tient was euthymic. Three months later, the patient signaled he felt very good and did not need cannabis anymore. After 12 months of treatment with aripiprazole the patient had not relapsed and did not use cannabis at all (urine screen did not reveal any THC). Moreover, his BMI was 24 and BPRS score decreased to 40.

This case report highlights different proposals about mechanism of action and side effects.

First, the concurrence of the diminution of cannabis consumption with the patient's treatment with aripiprazole suggests that aripiprazole contributed to the occurrence of this diminution. Different mechanisms may have played a part, such as aripirazole's partial agonism at dopamine D_2 receptors.[2] A similar observation was made with cocaine dependence.[3] Dopamine stimulation in the nucleus accumbens has been suggested to cause addictive behavior and aripiprazole's partial dopamine agonist effect in this area may reduce this behavior.[4] In addition, aripiprazole has a number of serotonergic actions that are not related to dopamine potentially modulating the response to THC.[5,6] Other mechanisms may be involved and Mr. A's rapid cessation of cannabis use after starting aripiprazole suggests the need to verify these mechanisms systematically and to plan controlled trials.

Second, several factors have to be considered when regarding why there may be a concomitant increase in substance abuse with the older antipsychotics. It was suggested that a strong antagonist effect at the dopamine D_2 receptors in the nucleus accumbens was involved in concomitant increase in substance abuse with old antipsychotics.[7] Moreover, unrelated to dopaminergic mechanisms, the use of certain antipsychotics with substantial side effects by schizophrenia patients may actually contribute to greater substance use in an effort to self-medicate the side effects.[8,9]

Finally, there could be at least two explanations for the weight loss. The reduction of cannabis use may have contributed to a reduction in eating,[10] and the switch in the antipsychotic to aripiprazole may have been instrumental in such a significant weight reduction, since aripiprazole is weight neutral and olanzapine is known to facilitate weight gain in patients.

To our knowledge, this is the first reported case of aripirpazole's effect on cannabis use. More research is needed to establish the benefits of aripiprazole in regard to cannabis. Some dual diagnosis patients may benefit from aripiprazole, which may reduce craving for and use of cannabis.

Martin Desseilles, M.D.
Department of Psychiatry, University of Liège, Belgium
Fernand Mathot, Pharm.D.
Michel Desseilles, M.D.
Psychiatric Hospital Centre "Le Petit-Bourgogne," Liège, Belgium

References

1. Ujike H, Morita Y: New perspectives in the studies on endocannabinoid and cannabis: cannabinoid receptors and schizophrenia. J Pharmacol Sci 2004; 96:376–381
2. Travis MJ, Burns T, Dursun S, et al: Aripiprazole in schizophrenia: consensus guidelines. Int J Clin Pract 2005; 59:485–495
3. Beresford TP, et al: Aripiprazole in schizophrenia with cocaine dependence: a pilot study. J Clin Psychopharmacol 2005; 25:363–366
4. Pilla M, et al: Selective inhibition of cocaine-seeking behaviour by a partial dopamine D3 receptor agonist. Nature 1999; 400:371–375
5. Kenna GA, McGeary JE, Swift RM: Pharmacotherapy, pharmacogenomics, and the future of alcohol dependence treatment, part 2. Am J Health Syst Pharm 2004; 61:2380–2388
6. Kenna GA: Rationale for use of aripiprazole for alcohol dependence treatment. Drugs Future 2003; 28:1227–1235
7. Warsi M, et al: Aripiprazole reduces alcohol use. Can J Psychiatry 2005; 50:244
8. McEvoy JP, et al: Haloperidol increases smoking in patients with schizophrenia. Psychopharmacology (Berl) 1995; 119:124–126
9. Siris SG: Pharmacological treatment of substance-abusing schizophrenic patients. Schizophr Bull 1990; 16:111–122
10. Kirkham TC: Endocannabinoids in the regulation of appetite and body weight. Behav Pharmacol 2005; 16:297–313

Clinical Correlates of Personality Changes Associated With Traumatic Brain Injury

To the Editor: Traumatic brain injury (TBI) is frequently complicated by alterations in temperament and character that have adverse consequences for day-to-day living, manifesting as poor decision-making, interpersonal problems, communication problems, and often overall poor quality of life.[1] Max et al.[2] have reported extensively on the correlates and predictors of personality changes after traumatic brain injury in children, but there is scant mention in the literature on adults. In this report, we describe the results of a preliminary study of the clinical correlates of personality change following traumatic brain injury in adults.

Analysis

Data are from a retrospective chart review of 54 subjects with closed head injury enrolled in an outpatient neuropsychiatry brain injury clinic. Patients with depressed skull fractures were excluded.

Every patient was evaluated and followed by a clinic psychiatrist. The assignment of personality change due to a general medical condition (TBI) diagnosis was based

on DSM-IV criteria. Pharmacological treatment was the primary mode of management. If records indicated that a patient had returned to "baseline," or had improvement in more than half the number of presenting symptoms, that patient was considered a "treatment responder." A patient who had absent or minimal improvement in symptoms was considered a "nonresponder."

About a third of the sample (n = 17, 32%) was diagnosed with personality changes.

The mean age of the sample was 41.1 years (SD = 12.26; range = 18–69). Most patients were male (N = 37, 68.5%), and 81.5% were Caucasians. The mean level of education was 13.4 years (range = 8–20). Forty-four percent of the patients had suffered mild traumatic brain injury, 20.4% moderate injury, and 35.2% had a severe injury, based on the Glasgow Coma Scale (GCS) scores at the time of the injury. Those with personality change were compared to those without personality change. The two groups did not differ in number of injuries, type of trauma, age, education, pre-traumatic brain injury marital, employment and legal status, substance abuse, and/or pre-traumatic brain injury history of mood disorder. However, those with personality change were disproportionately non-Caucasians, had lower scores on the Mini-Mental State Exam (MMSE), had more severe traumatic brain injury, were less likely to be employed post-TBI, and were more frequently judged to have failed pharmacologic treatment.

Discussion

In this study personality changes are associated primarily with severe traumatic brain injury, as was the case in an earlier study.[3] Association between personality change and mild traumatic brain injury has also been reported.[4] We are not surprised that our patients who had personality change did not benefit from pharmacological treatment since it is well established that multimodal regimens that include medications and psychotherapy are the best.[5] Our patients with personality change had worse cognitive function and were less successful in finding employment after traumatic brain injury but did not have a higher rate of substance abuse, legal problems, or mood disorder as has been reported by others.[4,6]

Conclusion

Our investigation relied on a highly selected sample and was limited in scope by the source of the data and the size of the sample. However, these observations are still helpful for guiding the development of future prospective studies.

Vani Rao, M.D.
Jennifer R. Spiro, M.S.
Sharon Handel, M.D.
Chiadi U. Onyike, M.D., M.H.S.
Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Md.

References

1. Warriner EM, Velikonja D: Psychiatric disturbances after traumatic brain injury: neurobehavioral and personality changes. Curr Psychiatry Rep 2006; 8:73–80
2. Max JE, Levin HS, Schachar RJ, et al: Predictors of personality change due to traumatic brain injury in children and adolescents six to twenty-four months after injury. J Neuropsychiatry Clin Neurosci 2006; 18:21–32
3. Golden Z, Golden CJ: Impact of brain injury severity on personality dysfunction. Int J Neurosci 2003; 113:733–745
4. Miller LJ, Donders J: Subjective symptomatology after traumatic head injury. Brain Inj 2001; 15:297–304
5. Prigatano GP, Klonoff PS, O'Brien KP, et al: Productivity after neuropsychologically oriented milieu rehabilitation. J Head Trauma Rehabil 1994; 9:91–102
6. Tateno A, Jorge RE, Robinson RG: Clinical correlates of aggressive behavior after traumatic brain injury. J Neuropsychiatry Clin Neurosci 2003; 15:155–160

Memantine in Major Depression with Catatonic Features

To the Editor: Catatonia is a movement disorder defined primarily by a cluster of signs including immobility, mutism, and withdrawal or refusal of food and water,[1] and its presence in the elderly patient can lead to rapid medical decompensation necessitating rapid and effective treatment for this condition. Catatonia may arise from GABA-$_A$ hypoactivity, dopamine (D_2) hypoactivity and possibly glutamate *N*-methyl-D-aspartic acid (NMDA) hyperactivity.[2]

Ms. A, an 80-year-old female, was admitted to a geriatric psychiatry residential unit presenting with depressive symptoms. She had been a widow for 3 years and was brought to the hospital at the request of her four sons because they felt that she could no longer care for herself. The patient had no prior episodes of depression or any other psychiatric disorder.

Her medical history was significant for hypertension, pacemaker placement, chronic renal failure, hiatal hernia, and gastritis. She was being treated for cognitive dysfunction. Medications prior to admission were enalapril, 20 mg/day, memantine, 10 mg/day, zolpidem, 10 mg/day, risperidone, 0.5 mg/day, and omeprazole, 20 mg/day.

A clinical and neurological exam showed no additional findings. Laboratory studies did not reveal significant abnormalities, creatinine was 1.2 on admission and remained at 1.1, and BUN was 41. CT of the brain was performed without con-