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Novel ayurvedic approach in the treatment of psychosis: An experimental study

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Introduction: Schizophrenia is a complex neuropsychiatric disorder with abnormalities involving multiple neurotransmitters like dopamine, serotonin, glutamate and GABA. Unmadgajakesari (UGK) is a herbomineral formulation claimed to possess antipsychotic activity. Hence the present study was carried out to assess the antipsychotic profile of UGK.

Aim: To evaluate the antipsychotic activity of UGK in animal models.

Materials & Method: After doing acute and sub-acute toxicity studies of UGK, it was evaluated for its effect on dopamine, serotonin, NMDA and GABA using following animal models: Inhibition of apomorphine induced climbing (dopamine), inhibition of 5-HTP induced head twitches (serotonin), Antagonism of MK-801 induced hyper locomotion (NMDA) in mice and Antagonism of PTZ induced convulsion (GABA) in rats. For studying each neurotransmitter, animals were divided into 6 groups, each group comprising of 6 animals. Group-I: Normal control, Group-II: Vehicle-control (ghrita), Group-III: Drug control (positive control). In test groups (IV-VI) UGK was administered in doses 100 mg/kg, 200 mg/kg and 400 mg/kg in mice and 70 mg/kg, 140 mg/kg and 280 mg/kg in rats. All the drugs were given orally for 8 days. Readings were taken on day 1and 8.

Results: UGK was found to be non-toxic up to dose 2000 mg/kg. Significant antidopaminergic and NMDA activity was obtained on day 1 which reduced by 8th day. Anti-serotonergic activity found on day 1 and continued to be seen on day 8. GABAergic activity was not seen on day one but was present on day 8, more than anti-serotonergic action. GABAergic activity was seen only in low dose. Efficacy of UGK was not increased with dose increment.

Conclusion: Present study concluded the multi-szte mechanism of UGK action in animal models of psychosis acting on dopamine, serotonin, NMDA and GABA receptors and activity changed over 8 days. GABA activity evolved over 8 days to become the major mechanism of action on long term use. None of the drugs presently available for the treatment of psychosis have such a changing profile and action on GABA.

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