ABSTRACT
Malaria is a life threatening infectious diseases transmitted by the bite of infected female Anopheles mosquito and responsible for high morbidity and mortality rates. Cerebral malaria is a complex neurological syndrome, whose pathology is mediated by inflammatory processes triggered by the immune system of the host following infection with P. falciparum. Coenzyme Q10 (CoQ10) is an obligatory cofactor in the electron transport chain. The reduced form of CoQ10 serves as a potent antioxidant additionally; CoQ10 has been identified as a modulator of gene expression, inflammation and apoptosis. However, the modulatory effects of CoQ10 during PbA infection process and risk occurrence of ECM have not been determined. In the present study we evaluated the effect of oral supplementation of CoQ10 in the host immunity and the outcome of experimental cerebral malaria caused by P. berghei ANKA in C57BL/6 mice. We observed that oral administration of CoQ10 for one month and after PbA infection prolonged the survival of mice during ECM. CoQ10 administration significantly reduced TNFα and IL-1β gene expression in brain samples and circulatory cytokines in PbA infected mice. The results also shows the ability of CoQ10 to reduce levels of CXCL9 and CXCL10 mRNA expression and cytoadhesion molecule ICAM-1 in the brain, resulting in significant reduction in the accumulation of pathogenic T cells and effector molecules in the brain and putatively improvement in the stability of the blood–brain barriers of PbA-infected mice. Furthermore, CoQ10 administration showed an enhanced upregulation of AAM gene expression. Moreover CoQ10 resulted in a significant decrease in liver aspartate aminotransferase [AST] an indicative of amelioration in liver inflammation. Our data collectively demonstrates the ameliorative function of CoQ10 on development of host inflammatory immune response, which provides an enhanced survival benefits in the murine ECM model.

Key words: Coenzyme Q10; Experimental cerebral malaria and Plasmodium berghei ANKA.