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Bronchopulmonary Dysplasia Remains a Significant Challenge in Neonatal Care

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Abstract

Bronchopulmonary dysplasia (BPD) is a common respiratory morbidity among premature infants. Nissen fundoplication may be performed on infants with BPD to protect the lungs from gastroesophageal reflux-related aspiration, but the indications and benefits associated with fundoplication are not well-defined. This study evaluated associations of Nissen with clinical outcomes in infants with severe BPD (sBPD), using propensity score matching to minimize bias and confounding. Bronchopulmonary dysplasia (BPD) is the most common pulmonary complication in preterm infants, where immature lungs are damaged by prolonged oxygen therapy and mechanical ventilation.

Keywords: Prematurity; Chronic Lung Disease; Oxygen Therapy; Alveolar Damage

Introduction

leading to inflammation, scarring, and abnormal lung development. BPD might result in persistent pulmonary hypertension (PPHN), asthma, chronic obstructive pulmonary disease, or neurodevelopmental impairment.6 One reason for this vulnerability is that preterm infants have notably immature lungs, making them susceptible to injury during caregiving. Multiple factors may disrupt alveolar growth and contribute to the pathogenesis of BPD, including prenatal infections, fetal growth restriction, patent ductus arteriosus, neonatal pneumonia/sepsis, and respiratory distress syndrome. Studies have also explored the association between infant characteristics or clinical treatment in neonatal intensive care units (NICUs)—such as gestational age (GA), birth weight, gender, APGAR score, oxygen toxicity, respiratory parameters, and mechanical ventilation—and BPD development.

Discussion

The pathogenesis of BPD is complex and multifactorial and also involves both genetic and environmental factors. However, the aforementioned factors are not sufficiently specific to guide clinicians in developing interventions for preventing BPD development. Accordingly, the present study was conducted to explore some specific and measurable factors in the NICU, including bradycardia episodes (BEs), hypoxemia episodes (HEs), and intubation within 3 days of birth, and their effects on BPD. Bronchopulmonary dysplasia (BPD) remains a significant challenge in neonatal care, the pathogenesis of which potentially involves altered lipid metabolism. Given the critical role of lipids in lung development and the injury response, we hypothesized that specific lipid species could serve as therapeutic agents in BPD [1-4].

Brain structural networks were constructed utilizing automated anatomic labeling mapping by tracing the fibers between each pair of regions in individual space. We calculated network metrics such as global efficiency, local efficiency, clustering coefficients, characteristic path length, and small-worldness. Then we compared the network metrics of these infants with those of 57 healthy term infants of comparable postmenstrual age at magnetic resonance imaging scan. Finally, network-based statistics was used to analyze the differences in brain network connectivity between the groups with and without BPD. Bronchopulmonary dysplasia (BPD) is a multifactorial disease with many associated co-morbidities, responsible for most cases of chronic lung disease in childhood. The use of imaging exams is pivotal for the clinical care of BPD and the identification of candidates for experimental therapies and a closer follow-up. Imaging is also useful to improve communication with the family and objectively evaluate the clinical evolution of the patient's disease. BPD imaging has been classically performed using only chest X-rays, but several modern techniques are currently available, such as lung ultrasound, thoracic tomography, magnetic resonance imaging and electrical impedance tomography. These techniques are more accurate and provide clinically meaningful information. We reviewed the most recent evidence published in the last five years regarding these techniques and analyzed their advantages and disadvantages. In the NICU, bronchopulmonary dysplasia (BPD) is a concerning common respiratory complication in preterm and low birth-weight infants. Clinical studies have confirmed that human milk has an important nutritional role for children with BPD, therefore, dentification of beneficial components in human milk that prevent BPD is urgently needed. Our previous work showed that human milk exosomes (HM-Exos) could inhibit apoptosis of alveolar type II epithelial cells (AT II), and the circular RNA (circRNA)-circABPD1 were highly expressed in preterm colostrum milk exosomes. Exosomes transport circRNAs that are stable and may exert anti-inflammatory and immune effects attracted the attention of researchers, but the role and mechanism of human milk exosome-derived circABPD1 in BPD remains unclear. Here, we constructed BPD in vivo and in vitro models through exposure to hyperoxia, verified the effect of circABPD1 and revealed its mechanism through rescue experiments. We found that circABPD1 had circRNA properties, and overexpression of circABPD1 could improve reduced alveolar number, enlarged the alveolar linear intercept in vivo models of BPD, promote cell proliferation, reduce oxidative stress levels and alleviate lung epithelial cell damage in vivo and in vitro models [5,6].

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These results suggest that circABPD1 can improve the pathologoical changes of bronchopulmonary dysplasia, promote cell proliferation, inhibit oxidative stress level, and alleviate lung injury by targeting the miR-330–3p/HIF1α axis, which provides a new idea for the prevention and treatment of bronchopulmonary dysplasia. Bronchopulmonary dysplasia (BPD) is the leading cause of chronic lung disease in infants and the commonest complication of prematurity. Advances in respiratory and overall neonatal care have increased the survival of extremely low gestational age newborns, leading to the continued high incidence of BPD. Pulmonary hypertension (PH) represents the severe form of the pulmonary vascular disease associated with BPD, and affects almost one-third of infants with moderate to severe BPD. PH responds suboptimally to pulmonary vasodilators and increases morbidity and mortality in BPD infants. An up-to-date knowledge of the pathogenesis, pathophysiology, diagnosis, treatment, and outcomes of BPD-PH can be helpful to develop meaningful and novel strategies to improve the outcomes of infants with this disorder. Therefore, our multidisciplinary team has attempted to thoroughly review and summarize the latest advances in BPD-PH in preventing and managing this morbid lung disorder of preterm infants. Bronchopulmonary dysplasia (BPD) is a common pulmonary injury among premature infants, which is often caused by hyperoxia exposure. Irisin is a novel hormone-like myokine derived mainly from skeletal muscles as well as adipose tissues. Many studies have indicated that Irisin a variety of properties against hyperoxia-induced inflammation and oxidative stress (OS).

Conclusion

Peripheral blood mononuclear cells were isolated from whole-blood samples obtained within a defined timeframe. Subsequently, mtDNA extraction and sequencing using next-generation sequencing technology were performed to identify mtDNA gene mutations. Among the cohort of ten extremely preterm infants with BPD, mtDNA sequencing revealed the presence of mutations in seven patients, resulting in a total of twenty-one point mutations. Notably, many of these mutations were identified in loci associated with critical components of the respiratory chain complexes, vital for proper mitochondrial function and cellular energy production. These findings suggest a potential association between mitochondrial dysfunction and the pathogenesis of BPD. Further extensive investigations are warranted to unravel the mechanisms underlying mtDNA mutations in BPD.

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