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MiR-200a/ZEB1 Pathway in Liver Fibrogenesis of Biliary Atresia

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Abstract : Objective: Biliary atresia (BA) is characterized by progressive liver fibrosis. Epithelial-mesenchymal transition (EMT) has been implicated as a key mechanism in the pathogenesis of organ fibrosis. MiR-200a has been shown to repress EMT. We aim to explore the role of miR-200a in the fibrogenesis of BA. Methods: We obtained the plasma samples and liver samples from patients with BA or controls to examine the role of miR-200a. Histological liver fibrosis was assessed using the Ishak fibrosis scores. Reverse transcription quantitative polymerase chain reaction (RT-qPCR) was performed to detect the expression of miR-200a in plasma. We also evaluated the expression of miR-200a in liver tissues using tyramide signal amplification fluorescence in situ hybridization (TSA-FISH). The expression of EMT related proteins zinc finger E-box-binding homeobox 1 (ZEB1), E-cadherin and α-smooth muscle actin (α-SMA) in the liver sections were detected by immunohistochemical staining. Results: We found that the expression of miR-200a was both elevated in the plasma and liver tissues from BA patients compared with the controls. The hepatic expression of ZEB1 and α-SMA were markedly increased in the liver sections from BA patients compared to the controls, whereas E-cadherin was downregulated in the BA group. Simultaneously, we noted that the hepatic expression of miR-200a, E-cadherin and α-SMA were upregulated with the progression of liver fibrosis in the BA group, while ZEB1 was downregulated with the progression of liver fibrosis in BA patients. Conclusion: These findings suggest EMT has a critical effect on the fibrotic process of BA, and the interaction between miR-200a and ZEB1 may regulate EMT and eventually influence liver fibrogenesis of BA.

Keywords : biliary atresia, liver fibrosis, MicroRNA, epithelial-mesenchymal transition, zinc finger E-box-binding homeobox 1
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