This study examines the effects of alcohol abstinence on H3k9me2 levels and its colocalization with IBA1 in key brain regions. Alcohol Use Disorder is prevalent and significantly impacts brain structure and function, also affecting immune signaling molecules in the brain (Microglia). This study focuses on the epigenetic marker H3K9me2, catalyzed by G9a, which is associated with gene silencing and implicated in the regulation of inflammatory responses and addiction. Utilizing a mouse model, male mice were treated with ethanol (EtOH) for 10 days, followed by a 28-day abstinence period. To determine our numbers, an Immunofluorescence experiment was conducted to analyze the expression of H3K9me2, levels of IBA1, and colocalization of H3K9me2 in IBA1. Results from the Immunofluorescence indicated a significant increase in H3K9me2 in the CeA (Central Nucleus of the Amygdala) in EtOH-treated mice compared to the controls. Notably, while some brain regions showed no significant change post-abstinence, increases in IBA1 and H3k9me2-IBA1 colocalization were observed across all studied regions, suggesting persistent microglial inflammation due to alcohol exposure, even after abstinence. These findings shed light and allow us to conclude more on the complex molecular pathways of alcohol addiction and support the continued investigation into H3K9me2's role in AUD, with implications for understanding the long-term effects of alcohol on the brain and the potential development of targeted therapies.