Periventricular-intraventricular hemorrhage

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Periventricular-intraventricular hemorrhage was first discovered by Abraham Towbin in 1968 [1)]

General information

This occurs primarily in premature infants. Alternate terms: subependymal hemorrhage (SEH), germinal matrix hemorrhage (GMH), periventricular-intraventricular hemorrhage (PIVH). Intraventricular hemorrhage (IVH) arises from the extension of SEH through the ependymal lining of the ventricle and occurs in 80% of cases of SEH [2)].

This intraventricular hemorrhage (IVH) is a common complication of preterm neonate.

Germinal matrix is a fragile portion of the brain that may be damaged leading to an intracranial hemorrhage known, which is the most common cause of second trimester spontaneous abortions.

Epidemiology

Periventricular-intraventricular hemorrhage Epidemiology

Classification

see Papile-Burstein classification

Etiology

see Periventricular-intraventricular hemorrhage etiology.

Pathogenesis

see Periventricular-intraventricular hemorrhage pathogenesis.

Risk Factors

see Periventricular-intraventricular hemorrhage Risk Factors

Clinical features

see Periventricular-intraventricular hemorrhage clinical features.

Diagnosis

see Periventricular-Intraventricular Hemorrhage Diagnosis.

Differential diagnosis of ventriculomegaly in PIVH

When ventriculomegaly is detected, it needs to be di erentiated from the following:

1. transient ventriculomegaly: occurs in the first few days after PIVH. This may not cause elevated ICP. As implied, it is self limited

2. progressive ventriculomegaly: occurs in 20–50%of cases (true hydrocephalus)

3. “hydrocephalus ex vacuo”: due to loss of brain tissue or maldevelopment. Is not progressive on serial U/S. OFCs may fall below normal due to lack of growing brain as stimulus for head growth

Treatment

see Periventricular-intraventricular hemorrhage treatment.

Complications

Periventricular-intraventricular hemorrhage complications.

Outcome

see Periventricular-intraventricular hemorrhage outcome.

Retrospective observational studies

Using the 2016-2019 National Inpatient Sample database, newborns with periventricular-intraventricular hemorrhage diagnosis. were identified using ICD-10-CM codes. Patients were stratified based on race. Patient characteristics and inpatient outcomes were assessed. Multivariate logistic regression analyses were used to identify the impact of race on extended LOS and exorbitant cost.

Of 1435 patients, 650 were White (45.3%), 270 African American (AA) (18.8%), 300 Hispanic (20.9%), and 215 Other (15.0%). A higher percentage of AA and Other patients than Hispanic and White patients were < 28 days old ($p = 0.008$). Each of the cohorts had largely similar presenting comorbidities and symptoms, although AA patients did have significantly higher rates of NEC ($p < 0.001$). There were no observed differences in rates of AEs, rates of mortality, mean LOS, or mean total cost of admission. Similarly, on multivariate analysis, no race was identified as a significant independent predictor of extended LOS or exorbitant cost.

The study found that in newborns with IVH, race is not associated with proxies of poor healthcare outcomes like prolonged LOS or excessive cost. Further studies are needed to validate these findings [3)].

Case series

Periventricular-intraventricular hemorrhage case series.

Experimental research studies

GMH/PHH was induced in 4-day-old mice using collagenase, blood, or blood serum injections. PHH severity was characterized 2 weeks later using magnetic resonance, immunofluorescence, and protein expression quantification with mass spectrometry. Ependymal restoration and wall regeneration after stem cell treatments were tested in vivo and in an ex vivo experimental approach using ventricular walls from mice developing moderate and severe GMH/PHH. The effect of the GMH environment on EpP differentiation was tested in vitro. Two-tailed Student t or Wilcoxon-Mann-Whitney U test was used to find differences between the treated and nontreated groups. ANOVA and Kruskal-Wallis tests were used to compare >2 groups with post hoc Tukey and Dunn multiple comparison tests, respectively.

Results: PHH severity was correlated with the extension of GMH and ependymal disruption (means, 88.22% severe versus 19.4% moderate). GMH/PHH hindered the survival rates of the transplanted neural stem cells/EpPs. New multiciliated ependymal cells could be generated from transplanted neural stem cells and more efficiently from EpPs (15% mean increase). Blood and TNFα (tumor necrosis factor alpha) negatively affected ciliogenesis in cells committed to ependyma differentiation (expressing Foxj1 [forkhead box J1] transcription factor). Pretreatment with mesenchymal stem cells improved the survival rates of EpPs and ependymal differentiation while reducing the edematous (means, 18% to 0.5% decrease in severe edema) and inflammatory conditions in the explants. The effectiveness of this therapeutical strategy was corroborated in vivo (means, 29% to 0% in severe edema).

Conclusions: In GMH/PHH, the ependyma can be restored and edema decreased from either neural stem cell or EpP transplantation in vitro and in vivo. Mesenchymal stem cell pretreatment improved the success of the ependymal restoration [4].

Animal models are needed to better understand the pathophysiology of IVH and test pharmacological treatments. While there are existing models of neonatal IVH, those that reliably result in hydrocephalus are often limited by the necessity for large-volume injections, which may complicate modeling of the pathology or introduce variability in the clinical phenotype observed. Recent clinical studies have implicated hemoglobin and ferritin in causing ventricular enlargement after IVH. Miller et al. developed a straightforward animal model that mimics the clinical phenotype of PHH utilizing small-volume intraventricular injections of the blood breakdown product hemoglobin. In addition to reliably inducing ventricular enlargement and hydrocephalus, this model results in white matter injury, inflammation, and immune cell infiltration in periventricular and white matter regions [5]

References

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