AMSER Case of the Month
September 2026

25yo F G3P2002 with no PMH (including substance use or exposures) who declined vaccinations presents for further evaluation after abnormal 20wk ultrasound showing echogenic bowel, subjectively prominent lateral ventricles and fetal growth restriction (FGR) with estimated fetal weight (EFW) <10th %ile). Cell free DNA screening: Low-risk, female.

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HEALTH
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ACR Appropriateness Criteria

Variant: 3 Second and third trimester screening for abnormal finding on ultrasound: soft markers. Next imaging study.

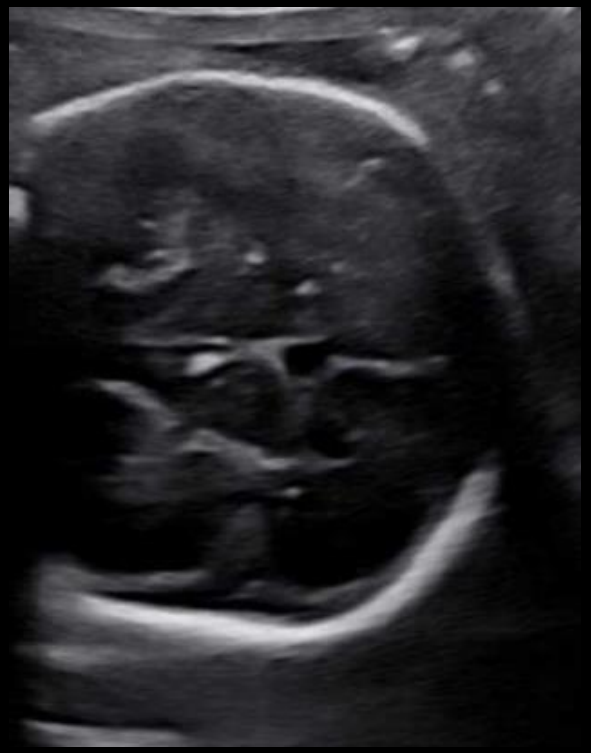
Procedure	Appropriateness Category	Relative Radiation Level
US pregnant uterus transabdominal detailed scan	Usually Appropriate	O
US pregnant uterus transabdominal follow-up	Usually Appropriate	O
US echocardiography fetal	May Be Appropriate	O
MRI fetal without and with IV contrast	Usually Not Appropriate	O
MRI fetal without IV contrast	Usually Not Appropriate	O

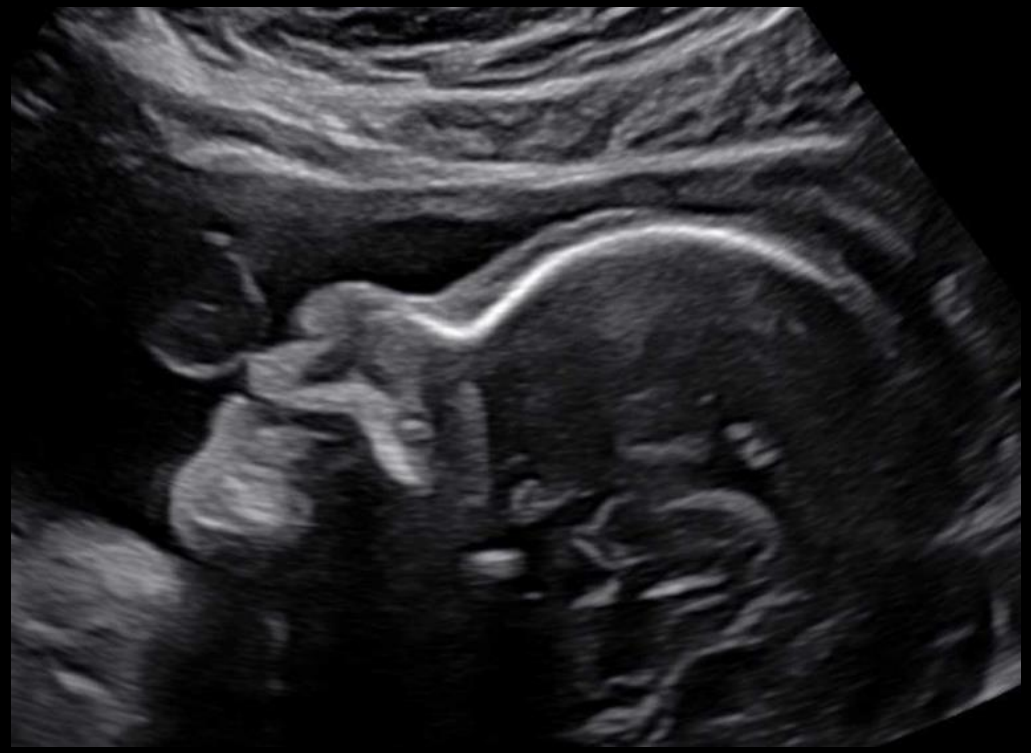
Source: American College of Radiology. ACR Appropriateness Criteria®: Second and Third Trimester Screening for Fetal Anomaly. Reston, VA: American College of Radiology; 2020. Accessed May 16, 2026. https://acsearch.acr.org/docs/3102400/Narrative/

Imaging Ordered: Detailed anatomic US
UA Doppler for FGR

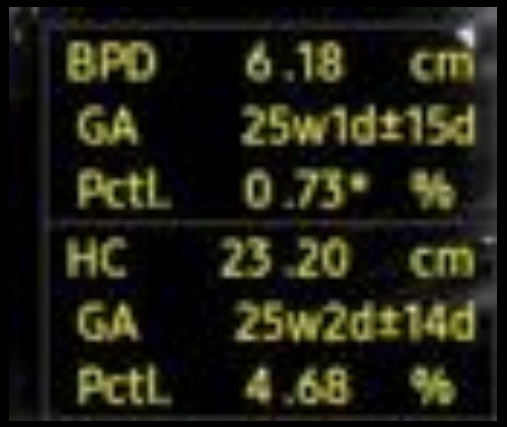
Findings at 27w5d

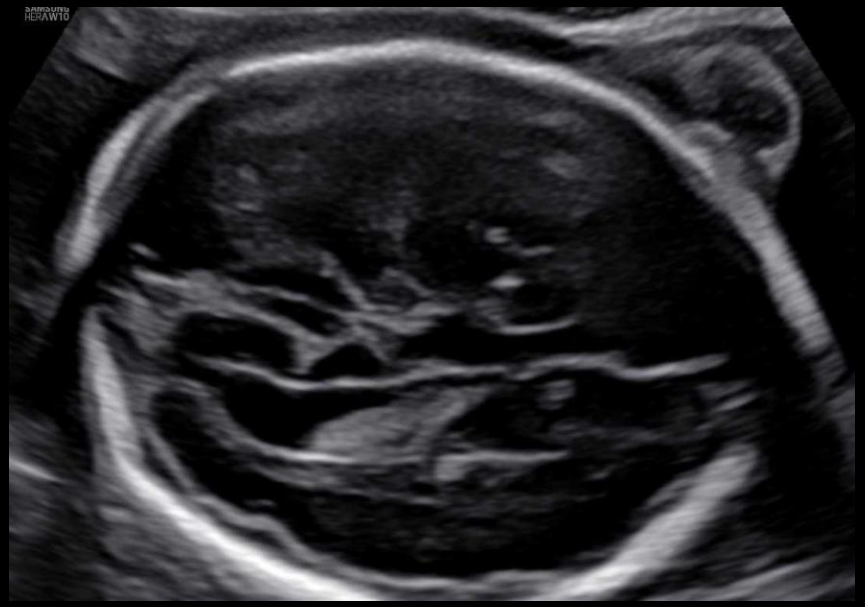
- Severe FGR ,now EFW< 1% and suboptimal interval growth
- Normal amniotic fluid volume
- UA doppler variable, some elevated systolic/diastolic (SD) ratios
- Liver and adrenal calcifications
- Enlarged cisterna magna (11.4mm) with small cerebellum
- Periventricular calcifications

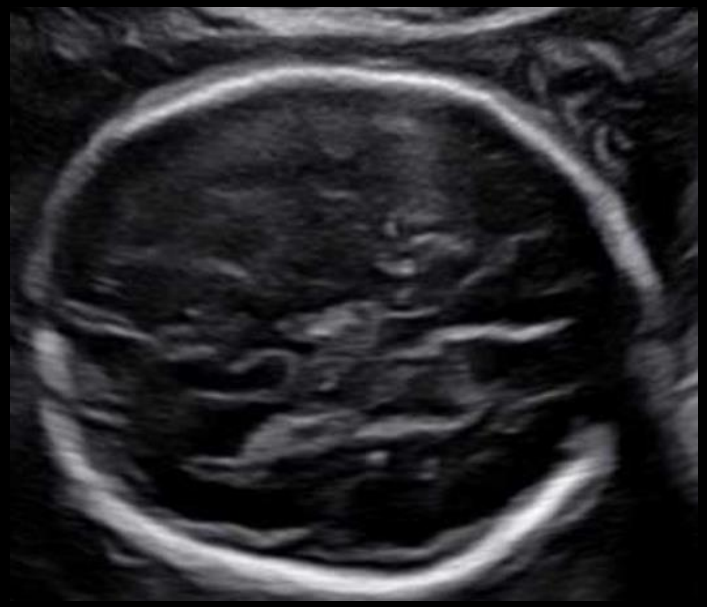


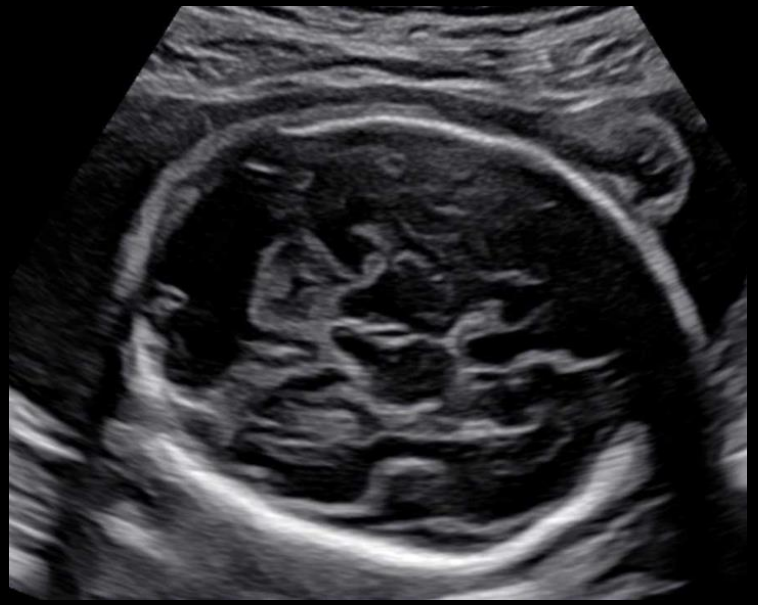


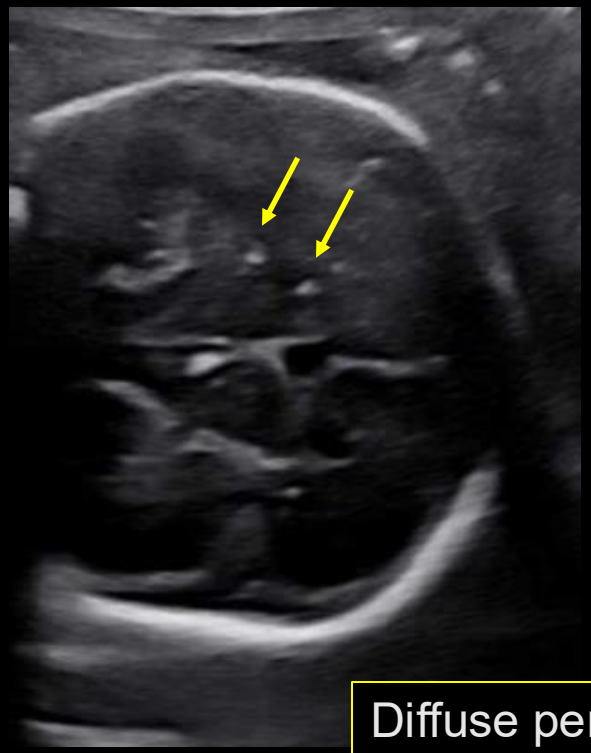
BPD : Biparietal diameter
HC: Head circumference
Pctl: Percentile for gestational age (GA)

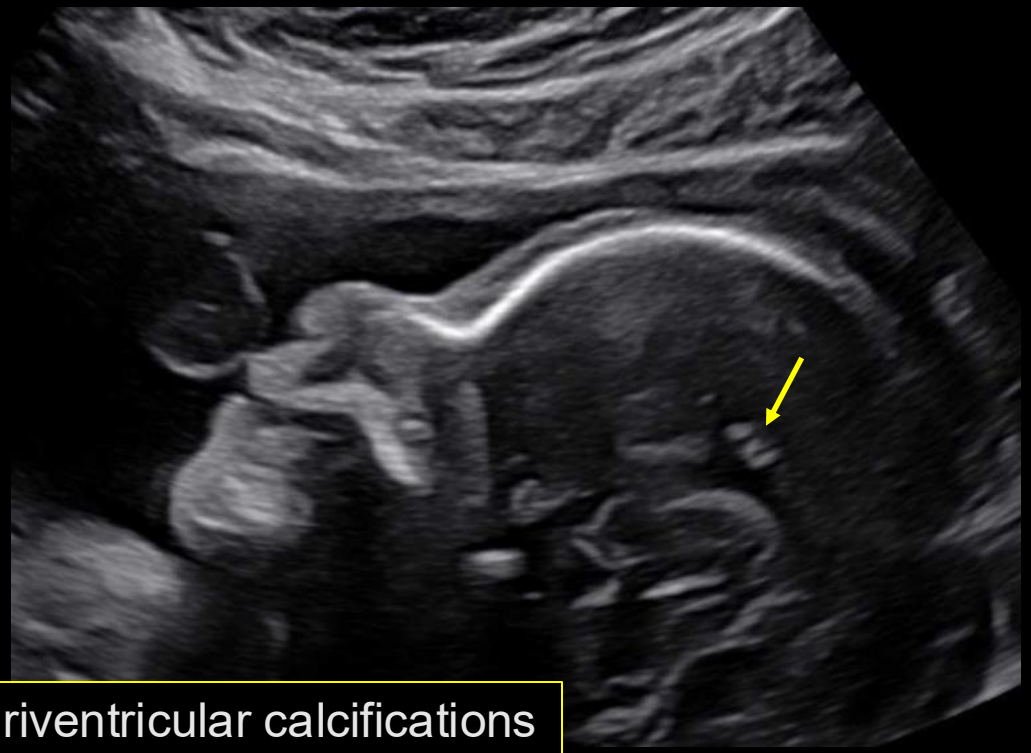




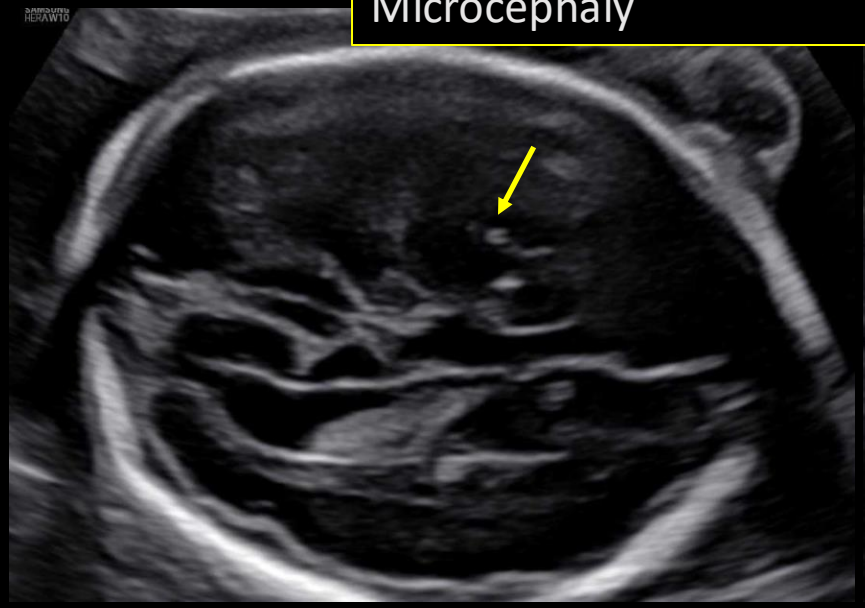


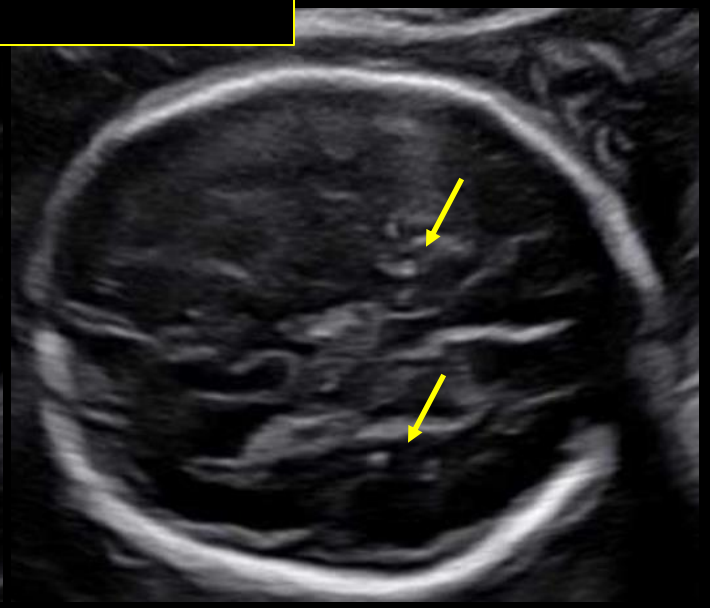






Diffuse periventricular calcifications
Microcephaly





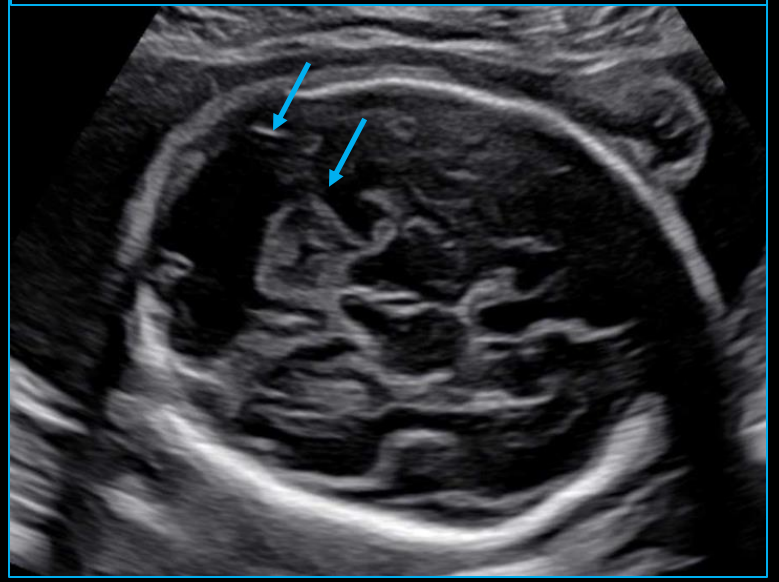
BPD : Biparietal diameter
HC: Head circumference

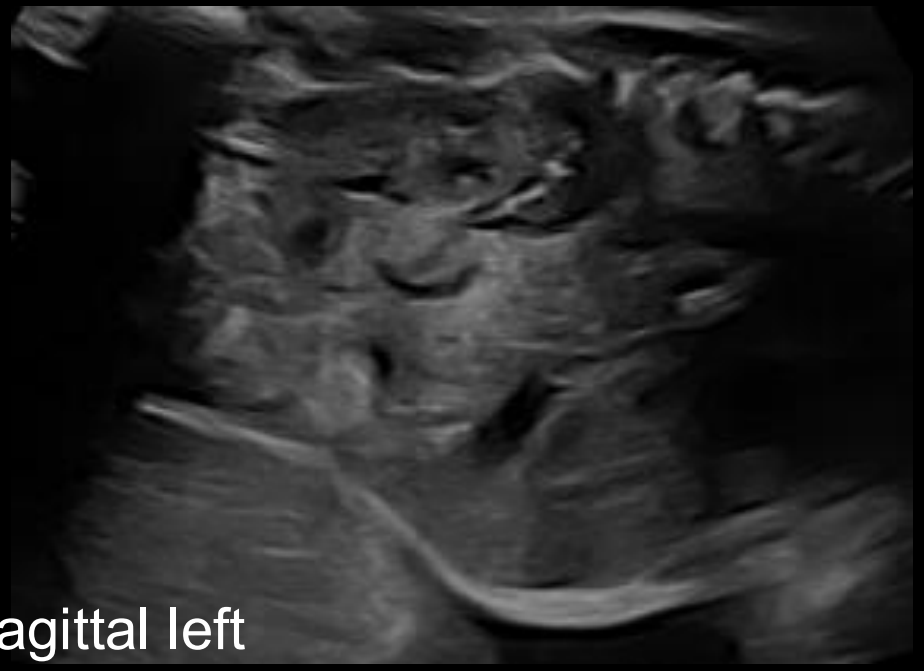
BPD	6.18	cm
GA	25w1d±15d	
PctL	0.73*	%
HC	23.20	cm
GA	25w2d±14d	
PctL	4.68	%

Z score -2.7

Z score -3.8

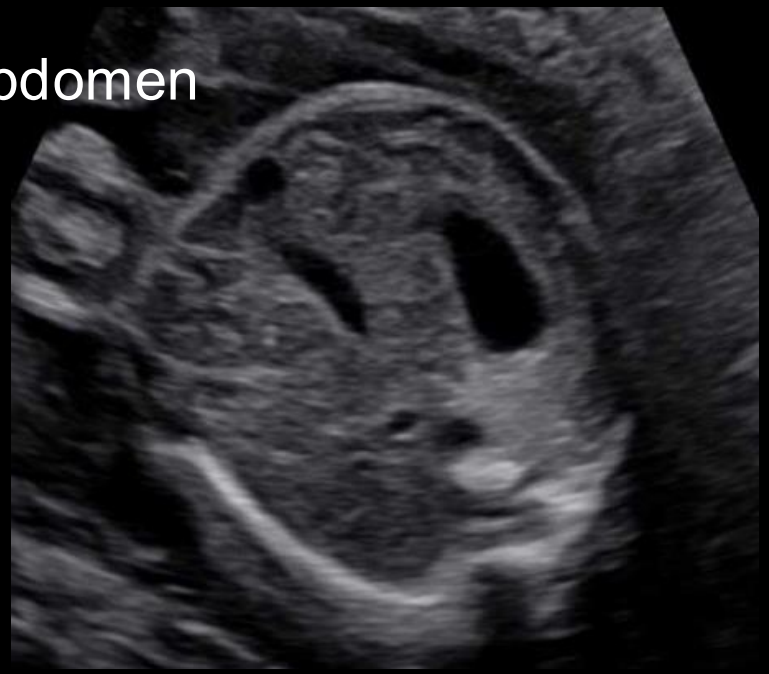
Enlarged cisterna magna
(11.4mm)
Small cerebellum

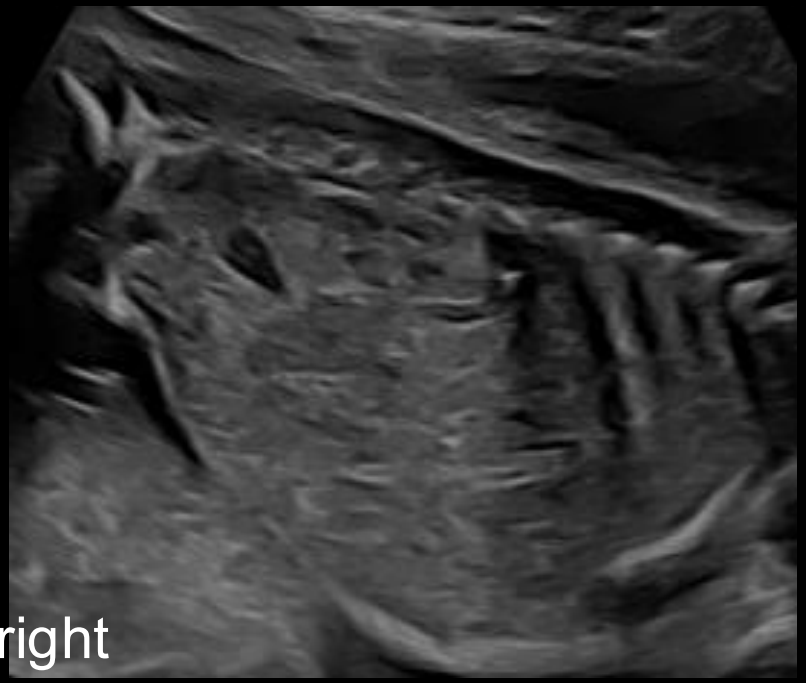




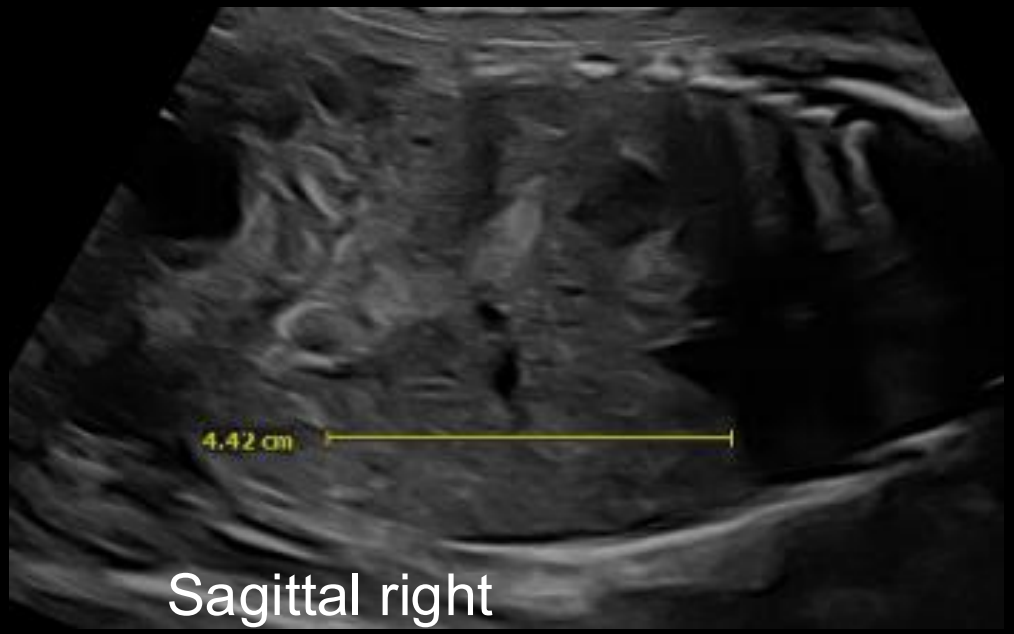
Sagittal left

Axial abdomen

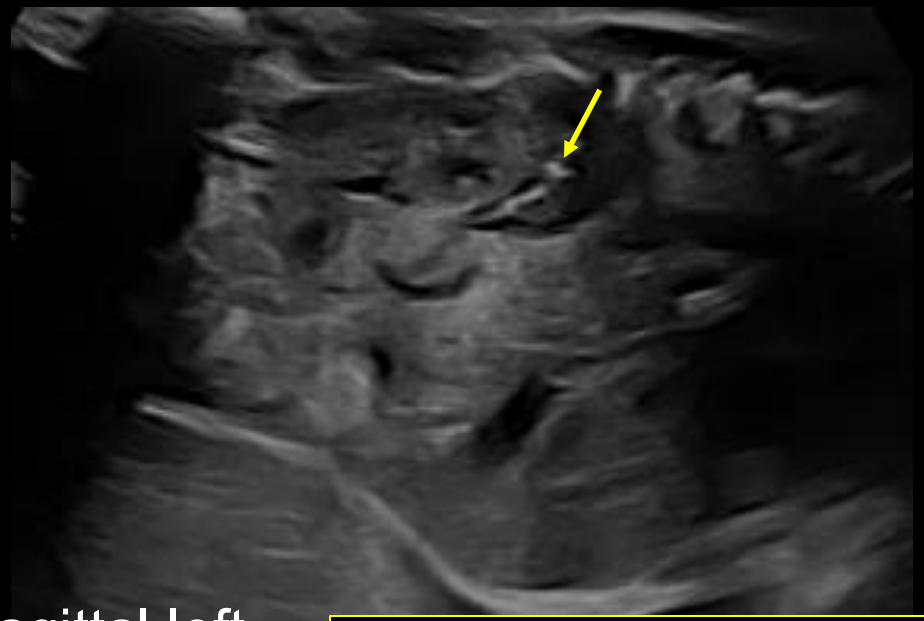




Sagittal right

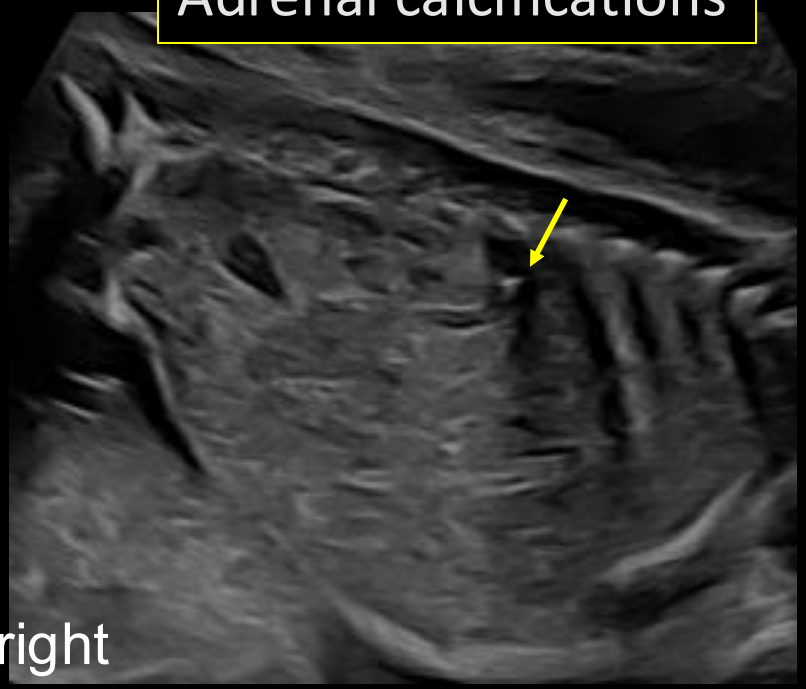


Sagittal right



Sagittal left

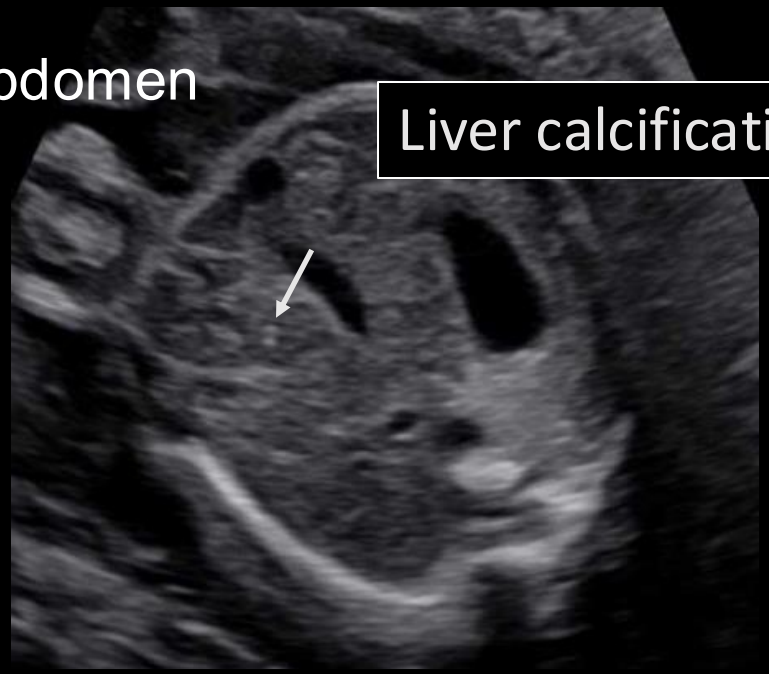
Adrenal calcifications



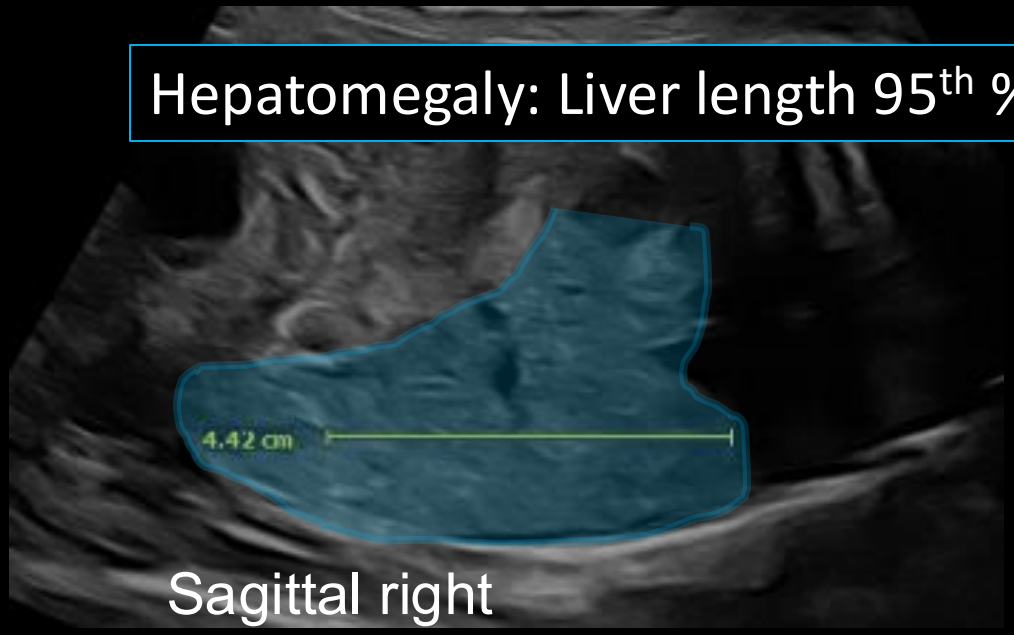
Sagittal right

Axial abdomen

Liver calcifications



Hepatomegaly: Liver length 95th %ile for GA



Sagittal right

ACR Appropriateness Criteria

Variant: 4 Second and third trimester screening for abnormal finding on ultrasound: major anomalies. Next imaging study.

Procedure	Appropriateness Category	Relative Radiation Level
US echocardiography fetal	Usually Appropriate	O
US pregnant uterus transabdominal detailed scan	Usually Appropriate	O
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MRI fetal without IV contrast	Usually Appropriate	O
MRI fetal without and with IV contrast	Usually Not Appropriate	O

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Imaging Ordered: Follow-up Ultrasound

Findings:

- 30w3d
- Severe fetal growth restriction with EFW 2%
- Elevated SD ratios in the umbilical artery

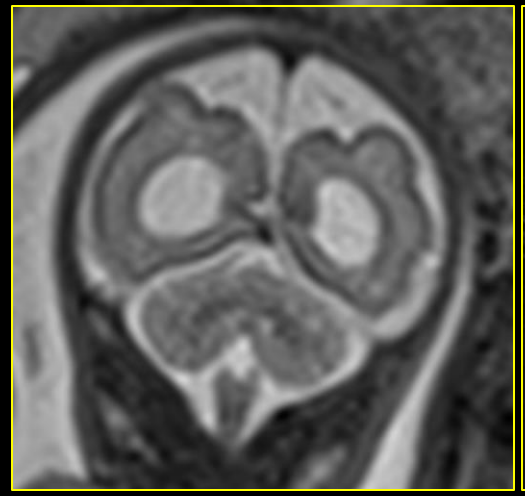
Imaging Ordered: Fetal MRI without IV contrast

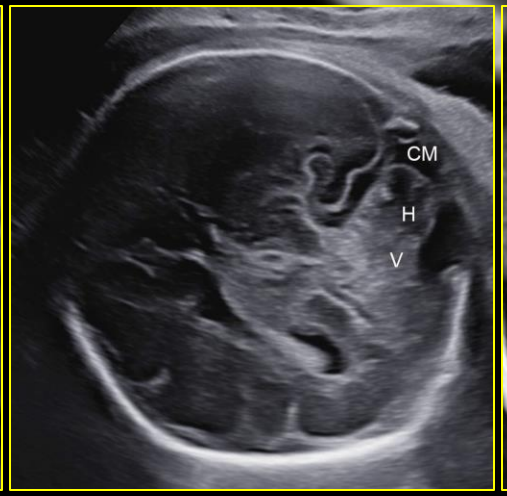
Findings:

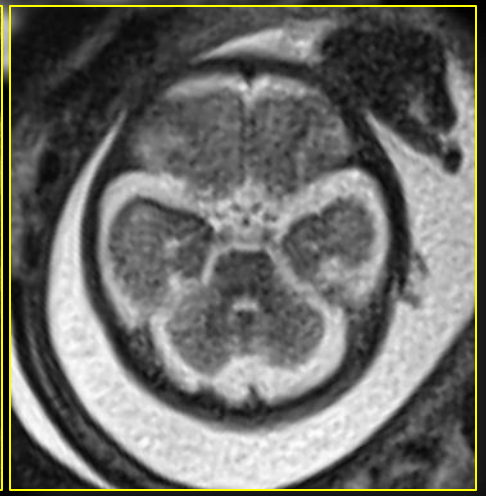
- Small, dysplastic cerebellum
- Suspected polymicrogyria involving the left cerebral hemisphere
  - Polymicrogyria: a brain development disorder where the surface has too many, excessively small folds (gyri) and a bumpy, thickened cortex with a fuzzy gray-white matter border
- Hippocampal atrophy

Normal age-matched images for comparison

H:Cerebeller hemisphere
V: Vermis
CM: Cisterna magna



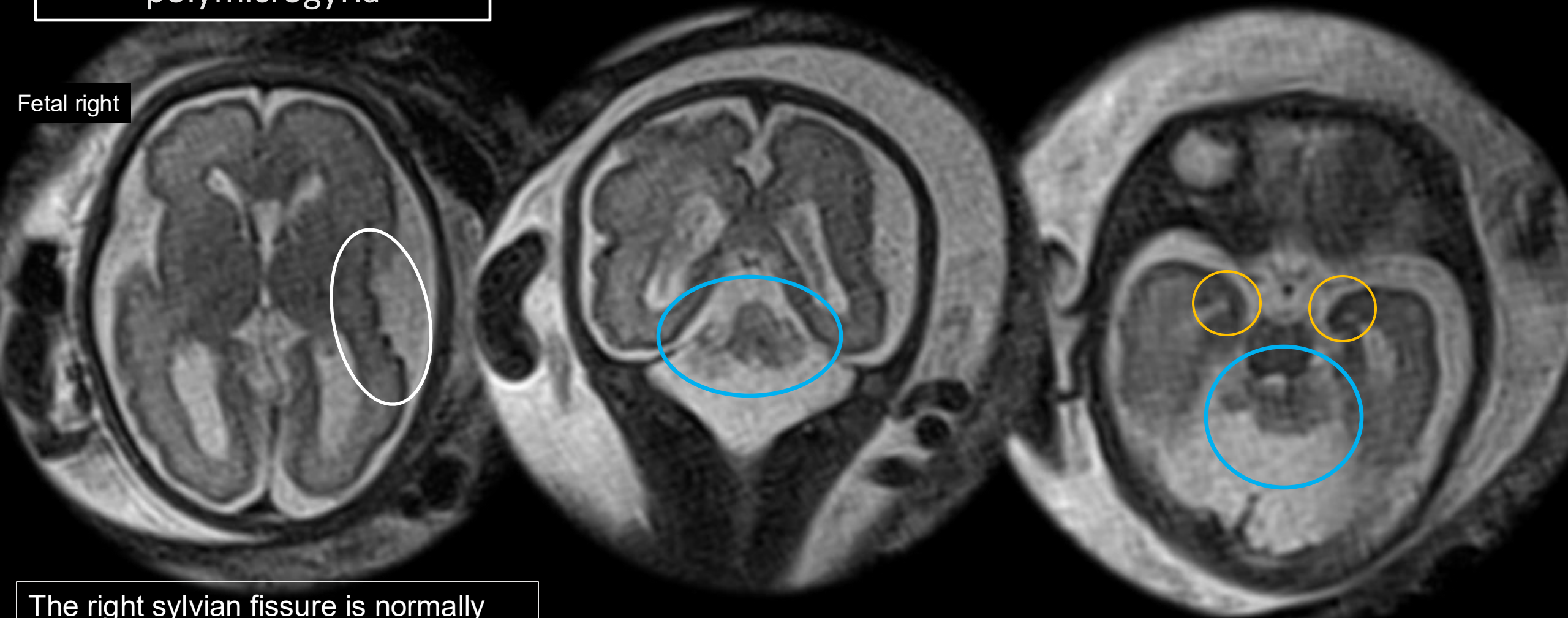




Cortical malformation/ polymicrogyria

Cerebellar dysplasia

Hippocampal dysplasia



The right sylvian fissure is normally formed with smooth cortex unlike the left where the fissure is abnormal in shape and the cortex has the “bumpy” appearance of polymicrogyria .

Severe loss of cerebellar tissue with resultant increase in size of the cisterna magna.
Hippocampi are thin, dysplastic and of abnormally low signal

Final Diagnosis

Congenital cytomegalovirus infection

Case Discussion

Congenital CMV (cCMV) infection is the most common congenital viral infection, affecting around 1 in 200 infants in high-income countries and 1 in 71 in low- and middle-income countries.[1,2] Reported prevalence of cCMV is 0.2% to 2.0% (average of 0.64%) of pregnancies.[3] Long-term sequala including sensorineural hearing loss and neurodevelopmental disability.

CMV infection can be acquired during pregnancy (primary infection) or be a reactivation of a prior CMV infection or a new infection with a different strain of the virus (non-primary infection).[2] Women with primary infection experience a higher transplacental transmission rate (30-40%) in comparison to women with a non-primary infection who experience a low transmission rate (1-2%).[4] Regardless of primary v. non-primary infection, the severity of cCMV is trimester specific with more severe neurologic sequelae occurring more frequently with infection in the first trimester (20–30%) when compared to infection in the second and third trimester.[4]

The most common source of CMV infection in pregnant women is the saliva and urine of young children. The majority of people with active CMV infection are asymptomatic with 10% experiencing symptoms like infectious mononucleosis.[2] Fetal diagnosis of cCMV is made by amniocentesis performed after 17 weeks of gestation and at least 6–8 weeks after the suspected maternal infection.[1]

Case Discussion

Ultrasound Findings[5]

- Fetal intracranial calcification
  - mainly periventricular
- Fetal hydrocephalus
- Heterogeneous brain parenchyma
- Microcephaly
- Intraventricular adhesions
- Fetal intrahepatic calcification
- Fetal hepatomegaly
- Fetal growth restriction (FGR)
- Echogenic bowel

MRI Findings[6]

- White matter hyperintensities
- Ventriculomegaly (unilateral or bilateral)
- Cysts/pseudocysts
- Ventriculitis
- Intraventricular septations/adhesions
- Cortical malformations/polymicrogyria
- Clefts (schizencephaly/porencephaly)
- Cerebellar Hypoplasia/dysplasia
- Hippocampal dysplasia
- Lenticulostriate vasculopathy
- Hepatomegaly, splenomegaly
- Effusions (pericardial, pleural, ascites)
- Skin edema
- Thickened placenta

Case Discussion

Prenatal treatment for cCMV is controversial. There is ongoing investigation of the role of valganciclovir and CMV hyperimmune globulin in treatment.

The prognosis predictors for infected fetuses are largely based on timing of infection, presence and type of fetal anomalies, and laboratory results.[2] Best next steps are case dependent on fetal findings and goals of care discussions between patients and providers.

References:

1. Jones CE, Bailey H, Bamford A, et al. Managing challenges in congenital CMV: current thinking. Arch Dis Child. 2023;108(8):601-607. doi:10.1136/archdischild-2022-323809
2. Khalil A, Heath PT, Jones CE, Soe A, Ville YG; Royal College of Obstetricians and Gynaecologists. Congenital Cytomegalovirus Infection: Update on Screening, Diagnosis and Treatment: Scientific Impact Paper No. 56. BJOG. 2025;132(2):e42-e52. doi:10.1111/1471-0528.17966
3. Rawlinson WD, Boppana SB, Fowler KB, Kimberlin DW, Lazzarotto T, Alain S, et al. Congenital cytomegalovirus infection in pregnancy and the neonate: consensus recommendations for prevention, diagnosis, and therapy. Lancet Infect Dis. 2017;17(6):e177-e188. doi:10.1016/S1473-3099(17)30143-3
4. Palmetti M, Venturini E, Bartolini L, Chiappini E, Lyall H, Galli L. Congenital CMV infection and central nervous system involvement: mechanisms, treatment, and long-term outcomes. Eur J Pediatr. 2025;184:381. doi:10.1007/s00431-025-06215-4
5. Radswiki T, Ibrahim D, Baba Y, et al. Congenital cytomegalovirus infection. Radiopaedia.org. Published 2010. Last revised October 12, 2021. Accessed May 18, 2026. https://doi.org/10.53347/rID-12647
6. Diogo MC, Glatter S, Binder J, Kiss H, Prayer D. The MRI spectrum of congenital cytomegalovirus infection. Prenat Diagn. 2020;40(1):110-124. doi:10.1002/pd.5591
7. Pinninti S, Boppana S. Congenital cytomegalovirus infection diagnostics and management. Dis. 2022;35(5):436-441. doi:10.1097/QCO.0000000000000874

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