论著

关木通对大鼠肾上腺的毒效应及其可能机制

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摘要 目的 观察关木通(AMK)对大鼠肾上腺的影响及其与肾损害的关系,并探讨其可能机制。方法 ig给予雌性大鼠40 g•kg⁻¹•d⁻¹ AMK水煎剂1,3及5 d,观测肾上腺和肾脏的重量,肾上腺和肾脏组织学及其超微结构变化,测定血清尿素氮、肌酐和皮质醇含量,免疫组化法检测8-羟基脱氧鸟苷(8-OHdG)和诱生型一氧化氮合酶(iNOS)的表达。结果 给药3 d后,大鼠血清皮质醇明显增高,肾上腺皮质明显增厚;连续给药5 d,大鼠血清尿素氮和肌酐明显增高,肾小管上皮细胞水肿,变性,坏死;给药组大鼠8-OHdG和iNOS明显表达。结论 AMK可引起肾上腺皮质增生和肾损害,肾上腺损害早于肾损害,其毒效应机制可能与DNA氧化损伤和一氧化氮增多有关。关键词 关木通 肾上腺 肾脏 8-羟基脱氧鸟苷 一氧化氮合酶

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Toxic effects and possible mechanisms of Aristolochia manshuriensis Kom on adrenals in rats

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Abstract

AIM To investigate the toxic effects and possible mechanism of *Aristolochia manshuriensis* Kom(AMK) on adrenals of rats and relationship between adrenal and kidney damage. **METHODS** Decoction of AMK 40 g·kg⁻¹·d⁻¹ was given ig to female rats for 1, 3 and 5 d, respectively. The observed items included absolute weight and relative weight, histology and ultrastructure of adrenals and kidneys, the serum level of urea nitrogen, creatinine and cortisol, and expression of 8-hydroxydeoxyguanosine(8-OHdG) and inducible nitric oxide synthase(iNOS). **RESULTS** After administration of AMK for 3 d, the serum cortisol level was markedly increased and adrenal cortex became thick. After administration of AMK for 5 d, urea nitrogen and creatinine significantly increased. Histologically, edema, denaturation and necrosis were observed in renal tubular epithelium. The 8-OHdG and iNOS positive cells obviously increased in treated groups. **CONCLUSION** AMK would induce adrenal hyperplasia and kidney damage, the occurrence of adrenal damage was followed by kidney damage. The possible toxic mechanism is related to oxidative damage of DNA and nitric oxide increase.

Key words Aristolochia manshuriensis Kom adrenal gland kidney 8-hydroxydeoxyguanosine nitric oxide synthase

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