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Study of the relation between biliary cirrhosis and hepatopulmonary syndrome and its reversibility among young rats submitted to common bile duct ligation

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Introduction: Biliary atresia is an important cause of childhood cirrhosis, accounting for most liver transplants at this age. Liver disease causes different organic alterations. Regarding the lungs, cirrhosis is associated to hepatopulmonary syndrome, which relates to increase in morbidity and mortality. This study aims to clarify the relation between biliary cirrhosis and onset of hepatopulmonary syndrome, while further analyzing its physiopathology.

Material and methods: 21-day-old Wistar rats were submitted to common bile duct ligation and allocated into groups A1-A5 and B1-B5. Animals in group A were sacrificed 2, 3, 4, 5 or 6 weeks after biliary obstruction, while those in group B were submitted to biliodigestive anastomosis 2, 3, 4, 5, or 6 weeks after the first procedure and sacrificed 3 weeks later. At the time of sacrifice, arterial blood was collected for analyses and samples from liver and lungs for histological and molecular analyzes. Liver and lung tissue were stained with hematoxylin-eosin. Gasometric parameters and expression in lung tissue of eNOS, NOS and endothelin were studied.

Results: We verified increased duct proliferation in liver tissue and reduced alveolar area in lung tissue, as passed the time from biliary obstruction. From the total of 42 blood samples, 15 showed hypoxia ($pO_2 < 85\text{mmHg}$) and 17 showed increased oxygen gradient ($p(A-a)O_2 > 18\text{mmHg}$), with no clear correlation with time of obstruction or any of the molecular mediators analyzed.

Conclusion: The present model is effective for producing lung injury, inflammation, and showed worsening of lung tissue alterations with biliary obstruction, without revealing clear effects of biliodigestive anastomosis. Thus, further studies are necessary.

Keywords: Hepatopulmonary syndrome; Biliary cirrhosis, Experimental animal model; Bile duct obstruction.