



Clinical analysis of 36 cases of hepatopulmonary syndrome

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Abstract

AIM: To analyse the causes and pathogenesis of Hepatopulmonary Syndrome (HPS) in patients with liver cirrhosis.

METHODS: Femoral artery blood samples of 102 cases with liver cirrhosis were taken right after their admission to the hospital to detect the PaO₂ and SaO₂.

RESULTS: PaO₂ and SaO₂ in patients with HPS were markedly lower than that without HPS ($P < 0.01$); PaO₂ and SaO₂ in patients with a liver function of child C were markedly lower than that of child A

($P < 0.01$); HPS incidence in patients with liver function of child C was markedly higher than that with child A ($P < 0.01$); The average PaO₂ and SaO₂ in patients with pylephlebotasis (> 1.4 in diameter) were markedly lower than that without pylephlebotasis ($P < 0.01$); HPS incidence in patients with pylephlebotasis was markedly higher than that without pylephlebotasis ($P < 0.001$); PaO₂ and SaO₂ in patients with cutaneous spider nevi were markedly lower than that without cutaneous spider nevi ($P < 0.05$); HPS incidence in patients with cutaneous spider nevi was markedly higher than that without cutaneous spider nevi ($P < 0.001$).

CONCLUSION: HPS might occur in any stage of patients with liver cirrhosis. The more severely the liver function was damaged, the more chances there were to suffer from HPS, and the more severe the symptoms would be.

Key words: Liver cirrhosis; Lung diseases/physiopathology; Lung diseases/diagnosis; Syndrome

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