

## PATHOGENESIS STUDY OF LEPTOSPIROSIS IN HAMSTERS INFECTED WITH *LEPTOSPIRA* ISOLATE FROM OUTBREAK AREA

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### Abstract

**Background:** Outbreaks of Leptospirosis had occurred in the northeastern part of Thailand during 1996-2003. High case fatality rate with atypical manifestation was found in leptospirosis patients. With this reason, pathogenesis of leptospirosis in hamsters infected with *Leptospira interrogans* serovar Pyrogenes isolated from a febrile patient in outbreak area was studied to reveal the pattern course of the disease.

**Methods:** Six control hamsters intraperitoneally injected with 0.5 ml PBS whereas the other 24 animals were injected with 0.5 ml of PBS containing  $1 \times 10^8$  leptospires/ml served as experimental group. Three of the control hamsters were sacrificed on day 2 and the last three were killed on day 5. Each three infected hamsters were sacrificed at 1 hour, 6 hours and on day 1, 2, 3, 4, 5 and 6 after inoculation. The kidneys, lungs, liver, gastrocnemius and hamstring muscles of all the sacrificed animals were removed and processed for histopathology.

**Results:** The infected kidneys showed degenerative changes of the renal tubular cells, including vacuolar degeneration, cellular swelling of proximal tubules, dilatation of the distal tubular lumen and necrosis. The glomeruli had congestion and swelling of the glomerular tuft, inflammatory cell infiltration, hemorrhage in the glomerular tuft and the urinary space. Interstitial nephritis and pyelonephritis were also found. In the lung, the alveolar and interalveolar capillaries were distended and engorged with red blood cells. A small number of alveoli were filled with inflammatory cells, which represented bronchopneumonitis. The interalveolar septum was thickened by accumulation of inflammatory cells, which was a sign of interstitial pneumonitis. The infected liver showed enlarged and vacuolated hepatocytes. Vascular and sinusoidal congestion, prominent Kupffer cells, and inflammatory cell infiltration in the hepatic parenchyma and hepatic sinusoids were demonstrated. The portal area showed a number of inflammatory cells. Hepatocellular necrosis was found scattered throughout the hepatic lobules, which was a sign of hepatocellular damage. In the gastrocnemius and hamstring muscles, dilation and congestion of blood vessels were shown in some hamsters in the infected groups. One hamster of the infected group showed inflammatory cell infiltration in the perimysium of the gastrocnemius muscle. Another one showed necrosis of some muscle fibers together with inflammatory cell infiltration, which were signs of muscular inflammation.

**Conclusions:** The results corresponded with previous similar studies, however, the pathogenesis of this study was quicker and the infection was more severe than in other studies. This might be due to the difference in the serovar studied.

**Keywords:** Leptospirosis, pathogenesis, hamster

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