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血小板源性生长因子受体在实验性蛛网膜下腔损伤中的作用 (PDF)

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Title: Role of platelet-derived growth factor receptor in early brain injury following subarachnoid hemorrhage

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关键词: [蛛网膜下腔出血](#); [早期脑损伤](#); [血小板源性生长因子受体](#); [凋亡](#); [脑水肿](#)

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摘要: 目的 通过研究大鼠蛛网膜下腔出血(subarachnoid hemorrhage, SAH)后脑水肿及细胞凋亡情况,初步探讨血小板源性生长因子受体(platelet-derived growth factor receptor, PDGFR)参与早期脑损伤的可能机制。方法 采用随机数字表法将成年雄性SD大鼠分配至假手术组、SAH组、SAH+Imatinib组、SAH+PDGF-BB组。Imatinib为PDGFR拮抗剂,PDGF-BB为其激动剂,每组18只。采用血管内丝线穿刺颈内动脉分叉处的方法,建立SAH模型。SAH+Imatinib组大鼠在建模前1 h,行腹腔注射Imatinib处理;SAH+PDGF-BB组大鼠建模前1 h,行侧脑

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室注射PDGF-BB处理。处理后24 h, 对各组动物的死亡率、神经功能学评分和脑水含量指标进行研究; RT-PCR、Western blot检测海马组织Caspase-3的表达。 结果 SAH+PDGF-BB组大鼠死亡率、脑组织含水量最高, 神经行为功能学评分最低 ($P<0.01$); SAH+Imatinib组与SAH组比较, 大鼠死亡率、脑组织含水量有所下降, 神经行为功能学评分有所提高 ($P<0.01$); 与其他组比较, SAH+PDGF-BB组大鼠海马组织Caspase-3 mRNA表达最高 ($P<0.01$), SAH+Imatinib组与SAH组比较, 海马组织Caspase-3 mRNA表达有所下降 ($P<0.01$); Western blot检测趋势与RT-PCR检测结果一致。 结论 PDGFR参与SAH后早期脑损害, 其拮抗剂Imatinib能在一定程度上减轻早期脑损伤。

Abstract: Objective To observe cephaloedema and cell apoptosis in rats following subarachnoid hemorrhage (SAH), and to explore the mechanism of platelet-derived growth factor receptor (PDGFR) involving in the development of early brain injury. Methods Adult male SD rats were randomly divided into 4 groups including a sham operation group, a SAH group, a SAH+imatinib group, and a SAH+PDGF-BB group ($n=18$). SAH mouse models were established by using intravascular silk puncture of the internal carotid artery crotch. The rats of the SAH+imatinib group were given an intraperitoneal injection of imatinib at 1 h before modeling, while those of the SAH+PDGF-BB group were given an intracerebroventricular injection of PDGF-BB at 1 h before modeling. After treatment for 24 h, the mortality, water content in brain and neurological function score were determined. Caspase-3 expression in hippocampus was detected by RT-PCR and Western blotting. Results The mortality, brain water content and caspase-3 expression level were the highest in the SAH+PDGF-BB group, but the neurological function score was the lowest ($P<0.01$). The parameters were reduced when SAH rats were injected with imatinib ($P<0.01$). Conclusion PDGFR may participate in the pathogenesis of early brain injury following SAH, and its antagonist imatinib can reduce brain injury to a certain degree.

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