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Therapeutic Effects of Tanshinone IIA on Severe Acute Pancreatitis in Mice Induced by Caerulein Combined with Lipopolysaccharide

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***SUMMARY*. This study aimed to investigate the therapeutic effects of tanshinone IIA on severe acute pancreatitis in mice induced by caerulein combined with lipopolysaccharide. Forty-five mice were randomly divided into normal, model, and treatment groups. The severe acute pancreatitis model induced by caerulein combined with lipopolysaccharidemice was established in model and treatment groups. Then, the treatment group was given 40 mg/kg tanshinone IIA by gavage, once a day, for seven consecutive days. At the end of experiment, compared with model group, in treatment group, the pancreatic mass index and serum amylase, lipase, tumor necrosis factor α and interleukin 6 levels were significantly decreased ($P < 0.05$), and the serum interleukin 10 level was significantly increased ($P < 0.05$); the pancreatic tissue superoxide dismutase and glutathione peroxidase levels were significantly increased ($P < 0.05$), and the malondialdehyde and reactive oxygen species levels were significantly decreased ($P < 0.05$); the pancreatic tissue nuclear factor kappa-B p65 and phosphorylated inhibitor of nuclear factor kappa-B α (IκB-α) protein expression levels were significantly decreased ($P < 0.05$), and the IκB-α protein expression level was significantly increased ($P < 0.05$). In summary, tanshinone IIA can inhibit the inflammatory response, oxidative stress and activation of NF-κB signal pathway, thus mitigating the severe acute pancreatitis in mice.**

RESUMEN. Este estudio tuvo como objetivo investigar los efectos terapéuticos de la tanshinona IIA en la pancreatitis aguda severa en ratones inducidos por caeruleína combinada con lipopolisacárido. Cuarenta y cinco ratones se dividieron aleatoriamente en grupos normales, de modelo y de tratamiento. El modelo de pancreatitis aguda grave inducida por caeruleína combinada con ratones lipopolisacáridos se estableció en los grupos de modelo y tratamiento. Luego, el grupo de tratamiento recibió 40 mg/kg de tanshinona IIA por sonda, una vez al día, durante siete días consecutivos. Al final del experimento, en comparación con el grupo modelo, en el grupo de tratamiento el índice de masa pancreática y los niveles de amilasa sérica, lipasa, factor de necrosis tumoral α e interleucina 6 disminuyeron significativamente ($P < 0.05$) y el nivel de interleucina 10 en suero aumentó significativamente ($P < 0,05$); los niveles de superóxido dismutasa y glutatión peroxidasa de tejido pancreático aumentaron significativamente ($P < 0.05$) y los niveles de malondialdehído y oxígeno reactivo disminuyeron significativamente ($P < 0.05$); el factor nuclear del tejido pancreático kappa-B p65 y el inhibidor fosforilado de los niveles de expresión de la proteína del factor nuclear kappa-B α (IκB-α) disminuyeron significativamente ($P < 0.05$) y el nivel de expresión de la proteína IκB-α aumentó significativamente ($P < 0,05$). En resumen, la tanshinona IIA puede inhibir la respuesta inflamatoria, el estrés oxidativo y la activación de la vía de señal de NF-κB, mitigando así la pancreatitis aguda severa en ratones.

KEY WORDS: mechanism, mice, severe acute pancreatitis, tanshinone IIA.

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