Periventricular-intraventricular hemorrhage etiology

Perinatal brain injury may lead to long-term morbidity and neurodevelopmental impairment. Improvements in perinatal care have resulted in the survival of more infants with perinatal brain injury. The effects of hypoxia-ischemia, inflammation, and infection during critical periods of development can lead to a common pathway of perinatal brain injury marked by neuronal excitotoxicity, cellular apoptosis, and microglial activation [1)]

Infants most at risk are those born before 33 weeks of gestational age, as after this time, the germinal matrix involutes.

The highly vascular germinal matrix is part of the primordial tissue of the developing brain and is the source of future neurons and glial cells. It is located just beneath the ependymal lining of the lateral ventricles, and undergoes progressive involution until 36 weeks gestational age (GA). Thus, the matrix may persist out of utero in premature infants. A disproportionate amount of the total CBF perfuses the periventricular circulation through these capillaries which are immature and fragile and have impaired autoregulation [2) 3)].

1)
Novak CM, Ozen M, Burd I. Perinatal Brain Injury: Mechanisms, Prevention, and Outcomes. Clin Perinatol. 2018 Jun;45(2):357-375. doi: 10.1016/j.clp.2018.01.015. Epub 2018 Mar 21. PMID: 29747893.
2)
Lou HC, Lassen NA, Friis-Hansen B. Impaired Autoregulation of Cerebral Blood Flow in the Distressed Newborn Infant. J Pediatr. 1979; 94:118– 121
3)
Milligan DWA. Failure of Autoregulation and Intraventricular Hemorrhage in Preterm Infants. Lancet. 1980; 1:896–898