عنوان مقاله:

Cell Death in Methamphetamine-Dependent Patients Based on Circulating Molecular Markers and Structural MRI

محل انتشار:

هشتمین کنگره علوم اعصاب و پایه و بالینی (سال: 1398)

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خلاصه مقاله:

Background and Aim : Although there are numerous animal studies showing that methamphetamine (MA) induces neuronal degeneration and cell death through all types of programmed cell death (PCD) mechanisms including apoptosis, autophagia and necroptosis, the effects of MA on human brain and the extent of induced neural degeneration is not well understood. We evaluated MA effects on active users considering various cell death mechanisms and structural MRI.Methods : Structural images of 19 active MA-dependent patients and 18 healthy controls were acquired on a 3 T Siemens MRI scanner and Voxel-based morphometry (VBM) analysis using SPM8. Socio-demographic and Addiction Severity Index (ASI) questionnaires were used to determine medical, drug use and family/social status. Blood samples of all subjects were collected. Human enzyme-linked immunosorbent assay (ELISA) kits were applied for the measurement of circulating molecular markers including Programmed cell death markers: caspase 3 for apoptosis - Mixed Lineage Kinase-Like pseudokinase (MLKL) for necroptosis and micro tube-associated protein 1A/1B Light Chain (LC3B) for autophagy. The data were analyzed using Graph Pad PRISM software.Results : Structural MRI analysis showed increased white matter volume in MA-dependent patients relative to the controls in right superior temporal gyrus, left temporal lobe, right frontal lobe and left medial frontal gyrus. Comparing the levels of blood markers between MA-dependent patients and controls showed no significant differences for the proteins involved in programmed cell death including Caspase 3, MLKL and LC3B. There was no significant correlation between pattern of MA use (years and amount of MA use) and Plasma molecular markers.Conclusion : Results showed that MA induced white matter hypertrophy without activation of apoptosis, .necroptosis, or autophagy mechanisms

کلمات کلیدی: