ory was relatively intact. On discharge, Mr. A continued to show improvement in his cognitive functions, and 2 months after the onset of NMS, his score on MMSE was 25/30 with persistent deficits in immediate and recent memory. He still had retrograde amnesia about the events surrounding the NMS and also had anterograde amnesia with deficits in learning new verbal information. Again, no deficits were noted in remote memory.

Comment

This patient met DSM-IV criteria for NMS. The cognitive deficits seen in this patient following NMS are reminiscent of an organic amnestic disorder, a rare entity described in patients recovering from NMS.[2,3] Of interest, among all the cognitive domains, memory impairment is the only one that has been consistently reported on recovery from NMS. Whether the concurrent urinary tract infection and the resultant delirium could have contributed to this presentation cannot be entirely ruled out. However, this seems less likely as this patient continued to exhibit cognitive deficits long after recovery from the urinary tract infection. Moreover, the Physicians' Desk Reference does not suggest any association of norfloxacin with cognitive deficits. Some investigators have speculated that these persistent deficits may be a consequence of complications of NMS, such as prolonged hypoxia and extreme hyperthermia.[1] This is supported by reports of memory deficit as a neuropsychological sequela of heat stroke.[4] In conclusion, more research is encouraged to explore why memory is preferentially involved in NMS. Excitotoxicity due to glutamate surge has been implicated in ECT-induced memory dysfunction,[5] and glutamate has also been hypothesized to play a role in NMS.[6] Thus, it would be interesting to decipher the role of this neurotransmitter in long-term cognitive sequelae of NMS.

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Nabilone Could Treat Chorea and Irritability in Huntington's Disease

SIR: Huntington's disease causes chorea and psychiatric abnormalities. Psychiatric symptoms were found in one study in 51 out of 52 patients.[1] Dysphoria, agitation, irritability, apathy, and anxiety were found in above 50% of the patients sampled.

Many sources postulate that cannabinoids could have a beneficial effect on the symptoms of Huntington's disease, especially on choreatic movements.[2–4] As well as providing possible symptomatic relief in Huntington's disease, there is also some evidence[5] that cannabinoids might have a neuroprotective effect which could delay the onset of symptoms by delaying or preventing the death of striatal neurons. This neuroprotective effect has also been postulated by other sources.[6–8]

To date there are only two reports on the use of cannabinoids in Huntington's disease in the literature. Cannabidiol, a nonpsychotropic cannabinoid, had no effect on chorea severity in 15 patients.[9] In one single patient, single dose, uncontrolled open clinical trial using nabilone, 1.5mg, the chorea increased significantly.[10] We present a case of a female patient with irritability, which improved after the introduction of cannabis. This improvement was maintained by treatment with nabilone.

Case Report

The patient was a 43-year-old female who died in December 2003. She developed symptoms of Huntington's disease at the age of 24 and her husband gave up paid employment to care for her in 1990 when she was 30 years old. In 1995, he reported difficulties in caring for his wife. These difficulties were related to personality changes due to her illness. She increasingly resisted help from professionals, especially care assistants, and refused any suggestion of short-term respite care. She became disinhibited and frequently undressed herself and walked around naked inside and occasionally outside the house. She exhibited a number of dangerous behaviors, such as leaving taps running and fires burning, and leaving burning cigarettes around. Her husband became concerned about the effect that the care for his chroni-

cally ill wife was having on his son, who was born in 1984.

The patient went into residential care in 1996. The patient's husband visited two or three times every week and he always took his wife out for a trip. These trips were difficult because of the patient's refusal to be strapped into the car or her wheelchair, which sometimes resulted in falls caused by violent choreic movements when he was unable to physically hold her in the chair because he was using his hands for some other purpose.

In 2001, he began to give his wife cannabis to smoke when he took her out on these regular trips. When he returned his wife to the nursing home after these visits the staff were aware of a significant difference in the patient. The cannabis appeared to improve her mood and she was calmer and more relaxed. Prior to the introduction of cannabis she was extremely impatient and would get angry if required to wait even a few minutes for a cigarette. After taking cannabis, she was able to wait a while without screaming and throwing things. The patient also willingly accepted the use of a car seat belt and wheelchair harness.

In December 2001, the local general practitioner prescribed a regimen of nabilone, a synthetic 9-keto cannabinoid, which the patient began taking, 1mg each day. The husband and the nursing home staff both reported improvements in behavior and reduction of chorea coinciding with the introduction of cannabis and maintained by daily taking nabilone.

Comment

This report has many limitations. It is a single case report and no measurements were taken at the time of the introduction of cannabis and nabilone. The information was obtained by interviewing the husband and staff from the care home in 2005. The symptoms of Huntington's disease do change over time and the movements are different in the later stages of the disease. However both the husband and the staff are sure that the introduction of cannabis was beneficial and greatly improved the patient's quality of life in her last years. There is need for further trials to establish the therapeutic use of cannabinoids in the symptomatic treatment of Huntington's disease.

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Essential Blepharospasm Responding to Haloperidol

SIR: Blepharospasm is a disorder of adulthood that is more common in women. It presents as a sudden involuntary bilateral eye closure that is often exacerbated by air pollution, wind, exposure to bright light, movement, and stress. However, to date it is not possible to correlate it with any psychopathology. If it presents as an isolated blepharospasm in adults, it is better termed as essential blepharospasm. It must be differentiated from Meige's syndrome which includes oromandibular dystonia along with blepharospasm.[1]

Below we describe a case of essential blepharospam that responded to low doses of haloperidol but not to other drugs.

Case Report

A 32-year-old married man presented with bilateral blepharospasms that lasted for 1 to 2 minutes. The spasms were provoked by light, embarrassment, and fatigue. The spasms would disappear in sleep. These complaints were of 5-month duration.

There was no history of any chronic physical illness including neurological illnesses such as parkinsonism, Wilson's disease, epilepsy, stroke, nor a history of ocular pathology (e.g., blepharitis, conjunctivitis or iritis), any psychiatric