

Effect of Taxifolin on Doxorubicin-Induced Oxidative Liver Injury in Rats: A Biochemical and Histopathologic Examination

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SUMMARY. Decrease in antioxidants is responsible for the increase in oxidants in hepatotoxicity induced by doxorubicin. Taxifolin is a flavonoid with strong antioxidant properties. This study was undertaken to investigate the effect of taxifolin on doxorubicin-induced potential oxidative liver injury in rats. Eighteen rats were divided into three groups of six each: healthy (HG), doxorubicin (DOXG), and taxifolin + doxorubicin (TDG). Taxifolin (50 mg/kg) was orally administered to TDG rats. After 1 h, DOXG and TDG received 5 mg/kg intraperitoneal doxorubicin. Treatment continued once daily for 7 days. On day 8, blood samples were taken from the rats and liver tissue was collected after euthanasia. The tissues were histopathologically assessed, and oxidant-antioxidant relationships were evaluated. Liver function tests were performed using blood samples. Taxifolin successfully suppressed tissue malondialdehyde, total oxidant status, and oxidative stress index caused by doxorubicin, as well as the increase in alanine aminotransferase, aspartate aminotransferase, and lactate dehydrogenase activity ($p < 0.001$). Additionally, it prevented the decline in total glutathione and total antioxidant status levels induced by doxorubicin in liver tissues ($p < 0.001$). Histopathological evaluation revealed that taxifolin alleviated doxorubicin-induced damage in liver tissue. Our experimental results support that taxifolin may be effective in preventing doxorubicin-induced hepatotoxicity.

RESUMEN. La disminución de los antioxidantes es responsable del aumento de oxidantes en la hepatotoxicidad inducida por la doxorubicina. La taxifolina es un flavonoide con fuertes propiedades antioxidantes. Este estudio se llevó a cabo para investigar el efecto de la taxifolina sobre la posible lesión hepática oxidativa inducida por doxorubicina en ratas. Dieciocho ratas se dividieron en tres grupos de seis cada uno: sanas (HG), doxorubicina (DOXG) y taxifolina + doxorubicina (TDG). Se administró por vía oral taxifolina (50 mg/kg) a ratas TDG. Después de 1 h, DOXG y TDG recibieron 5 mg/kg de doxorubicina intraperitoneal. El tratamiento continuó una vez al día durante 7 días. El día 8, se tomaron muestras de sangre de las ratas y se recogió tejido hepático después de la eutanasia. Los tejidos se evaluaron histopatológicamente y se evaluaron las relaciones oxidante-antioxidante. Las pruebas de función hepática se realizaron utilizando muestras de sangre. La taxifolina suprimió con éxito el malondialdehído tisular, el estado oxidante total y el índice de estrés oxidativo causado por la doxorubicina, así como el aumento de la actividad de alanina aminotransferasa, aspartato aminotransferasa y lactato deshidrogenasa ($p < 0,001$). Además, evitó la disminución de los niveles de glutatión total y del estado antioxidante total inducida por la doxorubicina en los tejidos hepáticos ($p < 0,001$). La evaluación histopatológica reveló que la taxifolina alivió el daño inducido por la doxorubicina en el tejido hepático. Nuestros resultados experimentales respaldan que la taxifolina puede ser eficaz para prevenir la hepatotoxicidad inducida por doxorubicina.

KEY WORDS: antioxidant, doxorubicin, hepatoprotective effect, oxidative damage, taxifolin,

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