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Background and aims: Bronchopulmonary dysplasia (BPD) is one of the most important chronic complications of premature birth. Although long term effects of BPD are more commonly known by the well-defined pulmonary consequences, cardiovascular sequelae related to BPD have also been reported. In the post-surfactant era data on the cardiovascular changes in new BPD patients is limited. In this study we aimed to investigate the role of myocardial tissue Doppler echocardiography in detecting cardiac pathology in pre-school BPD patients and to find out possible risk factors related to cardiovascular sequela.

Methods Prematurely born children with BPD (N=21, 4 severe BPD, 3 moderate BPD and 14 mild BPD) and without BPD (N=20) at 2 to 4 years of age were enrolled to the study. Conventional and myocardial tissue Doppler echocardiography studies were performed.

Results In conventional echocardiography; right ventricular fractional shortening, tricuspid E/A ratio, mitral late diastolic inflow velocity and pulmonary acceleration time were decreased; mitral E/A ratio, left and right ventricular myocardial performance indexes were increased in BPD group compared to controls. In myocardial tissue Doppler measurements; tricuspid annulus E'/A' ratio was decreased and interventricular septum systolic velocity was increased in BPD group. Low birth weight, disease severity and postnatal cumulative steroid dosage were related with echocardiographic changes.

Conclusion BPD affects global cardiac performances up to 2 to 4 year of age with regard to birth weight, disease severity and cumulative steroid dosage. Myocardial tissue Doppler examination did not have additional value to conventional echocardiography in demonstration of these changes.

600 EVALUATION OF INFLAMMATION IN BRONCHOPULMONARY DISEASE WITH TRANSCUTANEOUS CARBOXYHEMOGLOBIN MEASUREMENT- PRELIMINARY RESULTS

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Background and Aims Bronchopulmonary dysplasia (BPD) is an important chronic respiratory morbidity of premature infants. Increased carboxyhemoglobin (COHb) levels have been reported for chronic obstructive pulmonary disease, systemic inflammatory response syndrome and acute respiratory distress syndrome and also for mortality in premature infants. COHb levels increases as a result of oxidative stress and inflammation. Changes of COHb levels by the measurement of transcutaneous COHb levels may be informative for continuing inflammation levels of BPD. We aimed to evaluate inflammatory process in BPD with transcutaneous COHb.

Methods Twenty premature infants discharged from Ege University NICU with the diagnosis of BPD (Group 1), 20 premature infant without BPD (Group 2), 20 term healthy control (Group 3) infant were included in the study. Transcutaneous COHb levels were measured with Masimo radical set device following three months after discharge. Antenatal and neonatal characteristics of infants were recorded.

Results Mean transcutaneous COHb levels were significantly higher in group 1 than group 2 (p=0.000) at postnatal age 0. Mean SpCO values after first three months of discharge were higher in group 1 than group 2 (p<0.05) and group 3 (p<0.001). No difference was detected in the same groups' (Group 1 and 2) consequent measurements of SpCO.

Conclusion Our data support the ongoing persistent chronic inflammatory process after discharge in infants with BPD. The long term multisystemic morbidity, inflammatory mass could be minimized with early diagnosis and preventive treatments. Further investigations are needed in larger populations for early prediction of BPD among the risk group.

601 DOES BRONCHOPULMONARY DYSPLASIA RELATE TO REDOX STATUS IN INFANTS LESS THAN 29 WEEKS OF GESTATIONAL AGE?

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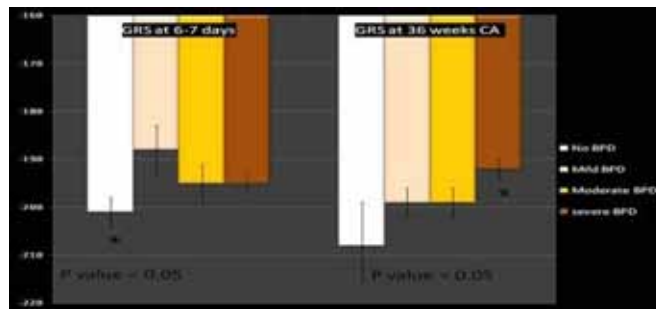
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Background Glutathione is the key molecule in detoxification of peroxides leading to an oxidized glutathione redox status (GRS). We hypothesizes that GRS plays an important role in the etiology of bronchopulmonary dysplasia (BPD).

Objective To test the relation between GRS at 6–7 days of life as well as at 36 weeks of corrected age and BPD. To identify perinatal factors affecting GRS.

Design/methods Whole blood GRS was measured at 6–7 days of life and at 36 weeks of corrected age (CA) in 51 infants less than 29 weeks of gestational age (GA). Perinatal clinical data that may affect the GRS were collected. The GRS was calculated using concentration of GSH and GSSG according to the Nernst equation (Schafer & Buettner, 2001).

Results Infants in our cohort had gestational age of 26±1 weeks with birth weight of 847±166 gm. Significant relation between GRS and BPD was confirmed with less risk of BPD in infants with most reduced GRS (day 6–7) and higher risk of BPD for infants with most oxidized GRS at 36 weeks CA.



Abstract 601 Figure 1 Relation between GRS and BPD

GA and BW were significantly related to GRS.

Abstract 601 Table 1 Different perinatal factors effect on GSR

	Test used	P value for GRS at 6–7 days	P value at 36 CA
Gestational age	Pearson correlation	0.01	0.08
Birth weight	Pearson correlation	0.04	0.93
Sex	T-test	0.47	0.89
Maternal preeclampsia	T-test	0.37	0.35
Chorioamnionitis	T-test	0.15	0.98
Small for gestational age	T-test	0.8	0.77

Conclusions There is a significant relation between GRS 6–7 days of life as well as at 36 weeks CA and BPD outcome in infants less than 29 weeks of GA. The significant impact of both GA and BW on GRS at 6–7 days of life is explained by the glutathione level that is correlated with gestational age.