

15. Periventricular Leukomalacia

Periventricular leukomalacia (PVL) is the most common ischemic brain injury of prematurity, occurring in up to one-fourth of these infants. Sequelae include cerebral palsy and mental retardation. PVL results from watershed hypoperfusion which progresses to infarction. Premature infants have collateral circulation between the meninges and cerebral arteries which protect the cortex from such infarction. Thus, the periventricular white matter, often specifically that adjacent to the atrial trigone or frontal horn, is most frequently involved. This produces a pattern of ischemia similar to that of chronic small vessel disease of the elderly but distinct from that of severe perinatal asphyxia (hypoxic ischemic injury) which involves the brainstem and deep (depths of sulci) subcortical white matter. MRI is rarely used acutely in pre-term infants suspected of PVL, but may easily detect mild to moderate disease missed on ultrasound. DWI, in particular, can be extremely sensitive for the detection of acute PVL. A follow-up MRI in symptomatic infants often confirms the diagnosis, demonstrating areas of increased SI on T2WI and FLAIR scans (Fig. 15.1 A) correlating with gliosis. Care must be taken to distinguish pathologic edema from the normal high water content of white matter, particularly in the occipital-parietal periventricular white matter where terminal zones of myelination exist through the first two decades of life. PVL may be further distinguished by the presence of ex vacuo dilatation resulting from white matter loss. Figure 15.1 B demonstrates a common appearance of such dilatation, showing the enlarged, somewhat irregular posterior aspects of the lateral ventricles encroaching abnormally closely to the cortical gyri and sulci. Marked callosal thinning may also be apparent on sagittal images. Chronically, the lesions of PVL may progress to cystic cavitation manifesting as high SI on T2WI.

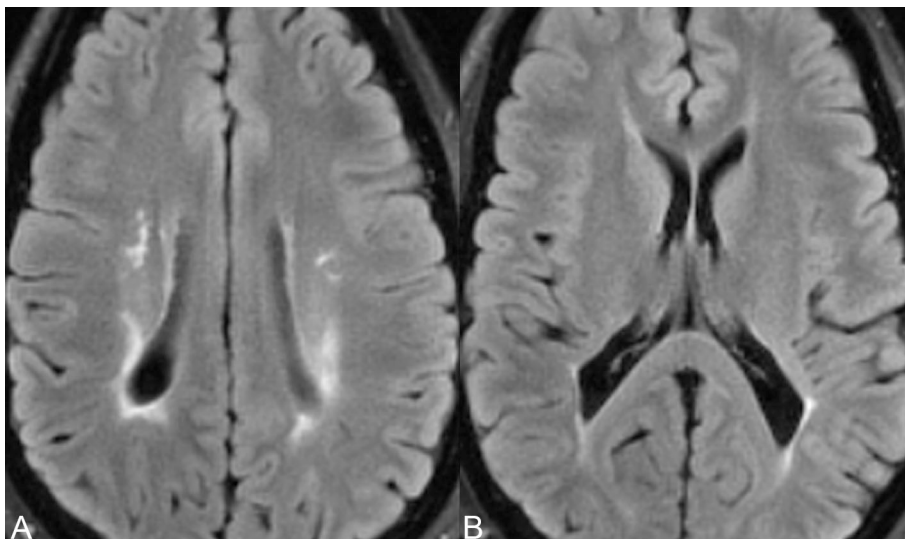


Fig. 15.1