

**PLACENTAL THROMBOTIC VASCULOPATHY WITH FETAL RENAL HEMORRHAGIC INFARCTION**

**FINAL DIAGNOSIS:** Third trimester fetal demise with placental pathology demonstrating fetal thrombotic vasculopathy – multiple placental foci of hemorrhagic endovasculitis and fetal villitis. Fetal autopsy demonstrating pre-mortem right renal thrombosis and hemorrhagic infarction with no other structural abnormalities of the kidneys found.

**HISTORY:** 31 week gestation referred for fetal MR based on outside ultrasound demonstrating an enlarged right kidney (image A) whose heterogeneous echotexture lacks corticomedullary differentiation and lacks discrete visualization of renal pyramids. Normal left kidney (image B) for comparison. Differential diagnosis at the time of presentation was mesoblastic nephroma, diffuse vascular insult, and Wilms tumor. One week prior to MR, outside ultrasound demonstrated fetal ascites that spontaneously resolved. Prior color Doppler ultrasound demonstrated a patent right renal artery. Normal karyotype.

**IMAGE A**



**IMAGE B**



Patient has two living children. Patient's most immediate prior pregnancy ended in a 29-week intrauterine fetal demise. Placental pathology at that time demonstrated placental thrombotic vasculopathy and umbilical cord stricture suggestive of pre mortem umbilical cord obstruction. Patient's coagulopathy workup is pending.

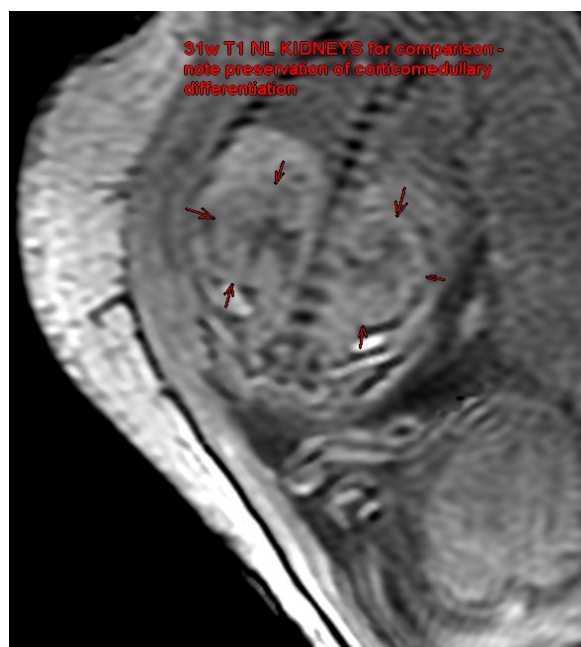
Image #1 is a coronal T1 pulse sequence (ps) of the right kidney demonstrating loss of cortical medullar differentiation and demonstrating multiple peripheral markedly dark (hyposignal intense) foci compatible with subacute blood by-products secondary to hemorrhagic infarction.

**IMAGE 1**



Image #2 is a normal coronal T1 ps gestational-age matched kidneys for comparison. Note normal corticomedullary differentiation and absence of any peripheral parenchymal markedly hyposignal intense foci.

**IMAGE 2**



The differential diagnosis for marked hypointense foci on T1 gradient-echo is subacute/chronic blood by-products, calcification and iron deposition. The marked darkness of these foci mitigates against fibrous dominant stromal foci of infiltration – a histologic feature of mesoblastic nephroma.

Image #3 is a sagittal T1 pulse sequence of the abnormal right kidney confirming in a second plane of section multiple hypointense foci.

**IMAGE 3**

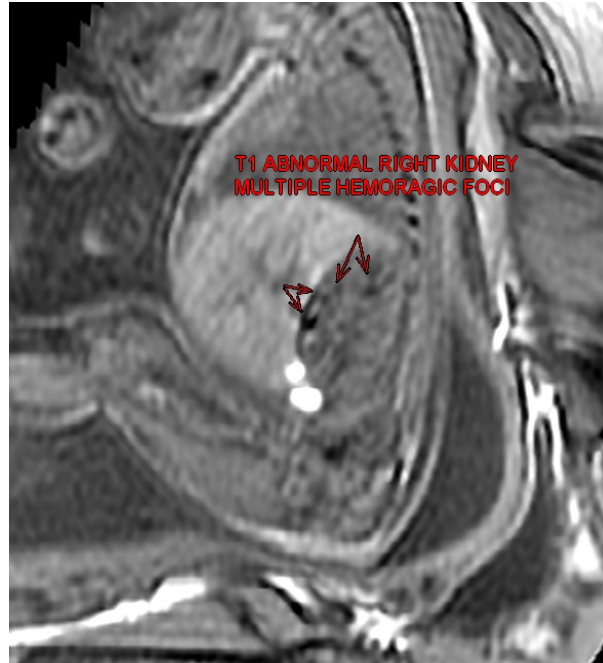


Image #4 demonstrates the spin echo T2 appearance of the abnormal right kidney, demonstrating heterogeneous loss of corticomedullary differentiation as well as persistence of multiple anterior peripheral dark foci compatible with the blood by-products accompanying hemorrhagic infarction.

**IMAGE 4**

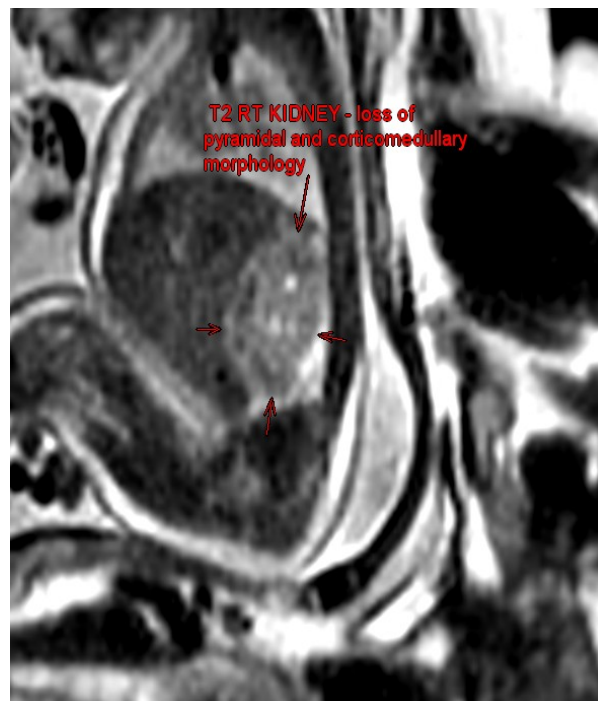


Image #5 is a gestational age matched normal T2 appearance of the kidneys for comparison. Note normal corticomedullary differentiation and note normal caliceal branching pattern.

**IMAGE 5**

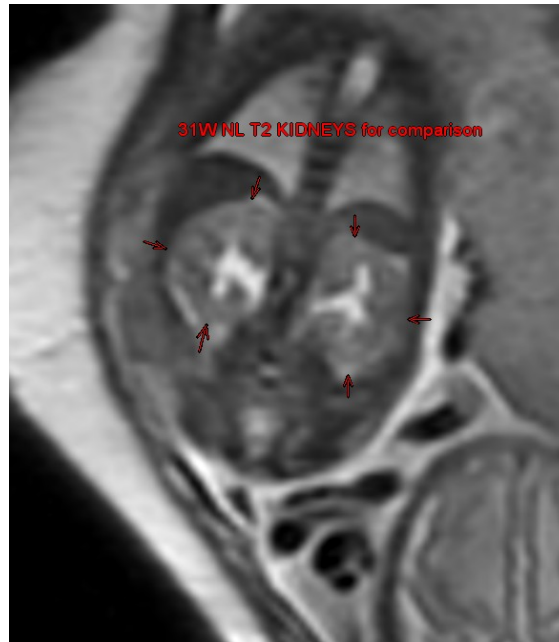


Image #6 is a gradient echo T1 sequence of the placenta demonstrating a 3 cm bright hypersignal intense focus compatible with intraplacental hemorrhage accompanying a thrombotic vasculopathy. The gradient echo T1 MR appearance of blood and blood by-products is variable given their age and the parenchyma in which they occur. Slow flow in veins along the chorionic and basal surfaces of the placenta appears as round and/or tubular bright signals.

**IMAGE 6**

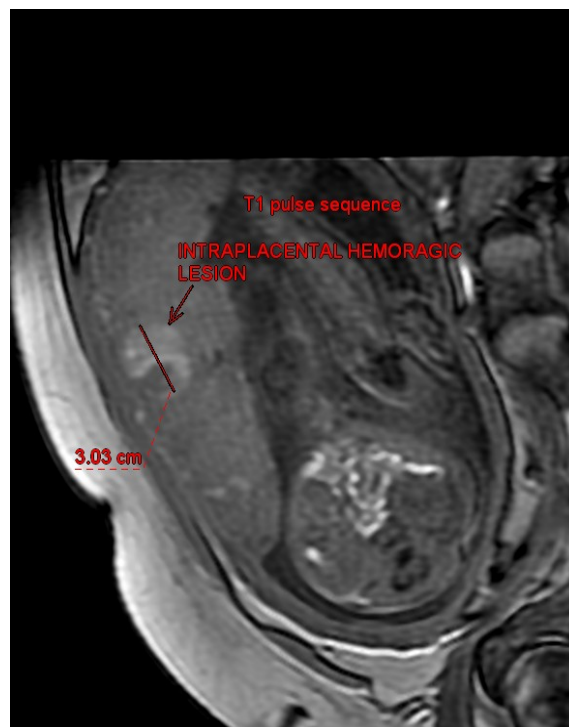


Image #7 demonstrates a second triangular site of placental hemorrhagic infarction.

### IMAGE 7

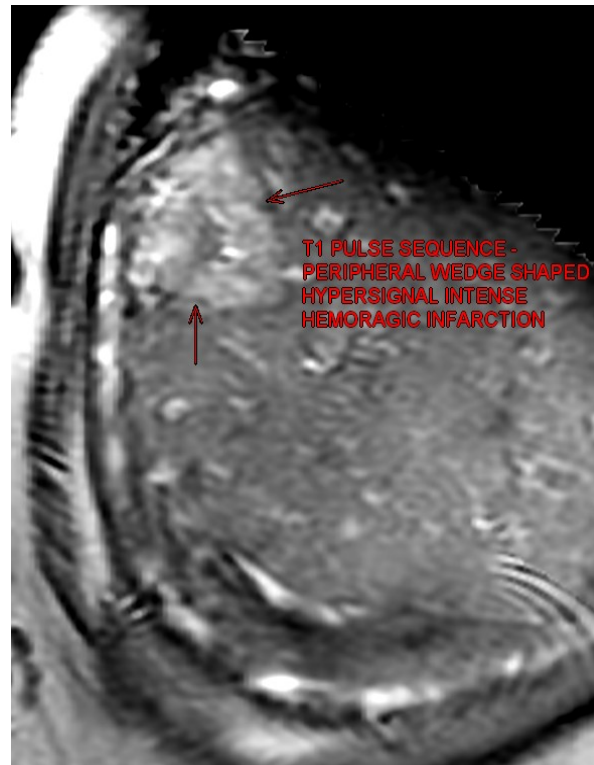


Image #8 is the corresponding T2 sequence. Note the significant diminution in conspicuity of the hemorrhagic infarction in the spin echo T2 sequence.

### IMAGE 8

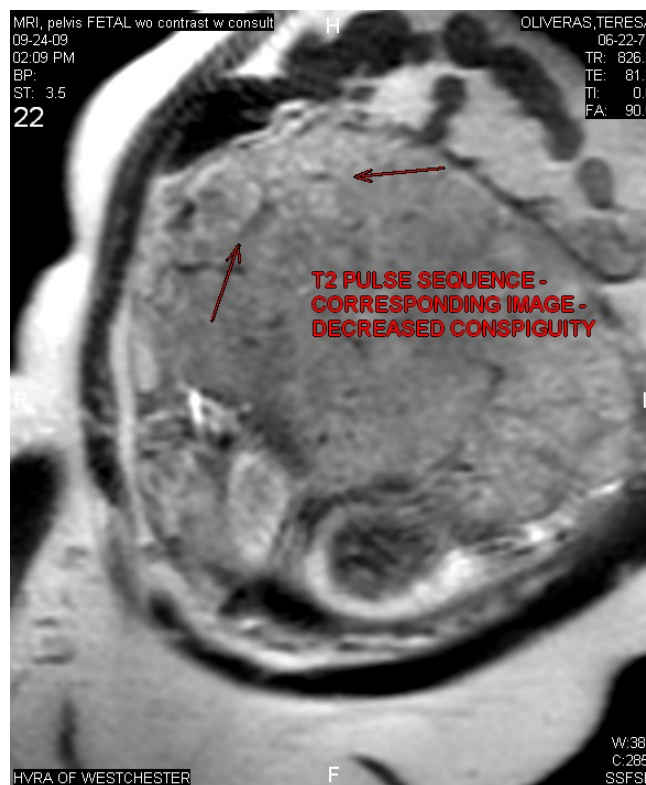


Image #9 is a spin echo T2 sequence demonstrating bilateral lower extremity subcutaneous edema that was not visible on ultrasound. This abnormality persisted on MR despite resolution of ascites on outside ultrasound.

### IMAGE 9



### DISCUSSION:

The current case focuses on the differential diagnosis and imaging evaluation of a unilateral enlarged kidney without dilatation nor cyst formation in a patient with a past obstetrical history of a third trimester demise whose placental pathology demonstrated a thrombotic vasculopathy.

The differential diagnosis for the enlarged abnormal right kidney is an unencapsulated infiltrative tumor such as mesoblastic nephroma versus an intrarenal parenchymal insulating process such as small vessel vasculopathy. The right renal artery and vein were reported as patent at time of ultrasound presentation. Wilms tumor – an encapsulated lesion - is considered most unlikely in the absence of a discrete mass.

### SUMMARY AND TEACHING POINTS:

This case exemplifies MR's uniquely sensitive and specific detection of infarction related blood by-products within the fetal kidney and placenta – not visible on ultrasound. MR's ability to detect intraparenchymal blood by-products is pulse sequence specific necessitating performance of the more time consuming and challenging gradient echo T1 pulse sequence. Blood and blood by-products in solid organs is poorly seen on the standard T2 single shot fast spin echo techniques upon which most fetal MR practitioners solely rely.

1. MR is a powerful tool to image fetal abdominal organs – its usefulness extends far beyond fetal CNS evaluation.
2. Gradient echo T1 sequence is essential for all fetal MR studies. The standard workhorse pulse sequence of fetal MR – the single shot fast spin echo T2 pulse sequence – poorly images blood and blood by-products and infarction patterns within solid organs, including the brain.
3. Consider fetal and placental MR in the evaluation of problematic pregnancies at risk for coagulopathy and vascular thrombotic disorders.
4. MR is more sensitive and specific in the detection of blood and blood by-products than ultrasound.
5. MR is more sensitive in the detection of subcutaneous edema than ultrasound – as in the current case, although ascites resolved on outside ultrasound, MR demonstrated persistent subcutaneous edema.

#### References:

Fetal Thrombotic Vasculopathy in the Placenta: Cerebral Thrombi and Infarcts, Coagulopathies and Cerebral Palsy. Kraus. Human Pathology. Vol. 30, Issue 7, pp 759-769. July 1999.

Placental FTV indicates a significant probability of thrombi in the fetus and represents an important, possibly under-recognized cause of perinatal mortality and neonatal injuries, especially cerebral palsy. Amongst 84 perinatal autopsies 16 (19%) exhibited FTV of the placenta. Six of the 16 demonstrated somatic thrombi involving cerebral, renal and pulmonary thrombotic emboli.

FTV: Clinical Significance of Extensive Avascular Villi. Redline. Human Pathology. Vol. 26, Issue 1, pp 80-85. January 1995.

Extensive avascular villi is associated with IUGR, acute and chronic monitoring issues, oligohydramnios, maternal coagulation disorders. Surviving neonates demonstrated major thrombotic events, umbilical artery PH less than 7.1, thrombocytopenia, transient hypoglycemia and increased nucleated red blood cell counts.

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