

Case Report: Fetal Neuroanomaly In A Diabetic Mother with DKA

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Case Report

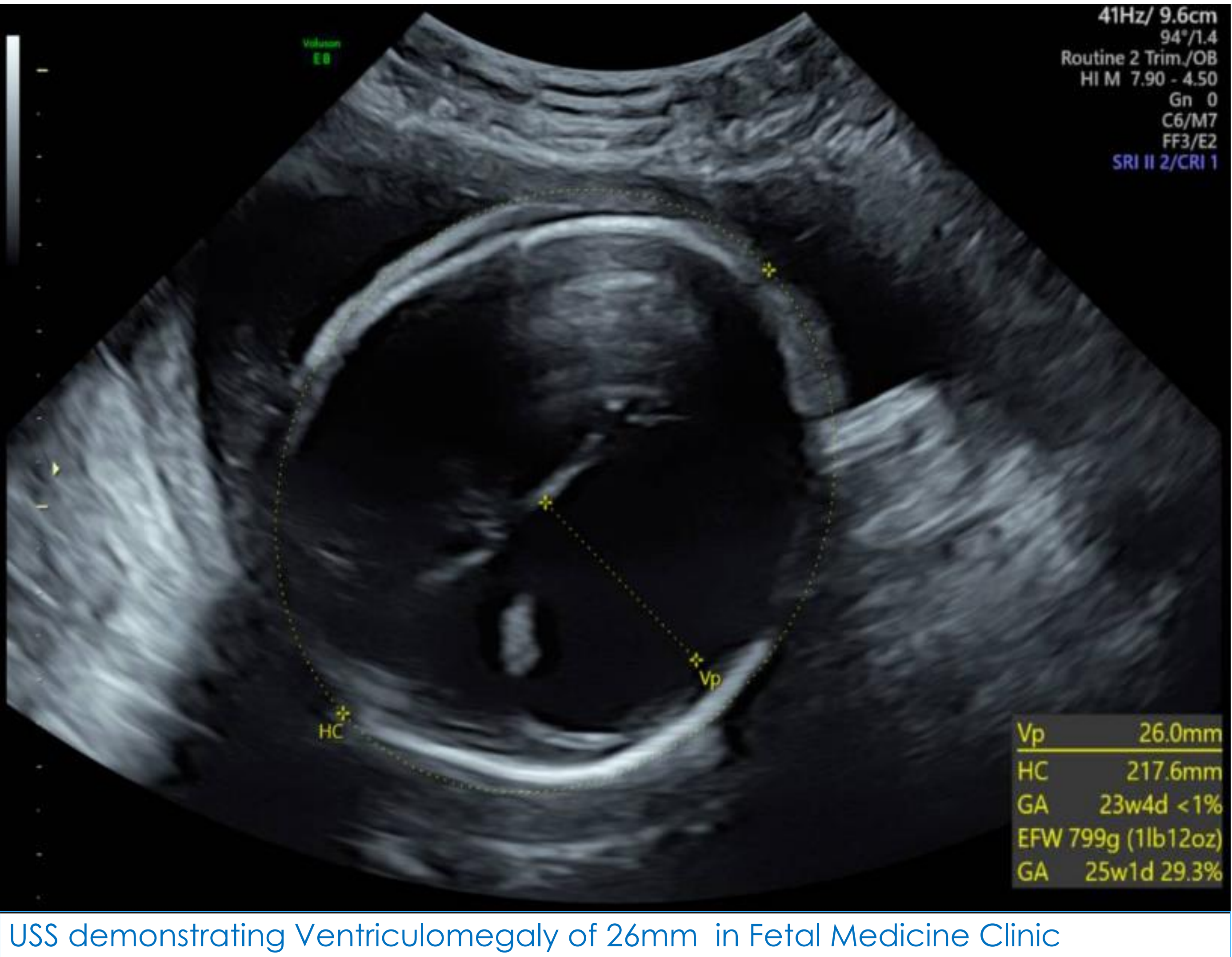
A non-English speaking 32 year old Slovakian woman was diagnosed with late onset poorly controlled T1DM nine months pre-conception. Three months prior to conception she was admitted to ITU with diabetic ketoacidosis (DKA) and was noted to have a HbA1c of 116.

The HbA1c was 94 at antenatal booking. Attendance to clinic appointments was poor and she had not seen a diabetologist in pregnancy. A translator was used for all appointments.

She had a further ICU admission at 18 weeks gestation with DKA. Fetal bradycardia was noted on admission which did resolve, however the duration of which is unknown.

Abnormal neuroanatomy was suspected on routine anomaly ultrasound scan at 19 weeks.

Fetal medicine clinic ultrasound showed the fetus had a small cerebellum, absent cavum septum pellucidum suggesting absent corpus callosum and mildly dilated lateral ventricles. PCR, microarray and exome sequencing showed no abnormality. Subsequent scans showed rapid progressive ventriculomegaly to 26mm and polyhydramnios with absent stomach. She subsequently went into preterm labour and delivered at 28 weeks gestation.



USS demonstrating Ventriculomegaly of 26mm in Fetal Medicine Clinic

Progressive Ventriculomegaly seen on USS	
Gestation	Lateral Ventricle (mm)
19+3	8.3
19+4	10.4
21+2	16.2
24+3	17.5
26+4	26

Discussion

We propose that the effects of poor glycaemic control and ketosis were contributing factors to the congenital abnormalities seen.

Congenital abnormalities are 2-3 times more likely in diabetic pregnancies than non-diabetics. It is well documented that blood glucose and ketones are teratogenic. Embryonal neural tissue takes up glucose equal to mother’s serum concentration at day 11-12 which may result in neural birth defects(1).

Hyperketonaemia has can be associated with cleft lip and palate, cardiovascular malformations, digestive and nervous system malformations including anencephaly and spina bifida(2). Sussman et al found that a ketogenic diet in mice caused a decrease of the cerebral cortex volume, corpus callosum and lateral ventricles of their offspring(3).

Acidosis adversely affects brain tissue and several studies have demonstrated this in labour. This effect may have been demonstrated in this case with ketoacidosis.

Conclusion

- We propose that maternal hyperglycaemia and ketoacidosis were the potential cause of this fetal abnormality.
- We would recommend further research into the impact of ketoacidosis on fetal brain tissue.
- The negative impact that socioeconomic status and language barriers can have on expectant mothers was also highlighted in this case.

References:
1. Bhandari J, et al. Diabetic Embryopathy. [Updated 2022 Aug 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan.
2. Qian M et al.. Effect of Elevated Ketone Body on Maternal and Infant Outcome of Pregnant Women with Abnormal Glucose Metabolism During Pregnancy. Diabetes Metab Syndr Obes. 2020 Nov 25;13:4581-4588. doi: 10.2147/DMSO.S280851. PMID: 33268998; PMCID: PMC7701151.
3. Sussman D,,et al. A gestational ketogenic diet alters maternal metabolic status as well as offspring physiological growth and brain structure in the neonatal mouse. BMC Pregnancy Childbirth. 2013 Oct 29;13:198. doi: 10.1186/1471-2393-13-198. PMID: 24168053; PMCID: PMC4231349.