Letter to Editor

New Feature of Tuberous Sclerosis or Acute Periventricular Leukomalacia?

Dear editor:

We read with interest the article by Langer et al., describing the MRI findings in a neonatal patient with tuberous sclerosis.[1] They describe the presence of periventricular white matter lesions in the centrum semiovale, with linear, circular, and semicircular shapes, with hyperintense margins, and isointense centers, and better seen on T1-weighted and T1 FLAIR sequences than on T2-weighted sequences. They propose that these "target"-like lesions have not been previously reported in patients with tuberous sclerosis.

We believe that what Langer et al. are showing is unrelated to tuberous sclerosis itself, and represents additional classical MRI findings of acute periventricular leukomalacia and acute white matter injury.[2,3] These can occasionally occur in the term neonate as well. This child was imaged on the 7th day of birth, so the DWI and ADC abnormalities may have diminished by that time, as they normalize faster than in an adult. Also, if looked carefully, one can see subtle T2 hypointensities in the periventricular white matter in Figure 1C of Langer et al's paper (though the legends and figures for 1C and 1D are reversed), a feature also see in acute white matter injury. Given the location of the lesions, and depending on the severity of the injury, the MRI lesion may progress to cystic change, diminish in conspicuity or even stay the same for some time, and the child may or may not have gross symptoms later on. The apparent frontal white matter edema on T1 and T2-weighted images could potentially be just very nonmyelinated normal neonatal white matter, which will invariably appear more mature, and "less edematous" in the two month follow-up. We often see the findings of acute injury to the brain in patients referred to our large pediatric neuroradiology practice and a similar example of these focal white matter lesions in another term neonate without tuberous sclerosis is shown in Figure 1.

Overall, we believe that the above findings represent the presence of a disease process in addition to tuberous sclerosis, rather than a new finding of tuberous sclerosis.

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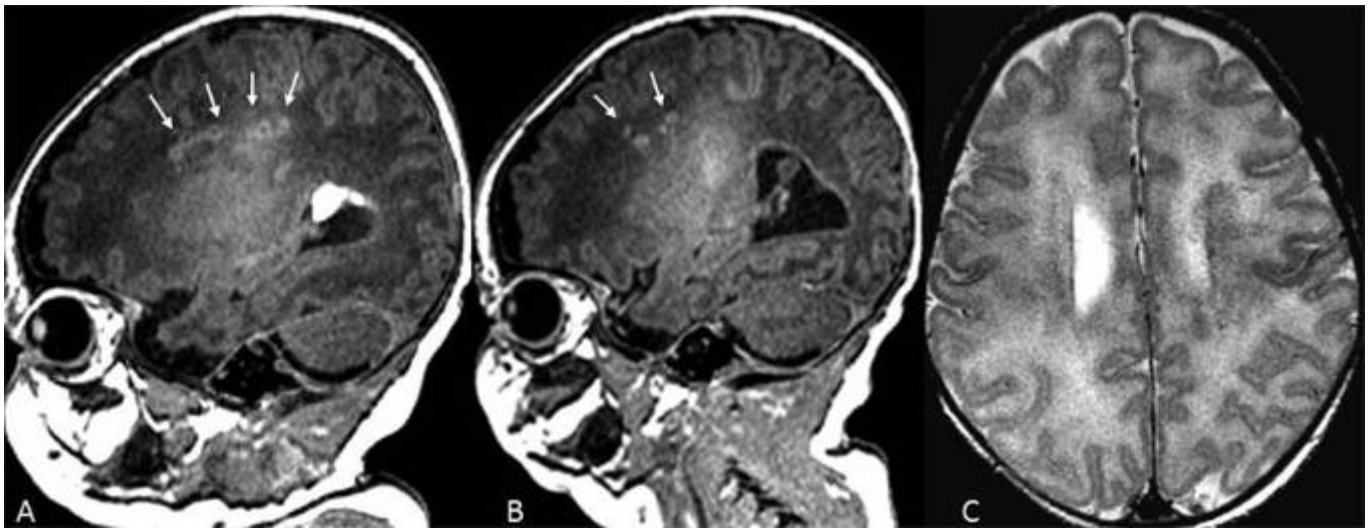

Fig. 1. Acute white matter injury and acute periventricular leukomalacia in a term neonate. **A and B.** Sagittal T1 weighted images show linear and circular small focal T1 hyperintensities in the periventricular white matter (arrows). There is also evidence of some intraventricular blood. **C.** T2-weighted images show subtle T2 shortening in the periventricular white matter, corresponding to the areas of T1 abnormalities.

References

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Authors Reply; Atypical Cerebral MRI Manifestations in Neonatal Tuberous Sclerosis

Dear editor:

We are gracious for the comments of Dr. Arastoo Vossough and Dr. Seyyed Ali Nabavizadeh. An acute periventricular leukomalacia is quite often seen in preterm infants and correlates with the prematurity. Peri-Ventricular Leukomalacia (PVL) is found in neonates after the 29th gestational week in a rate below 1%. In term babies, they should be well below 1%. This already yields a low probability of PVL in our case.

The case submitted under Figure 1 in your reply is a sagittal image of a quite typical PVL case with additional cortical lesions bilaterally along the central sulcus.[1,2] (Fig. 1 B). Our patient's history gave at least no indication of a perinatal hypoxia and as was mentioned in your reply, at the time of the examination no indication of an ischemia with diffusion-weighted imaging was present. Of course that does not exclude unrecognized prenatal asphyxia,[3] which would explain a normal DWI study. As you mentioned, 7 days postpartum is borderline for DWI and ADC to exclude ischemia for certain.[3-5] The findings in our case are not typical for PVL anyway.[1,2,4,6-11]

The subtle periventricular hypodensities in T2W images in our case are not characteristic for germinal matrix bleeding.[6]

I might mention that during the two-month observation time, the child did not develop neurologic deficits and the follow up MRI (2 months later) did not document the usual progression to defects as expected in PVL nor could bleeding retrospectively be ascertained.

In tuberous sclerosis, the white matter lesions are documented in the intrauterine stage[7,8,10] as well as in older children[9-11] and adults. More evidence of a quite high incidence of occult white matter lesions is documented with tensor imaging. These lesions must have precursors. In the very early postnatal time, only a very limited number of publications are available on MRI in TSC patients.

Last, not least, every case report has the inherited disadvantage of bad statistics and is still a valuable contribution in rare diseases. We thank Dr. Arastoo Vossough and Dr. Seyyed Ali Nabavizadeh for their contribution.

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