

개똥썩에서 分離된 artemisinin이 家兔
IgG에 의해 誘發된 생쥐의 腎毒性
血清絲球體腎炎에 미치는 影響

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ABSTRACT

The effect of artemisinin on the rabbit IgG accelerated nephrotoxic serum glomerulonephritis in mice

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Artemisinin, a new antimalarial to treat patients infected with strains of *Plasmodium falciparum*, derived from the plant *Artemisia annua* Linn, has immunopharmacologic actions such as enhance the PHA-induced lymphocyte transformation rate, increased the weight of spleen but reduced the weight of thymus, reduced phagocytic function of peritoneal macrophage, remarkably reduced the level of serum IgG and hemolysin forming capacity (sented with SRBC), inhibited the activity of Ts cells of donor mice by supraoptimal immunization(SOI), but enhanced activity of Ts cells of recipient mice by SOI. These results suggested that Ts cells may be the target cells of artemisinin. To the serum complement C3 level of plasmodium berghei-infected mice, artemisinin (i. m.) could remarkably increase it. The artemisinin also obviously reduced the prostaglandin E(PGE) in the mouse hind paw swelling induced by carrageenin. Numerous studies have demonstrated that pharmacologic doses of PGE attenuate the development of immunocomplex nephritis. Some autologous immune mechanisms may be involved in the pathogenesis of some types of glomerulonephritis. Glomerular abnormalities can be induced in animals by variety of immunological manipulations. The resulting disorder has many clinical and pathological similarities to the disease in human. Our purpose was therefore to test the ability of the artemisinin to lessen the severity of rabbit IgG accelerated nephrotoxic serum glomerulonephritis in mice model.

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Mice which had treated with rabbit IgG and NTS, administrated with saline, showed Significant increases of urinary protein, cholesterol level, and decrease of serum albumin in NS group. On the contrary, By i.g. administration of artemisinin at dose of 12.5, 25 and 50 mg/kg for 14 days after NTS injection, shown that artemisinin inhibited the nephritic changes in some parameters by means of urinary protein($p < 0.05$, $p < 0.01$) and serum cholesterol($p < 0.05$, $p < 0.01$) and albumin ($p < 0.05$, $p < 0.01$), blood urea nitrogen($p < 0.05$, $p < 0.01$), serum albumin($p < 0.05$, $p < 0.01$); Cyclophosphamide(i.p. 10mg/kg for 14d) had almost same effect as the artemisinin had.

Morphological studies shown that The picture of kidney from the mouse with NTS-nephritis accerated with rabbit IgG, treated with i.g. saline as the control, the mesangiocapillary were enlarged and proliferated; There were inflammatory cells infiltrating around the glomeruli; The ethelial cell were proliferated in the wall of Bowman's capsule.

Histopathological picture of kidney from the NTS-nephritis accerated with rabbit IgG mouse treated with i.p. 10mg/kg cyclophosphamide as the positive control. No significant histopathological evidence were found. Treaded with i.p. 12.5mg/kg artemisinine, the picture shown that mesangiocapillary were lightly proliferated; There were inflammatory cells infiltrating around the glomeruli; Treaded with i.p. 25mg/kg artemisinine, The picture shown that the mesangiocapillary were lightly proliferated; Treaded with i.p. 50mg/kg artemisinine, The picture shown that both the mesangiocapillary proliferated and the inflammatory cells infiltrating around the glomeruli are less than treated with saline, 12.5 and 25 mg/kg artemisinine.

On the basis of these studies we conclude that the artemisinin can relieve pathological change caused by NTS-nephritis accerated with rabbit IgG.