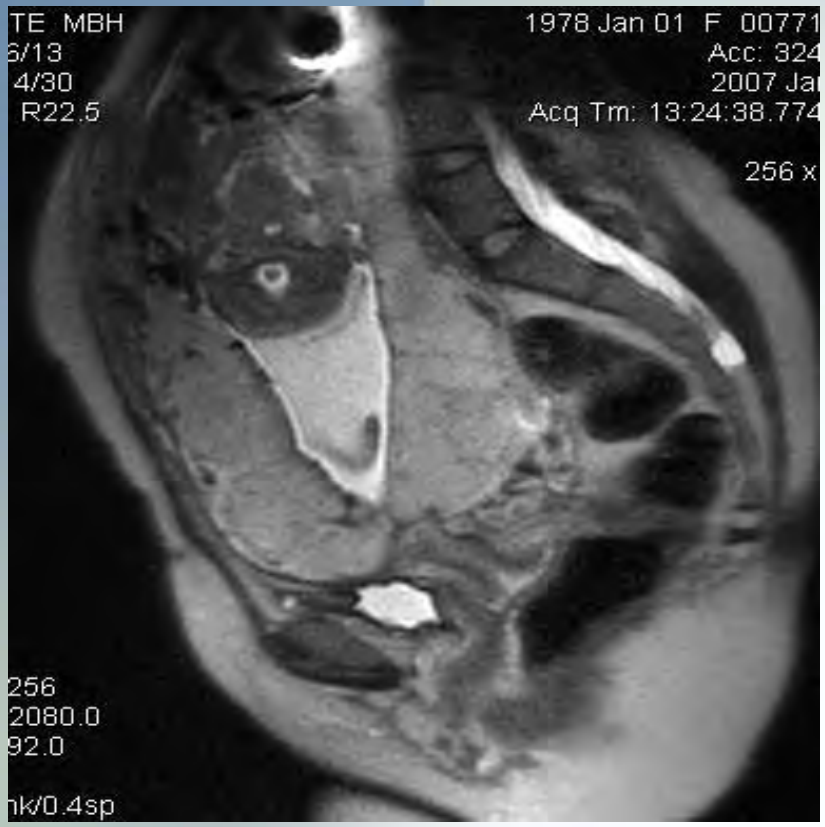
History: Complete placenta previa. Prior C-section.

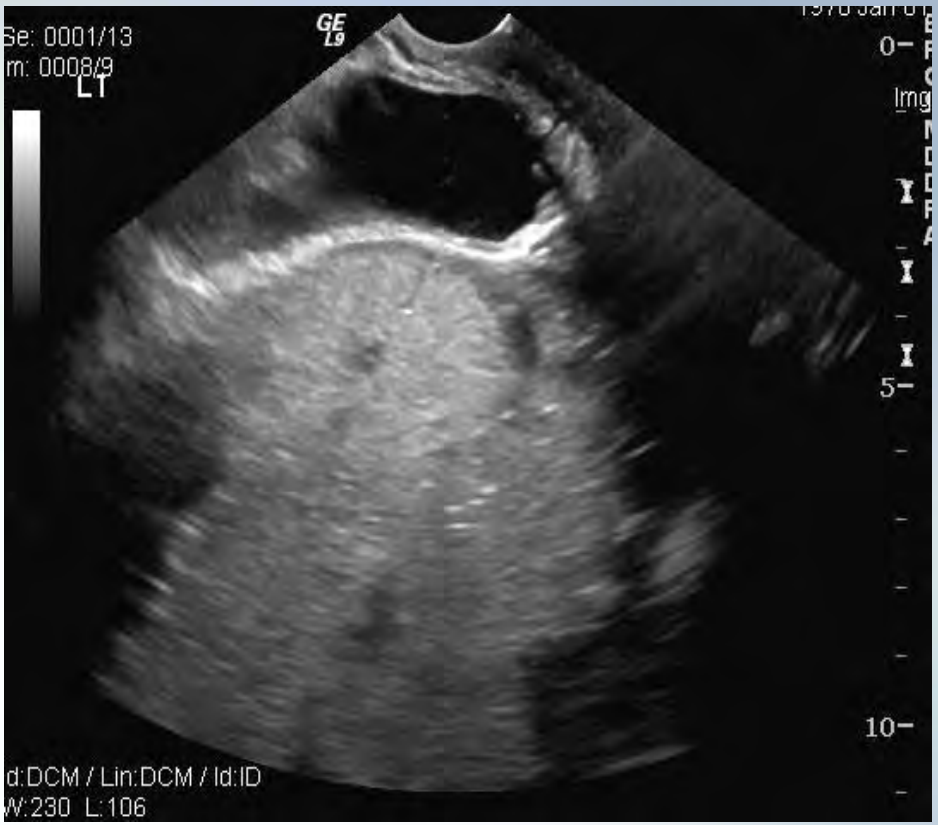
Findings: Ultrasound and MR demonstrate no signs of placenta increta/percreta.

OBSTETRIC MR

Adherent placentation – true negative for increta/percreta.

Complete previa.





Adherent placentation – true negative for increta/percreta.

Uterine Window.

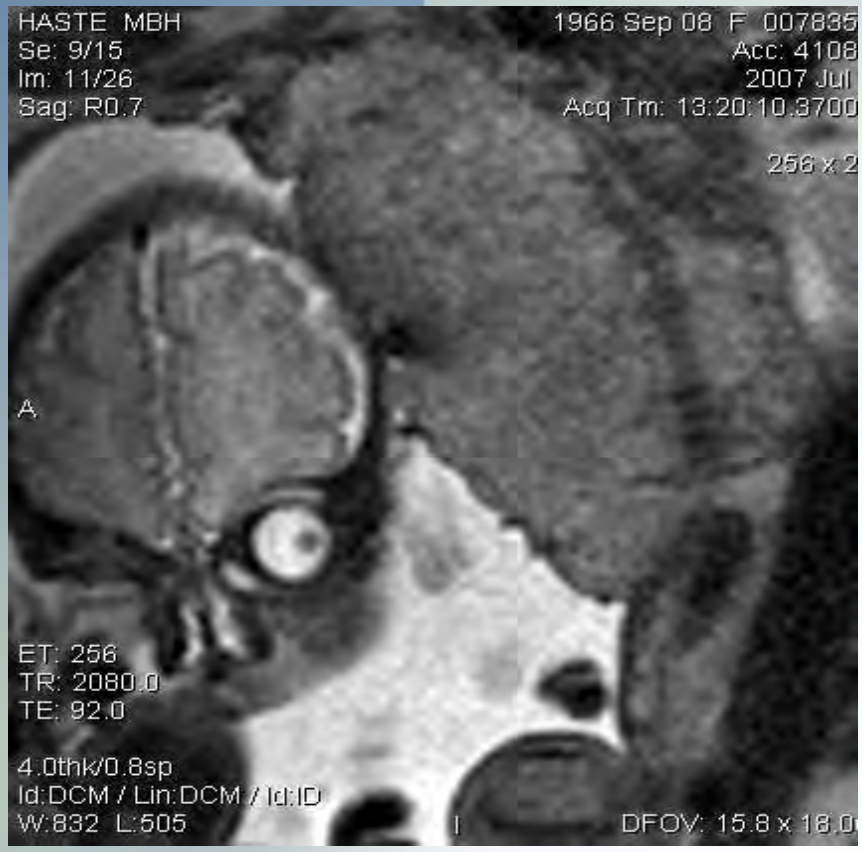
Elderly multigravida, status post prior pregnancy with placenta previa and increta requiring posterofundul CS incision.

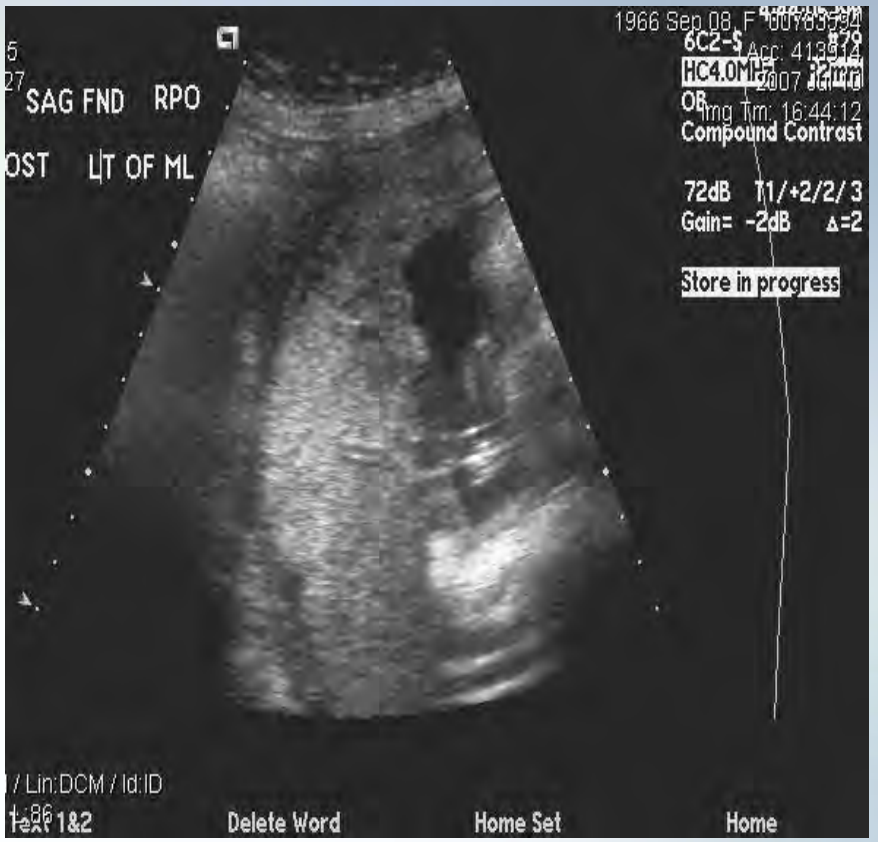
Imaging Diagnosis: Focal placental bulging into zone of thinned myometrium but low risk for increta/percreta.

Clinical Diagnosis: At time of elective c-section a large uterine window found at site of prior c-section corresponding to imaging findings.

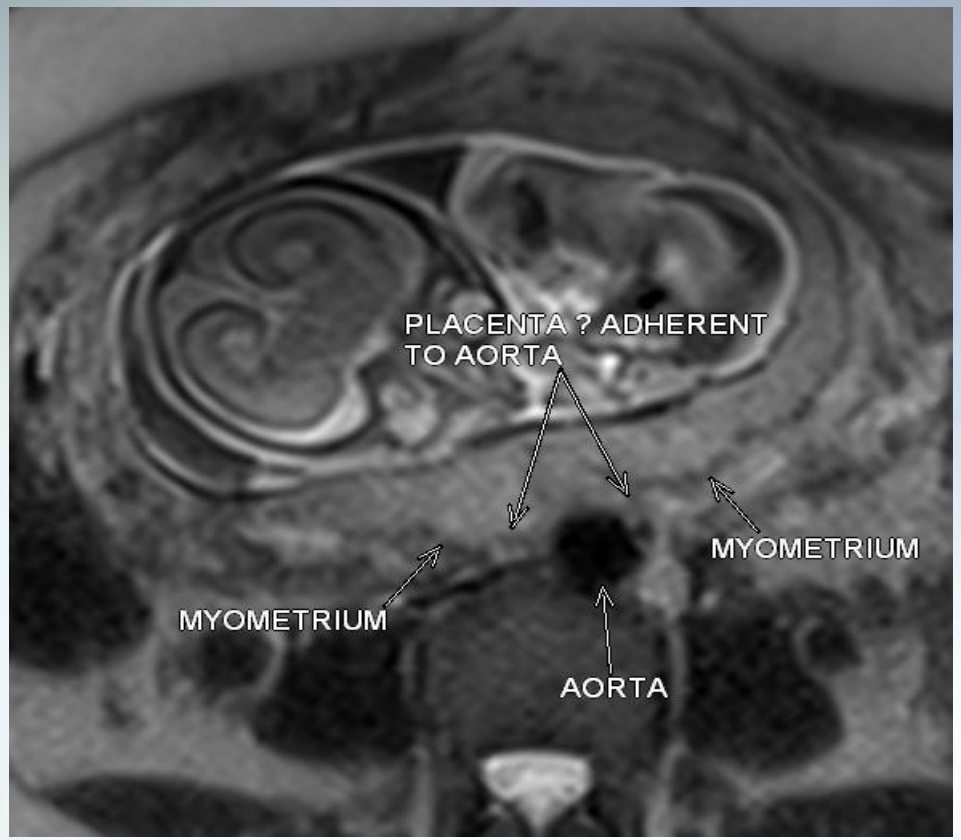
OBSTETRIC MR

Adherent placentation – true negative increta/percreta. Positive for post CS uterine "window".



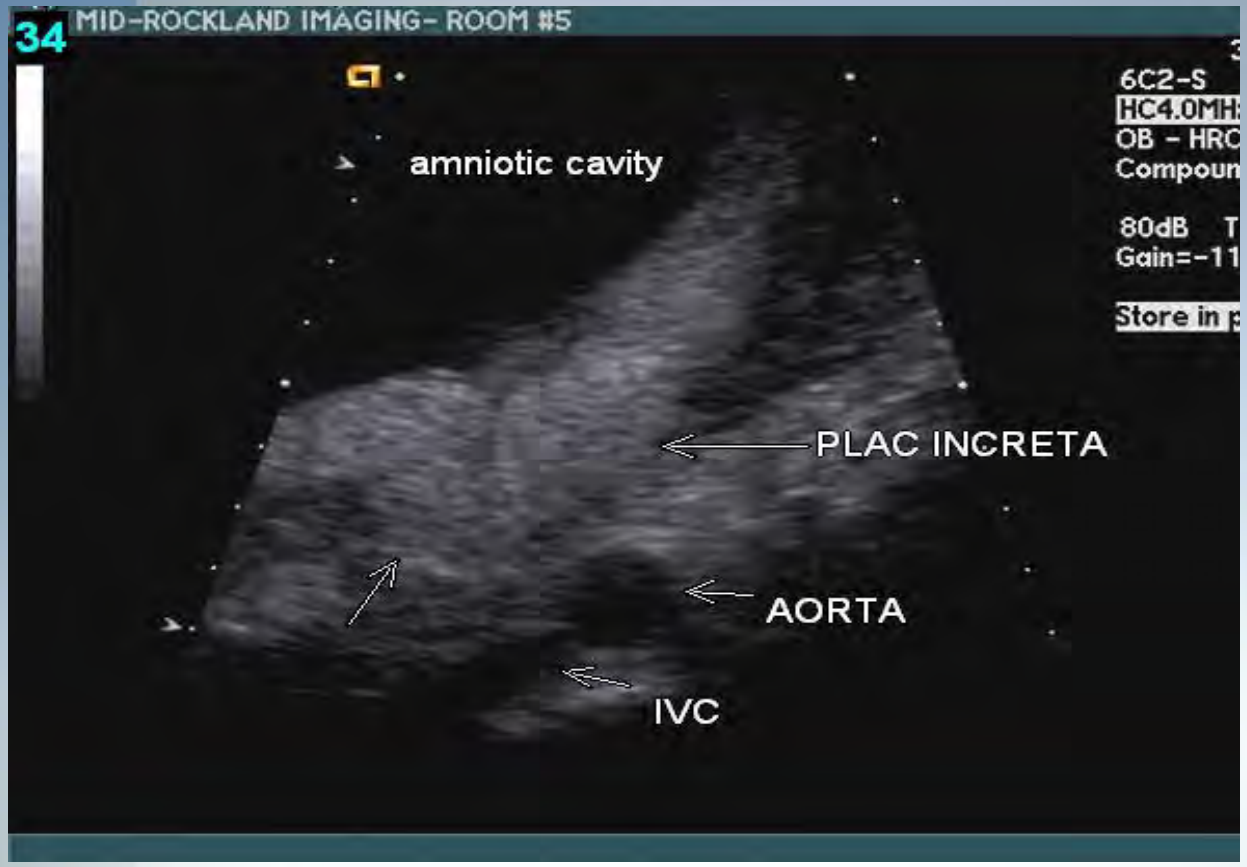


21w, same patient next pregnancy. At site of prior uterine window, focal bulge and effacement of myometrium but no lacunae.



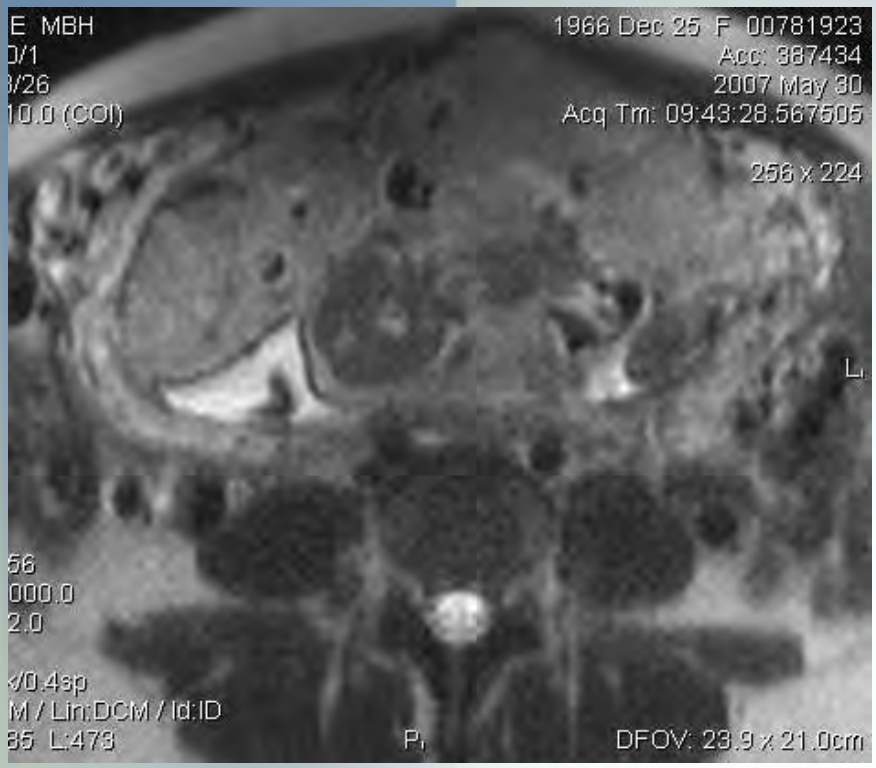
At 24w sudden pain, uterine rupture requiring hysterectomy. Path – Deep increta with microscopic percreta.

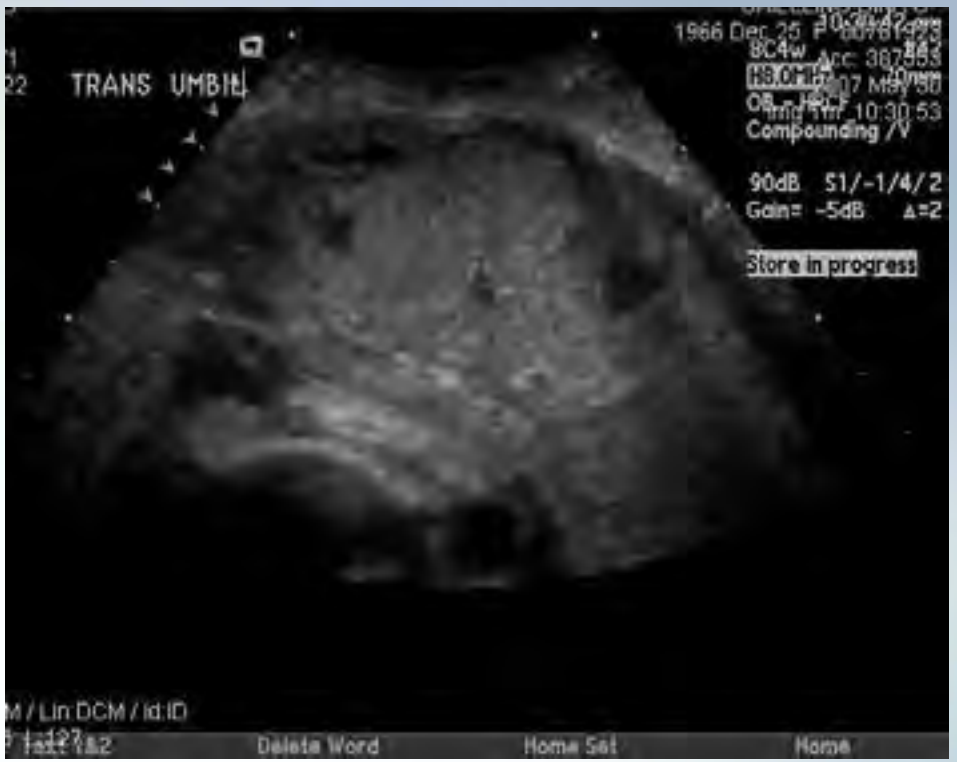
21w, same patient next pregnancy. At site of prior uterine window, focal bulge and effacement of myometrium but no lacunae.



At 24w sudden pain, uterine rupture requiring hysterectomy. Path – Deep increta with microscopic percreta.

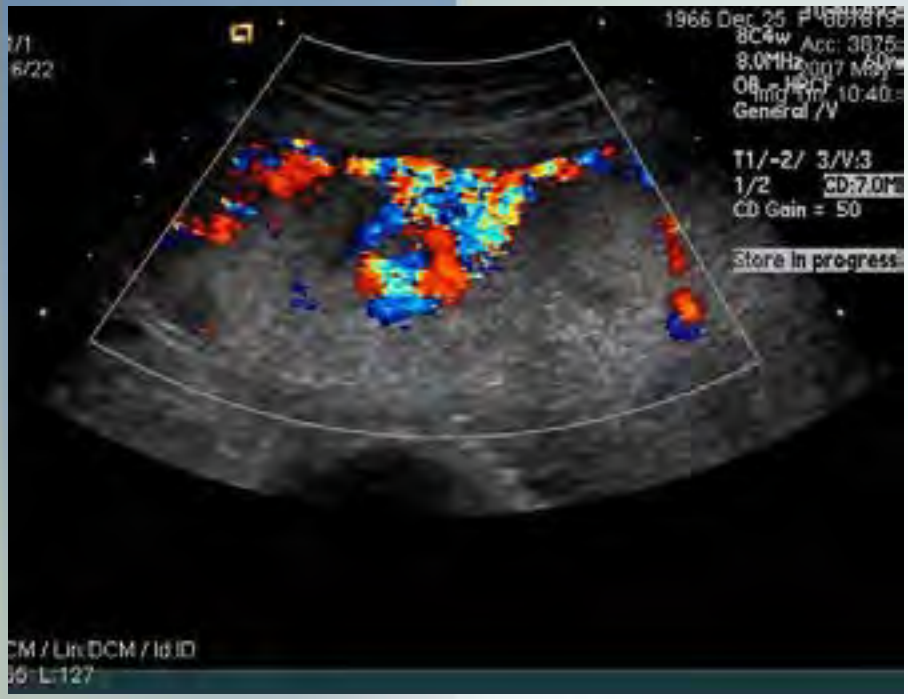
Adherent placentation – true positive placenta accreta.

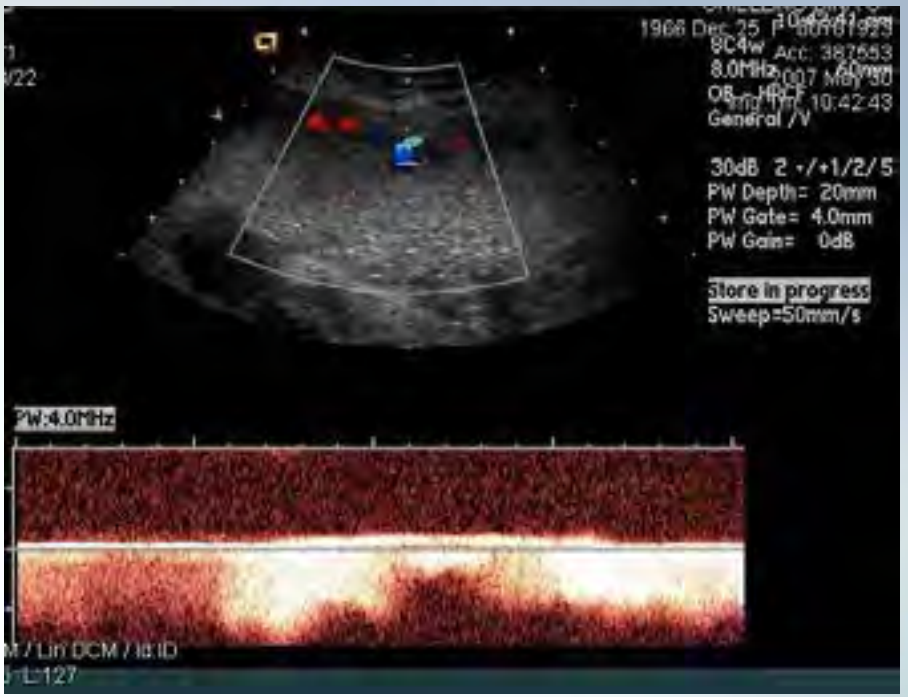




OBSTETRIC MR CLINICAL VIGNETTE

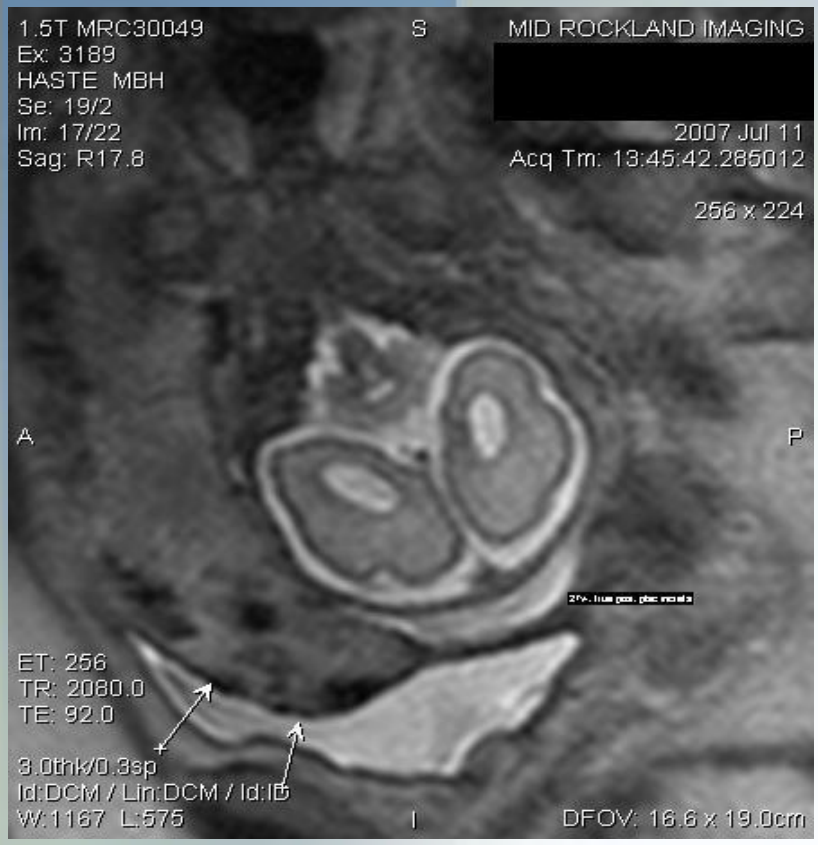
Adherent placentation – true positive placenta accreta.

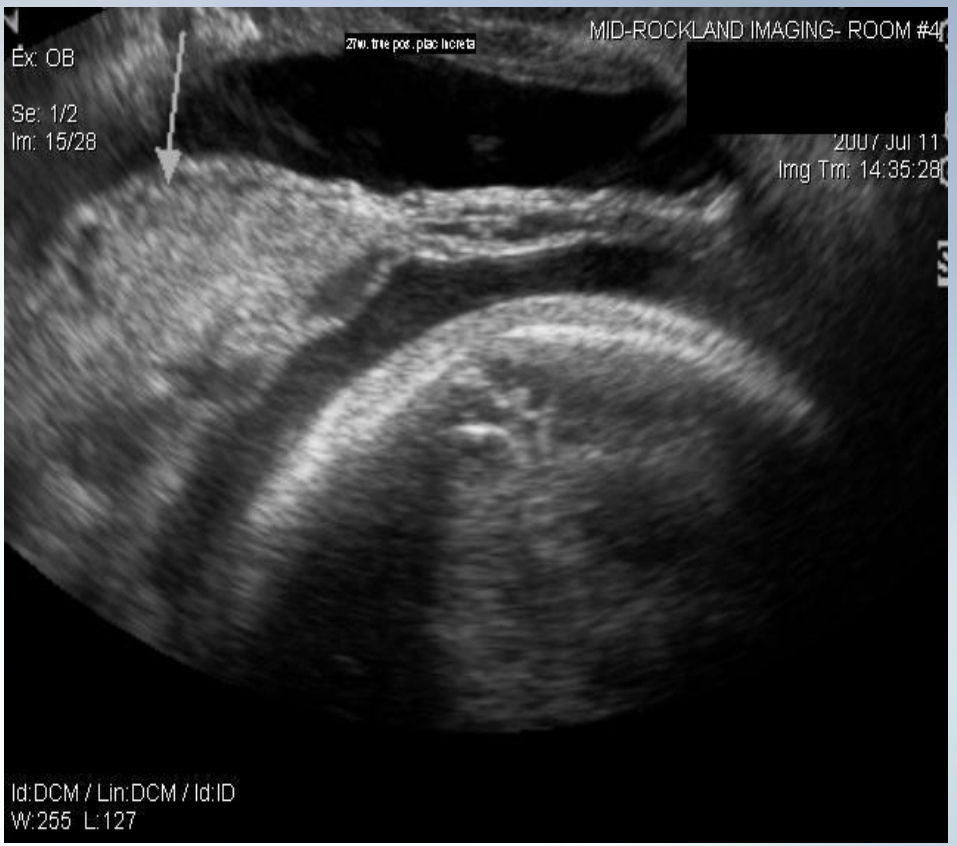




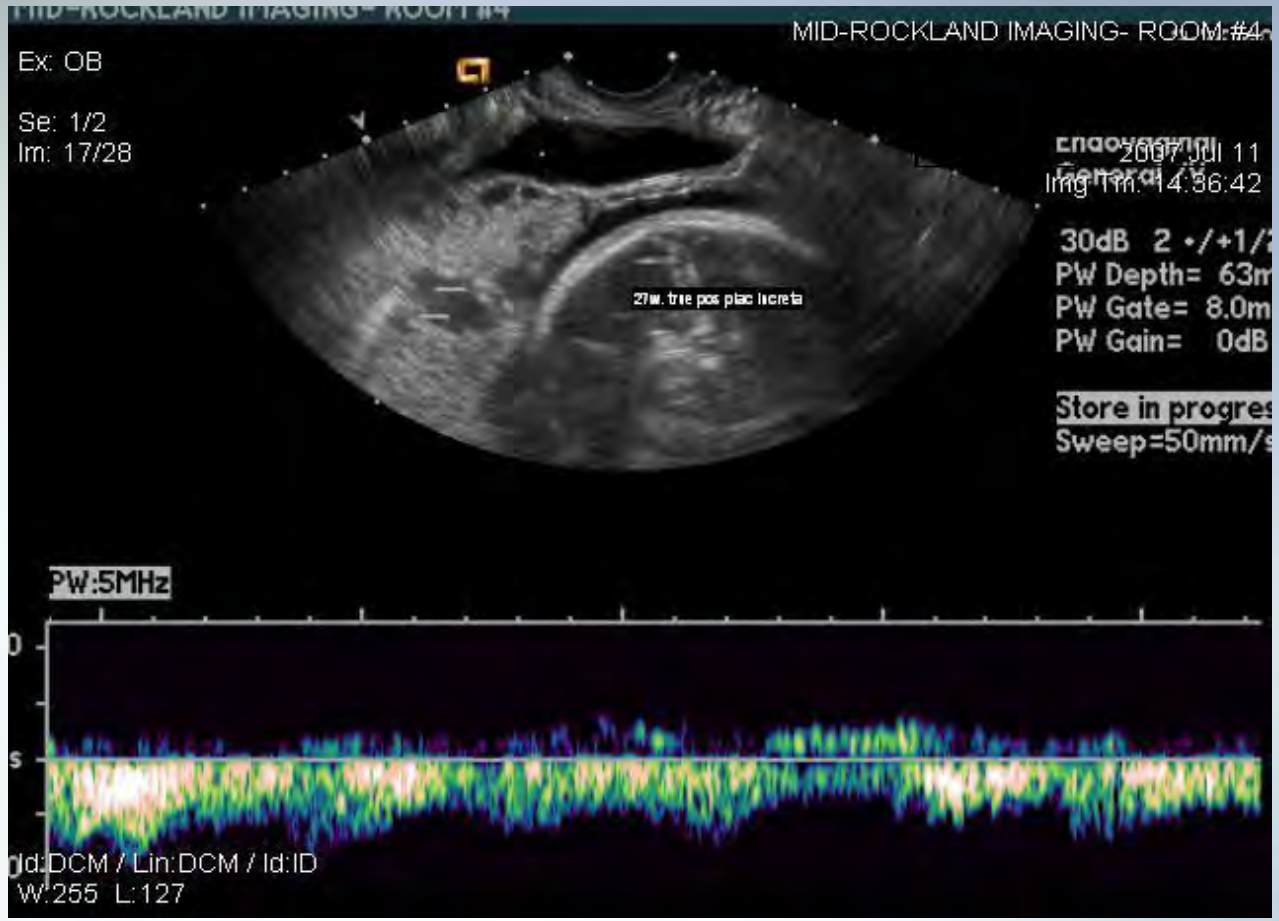
OBSTETRIC MR

Placenta Percreta – True positive. No previa.





Placenta Percreta – True positive. No previa.



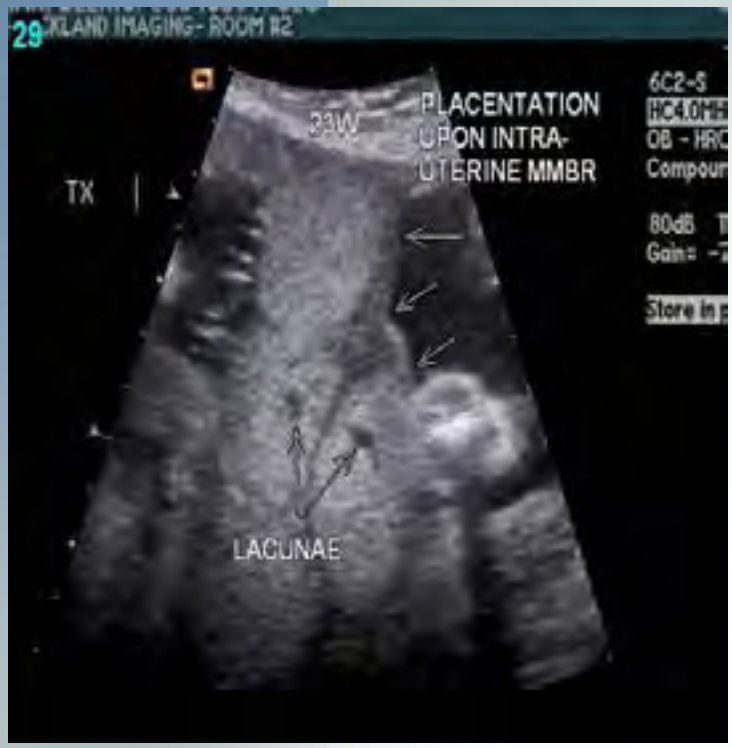
HISTORY: 23-week ultrasound demonstrates placentation upon an intrauterine membrane. s/p hysteroscopic resection of large intracavitary fibroid. Initial ultrasound demonstrates two small placental lacunae and velamentous cord insert.

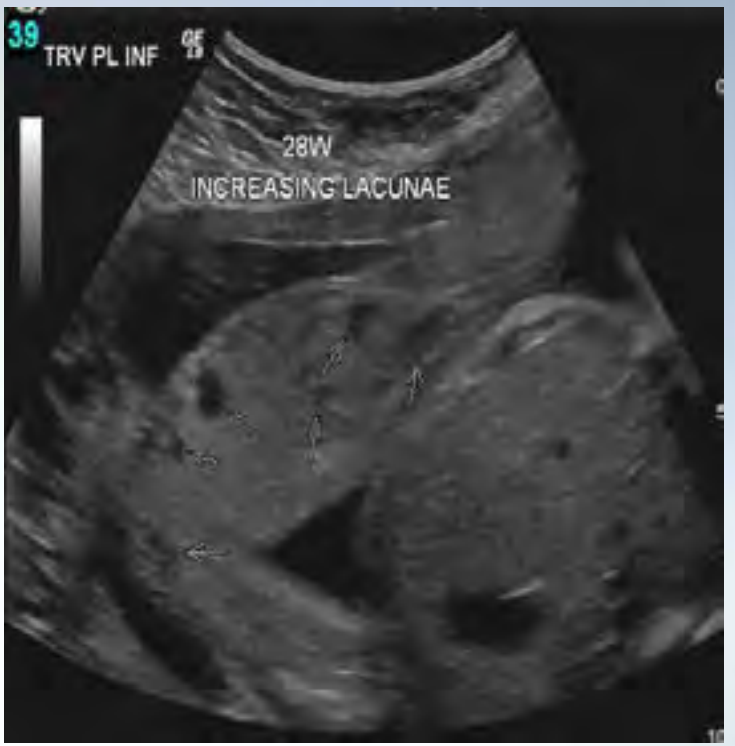
Follow up ultrasound at 28 weeks demonstrates increasing number of intraplacental lacunae worrisome for potentially clinically significant adherent placentation.

FINAL DIAGNOSIS: Placenta accreta requiring hysterectomy. Referring physician was prepared for this possibility.

TEACHING POINT: In addition to the history of prior C-sections, unusual patterns of placentation, velamentous cord insert and intraplacental lacunae are red flags increasing risk for adherent placentation.

Placentation upon intra uterine membrane. Placenta accreta requiring hysterectomy.





Personal perspectives on imaging for clinically significant adherent placentation

- time commitment to do it well in a busy practice
- depending upon pretest clinical risk factors, MR usually not contributory if trusted ultrasound demonstrates no lacunae, sharply defined basal plate with no focal bulges, intact subplacental myometrium and no focally bizarre angioarchitecture nor Doppler arterio-venous shunting.

Imaging challenges for ultrasound and MR – creating contrast in the subplacental myometrial zone

- discrete delineation of the subplacental myometrium is made difficult by the inherent thinning of the myometrium and the increasing engorgement of serosal vessels normally accompanying advancing gestational age

- thin/obscured subplacental myometrium in absence of lacunae, absence of focally bizarre angioarchitecture/arterio-venous shunting –

    - differential diagnosis favors absence of increta, but beware of false negative possibility

How does MR help in the imaging risk assessment for clinically significant adherent placentation?

- MR performed and reviewed before hands-on ultrasound optimizes multimodality evaluation of suspicious areas.

- MR provides a reassuring complete multiplanar documentation of placental-myometrial interface. Especially helpful in assessing patients whose risk factors are other than C-section – myomectomy, Asherman's, septate uterus.

- Provides the most global pelvic assessment for extent of percreta – abdominal wall, intestine, sidewall vascular encasement.

Placenta MR and Extreme Placental Pathologies

- "extreme" pathologies
  - massive perivillous fibrin deposition
  - fetal-maternal hemorrhagic vasculopathy
  - Maternal arterial malperfusion
  - Villitis of unknown etiology

Clinical indications

- unexplained second trimester growth restriction
- unexplained second trimester oligohydramnios

Especially if associated with:

- unexplained extreme trending of first and/or second trimester maternal serum biochemical analytes
- past history of stillbirth/neonatal death

Placental MR and placental pathology

- research is in its infancy
- only a few studies establishing predictive attributes published

Why placental MR for unexplained early growth restriction?

MR is underutilized in the detection of severe placental pathologies associated with early onset growth restriction –

- fetal-placental thrombotic (hemorrhagic) vasculopathy
- massive perivillous fibrin deposition (maternal floor infarction)
- maternal arterial malperfusion
- villitis of unknown etiology

Depending upon the specific pathology, significant association with

- recurrence risk
- preterm delivery
- abruption
- stillbirth and perinatal mortality
- cerebral palsy

Prenatal diagnosis of severe placental pathology indicates antenatal causation

Placental MR – placental insufficiency.

Pathophysiologic correlations

•decreased T2 signal (dark) placenta correlates with increased fibrosis, infarction, calcification

•diffusion weighted MR pulse sequences detect restricted water diffusion in the extracellular space as measured by decreased apparent diffusion coefficients (ADC)

•decreased ADC values correlate with restricted oxygen diffusion

Diffusion Weighted MR Imaging of the Placenta in Fetuses With Placental Insufficiency. Bonel. Radiology. December 2010

- 33 growth restricted fetuses
- Growth restriction defined as birth weight less than the 10th percentile, flattening of prenatal growth curve or fetal weight less than the 10th percentile.
- at $p<0.01$, ADC values of placentas of growth-restricted fetuses (146 +/- 10.63 mm/sec^2) were significantly different than normally grown fetuses (177 +/- 18.9 mm/sec^2)
- all 33 growth-restricted fetuses had decreased ADC values
- 9 of the 33 growth-restricted fetuses had normal prenatal ultrasound findings. 6 of these had positive histopathology. All 9 had decreased ADC values.

MR of second unexplained trimester IUGR – placental insuffiency

Case presentation – 19W IUGR refused karyotyping. SPROM/PTD at 28 weeks. Final pathologic diagnosis – extensive chronic villitis of unknown etiology (VUE).

- increased placental impedence mean uterine artery Doppler PI >1.45 Normal middle cerebral arterial Doppler and normal-appearing placenta on ultrasound
- normal fetal DNA in maternal serum screening
- normal infectious screening
- extreme trending of multiple first and second trimester biochemical analytes
  - PAPP-A 0.13 MoM (0.1 percentile)
  - free bHCG 12.75 MoM (99.9 percentile)
  - uE 0.16 MoM (0.1 percentile)
  - Inh 5.5 MoM (99.9 percentile)

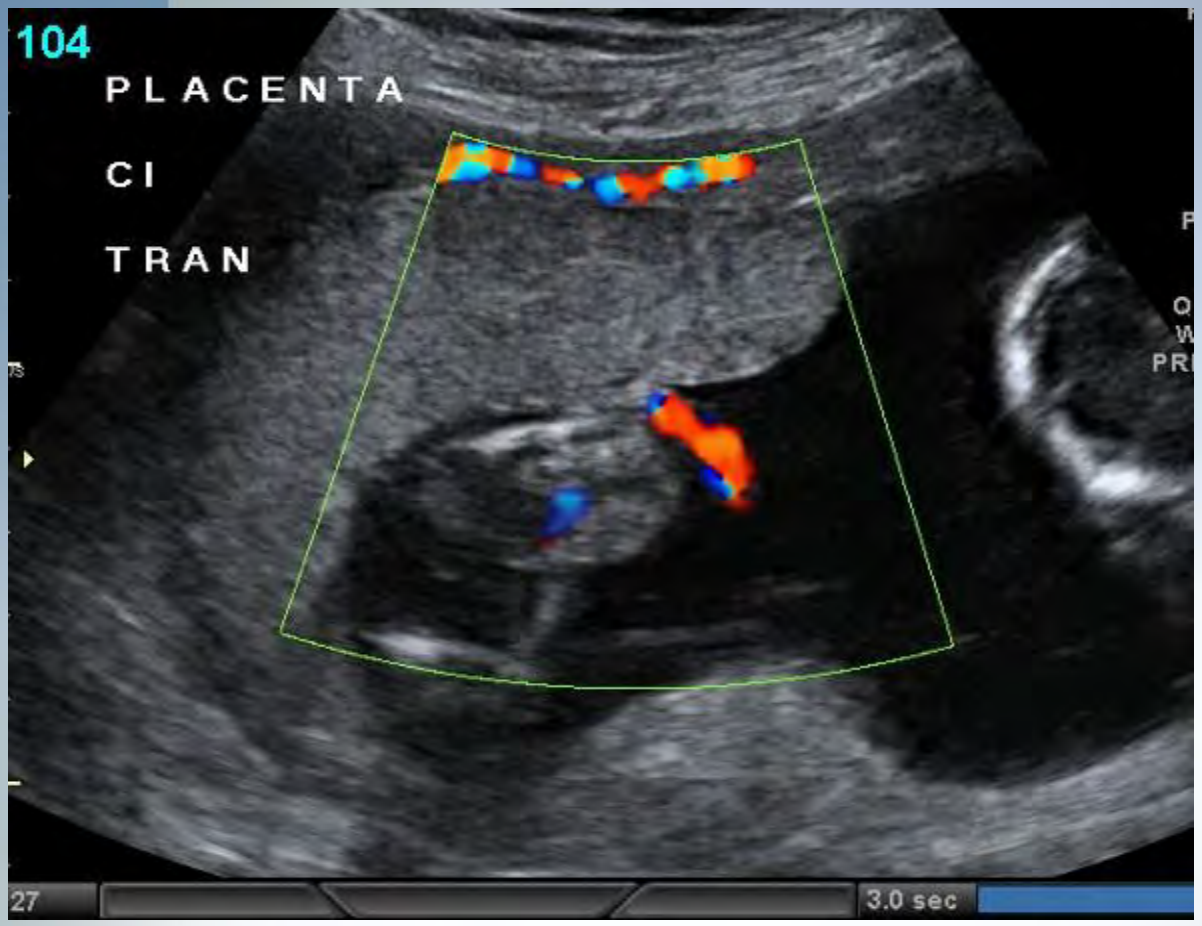
Case presentation – SPROM/PTD at 28 weeks.

MR of unexplained second trimester IUGR – placental insuffiency

Why do placental MR?

- patient refused karyotyping – etiology of IUGR uncertain
- attempt MR identification of extreme placental pathologies associated with high risk for fetal/neonatal morbidity and mortality.

19 W IUGR. Villitis of unknown ideology



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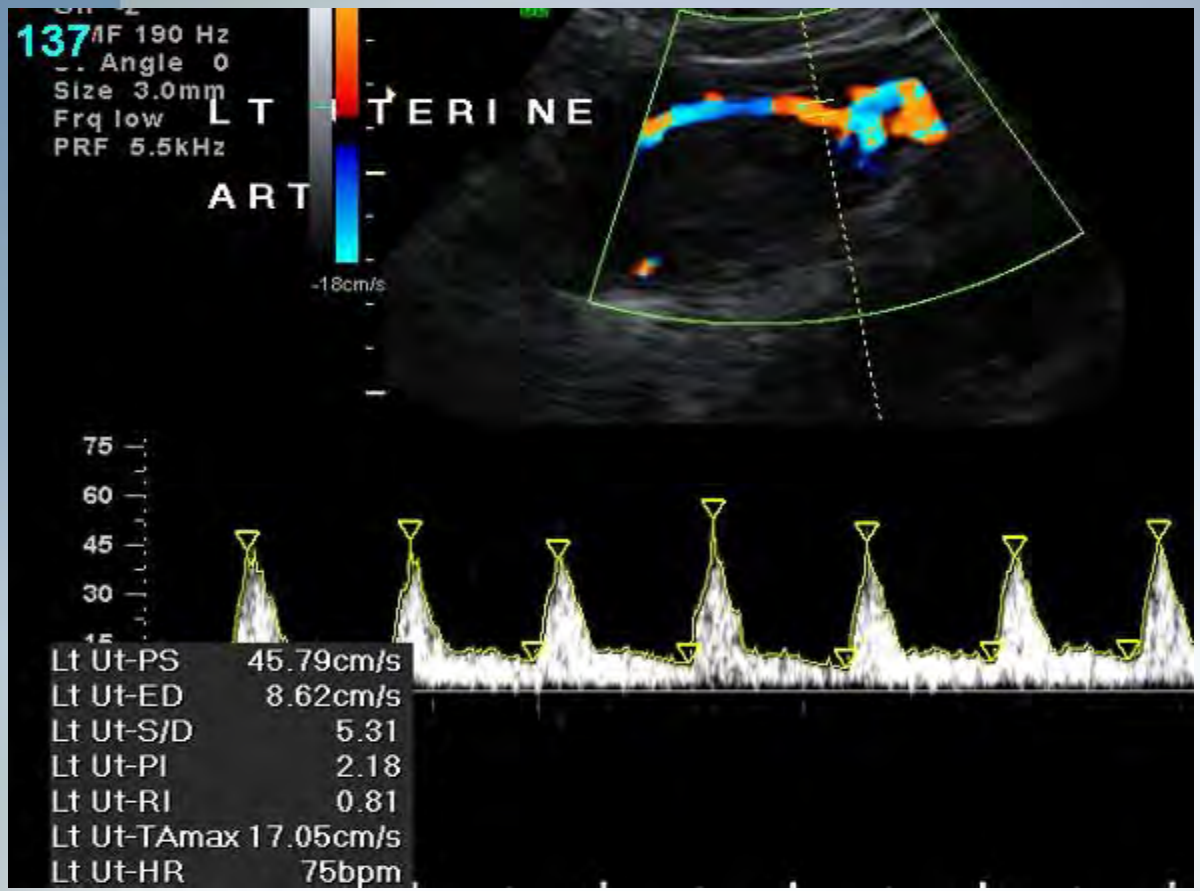

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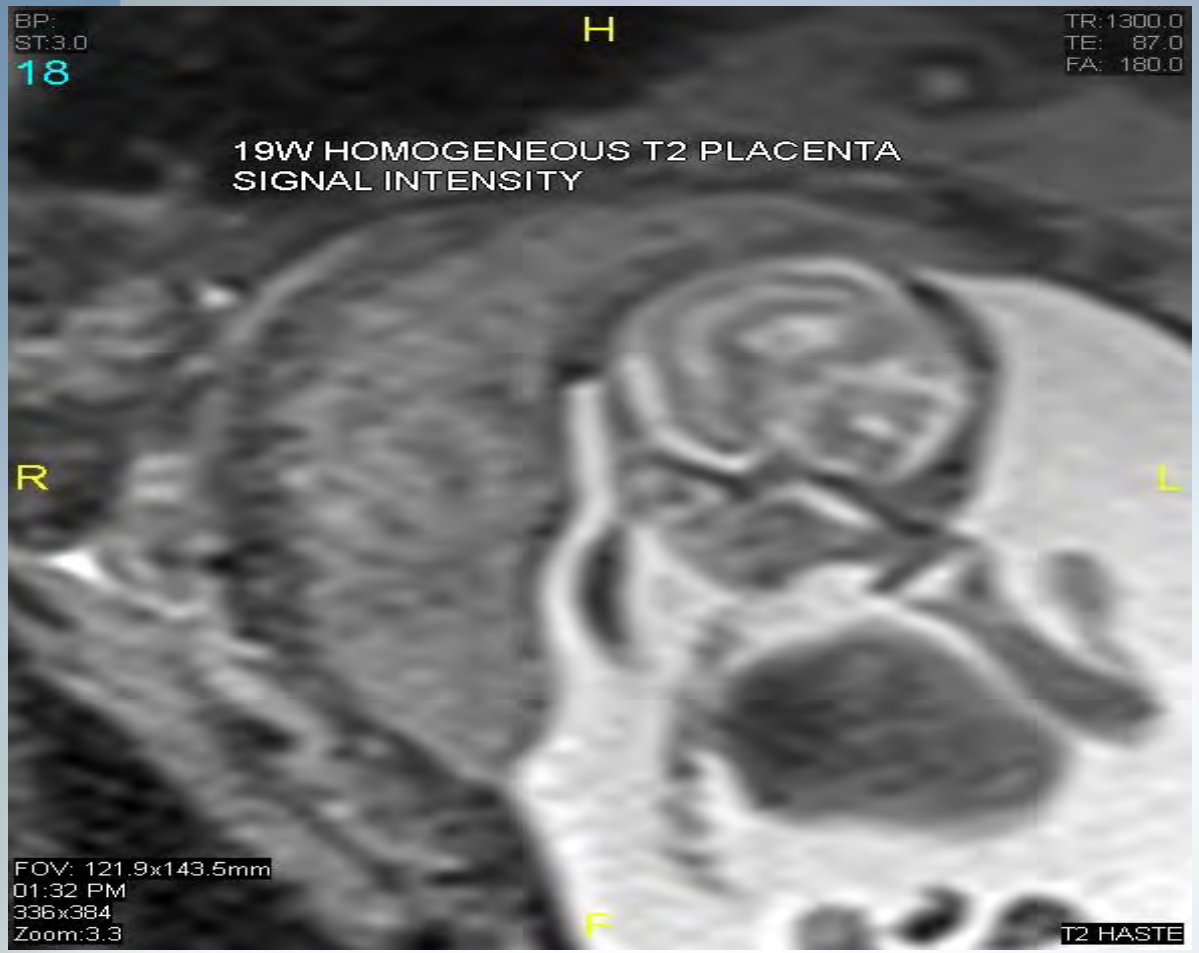

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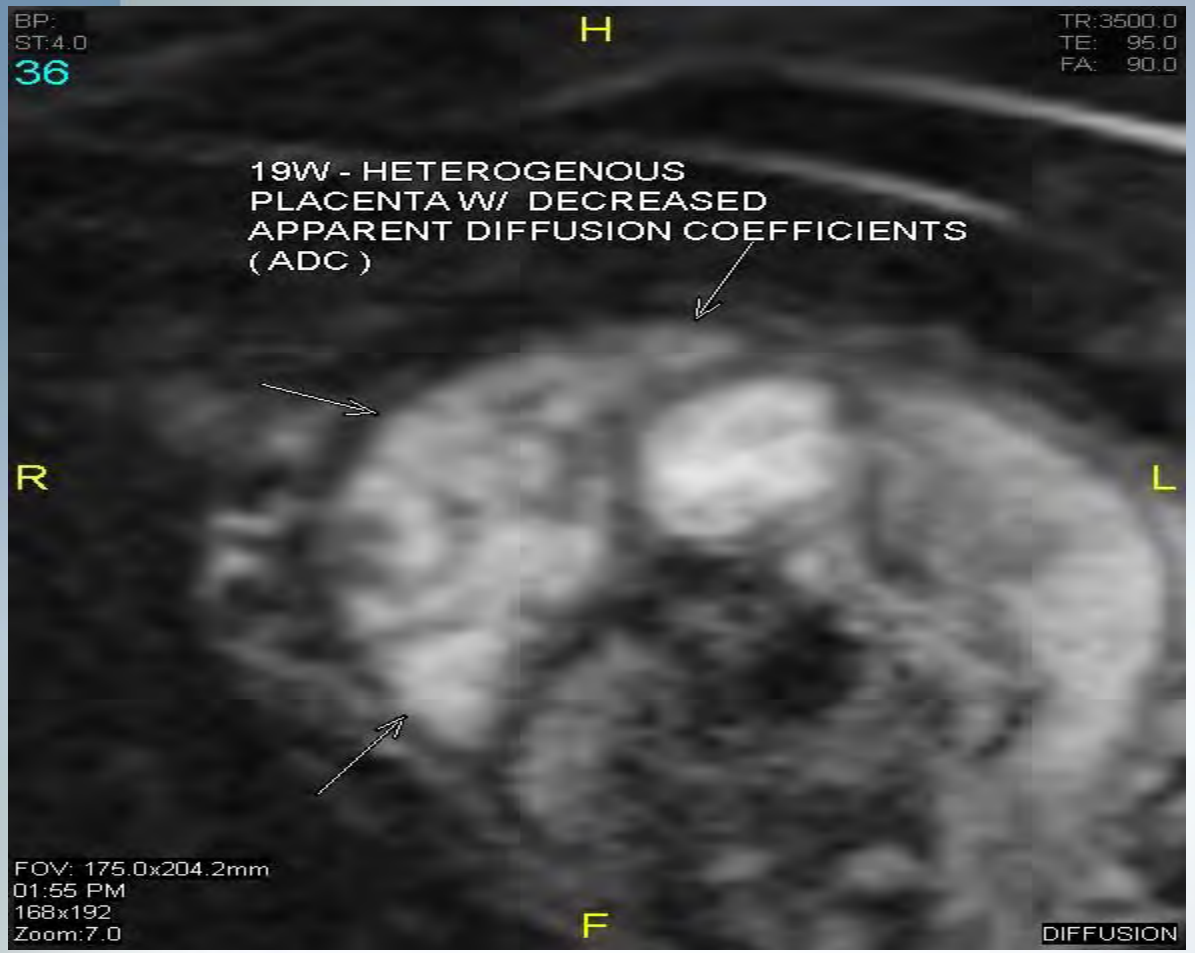


MR of unexplained second trimester IUGR – placental insuffiency

Case presentation – SPROM/PTD at 28 weeks

- clinical outcome – euploid neonate 1 lb, 4 oz with no structural malformations nor syndromic stigmata
- placental pathology – extensive chronic villitis of unknown etiology (VUE)
  - 191 g placenta less than the third percentile for gestational age; 3rd-10th percentile for neonatal BW
  - VUE: not part of impaired maternal-fetal perfusion spectrum

Pathologic diagnosis/clinical correlation

Villitis of unknown etiology

- widely believed to be a host – versus – graft response by mother directed at fetal antigens in the villous stroma

- major risk factor for CNS injury and stillbirth

- significant recurrence risk 20-30%

Redline. Human Pathol. October 2007; 38 (10): 1439-1446.

Abnormal placenta – IUFD 26w - massive perivillous fibrin deposition

History : 22w GA referred for suspected placenta increta. Patient has unexplained elevated maternal serum AFP.

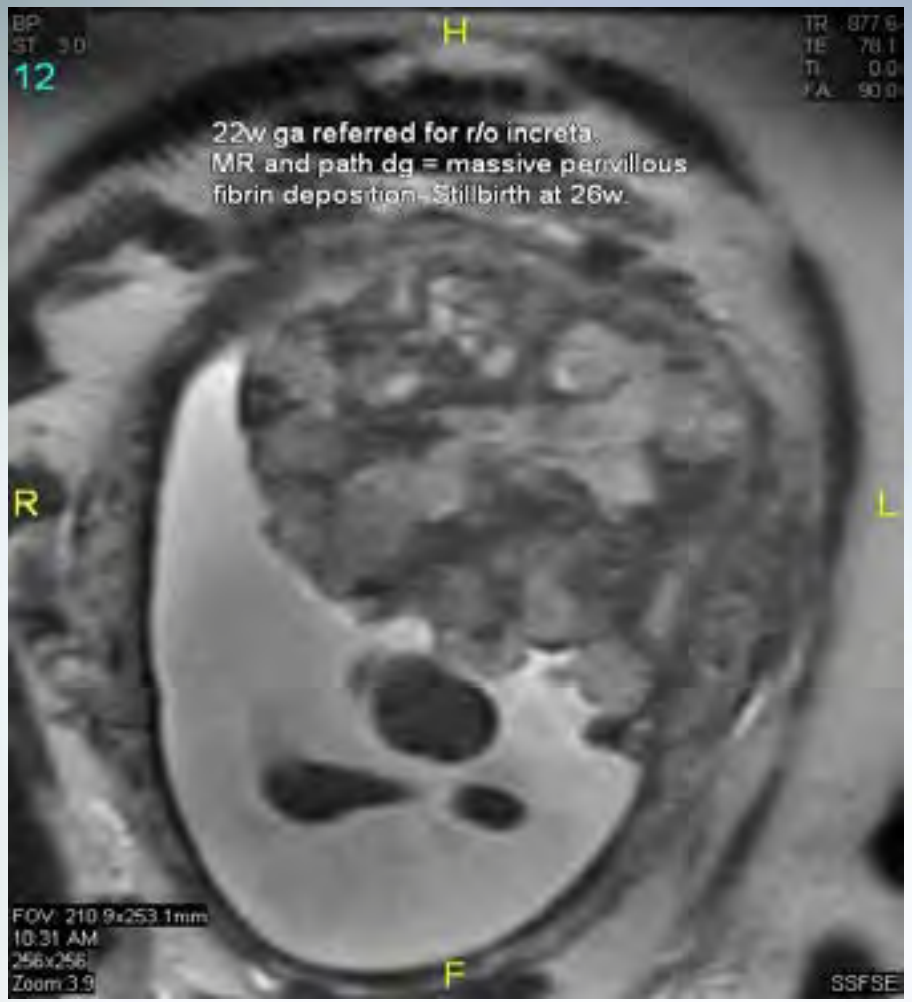
MR: Profoundly heterogeneous placenta with large curvilinear bands of hyposignal intense tissue. The distribution has an appearance similar in configuration to the convolutions of the cerebral cortex.

Clinical follow up: IUFD at 26w.

Teaching point: MR of the placenta can identify severe placental pathologies associated with early onset IUGR and marked morbidity/mortality such as hemorrhagic vasculopathy and massive perivillous fibrin deposition

MR establishes severe placental pathology unable to be seen on ultrasound

Abnormal placenta – IUFD 26w - massive perivillous fibrin deposition



Abnormal placenta – IUFD 26w - massive perivillous fibrin deposition

Pathologic diagnosis/clinical correlation

Massive perivillous fibrin deposition (maternal floor infarction)

risk factor for virtually all adverse outcomes from miscarriage to CNS injury

recurrence risk 50-75%

many affected women never achieve a successful pregnancy

Andres. Am J Obstet Gynecol. 1990; 163: 935-938.

Fetal MR Case Presentations

Last year HVRA performed 530 Obstetrical MR studies.

Daniel J. Cohen, M.D.

FETAL MR INTRODUCTION

The goal of our fetal MR is to optimize patient counseling and fetal/neonatal management by providing unique, clinically relevant contributions to fetal anatomic assessment above and beyond that which can be offered by high quality transabdominal/transvaginal ultrasound.

SPECIFICITY OF TERTIARY LEVEL FETAL IMAGING

- In general practice, MR and ultrasound are typically conducted by two different types of specialists – MR performed by radiologists who usually do not have a background in fetal medicine and who do not perform hands –on fetal ultrasound on a regular basis –

- Ultrasound often performed by physicians who do not have special skills in neuroanatomy and neuroimaging (Pilu, 1992-93)

SPECIFICITY CAN BE ENHANCED BY THE SAME PHYSICIAN PERFORMING BOTH MR EXAM AND TERTIARY LEVEL HANDS-ON ULTRASOUND IF NECESSARY.

Laurent Guibaud. *Prenatal Diagnosis* 2009, Vol. 29, pp 420-433.

MOST COMMON INDICATION FOR FETAL MR – INTRACRANIAL ABNORMALITIES ON ULTRASOUND

- Mild ventriculomegaly
- Nonvisualization or abnormal appearing cavum septum pellucidum.

"ISOLATED" MILD INTRACRANIAL VENTRICULOMEGALY

- Definition – lateral ventricles 10-12 mm, normal karyotype, no extracranial ultrasound abnormalities including normal fetal cardiac.
- MR will identify additional intracranial abnormalities that are clinically significant in approximately 10% of cases.

PATIENT COUNSELING FOR ISOLATED MILD VENTRICULOMEGALY

Definition of isolated mild ventriculomegaly: 10 -12 mm; versus 10-14 mm.

“Isolated” implies normal karyotype including microarray, normal fetal cardiac, no other malformations, non-progressive on follow up ultrasound.

PATIENT COUNSELING FOR MILD ISOLATED VENTRICULOMEGALY

When truly isolated, an optimistic prognosis can be provided. Outcome studies vary in their length of followup and in the type of neurodevelopmental and cognitive testing employed.

Less than 10% incidence of usually mild neurodevelopmental delays and/or learning challenges that are usually treated with early intervention.

PATIENT COUNSELING FOR ISOLATED MILD VENTRICULOMEGALY

Some authors feel better neurodevelopmental outcome in male than female fetuses
Better outcome in unilateral than bilateral mild isolated ventriculomegaly.

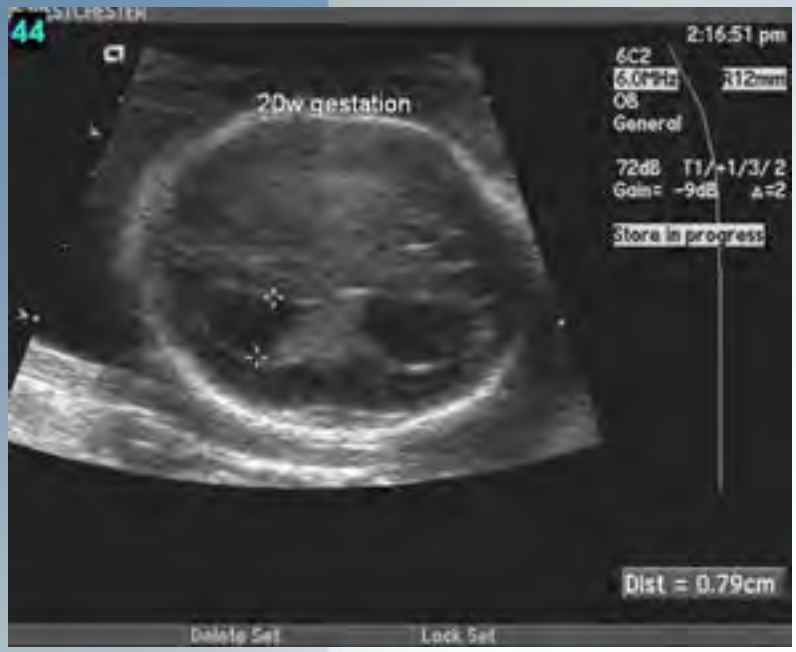
Intracranial Bleed Not Suspected on MFM Ultrasound

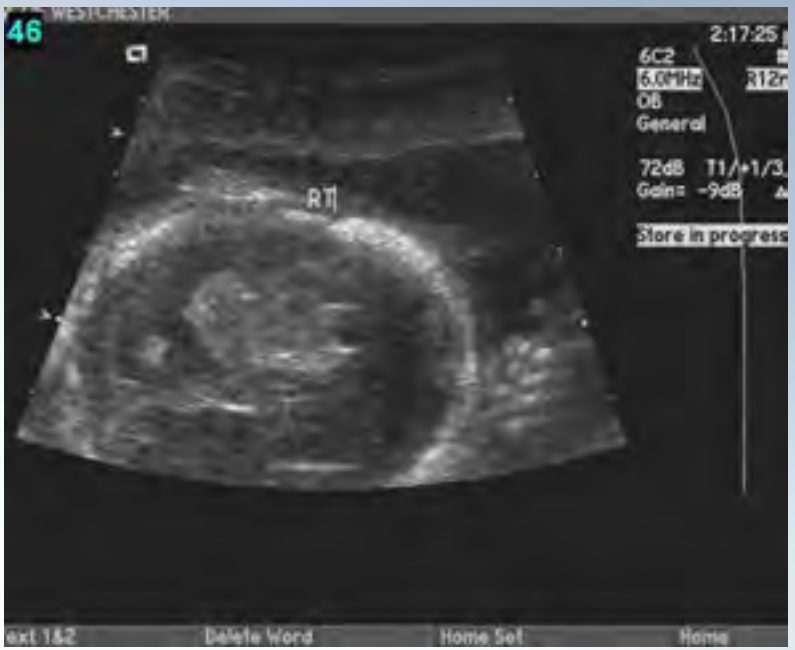
HISTORY: 20-week gestation with outside ultrasound studies describing prominent ventricles and single umbilical artery.

MR FINDINGS: Grade III germinal matrix bleed with diffusion pulse sequence demonstrating cerebral cytoxic edema.

OUTCOME: One-month follow up ultrasound demonstrated no calvarial growth consistent with microcephaly -profoundly poor prognosis.

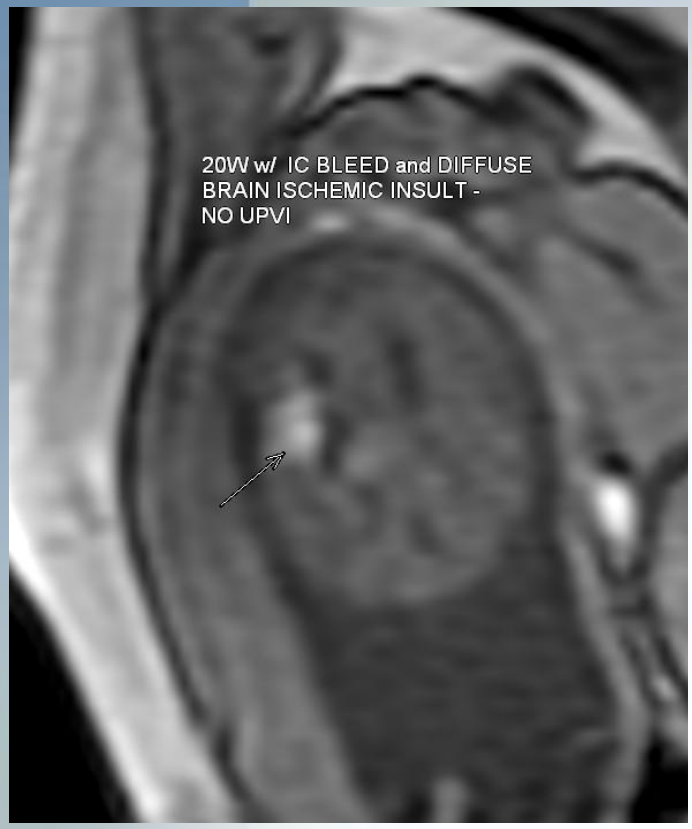
Intracranial Bleed Not Seen on MFM Ultrasound

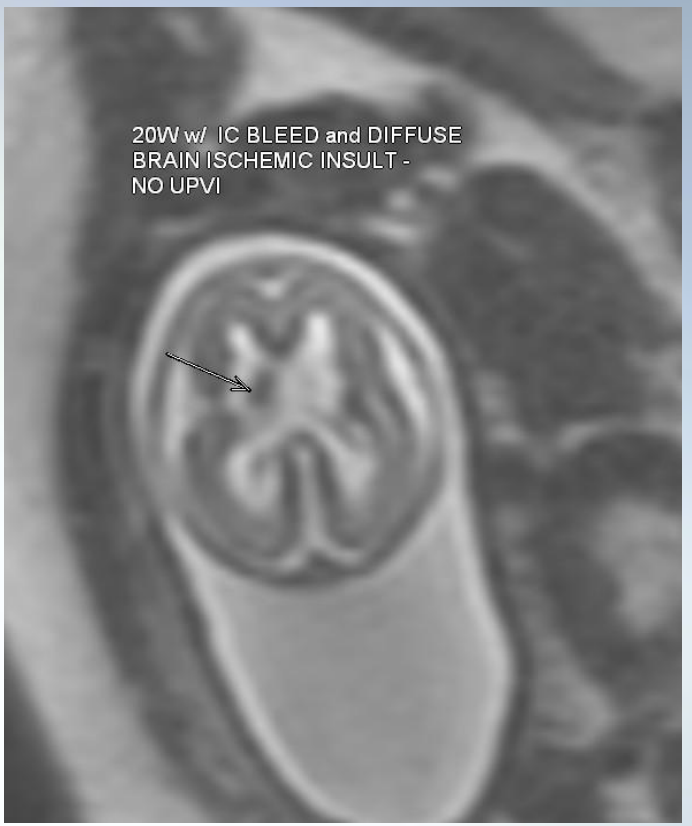




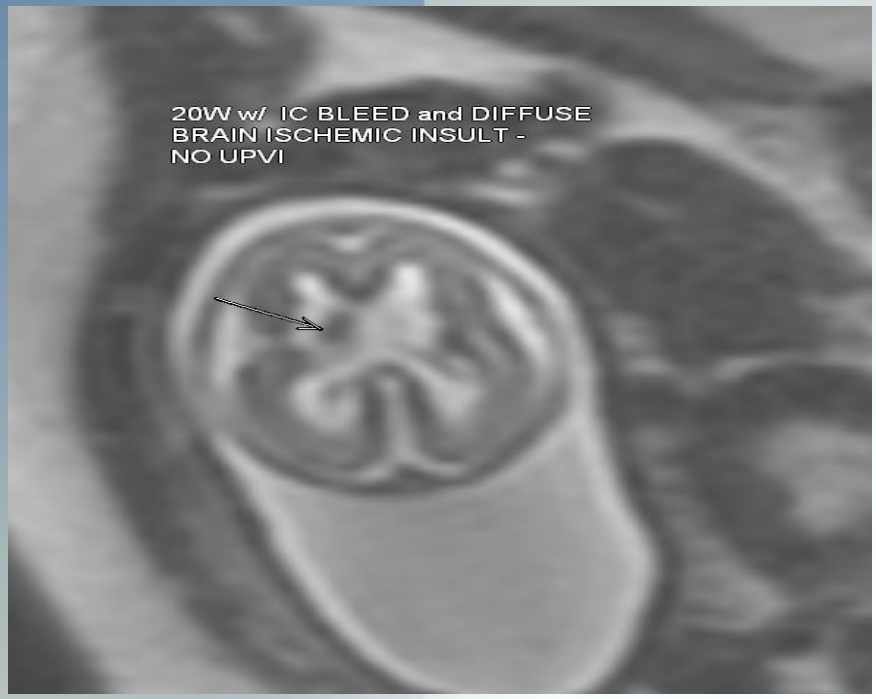
HUDSON VALLEY RADIOLOGY ASSOCIATES

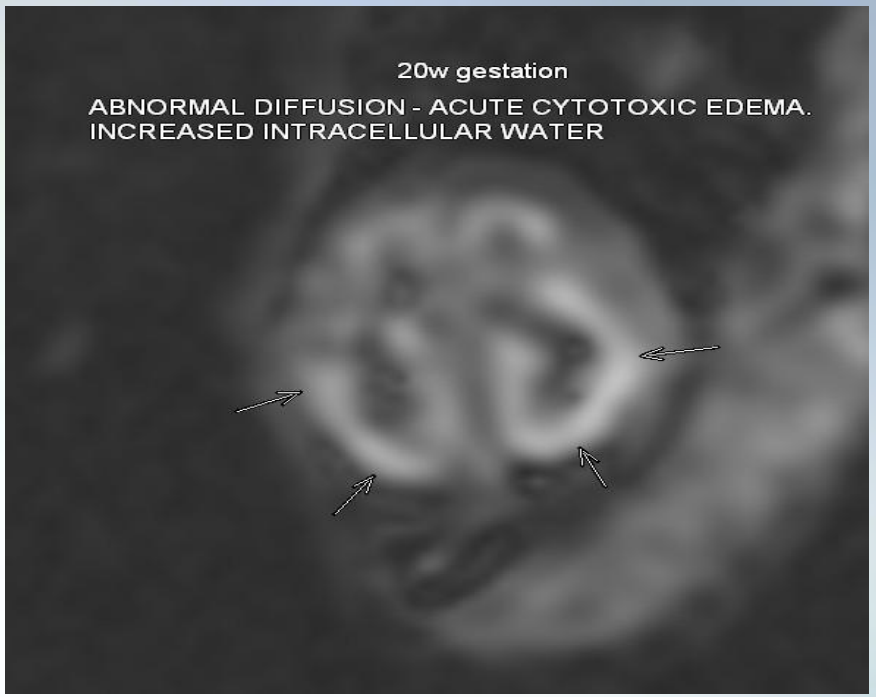
Intracranial Bleed Not Suspected on MFM Ultrasound





Cerebral cytotoxic edema accompanying intracranial bleed – poor prognosis





TEACHING POINTS:

- False-negative ultrasound for intracranial hemorrhage.
- Varying types of intracranial pathologies require different unique pulse sequences that are operator chosen and machine dependent.

HISTORY: 22-week gestation with outside ultrasound demonstrating mild ventriculomegaly.

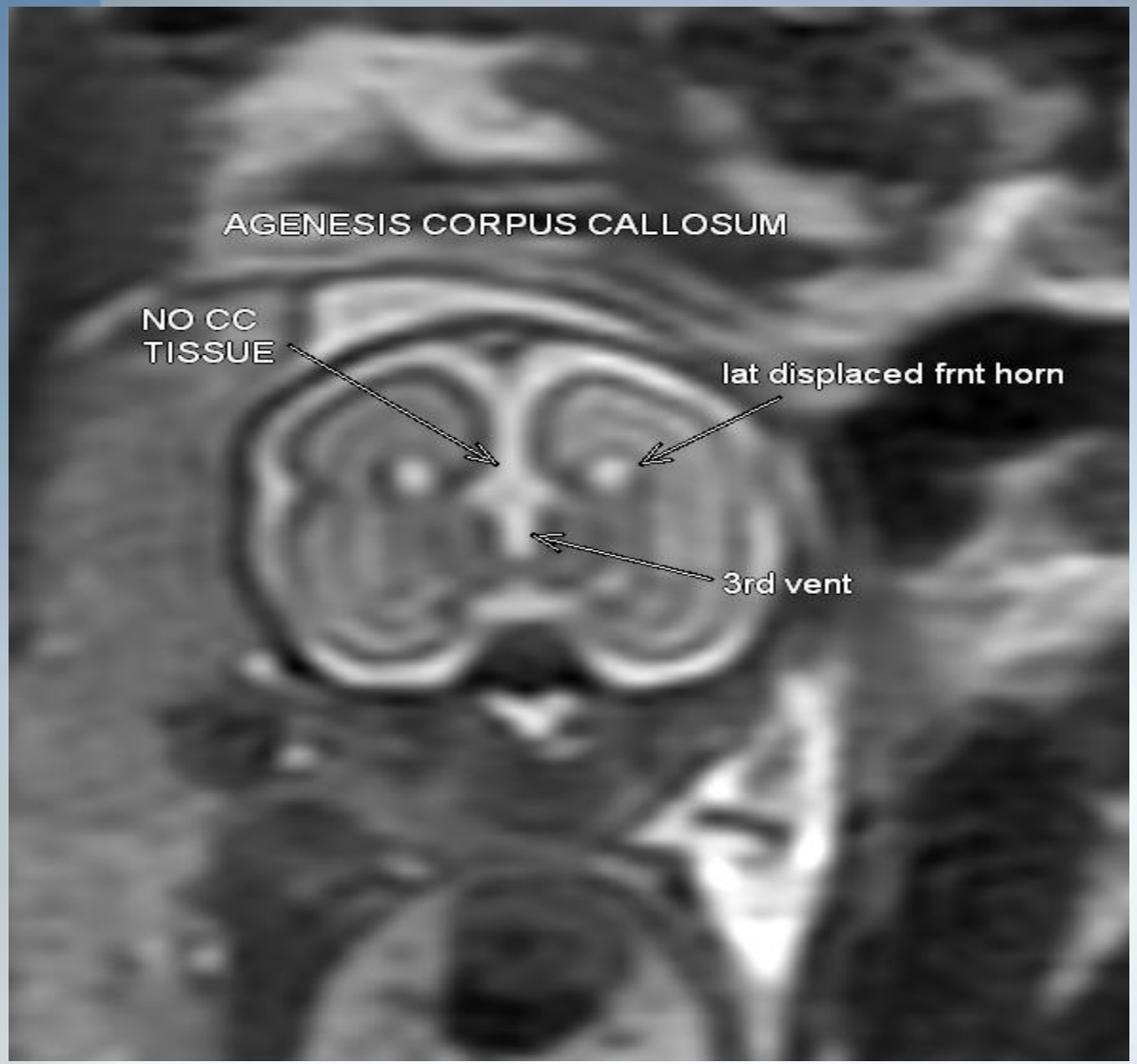
MR FINDINGS:

Complete agenesis of the corpus callosum and a schizencephalic defect – a malformation of cortical development (MCD).

TEACHING POINT:

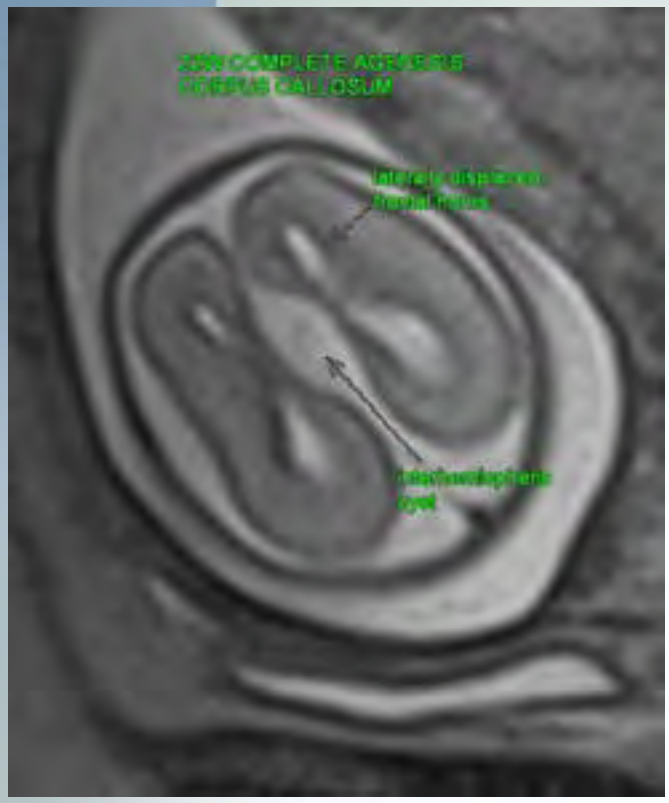
MR uniquely images MCD, conferring additional poor prognosis.

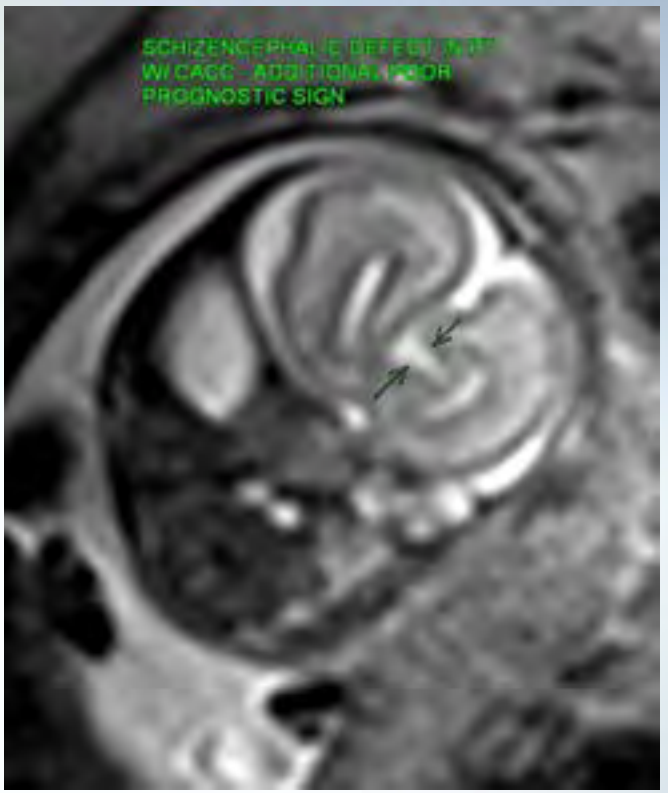
No Normal CSP - 2nd Most Common Indication



MR demonstrates pathology unable to be seen on ultrasound.

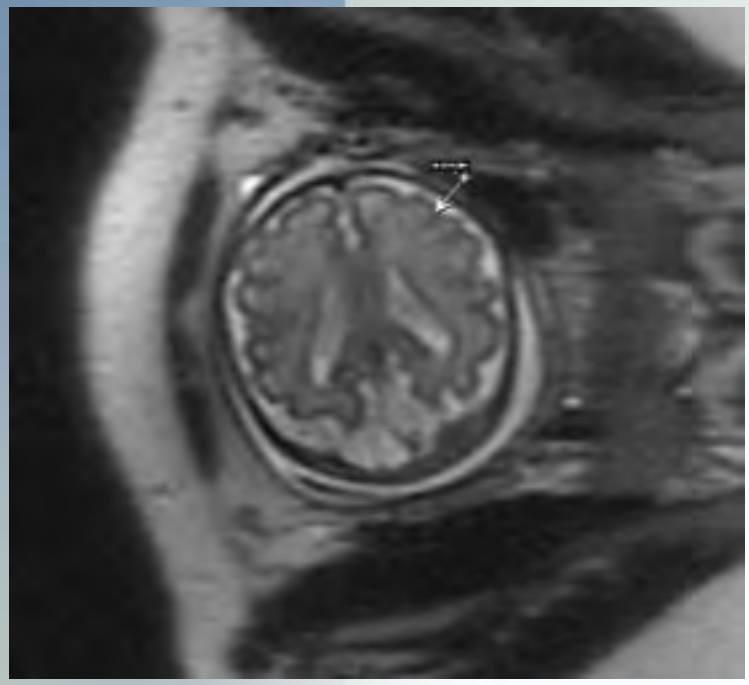
Agenesis of corpus callosum with schizencephalic defect – additional poor prognostic sign



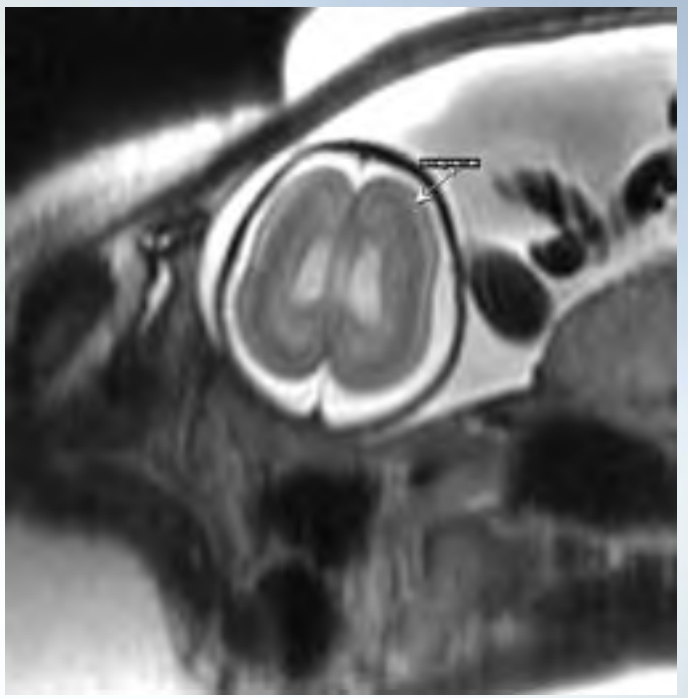


MFM ultrasound demonstrated 3rd trimester mild ventriculomegaly

MR dg = Lissencephaly
32 Weeks



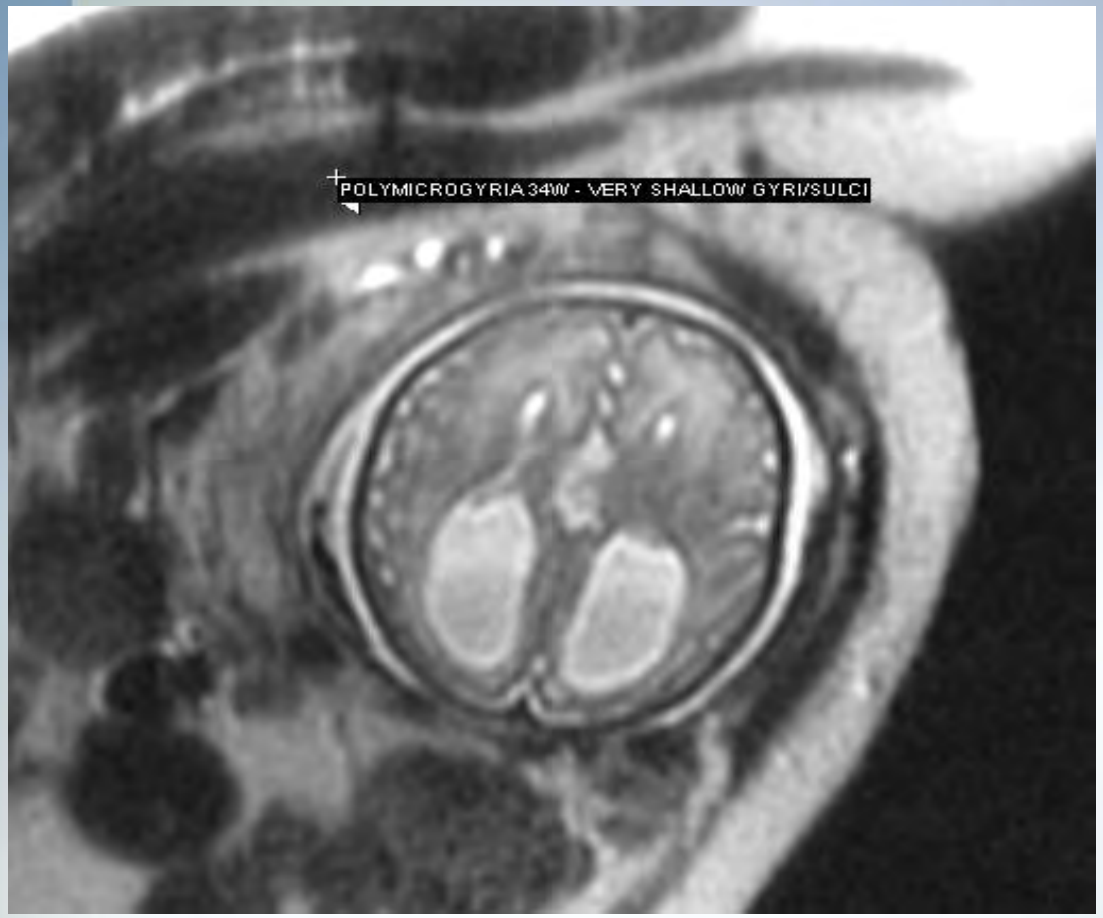
Normal gyral development



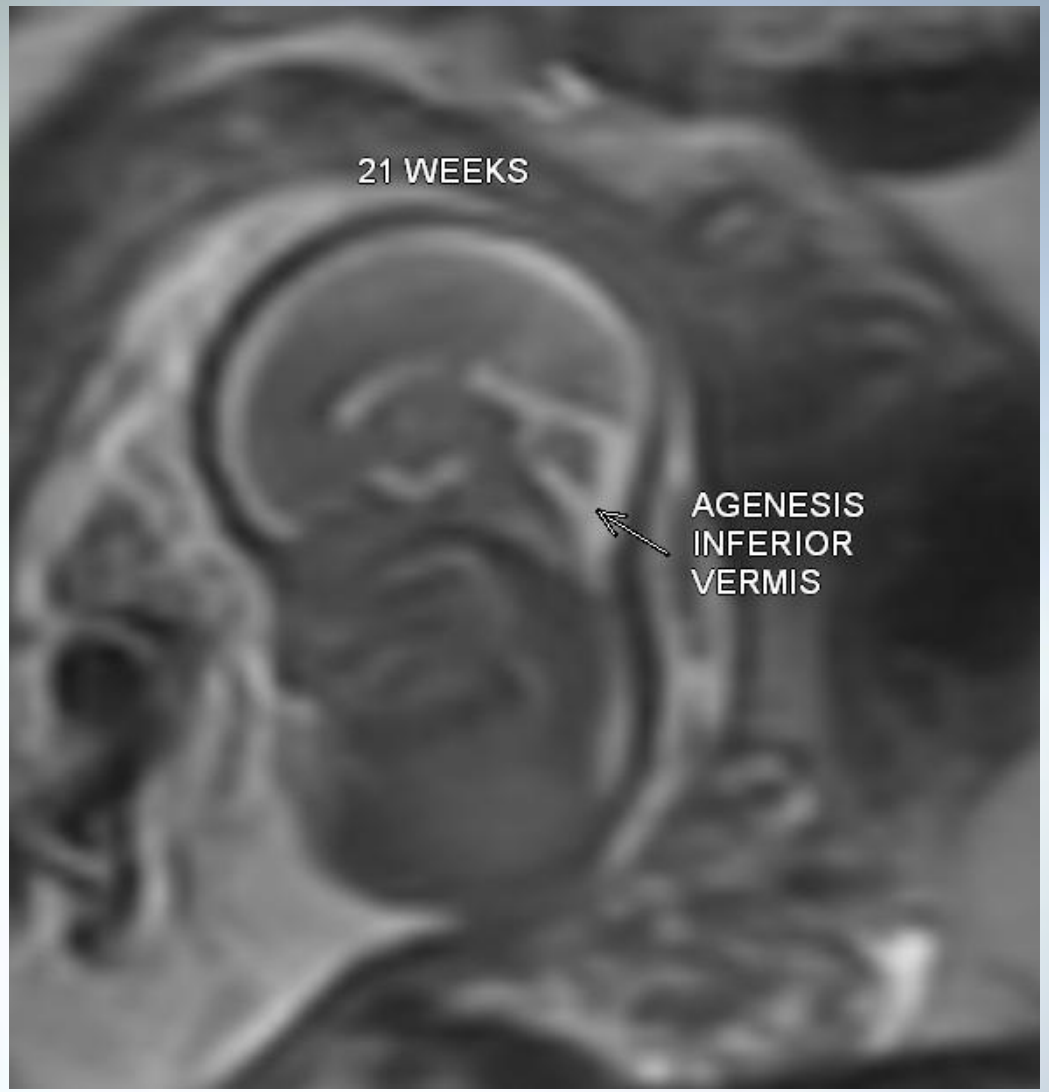
Complete agyria

MR demonstrates pathology rarely seen on ultrasound

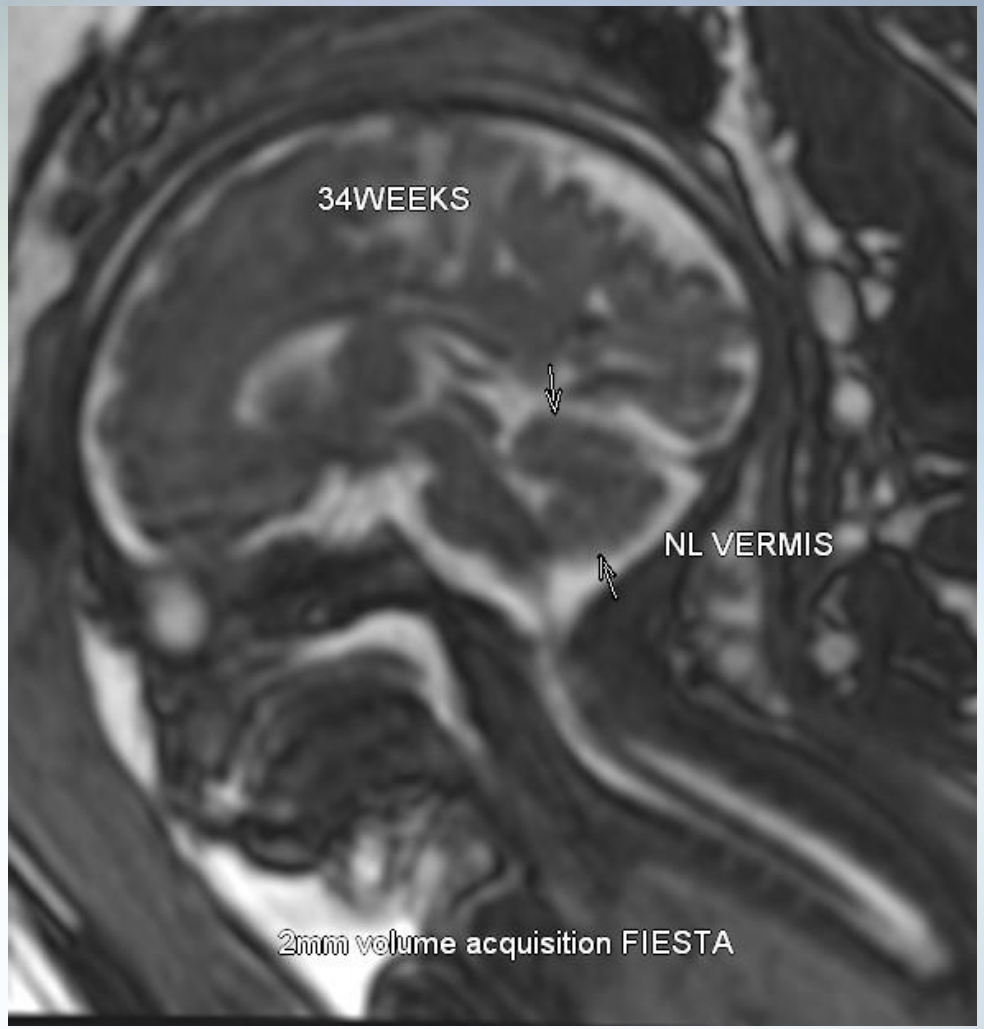
On US - 3rd TM onset of mild ventriculomegaly
MR dg = polymicrogyria



US dg – enlarged Cisterna Magna, R/O DWM
MR dg = NL Variant Agenesis Inferior Vermis



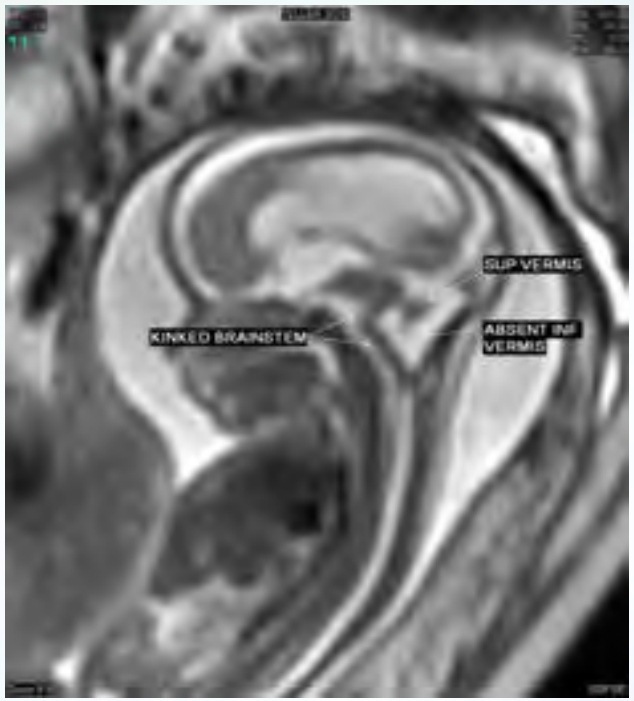
NL Variant Agenesis Inferior Vermis



MR demonstrates pathology – kinked brainstem- unable to be seen on Ultrasound

US dg - 20 w Hydrocephalus

kinked brainstem reflects late first trimester embryologic defect. Profoundly poor prognosis even if successfully shunted.



History:

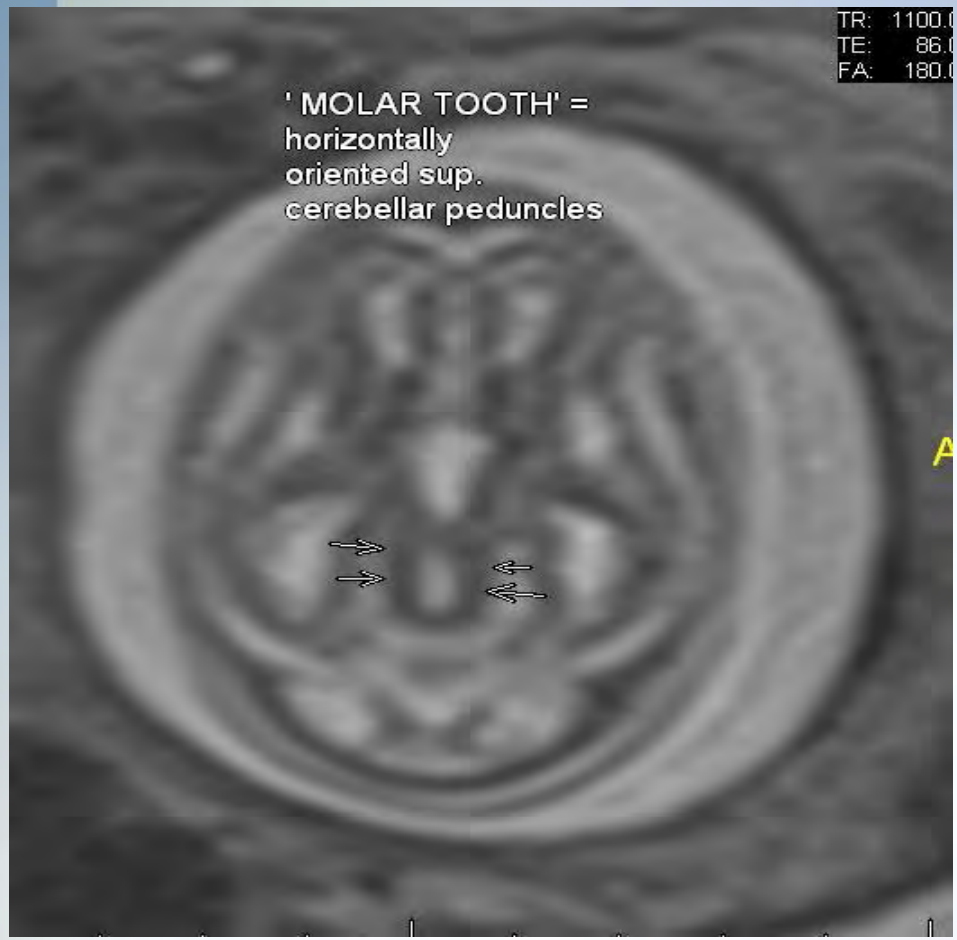
21 w GA whose MFM outside of ultrasound study demonstrated BPD and HC 12-14 days smaller than expected (2nd standard deviation) with non normalization of CSP, SUA, micrognathia and abnormal posturing of the lower extremities.

MR Findings:

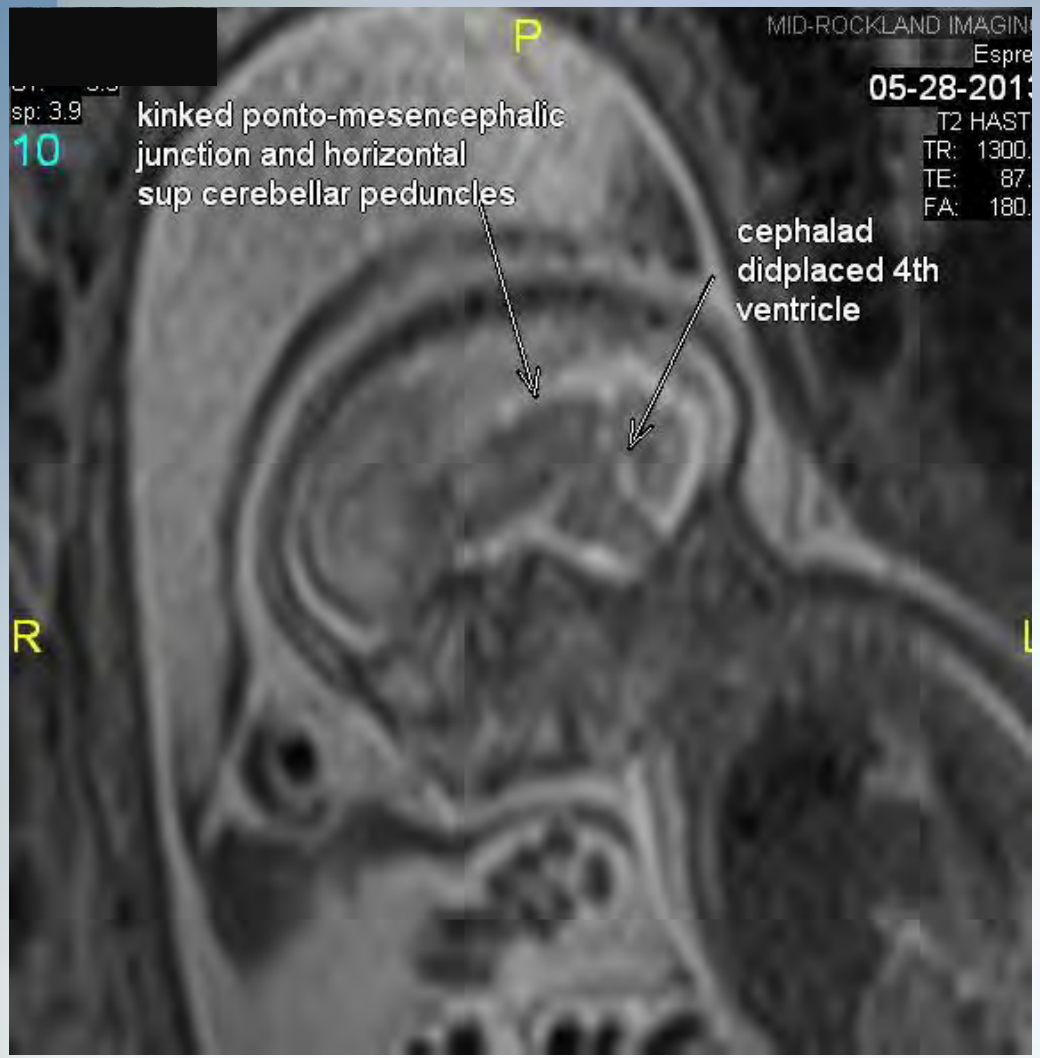
- Agenesis of the corpus callosum.
- Horizontal, thickened orientation to the superior cerebellar peduncles creating the molar tooth sign
- Cephalad displacement of the fourth ventricle with moderate brainstem kinking.
- Direct occipitofrontal brain diameter 3.5 standard deviations smaller than expected consistent with microcephaly.

MR demonstrates pathology unable to be seen on Ultrasound

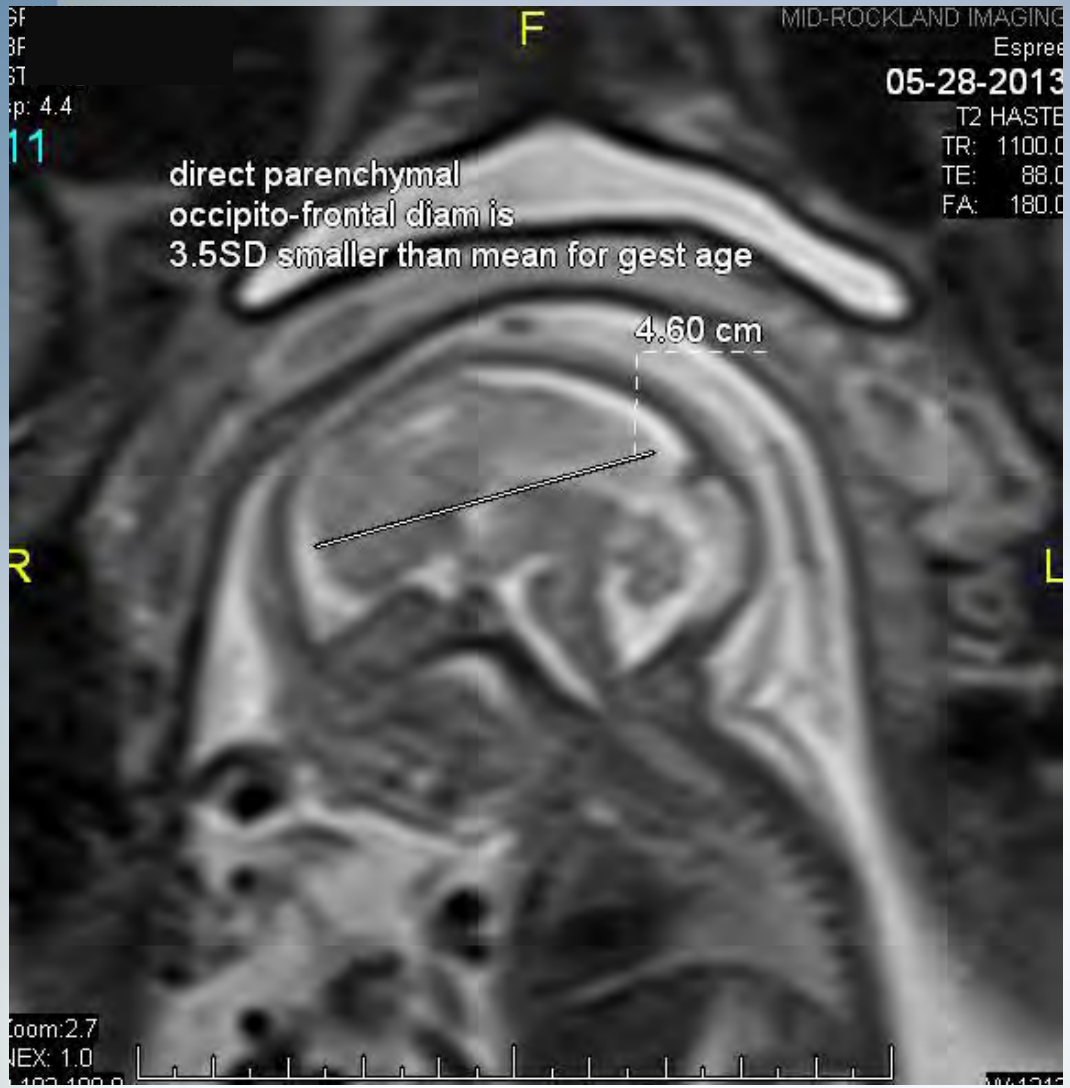
"Molar tooth"sign: ponto-mesencephalic (hindbrain-midbrain) dysmorphology



Ponto-mesencephalic dysmorphology
Not seen on ultrasound



MR images direct brain microcephaly before boney ultrasound measurements of BPD + HC



Clinical follow up: Patient terminated pregnancy. Whole -exome sequencing negative.

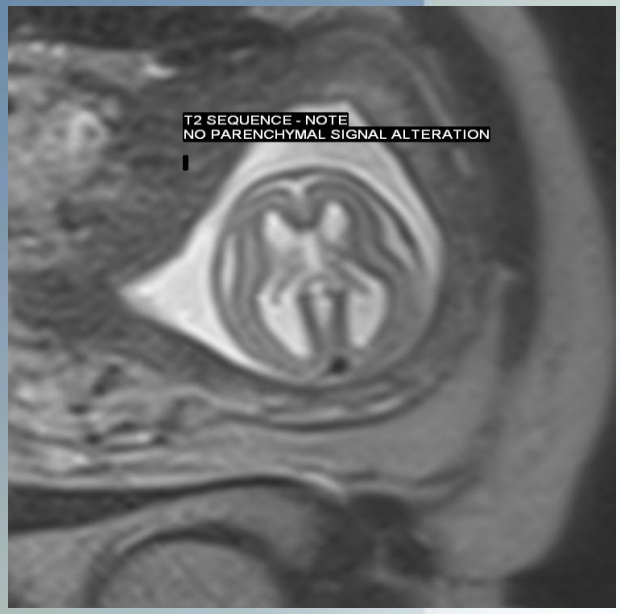
Teaching Point: MR's ability to diagnose multiple intracranial poor prognostic signs of types not able to be imaged on ultrasound.

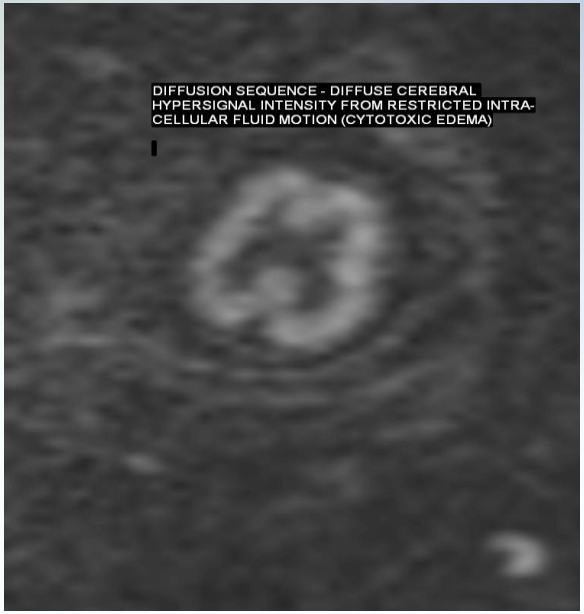
Consider whole- exome sequencing in cases with multiple malformations when metaphase cytogenetics and microarray are negative.

* Clinical Whole –exome Sequencing for the Diagnosis of Mendelian Disorders. NEJM Oct 2, 2013. Yang -

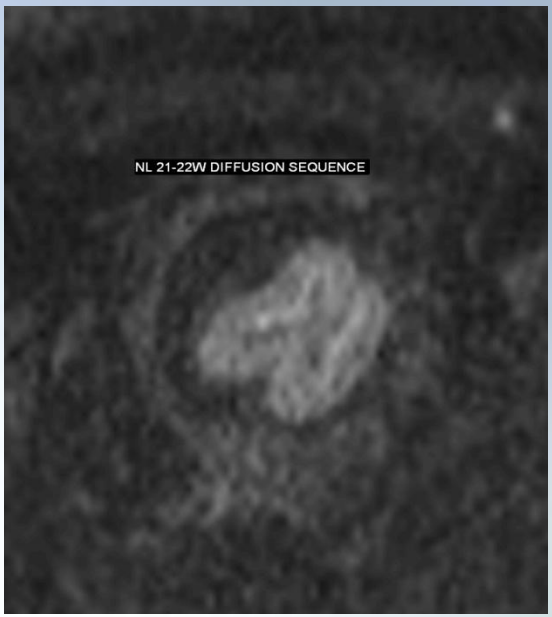
Abstract Conclusion: Whole –exome sequencing identified the underlying genetic defect in 25% of consecutive patients referred for evaluation of a possible genetic condition.

CMV FETOPATHY





DWI sequence at the level lateral ventricle demonstrating global cerebral hypersignal intensity not present on the axial T2 image for comparison. (Image 11)



Age matched normal DWI for comparison.

HISTORY: 23-week gestation with outside ultrasound identifying cerebellar hypoplasia. Normal karyotype. Isolated cerebellar hypoplasia is worrisome but not specific for bad outcome.

FINDINGS: MR identifies not only cerebellar but also significant pontine hypoplasia. Direct cerebral parenchymal biometry is at the fifth percentile.

MR IMAGING DIAGNOSIS:

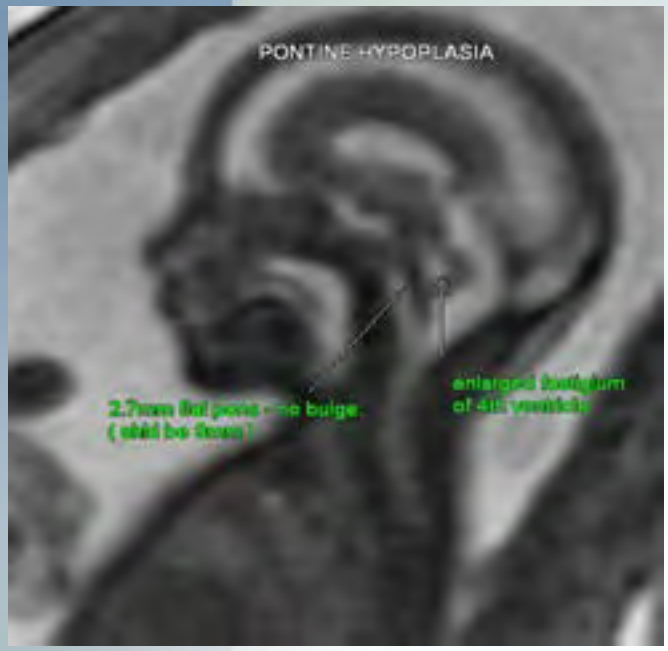
Hypoplasia not only of the posterior fossa anatomy but also of the cerebral hemispheres -greater specificity for poor prognosis.

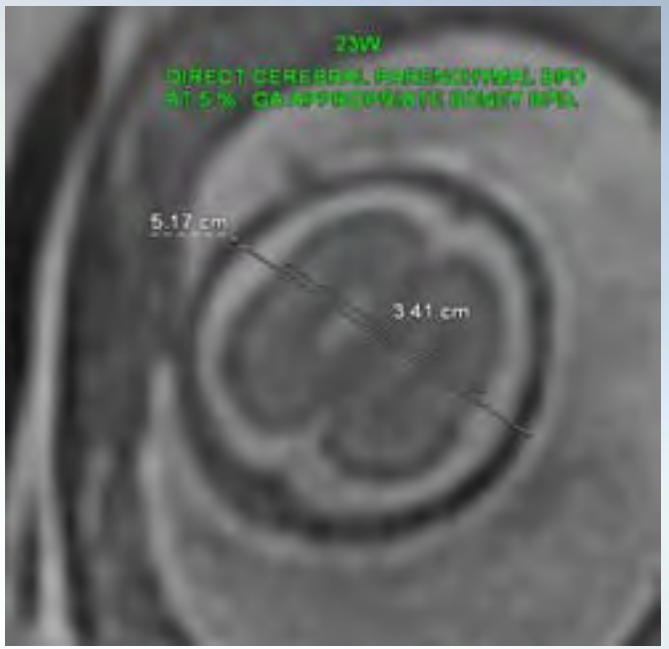
TEACHING POINTS:

MR can provide a direct brain parenchymal measurement in the assessment of impending microcephaly prior to diminution in measurements of the bony calvarium.

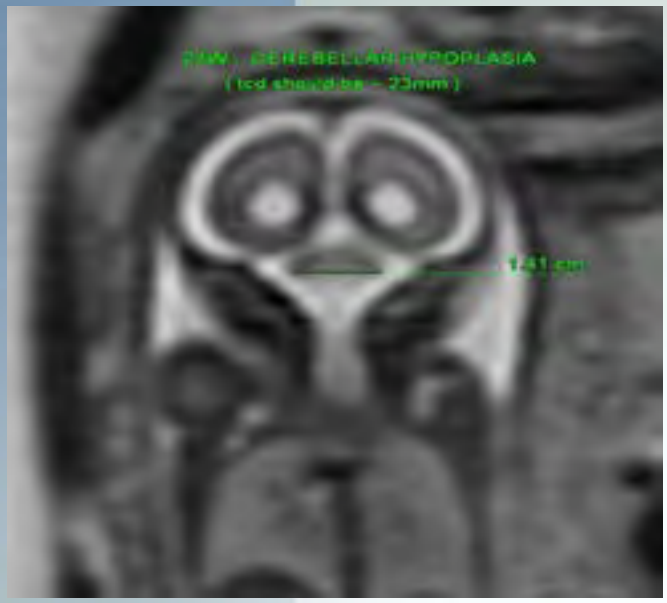
MR uniquely visualizes regions of brain anatomy unable to be seen on ultrasound and necessary to optimize patient counseling.

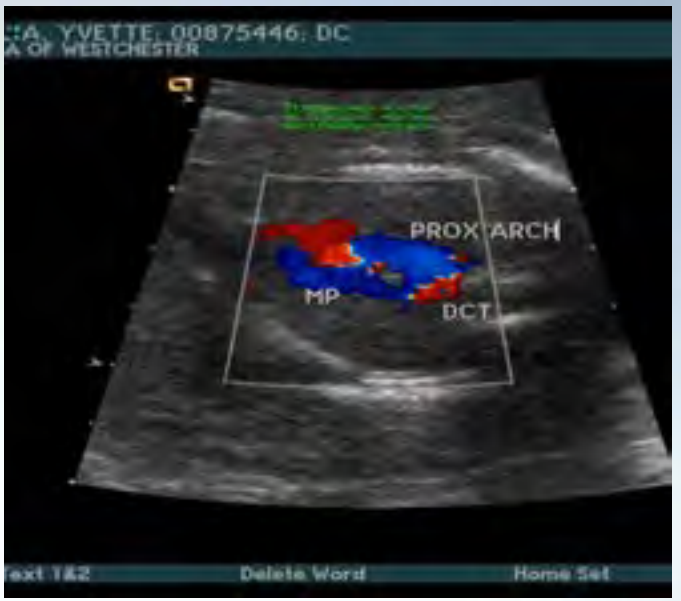
Hypoplasia of cerebellum, pons and cerebral hemispheres.



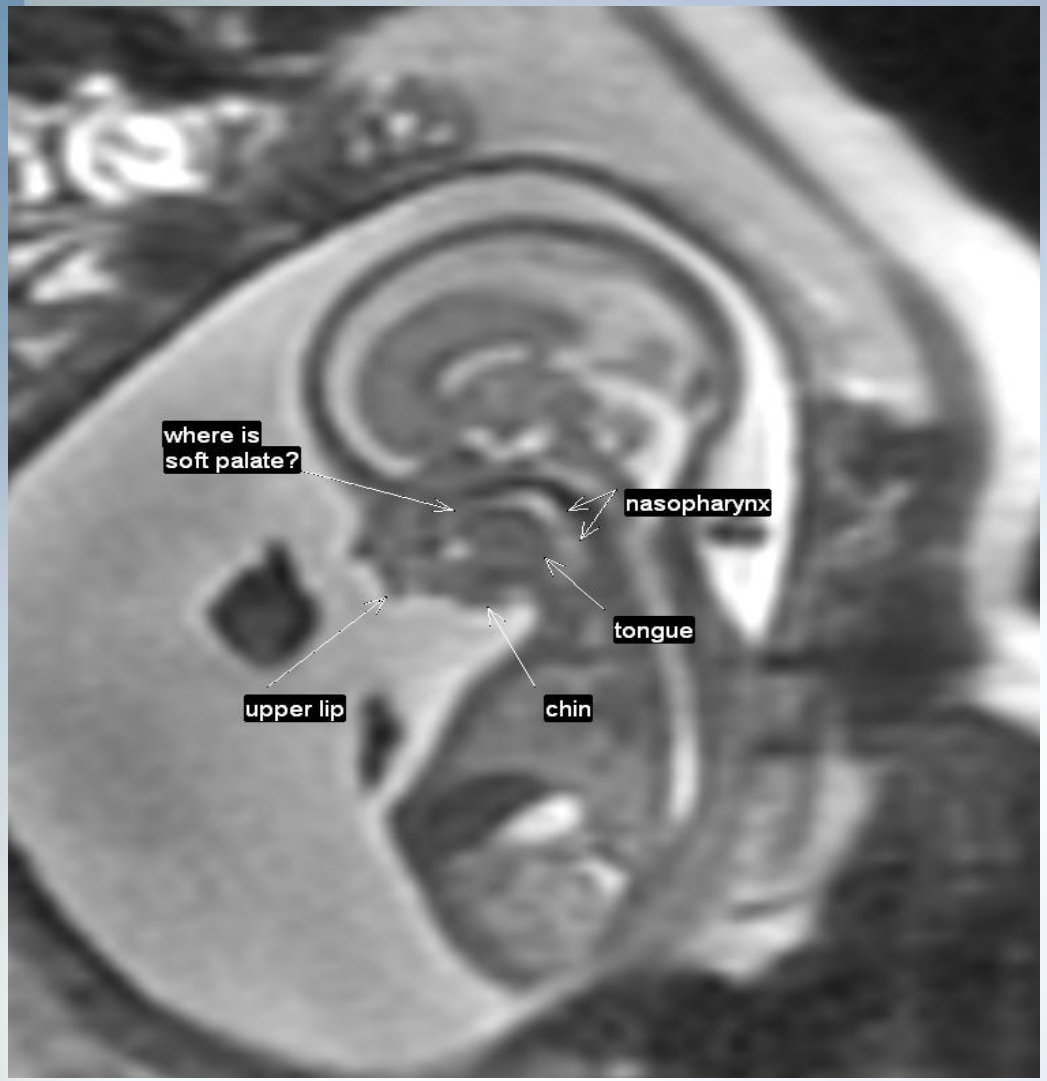


Virtually all fetuses requiring MR also require fetal cardiac ultrasound to complete and optimize enumeration of dysmorphology.





20 W Recurrent Pierre Robin syndrome, micrognathia. large soft palate defect.



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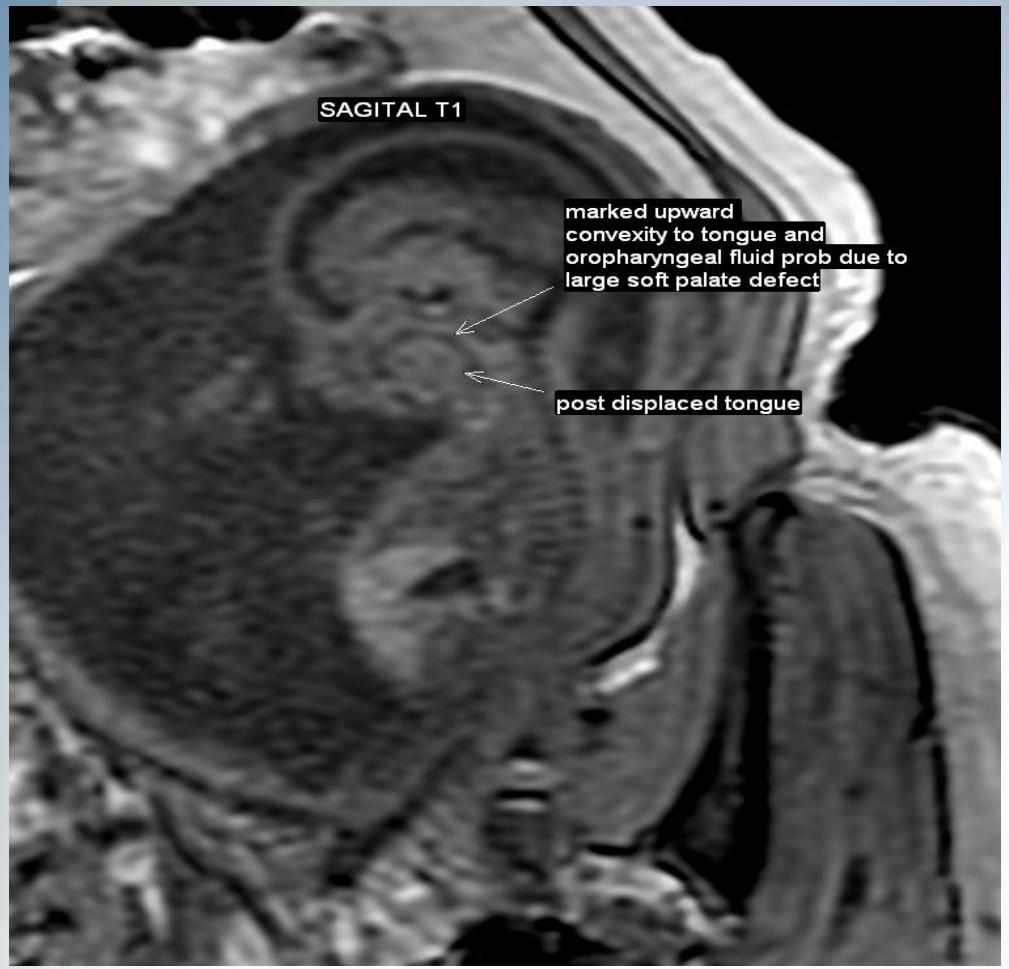
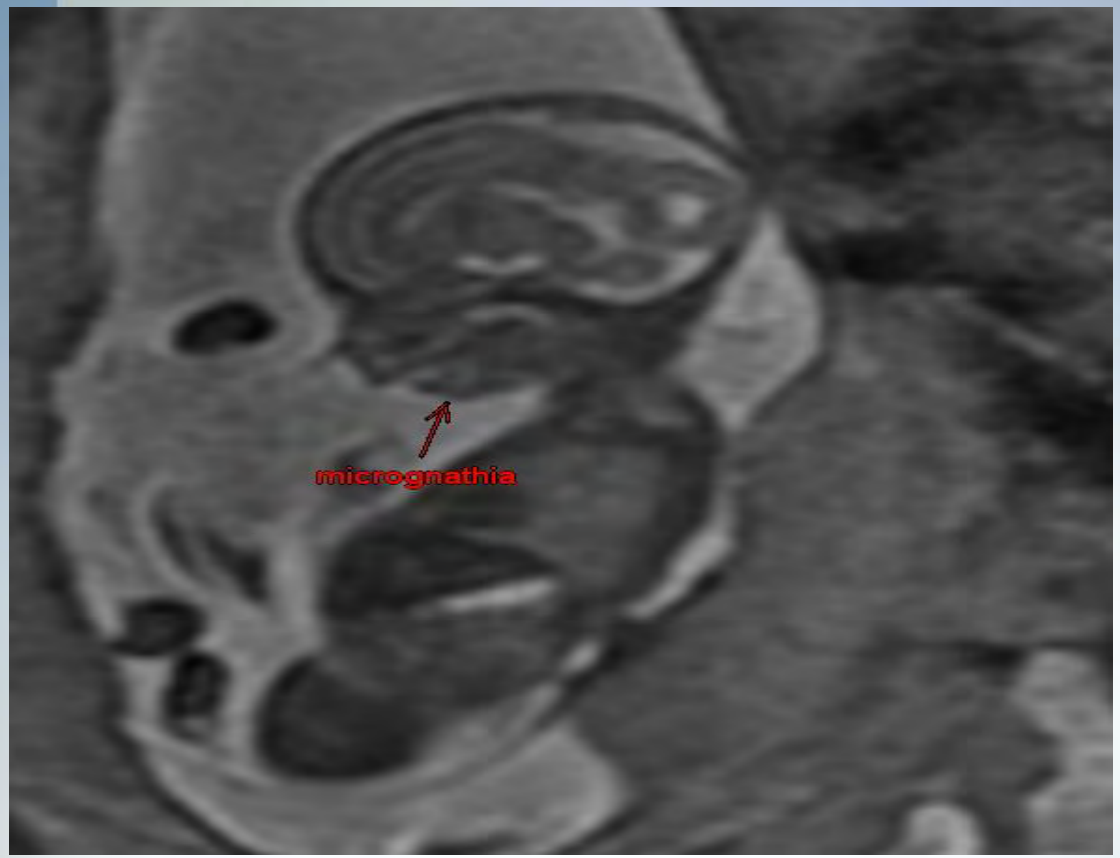


GOLDENHAR SYNDROME
MFM Ultrasound Identified an Arachnoid Cyst. MR Identifies Additional Pathologies as Clues to Syndromic Condition



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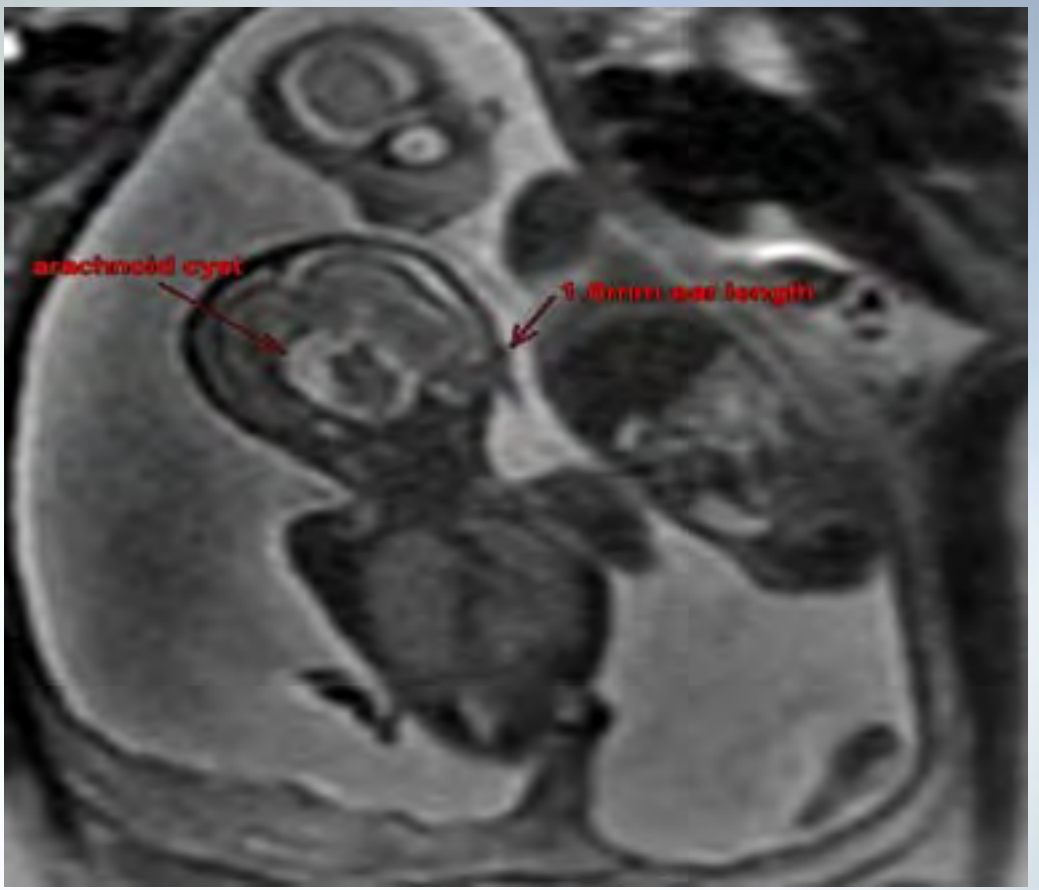
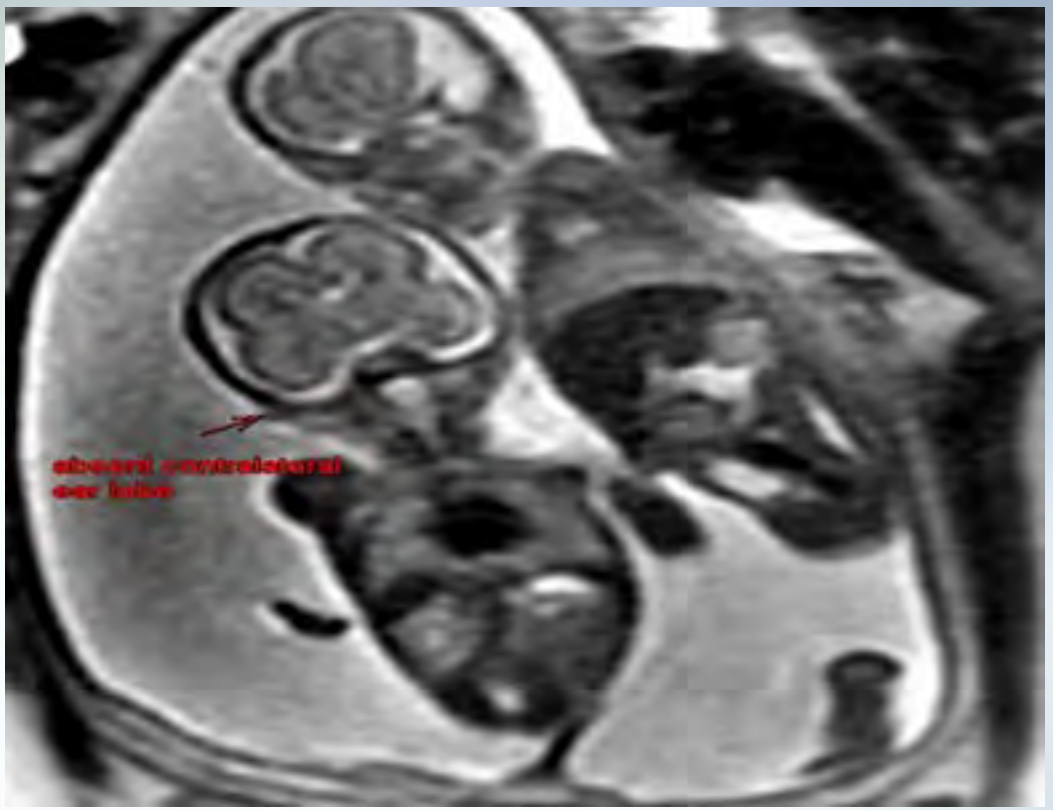


GOLDENHAR SYNDROME
Absent ear

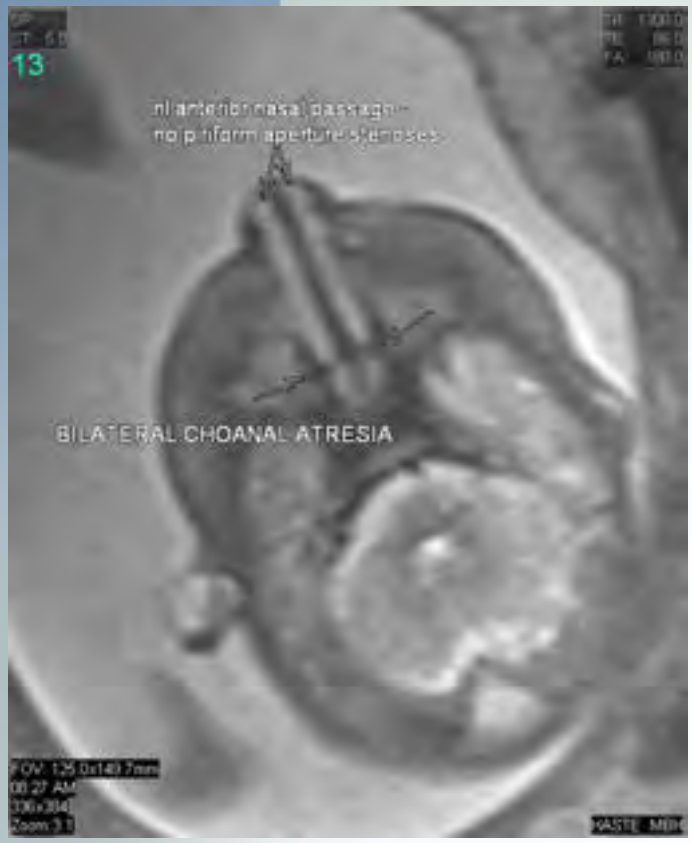


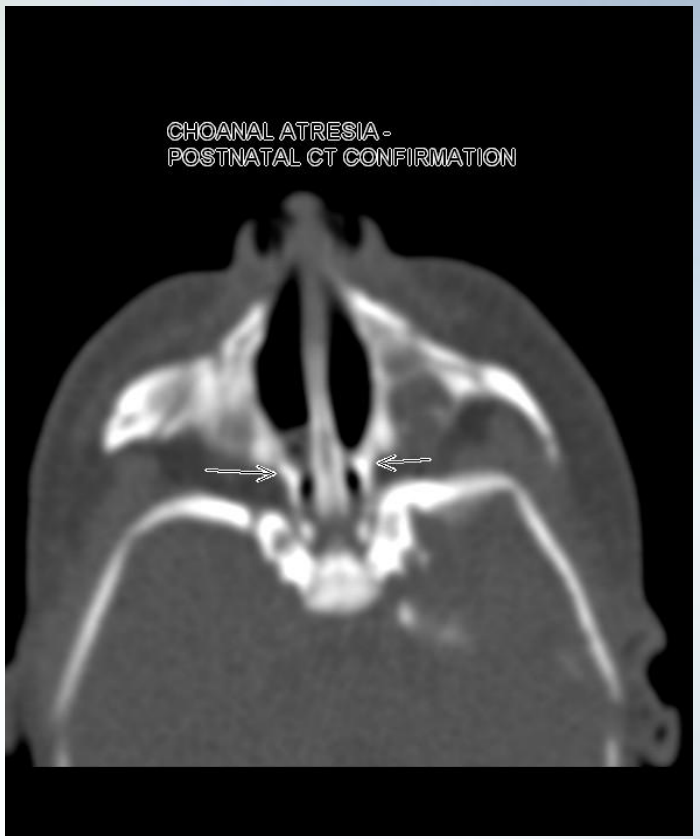
MR demonstrates pathology unable to be seen on ultrasound

29w ga referred for mild ventriculomegaly

nasal passage obstruction requiring neonatal surgery

Choanal atresia. CHARGE syndrome





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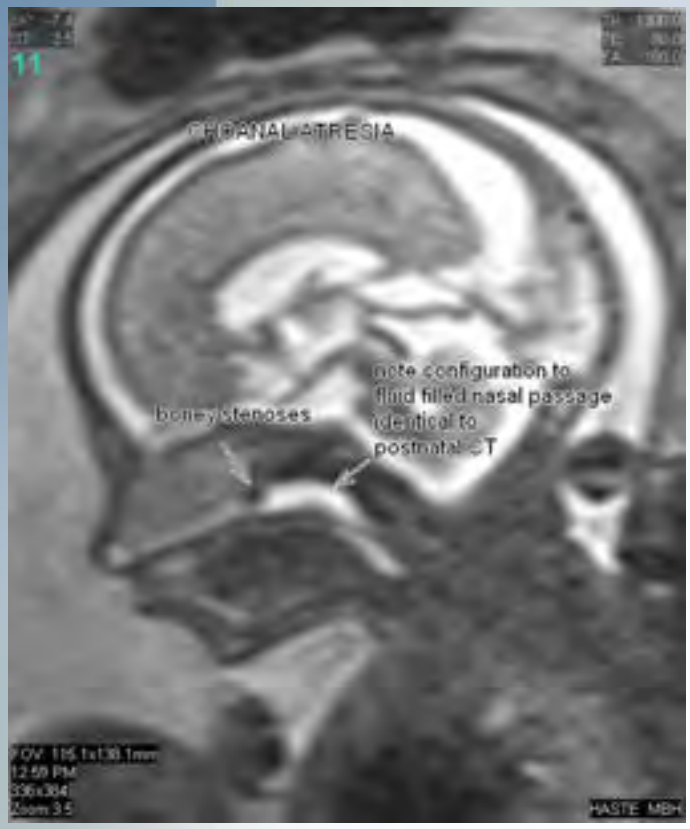
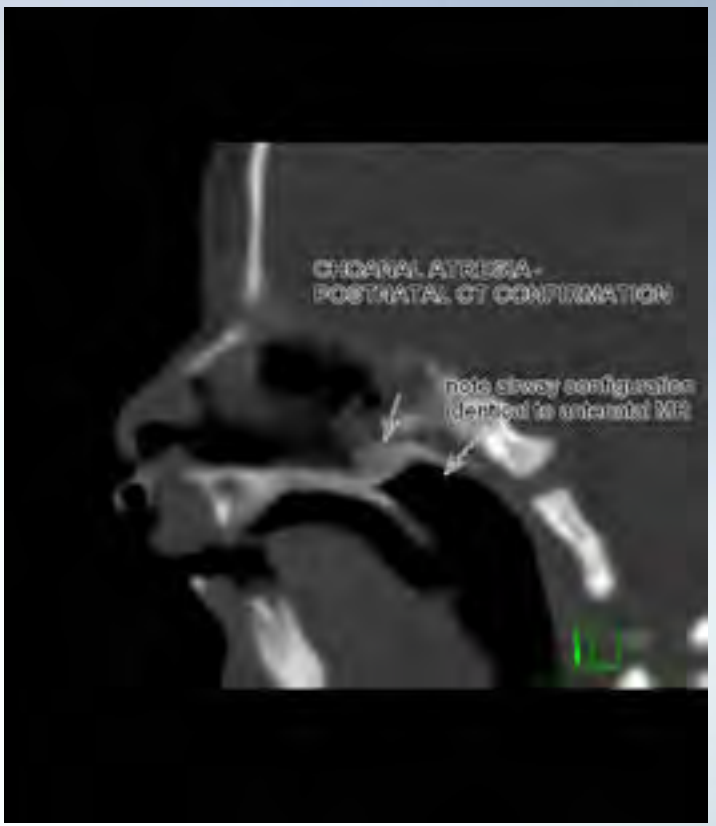




30 W Twin B referred for mild ventriculomegaly and non normalization of corpus callosum.

Final Diagnosis – Nasal passage obstruction due to *pyriform aperture stenosis and basal frontal encephalocele with dermoid* requiring intubation and tracheostomy.

HISTORY: IVF pregnancy with normal karyotype.

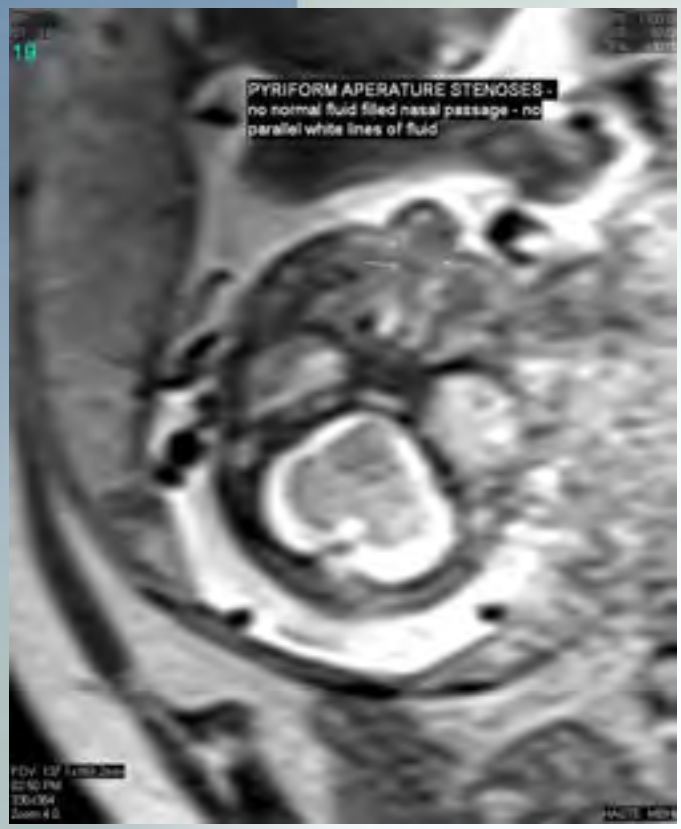
Fetal MR confirmed complete agenesis of the corpus callosum and demonstrated no other identifiable intracranial abnormalities.

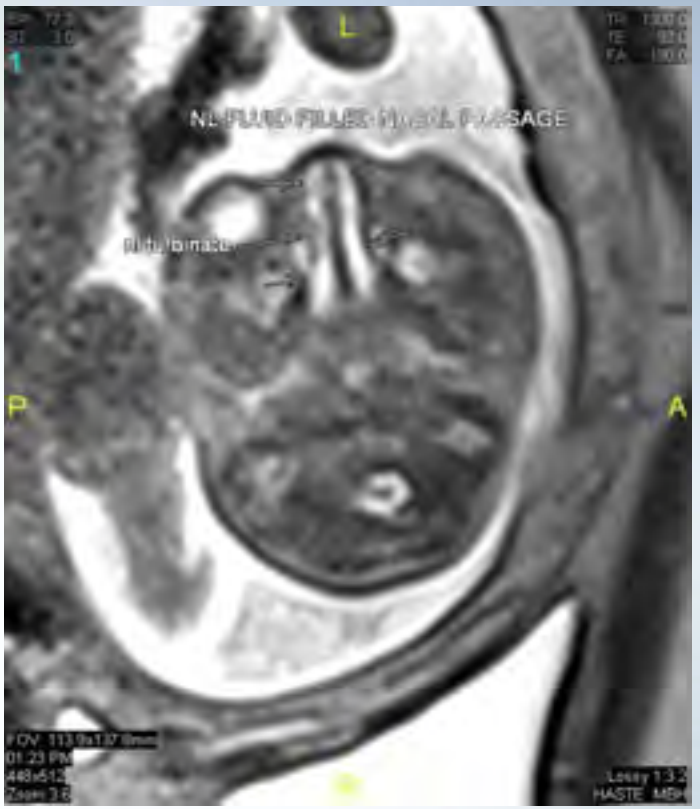
The twin with agenesis of the corpus callosum (ACC) had difficulty breathing and feeding, Requiring intubation and then tracheostomy. Postnatal MR and CT for the evaluation of upper airway issues identified two sources of nasal passage obstruction – pyriform aperture stenosis (PAS) and a small 6 x 4 mm anterior nasal encephalocele with an accompanying fatty dermoid component. Clinical exam and imaging demonstrated a large single midline maxillary incisor – a frequent accompaniment to PAS.

Pyriform aperture stenosis (PAS) creates an obstruction to the anterior aspect of the nasal passage (in comparison to choanal atresia which is obstruction to the posterior aspect of the nasal passage).

MR demonstrates pathology unable to be seen on ultrasound

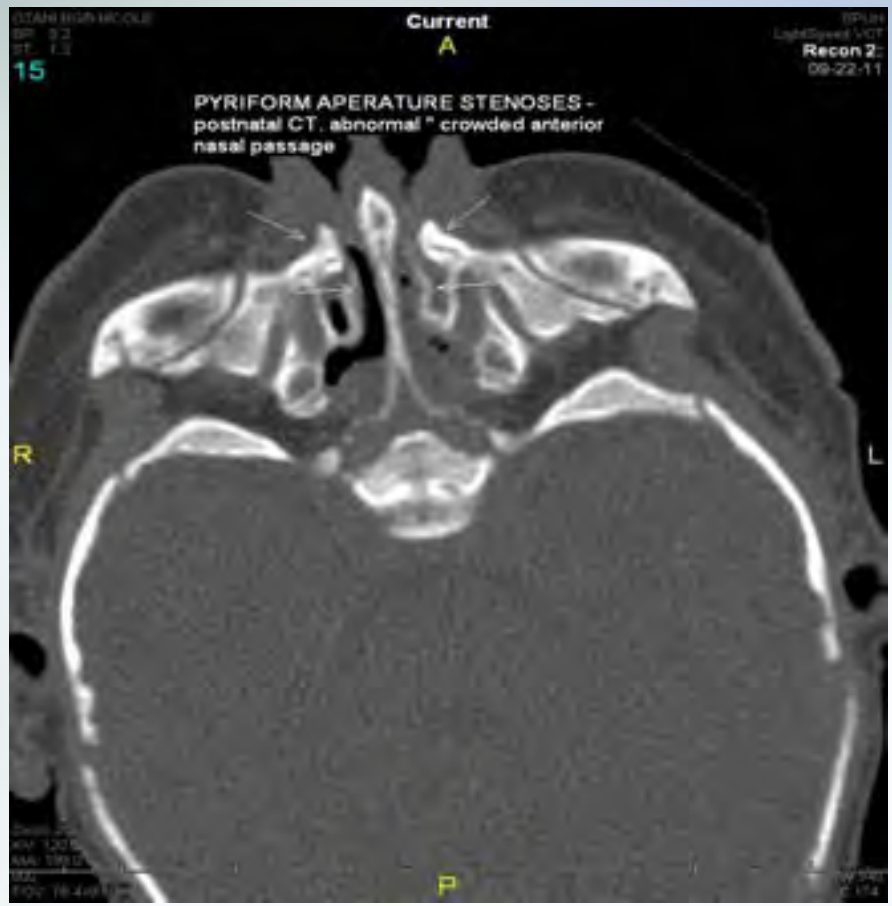
Pyriform aperture stenoses.
nasal passage obstruction requiring intubation and tracheostomy.





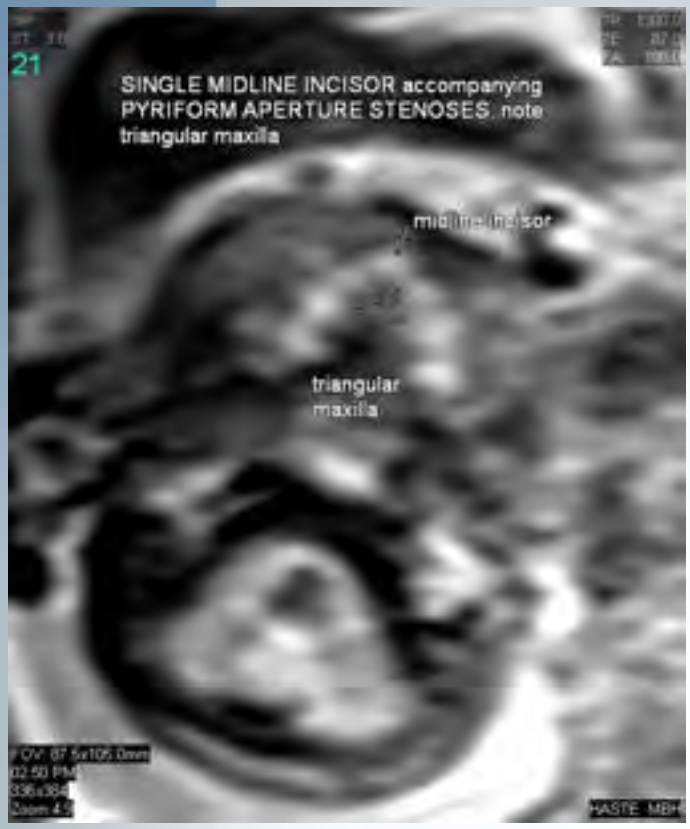
MR demonstrates pathology unable to be seen on ultrasound

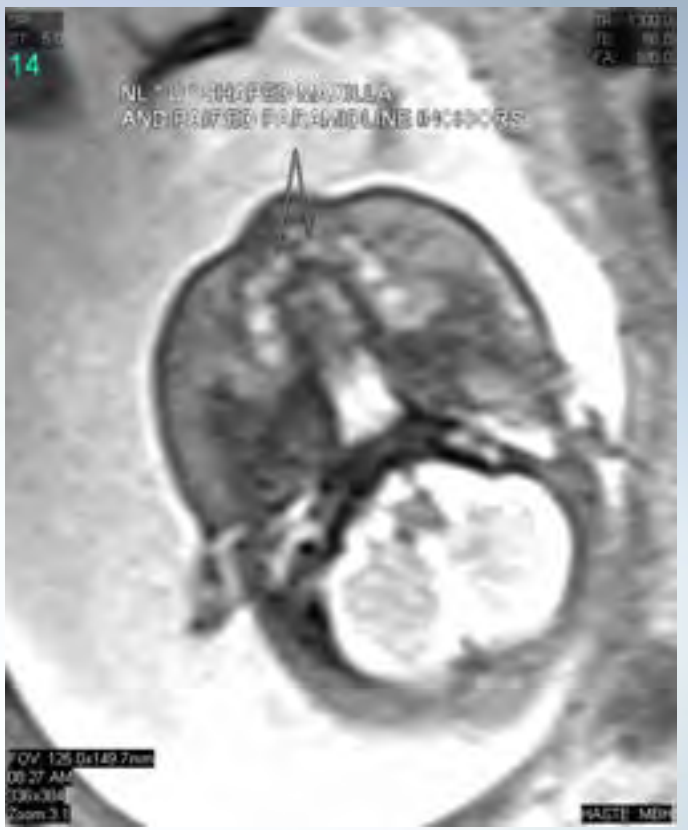
Pyriform aperture stenoses.
nasal passage obstruction requiring intubation and tracheostomy.



MR demonstrates pathology unable to be seen on ultrasound

Pyriform aperture stenoses with midline maxillary incisor nasal passage obstruction requiring intubation and tracheostomy.

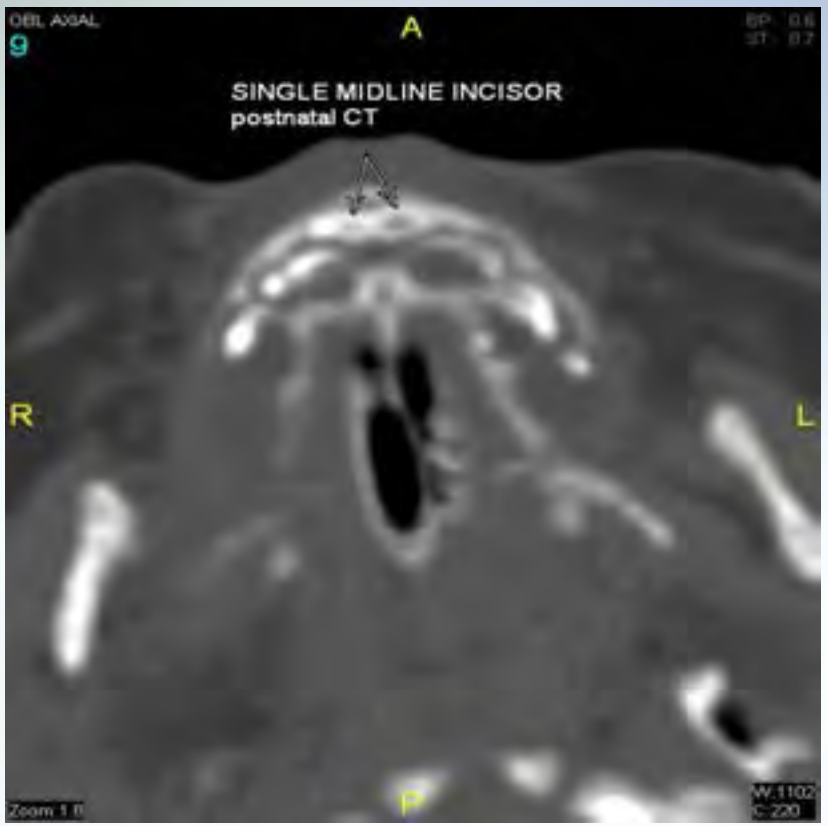




MR demonstrates pathology unable to be seen on ultrasound

Pyriform aperture stenoses with midline maxillary incisor

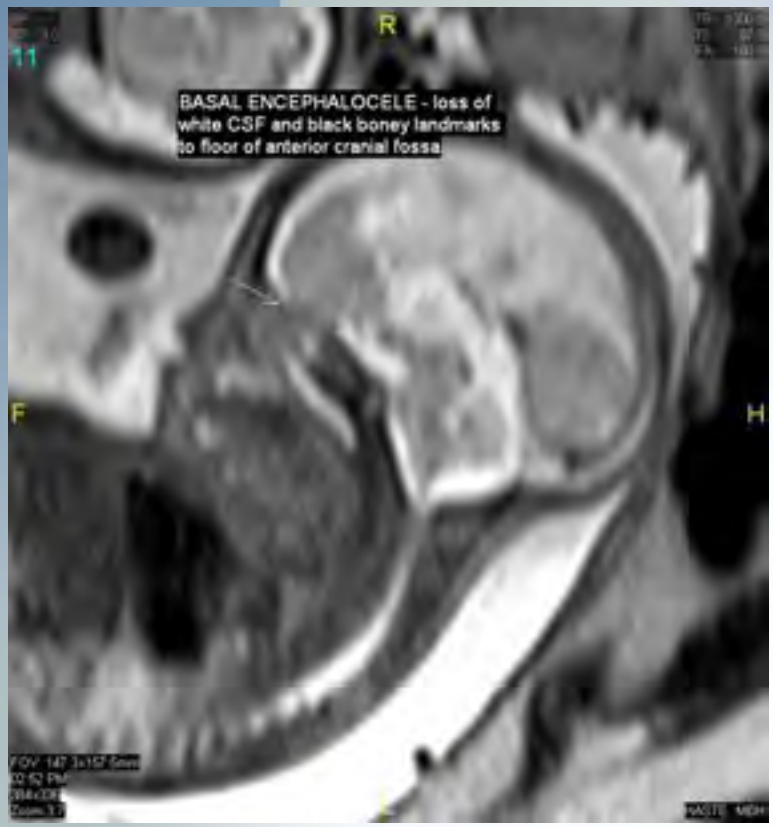
nasal passage obstruction requiring intubation and tracheostomy.

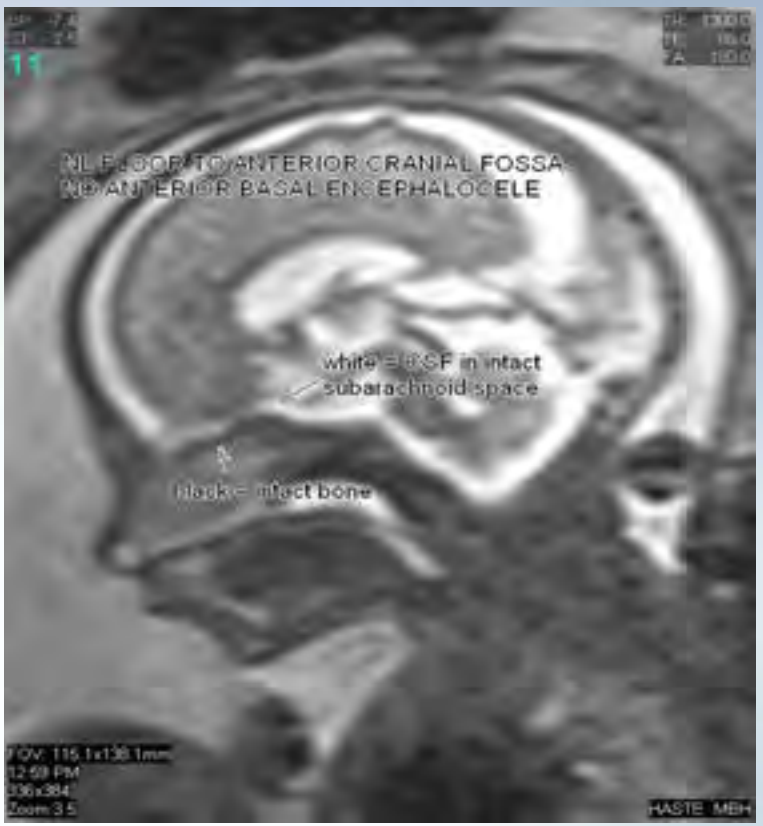


MR demonstrates pathology unable to be seen on ultrasound

Basal encephalocele with dermoid

nasal passage obstruction requiring intubation and tracheostomy.



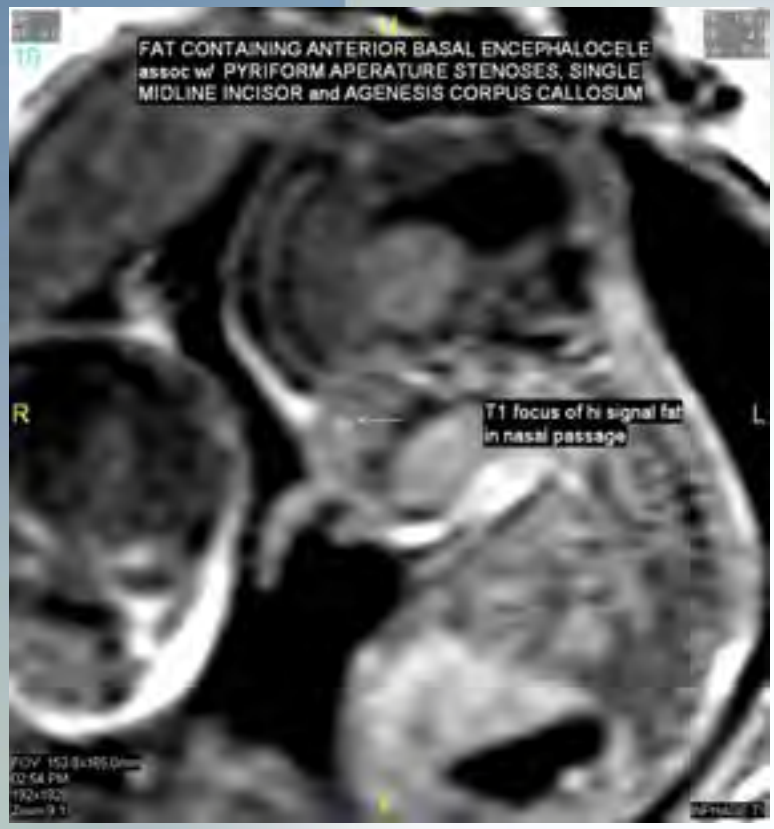


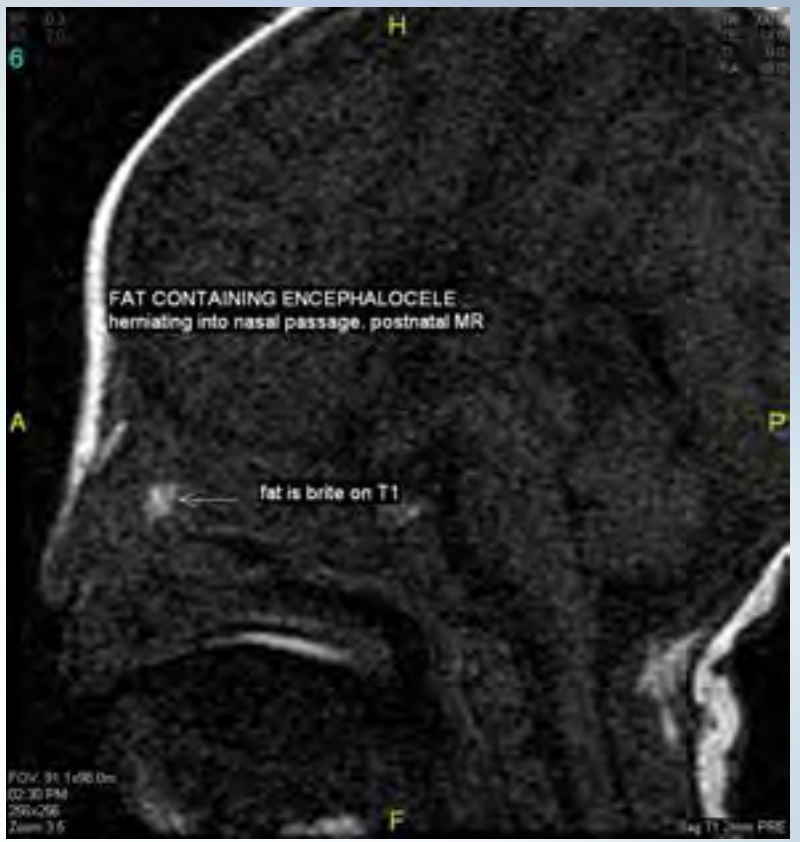
HUDSON VALLEY RADIOLOGY ASSOCIATES

MR demonstrates pathology unable to be seen on ultrasound

Basal encephalocele with dermoid

nasal passage obstruction requiring intubation and tracheostomy.





HUDSON VALLEY RADIOLOGY ASSOCIATES

Bilateral atresia of external auditory canal with middle ear dysplasia – trisomy 18

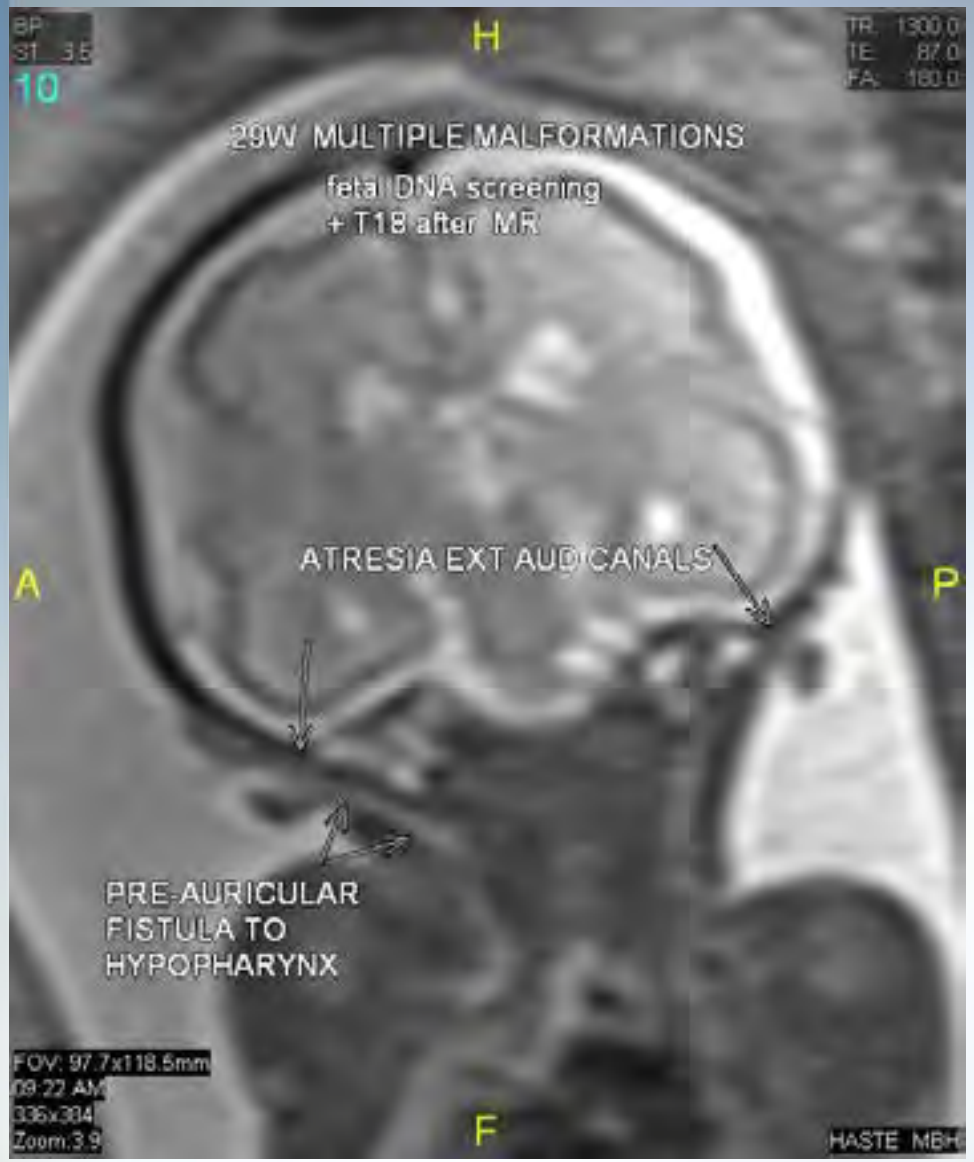
History: 29w late registrant whose outside MFM ultrasound demonstrated multiple malformations including enlarged cisterna magna, mild ventriculomegaly, abnormal hand and feet posturing and polyhydramnios. *At the time of MR, fetal DNA screening results were not known.*

MR demonstrated:

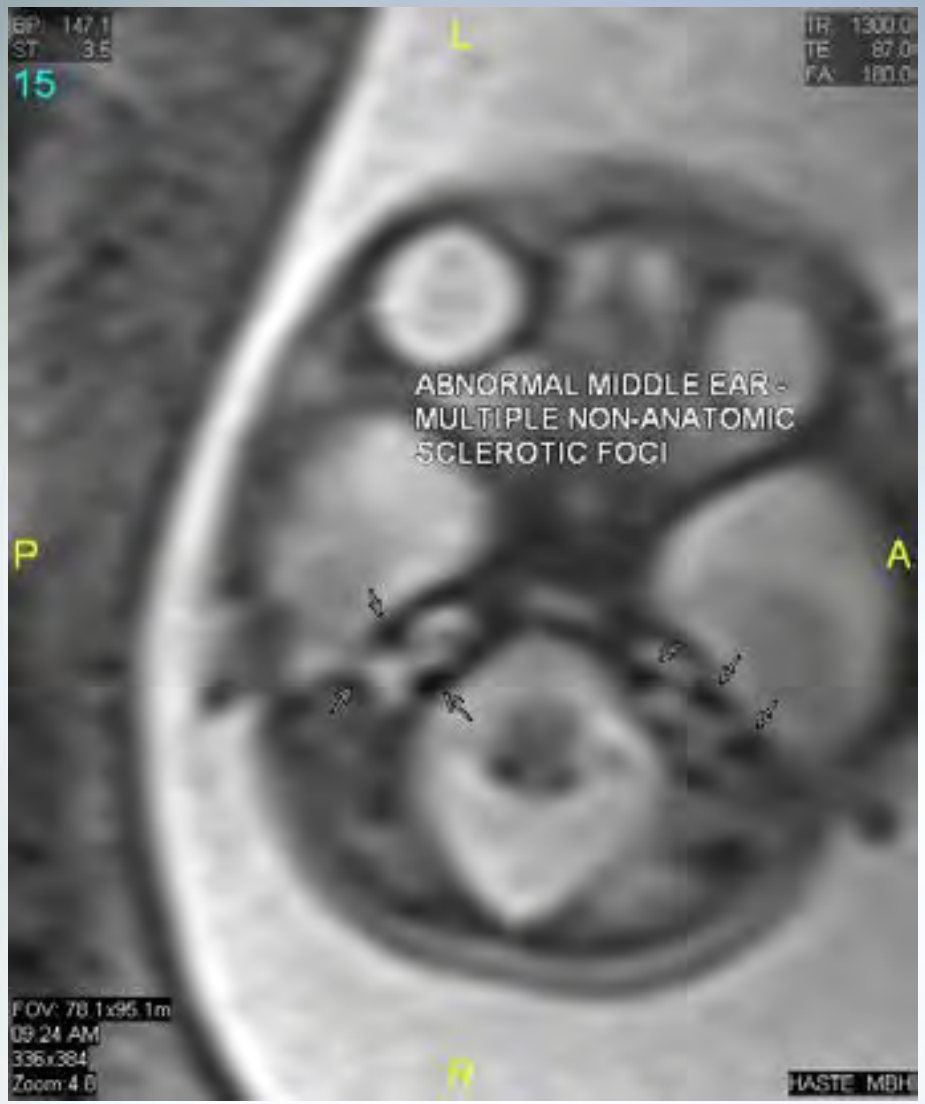
Cerebellar and pontine hypoplasia with intact vermis. No DWM.
Bilateral atresia of external auditory canals with bilateral preauricular fistula to hypopharynx.
Bilateral abnormal mesotympanum with multiple non anatomic sclerotic foci.

One week after MR, fetal DNA in maternal serum screening was positive for trisomy 18.

MR demonstrates malformations of the auditory anatomy unable to be seen on ultrasound



MR demonstrates auditory malformations unable to be seen on ultrasound

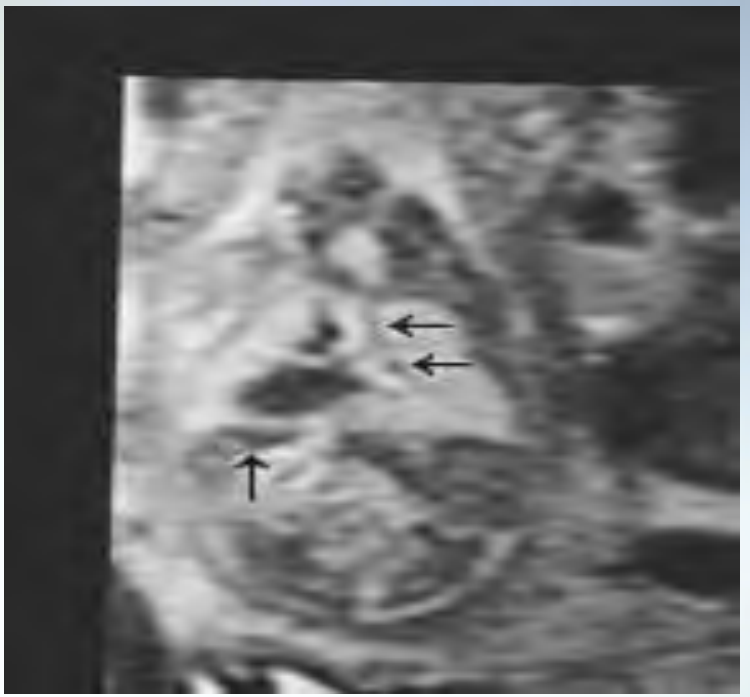


MR changes diagnoses from guarded optimism to lethal

MFM Ultrasound Diagnosed Bilateral Congenital Cystic Adenomatoid Malformation (CCAM)

MR dg = Laryngeal Atresia

Dilated trachea and major bronchi, bilateral enlarged lungs with flattened diaphragm consistent with upper airway obstruction most commonly laryngeal atresia.

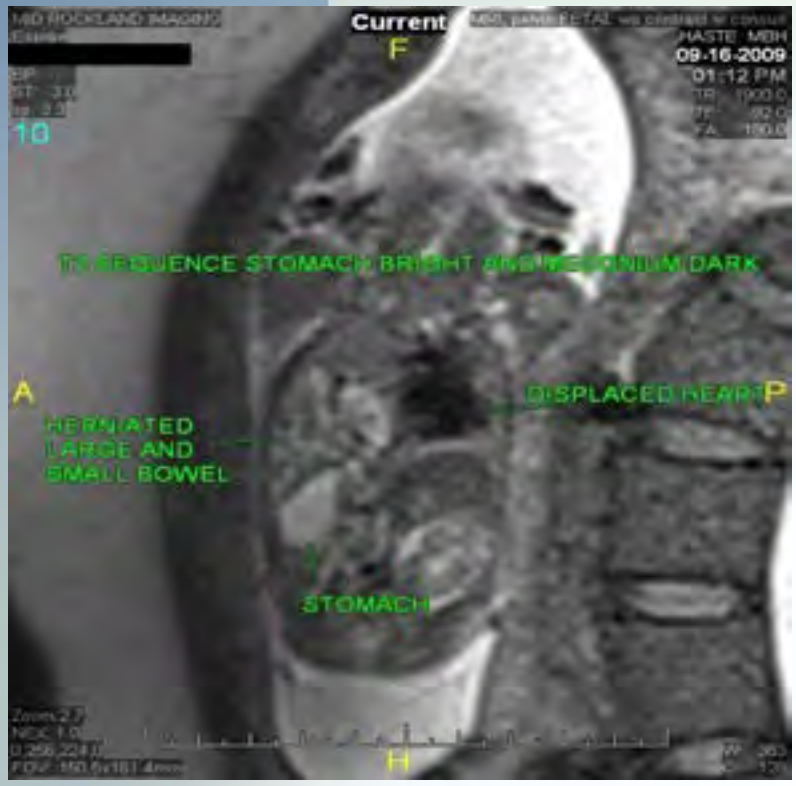


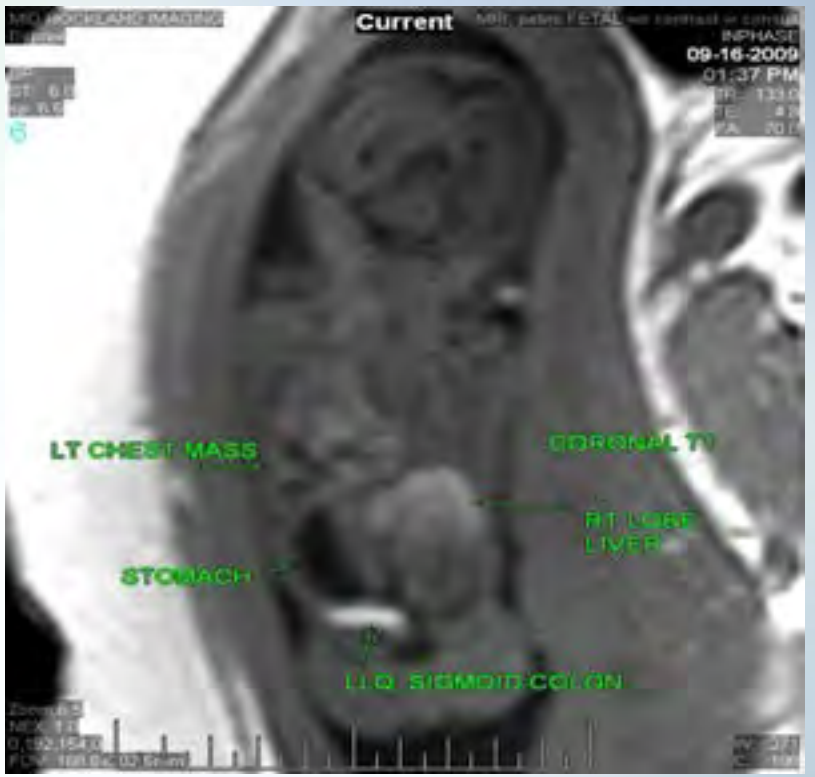
- **Teaching Point**: MR is the modality of choice for evaluating fetal chest masses. In this case MR was able to distinguish laryngeal atresia from bilateral congenital cystic adenomatoid malformations.

MR changes Ultrasound Diagnosis

HISTORY: 21-week gestation with outside ultrasound studies demonstrating rightward cardiac displacement and echogenic left thoracic mass – CCAM vs BPS

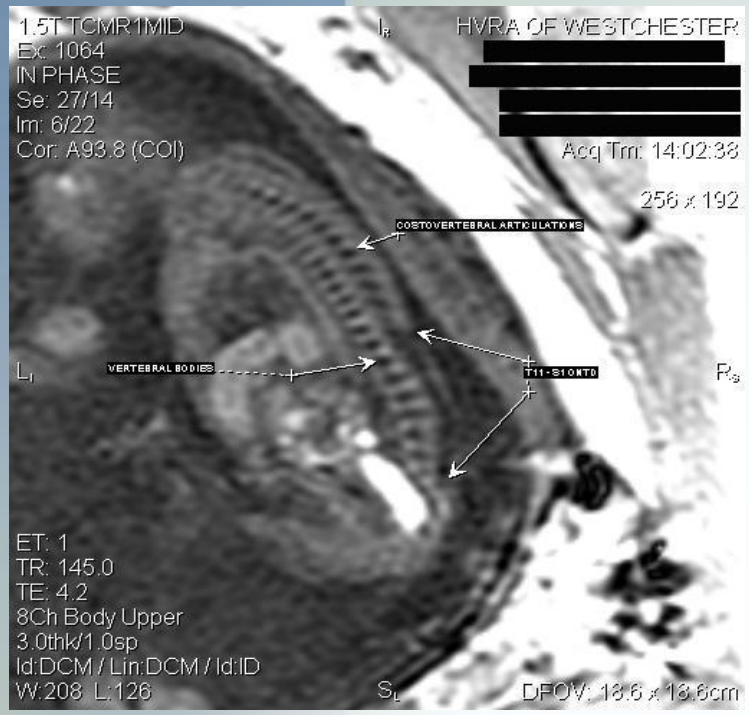
DIAGNOSIS: Left congenital diaphragmatic hernia. Intra- abdominal stomach.

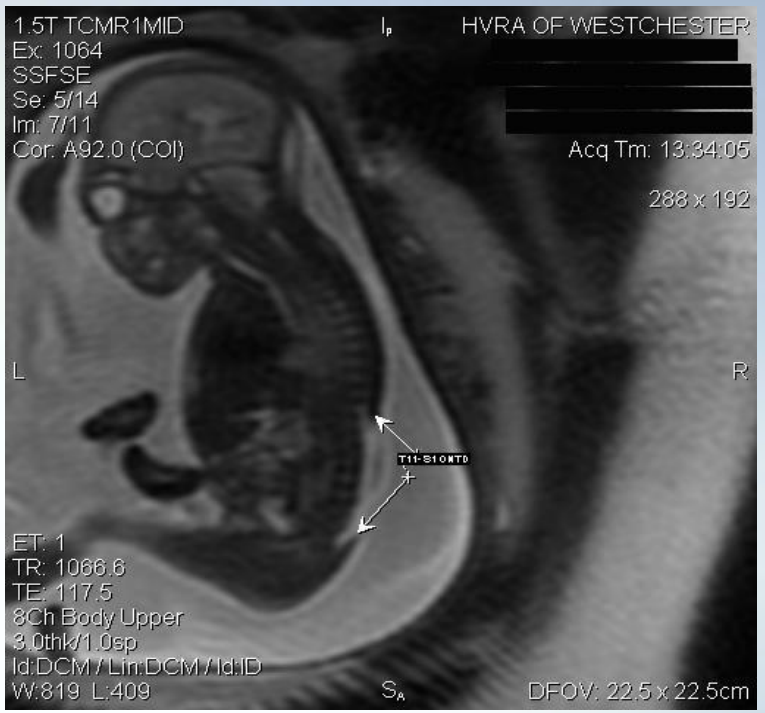




T12 – SI ONTD with Anal Atresia

Ultrasound Uncertain As to Length of ONTD. MR diagnoses additional pathology not seen on ultrasound

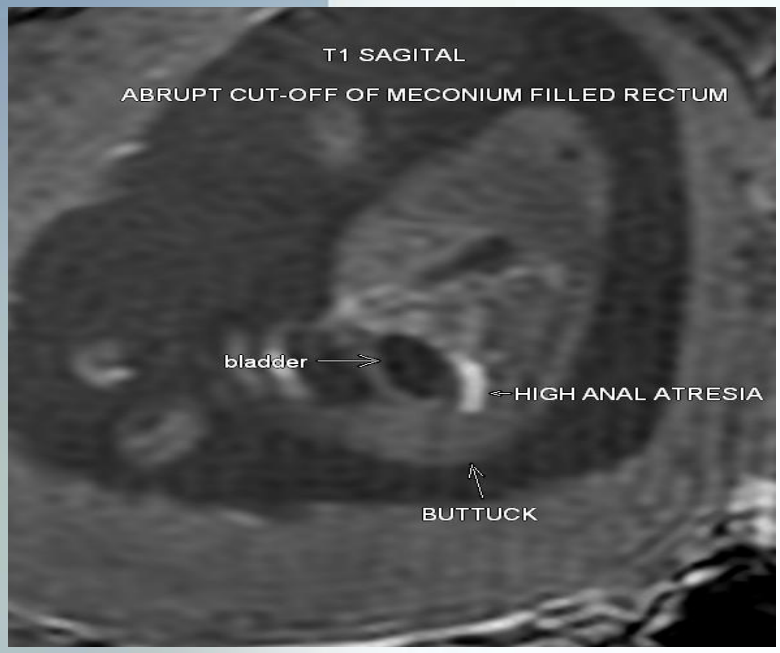


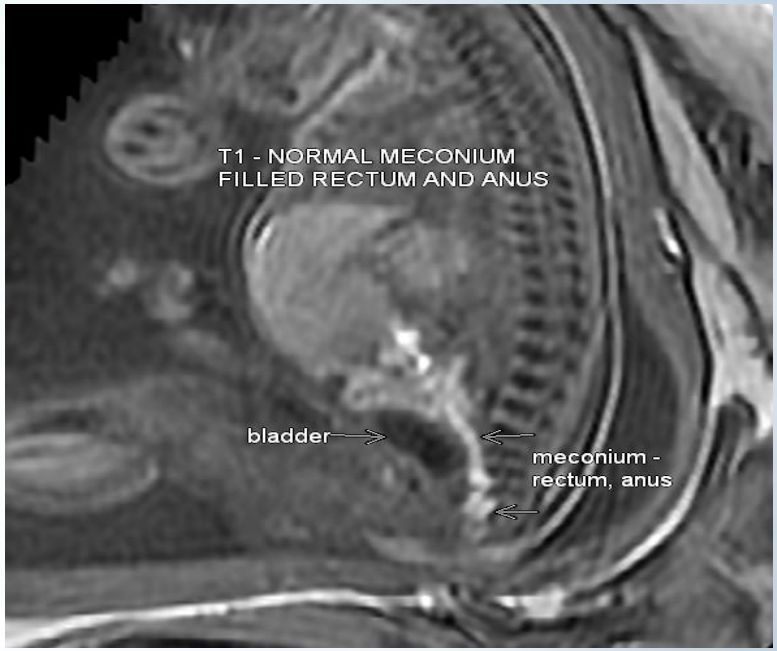


MR identifies significant pathology unable to be seen on ultrasound
Anal atresia in patient with closed sacral NTD

HISTORY: 20-week gestation with outside ultrasound studies suggesting a sacral spinal defect. Our ultrasound studies confirmed a skin-covered sacral spina bifida "occulta" with male appearing phallus with shawl-type scrotum.

Unexpected MR Findings: High anal atresia





Anal atresia with recto-urethral fistula

History: 21w GA whose outside ultrasound studies identified dilated intestine- large versus small bowel uncertain, etiology uncertain.

MR findings: T2 imaging demonstrated a dilated rectosigmoid that did not extend caudal to the anal region consistent with anal atresia. A fistulous tract was identified extending to the urethral region.

Ultrasound and MR evidence of fistulous admixture of urine and meconium.

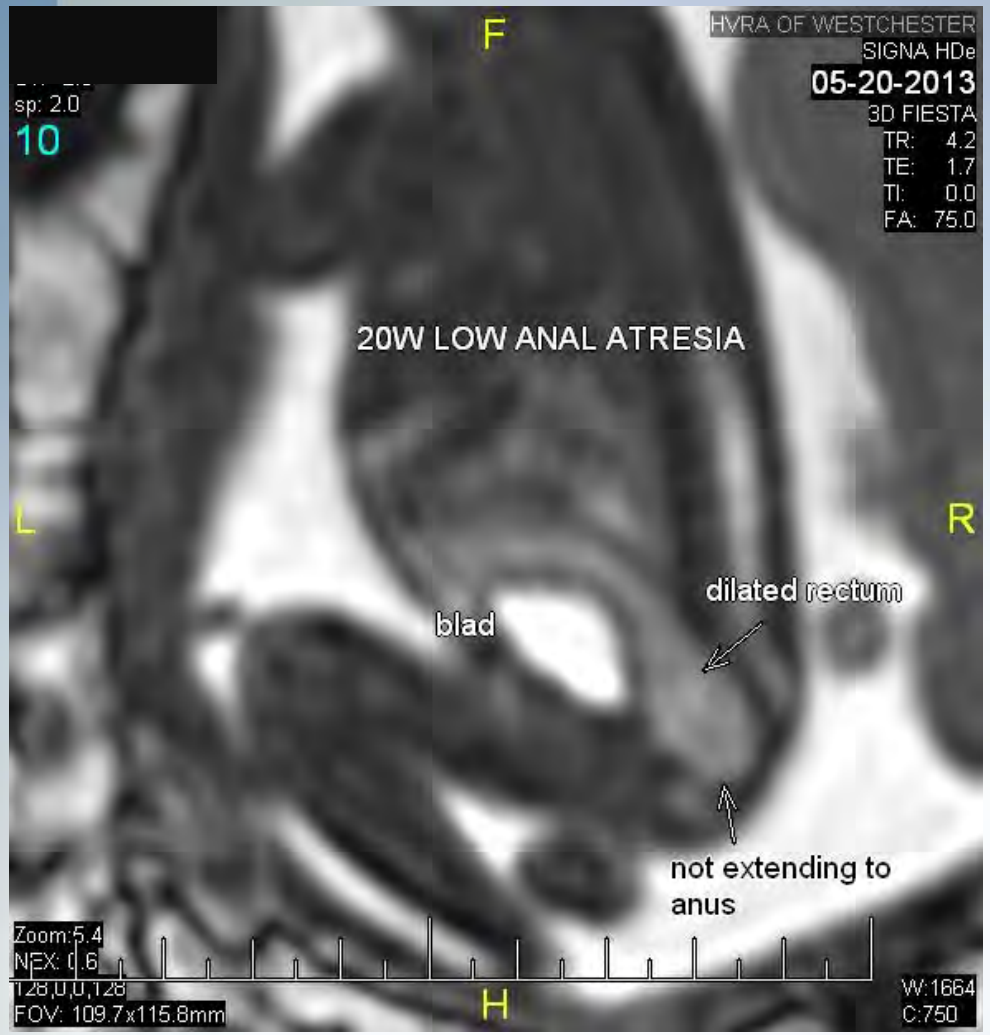
- Absence of T1 meconium hypersignal intensity
- Presence of an enterolith (calcification) within the stool of the dilated colon

Clinical outcome: Patient was born full term with anal atresia. Urine analysis confirmed fecal content consistent with fistula. VCU pending.

Patient treated with diverting colostomy pending definitive surgery when older.

Teaching point: MR is complementary and often superior to ultrasound in diagnosing GI and GU pelvic pathology.

MR establishes a Dg rarely made on ultrasound
Anal atresia with recto-urethral fistula



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MR establishes a Dg rarely made on ultrasound
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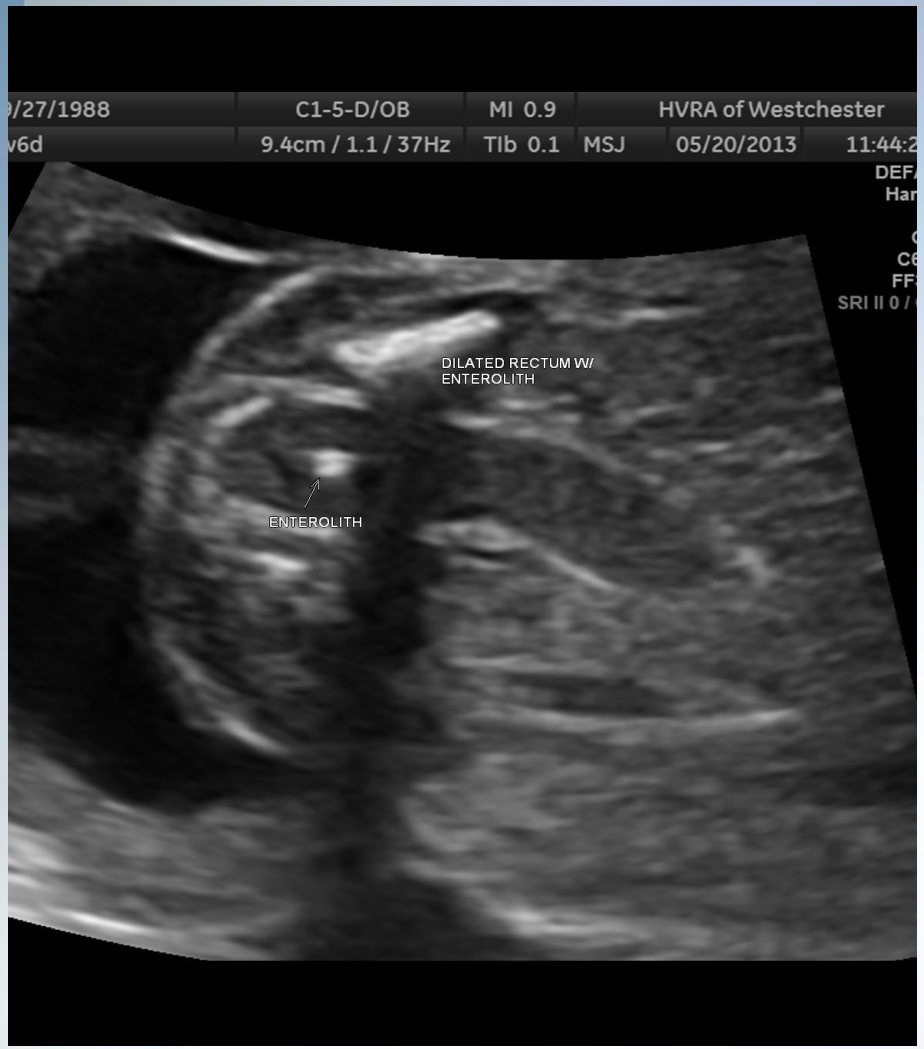


Esophageal atresia with TE fistula

History: 32w GA whose outside ultrasound demonstrated a small stomach. Patient was screened positive for T21 but declined karyotyping.

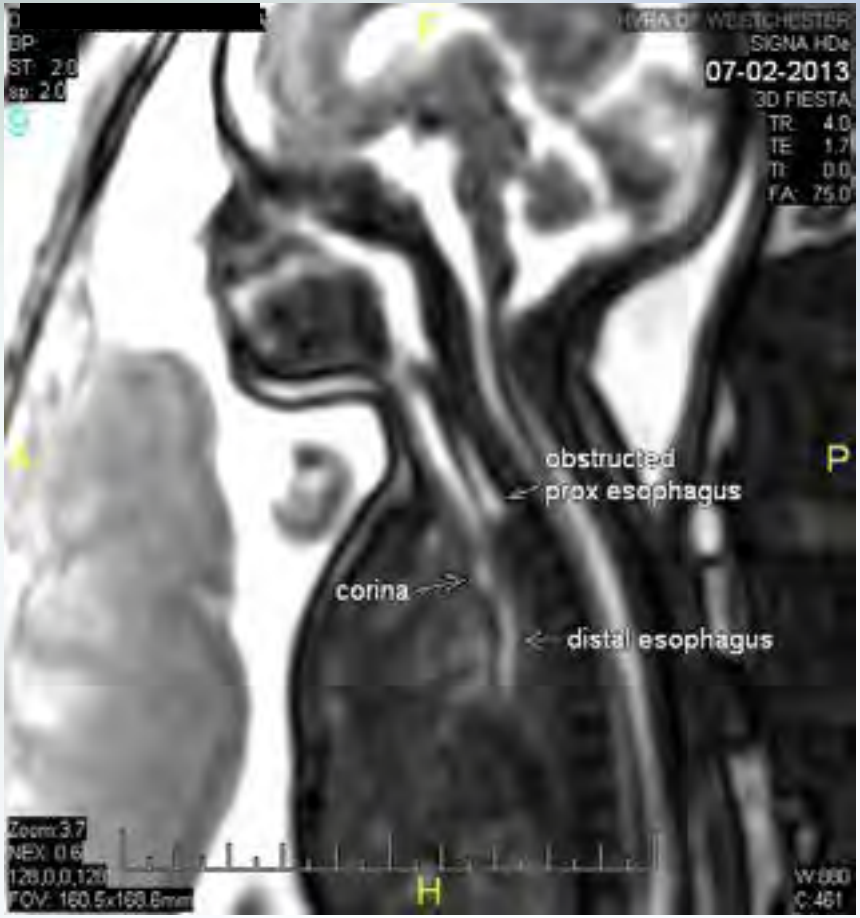
MR demonstrates transient dilatation of the proximal esophagus, the “pouch” sign and
visualizes the TE fistula between the carina and the distal esophageal segment.

Teaching point: Consider MR in the evaluation of otherwise unexplained polyhydramnios and in the evaluation of malformations for which esophageal atresia may coexist in syndromic association –

- VACTERL
- CHARGE

MR establishes a Dg rarely made on ultrasound

Esophageal atresia with TE fistula



Fetal MR for esophageal atresia

total # fetuses studied – 15

indications - outside US studies demo' one or more of the following : small stomach; unexplained polyhydramnios; intestinal dilitation; extra-intestinal dysmorphology that might be part of a chromosomal or non chromosomal syndromic association (ex - VACTERL; CHARGE)

Technique - all fetuses evaluated by sagital 2mm T2 volume acquisition continously repeated over 10minutes in an effort to capture swallowing.

Results

True positive	2
True negative	10
False negative	3

Discussion - One case an interpretive error early in my experience; one case at 20wks – early gest age may be contributory. One case with multiple malformations including cardiomegaly and vascular ring surrounding trachea thereby possibly contributing to interpretive challenges.

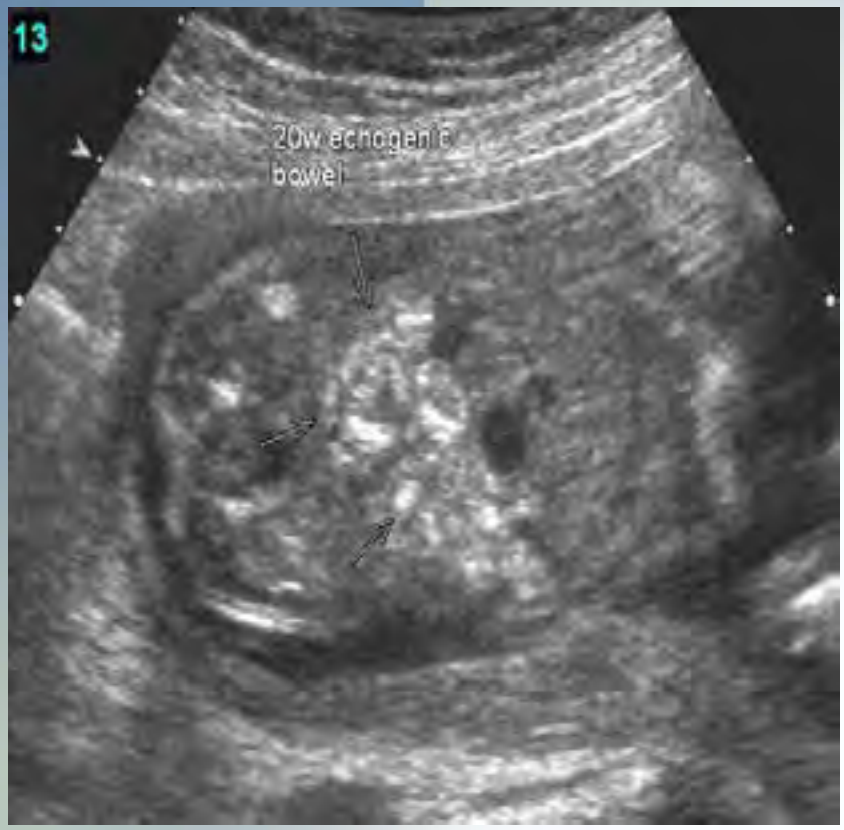
Fetal bowel dilation - small vs. large bowel on Ultrasound, uncertain

History: 21w GA with detailed ultrasound demonstrating a single 8 mm dilated loop of bowel and large amount of non dilated echogenic bowel. Aneuploidy, infectious and cystic fibrosis screening studies were negative.

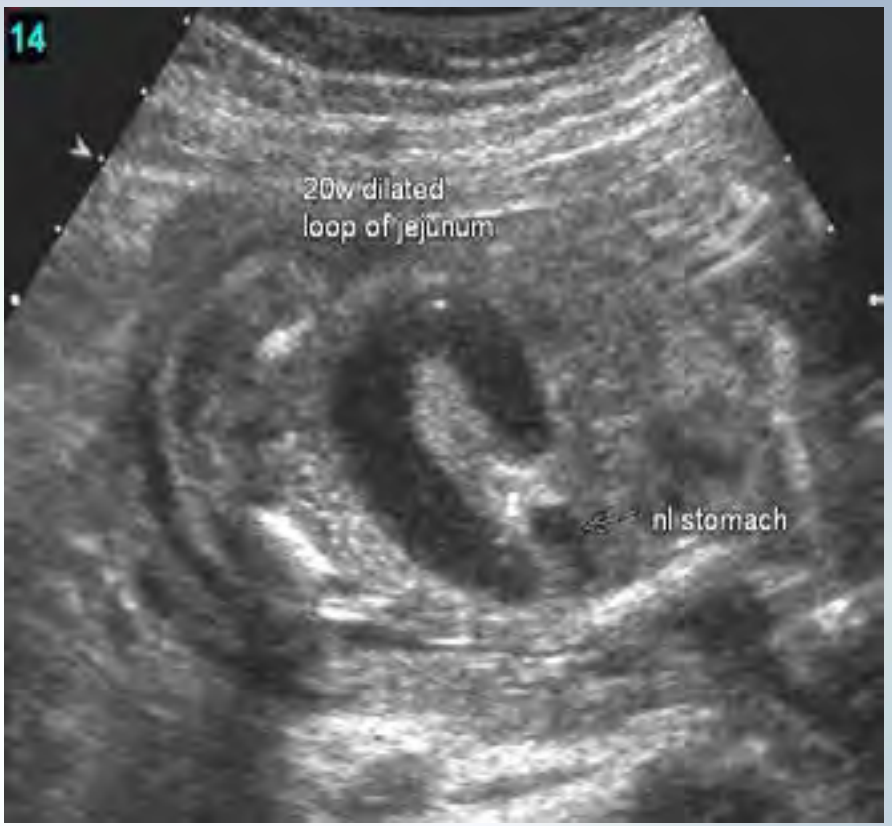
MR demonstrated dilated loop to be of small bowel origin with small, but intact meconium-filled rectum being traced to the anal region (anorectal atresia excluded).

Followup US at tertiary care center demonstrated unchanged small bowel dilatation until 32 weeks, at which time a dilated stomach developed.

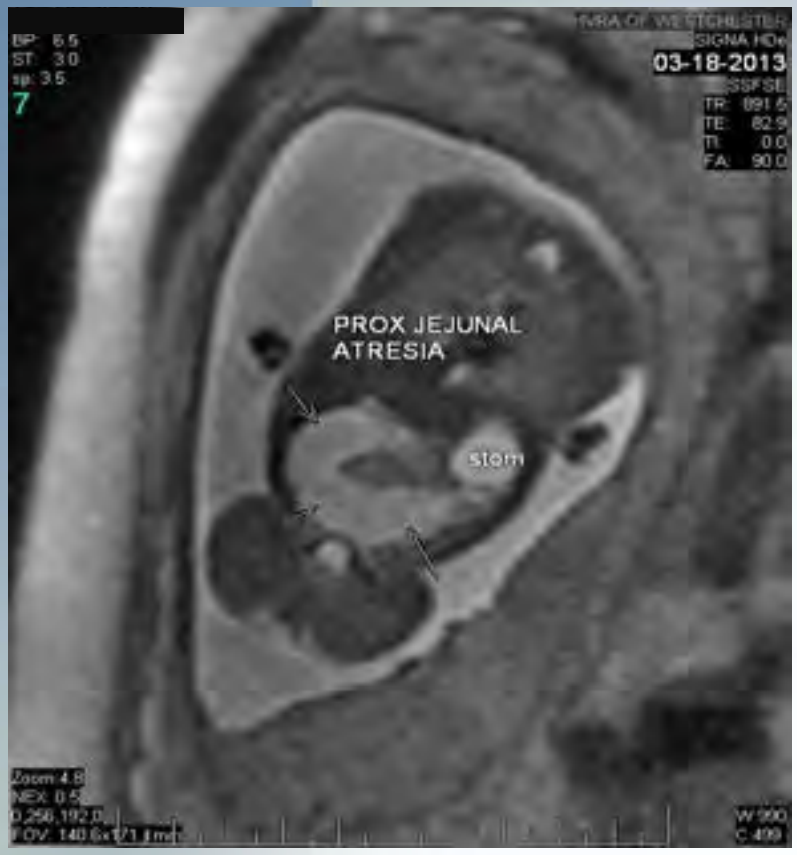
Large vol. of echogenic bowel and dilated jejunum suggests meconium ileus.

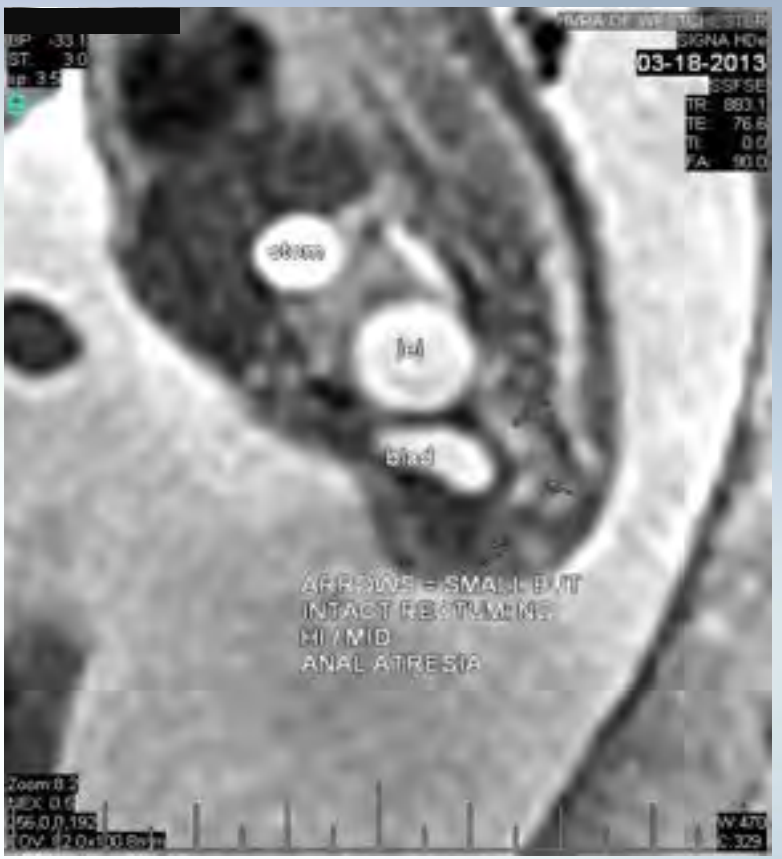


Ultrasound demo - Fetal bowel dilatation small vs. large bowel?



Fetal bowel dilatation - MR assigns organ of origin and answers the question- “is there anal atresia”?





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At surgery – gastric antral atresia with 7 sites of small bowel and colonic atresias.

Clinical workup consistent with combined immunodeficiency syndrome.

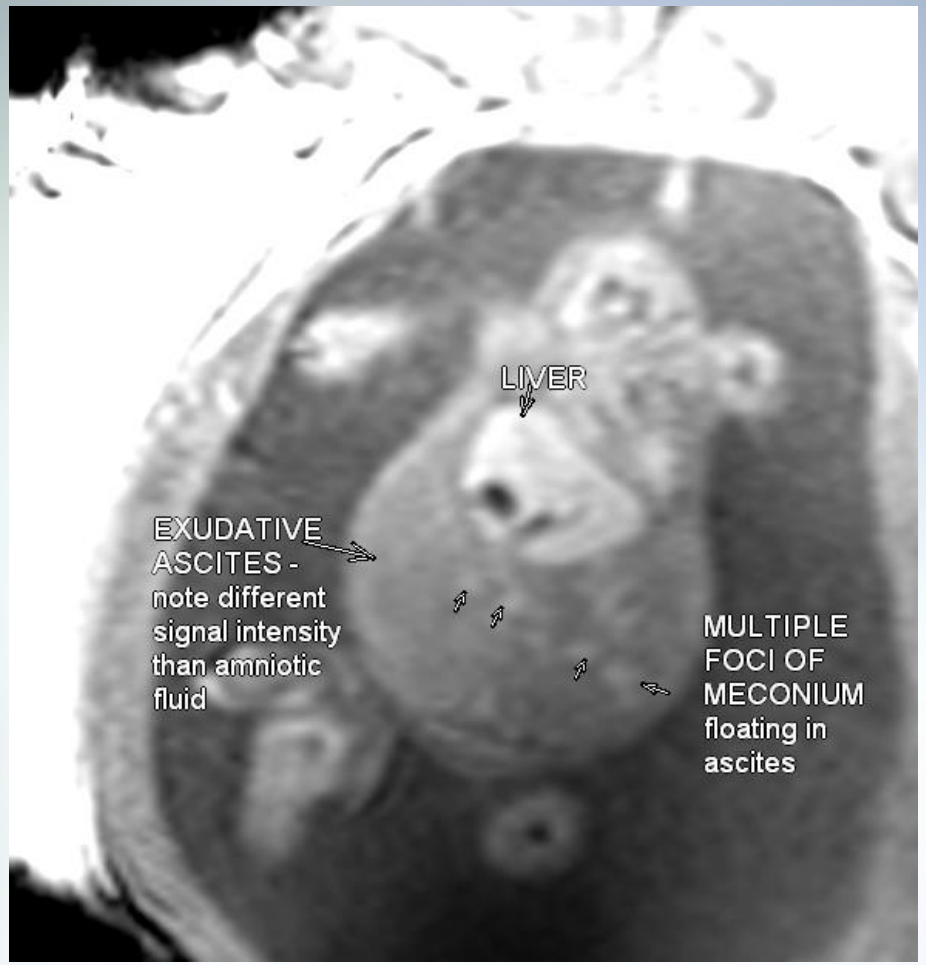
Two months after surgery, the neonatal GI tract remains nonfunctioning. Poor prognosis. ? bone marrow transplant: ? intestinal transplant.

Teaching point:

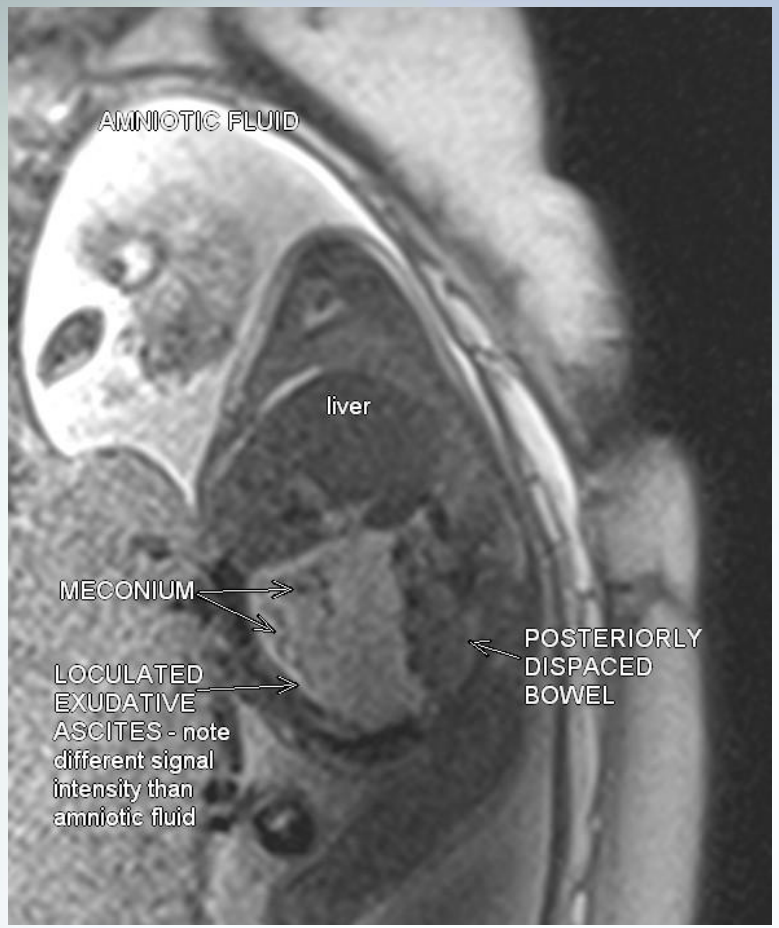
1.When ultrasound identifies dilated bowel, MR is the modality of choice to establish small versus large bowel organ of origin and generate differential diagnosis.

2. If imaging demonstrates multiple sites of bowel dilatation, immunodeficiency syndromes should be considered in the differential diagnosis.

26 w US demo. ascites of unknown etiology. MR diagnosis – meconium peritonitis



26 w US demo ascites of unknown etiology. MR diagnosis – meconium peritonitis



MR establishes a Dg unable to be made on US – hemosiderosis

Anhydramnios and fetal hydrops of unknown etiology.

History : 23w GA with MFM outside studies demonstrating anhydramnios, nonnormalized kidneys, ascites and hydrops of unknown etiology.

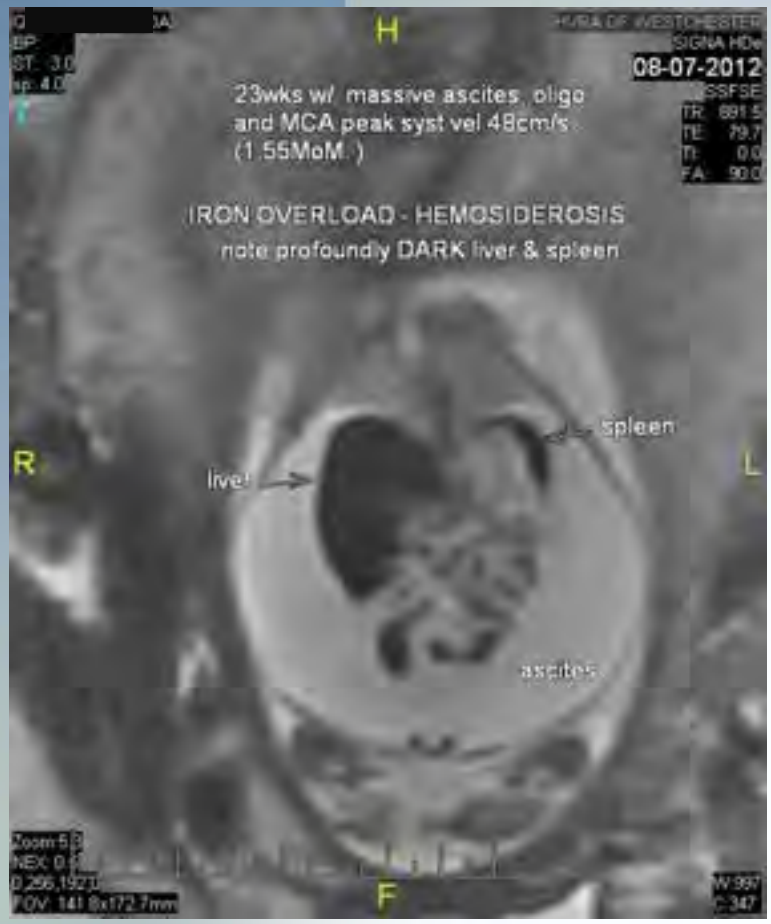
At the time of MR study results of infectious workup were not known. Negative maternal workup for immune incompatibility. Normal fetal echocardiography during MR appointment. Middle cerebral peak systolic velocity 48 cm/second (1.55 MoM) cw severe anemia.

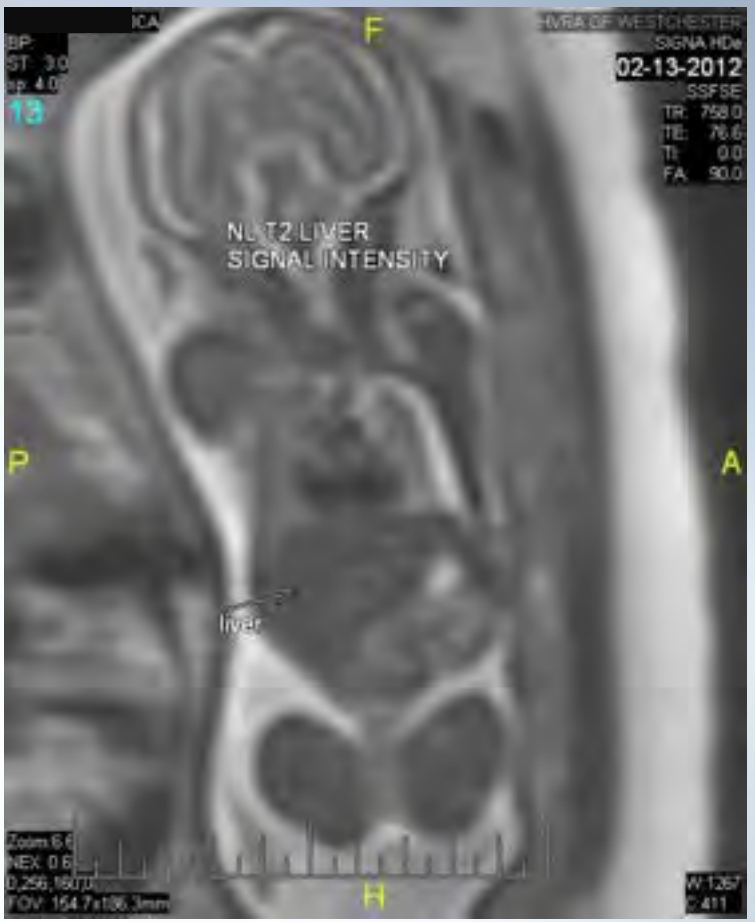
MR demonstrated normal kidneys, featureless transudative-appearing ascites and marked hepatic hyposignal intensity.

Differential diagnosis for marked decreased fetal liver signal intensity statistically favors iron overload (hemosiderosis) due to either various etiologies of fetal anemia (hemolysis) or congenital alloimmune hepatitis (fetal/neonatal hemochromatosis).

MR establishes a dg unable to be made on ultrasound – hemosiderosis

Anhydramnios and fetal hydrops of unknown etiology.





HUDSON VALLEY RADIOLOGY ASSOCIATES

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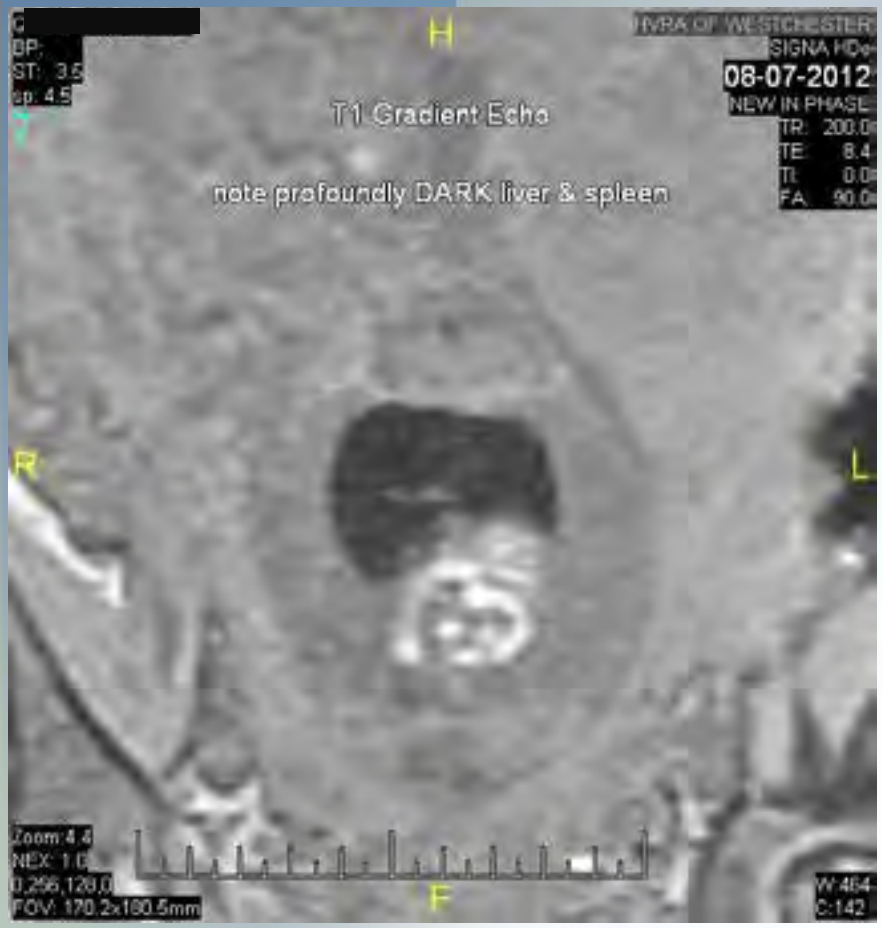

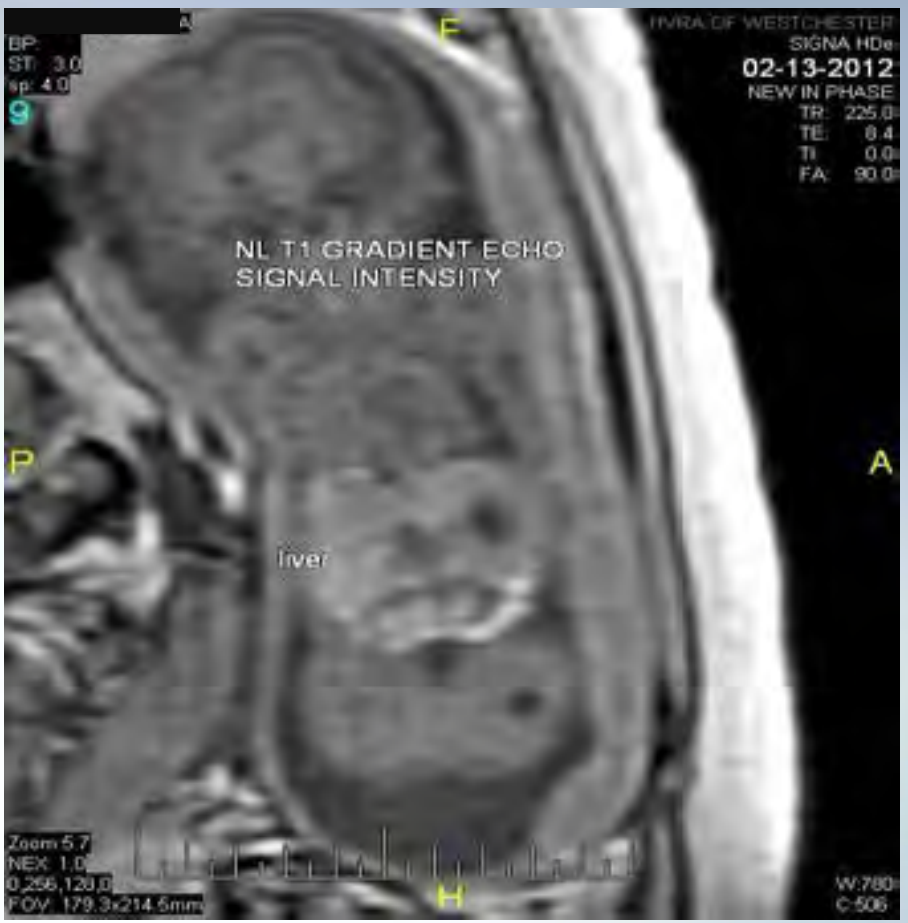

MR establishes a Dg unable to be made on US – hemosiderosis

Anhydramnios and fetal hydrops of unknown etiology.

Clinical outcome: IUFD after fetal transfusion.
After demise lab workup returned + CMV.

Teaching point:

1. With oligohydramnios/anhydramios, MR is superior to ultrasound in evaluation of fetal anatomy.

2. In the context of ascites/hydrops of unknown etiology, MR's ability to diagnose hepatic iron overload is unique. *(Congenital hemochromatosis is the most common cause for neonatal liver failure and has a high rate of recurrence in future pregnancies.)*