



INCREASED EXPRESSION OF TGF-B IN THE LUNG OF ARTHRITIC RATS

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BACKGROUND

There is evidence of the involvement of the renin-angiotensin system in extra-articular manifestations of rheumatoid arthritis (RA). The objective of the present study was to investigate the lung manifestation of rheumatoid arthritis in rats and the involvement of angiotensin II in the mechanism of the disease.

MATERIALS AND METHODS

Male Wistar rats (200 g), ingesting low salt diet, were distributed in three groups (8 rats/group): Control, Arthritis and Arthritis + AT1 blocker (losartan). The arthritis was induced by the injection of 100 µL of an emulsion of dissected Mycobacterium tuberculosis (50 mg/mL) on the intradermal paw. The control group received the vehicle (mineral oil emulsion). Treatment with losartan was initiated on the first day of immunization by subcutaneous injection daily (1 mg/Kg daily). After confirmation of systemic arthritis, the animals were sacrificed 60 days post immunization or vehicle. It was evaluated the plasma concentration of angiotensin II by ELISA assay, and the following parameters in the lung: histopathological analysis; immunohistochemistry for TGF-B, iNOS (oxidative stress) and AT1/AT2 receptors.

RESULTS

Although there was no significant difference in lung inflammation among groups, the arthritic rats showed elevated plasma levels of angiotensin II as well as enhanced TGF-B lung expression. The losartan treatment reduced this TGF-B expression. No difference was observed in lung AT1/AT2 receptors or iNOS expression among groups.

CONCLUSION

Experimental RA causes increased expression of TGF-B in the lung and this alteration is related to angiotensin II.