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小鼠感染弓形虫Prugniaud株后的组织病理学变化

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Histopathology Changes in Mice Infected with *Toxoplasma gondii* Prugniaud Strain

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摘要

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摘要 目的 观察ICR小鼠感染Prugniaud株弓形虫后的症状和组织病理学动态变化。方法 46只ICR小鼠随机分为感染组(30只)和对照组(16只), 感染组小鼠经腹腔注射弓形虫Prugniaud株包囊(10个/鼠, 悬于0.5 ml PBS中), 对照组小鼠注射等量PBS。每天观察小鼠发病情况, 并于感染后第5、10、15、20、25、30、60和90天, 分别处死感染组小鼠3只和对照组小鼠2只, 取小鼠肝、脾、肺、肾、心和脑组织制作切片, HE染色和免疫组织化学检测。结果 感染组小鼠于第6天开始出现食欲减退、耸毛、抖动和腹泻等症状, 死亡率为20.0%。HE染色镜检发现第5天至20天, 肝组织结构破坏, 少数肝细胞水肿, 气球样变和小灶性肝细胞坏死, 肝窦扩张充血伴有炎症细胞浸润等; 脾脏可见脾小体破坏、消失, 脾窦扩张充血, 红髓增宽白髓萎缩等。肺组织结构破坏, 出现间质性肺炎等病理改变。感染第20天后上述组织病理变化逐渐减轻至恢复。脑组织从感染第10天起出现神经元变性、坏死, 第15天~90天出现神经胶质结节、血管袖套现象和蛛网膜下腔炎症细胞浸润等, 并可见弓形虫包囊, 第90天蛛网膜下腔内见肉芽组织。免疫组织化学法检测结果显示, 感染第5天内脏器器官即出现弓形虫抗原, 第10天最强, 后逐渐减弱直至转阴, 脑组织从感染10天起至90天均可见弓形虫抗原。结论 ICR小鼠感染弓形虫Prugniaud株后早期出现速殖子所致的非特异临床症状表现及多脏器组织变性坏死和炎症细胞浸润等病理改变, 此后表现为非特异性的脑组织感染与弓形虫包囊共存。

关键词: 弓形虫 Prugniaud株 病理学 免疫组织化学

Abstract: Objective To observe the symptoms and dynamic changes of histopathology in the organs from ICR mice infected by *Toxoplasma gondii* Prugniaud strain. Methods Thirty ICR mice were infected intraperitoneally with cysts, 10 cysts per mouse. 16 mice were injected with PBS. Incidence of the mice was observed. Three mice from the infected group and two mice from the control group were sacrificed, and the liver, spleen, lung, brain, kidney and heart were collected for pathology and immunohistochemistry examinations on the day 5, 10, 15, 20, 25, 30, 60 and 90 post-infection. Results The infected mice began to fall ill at 6 d post-infection, symptoms including decreased appetite, piloerector fur, sloth, shakes and diarrhea, with a mortality rate of 20%. From 5 d to 20 d post-infection, microscopic examination for HE stain-slides showed the destroyed liver structure, cellular edema, ballooning change, focal necrosis, sinus hepaticus expansion and hyperemia, and inflammatory infiltration. Splenic corpuscles demolished and disappeared, red pulp widened and white pulp atrophied, splenic sinusoid extended with hyperemia. Lungs showed destruction of the structure and pathological changes of interstitial pneumonia. The pathological changes began to alleviate until recovery after 20 d post-infection. In the brain, neuronal degeneration and necrosis were found at 10 d post-infection. Some neuroglial cell tubercle, blood vessel sleeve cuffing, inflammatory cell infiltration on cavitas subarachnoidealis and cysts were observed from 15 d to 90 d. Granulation tissue was seen at 90 d post-infection. By immunohistochemistry test, internal organs showed toxoplasma antigen at 5 d post-infection, and the positive reaction was remarkable at 10 d post-infection, then began to taper until negative. Toxoplasma antigen was revealed in the brain from 10 d to 90 d post-infection. Conclusion Non-specific clinical manifestation and the degeneration, necrosis and inflammatory cell infiltration in poly-organs appear in earlier period of toxoplasma tachyzoite infection in the ICR mice, followed by the co-existing phenomenon of non-specific infection with cysts in the brain.

Keywords: *Toxoplasma gondii* Prugniaud strain Pathology Immunohistochemistry

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