Astrocytes are involved in most neuroinflammatory and neurodegenerative diseases, and accumulating evidence indicates that they may play important roles in regulating the progression and resolution of these disorders. Upon brain insult, substantial changes in the metabolic profile of astrocytes have been reported, however, whether and how these changes contribute to brain tissue remodelling in disease settings is unclear. Here, we report that astrocyte perivascular end-feet, which are hotspots of $Ca^{2+}$-mediated signalling pathways regulating neurovascular coupling, are naturally enriched in mitochondria-endoplasmic reticulum (ER) contact sites *in vivo*. Following acute brain injury, these two organelles undergo a marked remodelling, mirroring the emergence of typical hallmarks of astrocytic reactivity. Importantly, we found that the formation of a prominent and lasting mitochondrial-enriched compartment in perivascular end-feet is part of an adaptive metabolic response to injury regulated by fusion dynamics. Conditional deletion of Mitofusin 2 (*Mfn2*) prevented this mitochondrial enrichment and resulted in altered mitochondrial-ER tethering domains in end-feet. Proteomic analysis of MFN2-deficient reactive astrocytes isolated from injured mouse cortices, revealed not only a significant impairment in OXPHOS, but also an up-regulation in several important metabolic pathways, including amino acid catabolism, glycolysis, cholesterol biosynthesis and $Ca^{2+}$ transport regulation. Physiologically, changes in spontaneous $Ca^{2+}$ activity in injured astrocytes lacking MFN2 were validated by two-photon imaging experiments *ex vivo* and *in vivo*, which revealed a compromised mitochondrial $Ca^{2+}$ uptake leading to abnormal cytosolic $Ca^{2+}$ transients in astrocytic end-feet. At the tissue level, a severe impairment in microvasculature recovery following injury was rescued by experimentally boosting perivascular mitochondrial enrichment in astrocytes lacking fusion dynamics. These data reveal a key role of astrocyte mitochondrial fusion dynamics in local metabolic signalling and offer the opportunity for developing targeted approaches to ameliorate vascular network repair in the injured brain.