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Case Report

Vulnerability to Psychosis in Cannabis Users: The Case of Two Pairs of Brothers

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Abstract: In some recent papers, it have been suggested the influence of genetic factors as an important factor into the development of psychotic symptoms and/or psychotic disorder in patients with drug use. In the current paper, we show the history of two pairs of brothers using drug, with psychotic symptoms and /or psychotic disorder and distinct outcome after several years

Keywords: Cannabis, Genetics, Psychosis, Schizophrenia

1. Introduction

The relationships between cannabis and psychotic disorders have been proved in several cohort studies of general populations (Van os et al, 2002; Arseneault et al, 2002) Moore et al (2007) concluded in their metanalisys of the lliterature that cannabis use is just one of the factors involved in the development of psychotic disorder. (Täschner (1983) suggests that cannabis psychosis coul only be "the first sept to schizophrenia in predisposed subjects"(vulnerable patients?) Another factors could be involved in this relationship, as genetic aspects, time of exposition to drug use, previous personality trais, etc.

In relation with the presence of previous abnormal personality traits, Núñez and Gurpegui (1998, unpublished data) found that cannabis users, with a previous history of cannabis-induced psychosis and long-term exposure, could develops a functional psychosis (schizophrenia or schizoaffective disorder), specially in subjects with some abnormal personality traits (antisocial, for example) Ungerleider et al. (1968) and Negrete (1984) found a high score in Sc scale in MMPI among subjects with cannabis-induced psychosis.

Related to genetic factors, Tsuang et al. (1982) and Mc Guire et al. (1995) found a high frequency of schizophrenia among relatives of patients suffering cannabis psychosis. Varma and Sharma (1983) describe a high prevalence of cannabis dependence among the relatives of schizophrenic patients.

More recently, variations of COMT (Caspi et al., 2005) and AKT1 (Di Forti et al, 2012) genes has been suggested as modulators to the response of cannabis use, leading to apparition of psychotic symptoms and/or psychotic disorders. The first hypothesis could be not real after the results form Zammit et al (2011) in a large sample of psychotic patients. The second one has been replicated and confirmed the previous results Morgan et al., 2016)

In this issue, the author present two pairs of brothers with different evolutions after long term cannabis exposure, suggesting the presence of genetic vulnerability.

2. Families Descriptions

FAMILY A

Familiar precedents of psychiatric disorders: No

BROTHER 1:

Male, 25 years old, previous schizoid traits. Bad academic results, unemployed.

History of drug (cannabis) use since 12 years old, daily from 17 years, 10-15 joints per day.

First psychiatric treatment with 18 years: He was diagnosed as cannabis-induced psychosis, complete recovery after haloperidol treatment during 3 months.

He returned to cannabis use again one year later and he was admitted in the psychiatric unit. Diagnosis: schizophrenia, partial recovery after high doses of neuroleptic.

At the current moment, he maintain the psychopharmacological treatment, with the presence of some residual symptoms (social isolation, apathy).

BROTHER 2:

Male, 22 years old, student, no abnormal personality traits.

History of cannabis use since 13 years, LSD and cocaine eventually, alcohol abuse at weekends. 10 joints per day.

First psychiatric treatment with 17 years, with the diagnosis of cannabis-induced psychosis. He got a total recovery after neuroleptic treatment.

In the present moment, eventual cannabis use, with no psychological consequences and not psychiatric treatment.

FAMILY B

Familiar precedents of psychiatric disorders: Grandfather: suicide; father: alcohol dependence; third brother heroine dependence.

BROTHER 1:

Male, 34 years old, divorced, unemployed.

History of cannabis use since 29 years old (after marital divorce), 4-5 joints by day, alcohol abuse at weekends. Cocaine and amphetamine eventually.

With 31 years (after divorce and lost of his job), he needed go to psychiatric service, where he was diagnosed as cannabis-induced psychosis with complete recovery after antipsychotic treatment during 6 months.

After left treatment, first admittance in psychiatric unit: diagnosis schizoaffective disorder, with complete recovery. After that, 3 new admittances after cannabis and alcohol abuse, with complete recovery.

BROTHER 2:

Male, 25 years old, married, unemployed. Antisocial personality traits.

Cannabis use since 12 years old, from 16 daily use, 20-25 joints per day. Alcohol abuse at weekends, ocasionally LSD, cocaine and heroine.

With 18 years, he presented a cannabis-induced psychosis, with complete recovery after treatment. New psychotic episode after cannabis abuse one year later, with postpsychotic depression. Two cannabis intoxications and one more depresive episode. Diagnosis: Depresive disorder.

3. Discussion

Both pairs of brothers present similar characteristic: non psychotic antecedemts in relatives, long-term drug exposure, especially cannabis; older brother, with lesser cannabis use, lesser frequency of use and one previous psychotic episode have developed a functional psychosis (schizophrenia and schizoaffective disorder, respectively); younger brothers, with higher cannabis use, frequency of use and longer cannabis use do not present any other psychotic episode in abstinence.

Gold and Bowers (1978) and Sato (1992) suggest that drug use modifies the normal cerebral homeosthasis, producing this "vulnerability to psychosis". Fernández-Ruiz et al. (1996) have observed a malfunctioning in synthesis and turn-over of dopamine, as well as cerebral dopamine receptors in rats whose mothers have been treated with cannabinoids. These subjects could present a new psychotic episode after some stressful conditions, even social.

Van Os and Marcelis (1998) have proposed that individual with a high risk to psychosis are more prone to drug use; in these cases, the mixture of these two factor provokes the ocurrence of a schizophrenic disorder. Something similar occurs in both families.

In the current families, we may suppose the concomitant influence of the two factors, perhaps a previous genetic predisposition to psychosis and the psychotic-like effects of cannabis use, leading to the development of a full psychotic disorder, with full recovery after antipsychotic treatment and abstinence of drug use. In older borthers, the new use of drug and /or the presence of stressful events lead to the development of a chronic psychosis.

4. Conclusions

Drug use is one factor for the occurrence of psychotic symptoms and/or psychotic syndrome in vulnerable people. The presence of genetic factors must be included in the study of these patients.

References

[1] Arseneault L, Cannon M, Poulton R, Murray R, Caspi A, Moffitt TE. (2002) Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. BMJ. 23; 325 (7374): 1212-3

[2] Bowers MB Jr. (1998) Family history and early psychotogenic response to marijuana. J Clin Psychiatry. 1998 Apr; 59 (4): 198-9

[3] Bowers MB, Kantrowitz JT (2007) Elevated Plasma Dopamine Metabolites in Cannabis Psychosis. Am J Psychiatry Psychosis, 164 (10): 1615-1616

[4] Bowers MB: (1996) Chronic substance-induced psychotic disorders: state of the literature. J Neuropsy Clin Neurosc, 8 (3): 262-269

[5] Caspi A, Moffitt TE, Cannon M, McClay J, Murray R, Harrington H, Taylor A, Arseneault L, Williams B, Braithwaite A, Poulton R, Craig IW. (2005) Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: longitudinal evidence of a gene X environment interaction. Biol Psychiatry, 15; 57 (10): 1117-27

[6] Di Forti M, Iyegbe C, Sallis H, Kolliakou A, Falcone MA, Paparelli A, Sirianni M, La Cascia C, Stilo SA, Marques TR, Handley R, Mondelli V, Dazzan P, Pariante C, David AS, Morgan C, Powell J, Murray RM. (2012) Biol Psychiatry. 15; 72 (10): 811-6

[7] Fernández-Ruiz JJ, Romero J, García L, García Palomero E, Ramos JA (1996) Dopaminergic neurons as substrate of neurobehavioral effects of marihuana: Developmental and adult studies. En: Beninger RJ, Palomo T, Archer T, eds. Dopamine disease states. Editorial CYM, Madrid

[8] Gold MS, Bowers MB (1978) Neurobiological vulnerability to low-dose amphetamine psychosis. Am J Psychiatry, 135: 1546-1548

[9] McGuire PK, Jones P, Harvey I, Williams M, McGuffin P, Murray RM (1995) Morbid risk of schizophrenia for relatives of patients with cannabis-induced psychosis. Schizophr Research, 15: 277-281.

[10] Morgan CJ, Freeman TP, Powell J, Curran HV (2016) AKT1 genotype moderates the acute psychotomimetic effects of naturalistically smoked cannabis in young cannabis smokers. Transl Psychiatry. 2016 Feb 16; 6: e 738. doi: 10.1038/tp.2015.219

[11] Moore TH, Zammit S, Lingford-Hughes A, Barnes TR, Jones PB, Burke M, Lewis G. (2007) Cannabis use and risk of psychotic or affective mental health outcomes: a systematic review. Lancet. 28; 370 (9584): 319-28

[12] Negrete JC (1984) Clinical psychiatric complications of cannabis use: an update. Marihuana Proceedings of the Oxford Symposium on cannabis, Oxford, septiembre: 482-486

[13] Sato MA (1992) A lasting vulnerability to psychosis in patients with previous metamphetamine psychosis. Ann NY Acad Sci, 654: 160-170

[14] Täschner KL (1983) Zur Psychopathologie und differentialdiagnose sogenannter cannabispsychosen. Forrschr Neurol Psychiatr, 51: 235-248

[15] Tsuang MT, Simpson JC, Kronfold Z (1982) Subtypes of drug abuse with psychosis. Arch Gen Psychiatry, 39: 141-147.

[16] Ungerleider JT, Fischer D, Fuller M (1968) The “bad trip”: the etiology of the adverse LSD reaction. Am J Psychiatr, 124: 1483-1490

[17] van Os J, Bak M, Hanssen M, Bijl RV, de Graaf R, Verdoux H. (2002) Cannabis use and psychosis: a longitudinal population-based study. Am J Epidemiol, 15; 156 (4): 319-27

[18] van Os J, Marcelis M. (1998) The ecogenetics of schizophrenia: a review. Schizophr Res. 27; 32 (2): 127-35

[19] Varma SL, Sharma I (1993) Psychiatric comorbidity in first-degree relatives of schizophrenic patients. Br J Psychiatry. 162: 672-678

[20] Zammit S, Owen MJ, Evans J, Heron J, Lewis G. (2011) Cannabis, COMT and psychotic experiences. Br J Psychiatry; 199 (5): 380-5