

**Pathology and pathogenesis of human leptospirosis: a commented review.**

**Thales De Brito<sup>1,2</sup>, Ana Maria Gonçalves da Silva<sup>1,2</sup>, Patrícia Antonia Estima Abreu<sup>3</sup>.**

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*Page 5, 1<sup>st</sup> column, line 49, where it reads:*

Recently, it was also shown, in normal guinea pigs that Lp25, a surface protein of pathogenic leptospires, was partially responsible for hyperkalemic pre-renal acute kidney manifestations induced by rhabdomyolysis.

*Should be read:*

Recently, it was also shown, in normal guinea pigs that Lp25, a surface protein of pathogenic leptospires, was partially responsible for hyperkalemic and oliguric acute kidney manifestations induced by rhabdomyolysis.

*On page 7, 1<sup>st</sup> column, line 39, where it reads:*

Immunohistochemical exams in autopsy material confirmed a primary lesion of the proximal convoluted tubules when a decrease of the endogenous sodium/hydrogen exchanger isoform 3 (NH3), aquaporin 1 and 2 and  $\alpha$ -Na<sup>+</sup> K<sup>+</sup> ATPase are found.

*Should be read:*

Immunohistochemical exams in autopsy material confirmed a primary lesion of the proximal convoluted tubules when a decrease of the endogenous sodium/hydrogen exchanger isoform 3 (NH3), aquaporin 1 and  $\alpha$ -Na<sup>+</sup> K<sup>+</sup> ATPase are found.

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