In the aftermath of neuronal injury metabolic imbalance occurs and leads to the loss of neuronal tissue. During this process glycolysis is elevated and enhanced levels of methylglyoxal (MG) are formed. Methlyglyoxal induces the expression of proinflammatory cytokines leading to loss of protein function. Especially, neurons shows high susceptibility to MG toxicity. Glyoxalase 1 (Glo1) as the ubiquitous endogenous detoxification system catabolises MG. Astrocytes present high Glo1 levels compared to neurons. Glo-1 underlies changes in ageing and neurodegeneration. However, data about the role of Glo-1 secondary injury are still rare. Here, we investigated the temporal dynamics of Glo1 distribution and expression by immunohistochemistry and Western Blot analysis. Organotypic hippocampal slice cultures were excitotoxically (N-methyl-D-aspartate, 50µM for 4 hours) lesioned in vitro (5 minutes to 72 hours), scratch-wound model of astrocyte monolayers treated with the Glo1 inhibitor ethyl pyruvate (EP; 0.84µM, 8.4µM, 84µM; 0 hours to 48 hours) and permanent middle cerebral artery occlusion were performed (75 minutes to 60 days). We found i) a time-dependent up-regulation and re-distribution of Glo1 in neurons and astrocytes ii) a strong increase in Glo1 dimers after injury (24h-72h) when compared to monomers of the protein after acute neuronal injury and a diminished astrocytic scar formation after EP treatment. In conclusion, the temporal and spatial dynamics following the process of neuronal injury may present Glo1 as important player in the process of secondary neuronal injury.