TRIFLUSAL ADMINISTRATION REDUCES GLIAL REACTIVITY AND COX-2 EXPRESSION IN A VIRAL MODEL OF MULTIPLE SCLEROSIS.

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Administration of Triflusal diminishes the neuronal death and glial reactivity associated to an inflammatory process. The aim of this study was to elucidate the effects that Triflusal administration exerts in the course of Theiler's murine encephalomyelitis with special emphasis in glial reactivity. Susceptible mice SJL/J (4 week old) were infected with DA strain of Theiler's virus. Sixty days after infection, animals were administrated orally with Triflusal (30 mg/kg) daily during 15 days until sacrifice. In addition, a group of infected animals received vehicle solution instead Triflusal. A number of non-infected animals with and without Triflusal administration were used as control. Evaluation of coordination and motor activity was performed in all animals just before, during and at the end of the administration, using activity box and rotarod tests. Animals were sacrificed and spinal cord and brainstem were processed for GFAP and COX-2 immunohistochemistry, tomato lectin (TL) histochemistry and Western Blot for COX-2. Our results revealed that, when compared to control non-infected animals, those infected with Theiler's virus, in addition to motor function deterioration, showed a remarkable astroglial and microglial reactivity that correlated with an increase in COX-2. Whereas in grey matter astrocytes displayed a GFAP increase, this expression decreased in white matter. Morphological changes in microglial cells were associated with an increase of TL binding. Triflusal administration improved the coordination and motor activity in the majority of infected animals. This improvement was accompanied by reduction of COX-2 expression and decrease of astroglial and microglial reactivity in around 50% of Triflusal treated mice, in which glial cells showed a morphology and distribution similar to non-infected controls. These results show that Triflusal administration produce an improvement in the course of this viral-induced autoimmune disease model. Further studies are necessary in order to elucidate the molecular mechanisms underlying this drug interaction in glial activation pathways.