CASE REPORT

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Marihuana as a therapeutic agent for muscle spasm or spasticity

Marihuana has been used as a medication since ancient times, but potential uses for derivatives from the plant have only recently become the subject of great interest in Western medicine. Reports concerning benefit in treating glaucoma[1] and in controlling nausea associated with cancer chemotherapy[2] have led to major research studies with cannabis and synthetic cannabinoids. The broad range of potential uses of cannabis has been the subject of several excellent review articles.[3-5] Since the National Institute on Drug Abuse in 1977 reported an estimated 16 million Americans over the age of 12 use cannabis regularly, other claims from patients can be expected in the future. The following two cases suggest beneficial effects that have not been widely reported.

Case 1

A 30-year-old woman received multiple injuries in an automobile accident in 1970, including fractures of the pelvis, right patella, L-5 vertebra, and multiple ribs. Her recovery was complicated by persistent, severe pain and muscle spasms of the neck and low back, which were treated using medication including meperidine, pentazocine, and diazepam. When we first saw her in consultation in 1974, her neurologic examination was normal except for bilateral tenderness of the paraspinal muscles in the neck and a subtle sensory loss to light touch over the C-5 dermatome on the left. She complained that the medication caused excessive sedation, interfering with her work. In addition, the long-term consequences of combination drug use concerned her. We discussed alternative therapy with conventional drugs. Since the occurrence of the automobile accident, the woman had abstained from cannabis use.

Over a period of three months, the woman eliminated use of medication for pain and muscle spasm and substituted cannabis when symptoms occurred. She reported that the cannabis produced both analgesic and muscle-relaxant effects without the drug side-effects that had disturbed her. Nor did it produce drowsiness. In four years of intermittent use, she claims her cannabis pattern has not changed; she uses up to one cannabis cigarette every second or third evening.

Case 2

A 27-year-old man was initially referred to the neurology clinic for treatment of spasticity. Five years prior to the visit, he had experienced vision loss in his left eye. Vision gradually returned to normal in three months. One year following the first episode, he noted weakness and numbness of both legs. The weakness prevented him from climbing stairs without assistance and was made worse when he became overheated. A diagnosis of multiple sclerosis was made by a neurologist after visual evoked potential and spinal fluid studies. Again, the patient improved without treatment over several months. The only residual complaint was easy fatigability of the legs. On occasion he experienced nocturnal leg spasms. The leg symptoms associated with spasticity were not relieved with aspirin or acetaminophen. His family physician prescribed diazepam, which decreased the leg spasms but caused excessive daytime sedation. Dantrolene produced no perceptible benefit after three months.

At the suggestion of a friend, the patient tried smoking cannabis. He would smoke a single cannabis cigarette in the evening when he felt leg fatigue or spasms. Within five minutes, his symptoms would be relieved. If he did not have symptoms, he avoided cannabis. Both the patient and the referring physician were concerned about the possible

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hazard this unconventional therapy might present in a patient with multiple sclerosis.

On examination, positive physical findings were limited to the nervous system. Funduscopic examination revealed left optic pallor. The color red was noted to be pale when viewed by the left eye alone, yet confrontation acuity was normal in both eyes. Pursuit and saccadic eye movements were normal. The upper extremities were normal in strength and tone. Signs of lower extremity spasticity were prominent, including increased resistance to passive movement at the knee, brisk tendon reflexes with spread, and extensor plantar responses. Gait was within normal limits and Romberg's sign was negative. The history and examination were consistent with multiple sclerosis with involvement of the visual pathway and spinal cord. The disease was stable, with only residual leg spasticity. We asked the patient to refrain from using cannabis and to return to the clinic in six weeks.

At the time of the second visit, he described an increase in symptoms to the point where he had leg pains, increased clonic activity, and uncontrolled spasms every night. More disturbing to him was urinary incontinence, which occurred on two occasions during leg spasms. On examination, more prominent signs of spasticity were elicited, including the clasp-knife response on knee flexion and sustained ankle clonus. At the patient's request, he left the clinic and returned one hour later. This second examination was remarkable in that the earlier findings of moderate spasticity could not be elicited. Deep tendon reflexes were brisk but without spread, ankle clonus was absent, and the plantar response was flexor on the left and equivocal on the right. He admitted to having smoked a cannabis cigarette in the interval between examinations. Recalling the experience of the first case reported here, we discussed with him the known health risks from chronic cannabis use and the range of alternative therapy with conventional agents, such as dantrolene and baclofen. Despite all the unanswered questions regarding cannabis, the patient decided to continue using it. To date, he has used cannabis almost daily for more than three years without perceptible decrement in function and without exacerbation of demyelinating disease.

Discussion

When viewed objectively, the two cases presented share little in common to suggest a mechanism of action for cannabis. After we observed these two cases, four other patients with muscle spasm following trauma, and eight others with spasticity associated with multiple sclerosis, described similar benefit from cannabis.

In the 19th century, O'Shaughnessy[6] used a hemp extract in treating muscle spasms associated with tetanus and rabies. Reynolds,[7] in 1890, reported using cannabis for muscle spasm, as well as for epilepsy, migraine, and other indications. Research was temporarily stifled by the Marijuana Tax Act of 1937, but animal studies resumed in the 1940s and studies of cannabis in humans became widespread as social use grew in the 1960s.

Publications that address the potential therapeutic benefit of cannabis in diseases involving the neuromuscular system remain scant, however. A survey of ten spinal-cord-injured males who admitted using cannabis after their injury found an inconsistent effect on symptoms such as phantom pain, urinary retention, and bladder spasms. Five patients reported reduced spasticity, three patients noted no effect, and two patients did not suffer significant spasticity.[8]

The medical literature, both in animal and human studies, does indicate that cannabis produces effects on the motor system. Studies in healthy adults indicate that cannabis has an effect on performance of skilled motor tasks such as driving. Experienced cannabis users tend to show improvements in performance, which may suggest tolerance to the effects.

Along with drug tolerance, other issues that remain unresolved include the long-term effects of cannabis on memory and intellect, its pulmonary effects, and risks in pregnancy. In cases of cerebral infarction causing spasticity, the cognitive impairment induced by cannabis may cause decompensation. In addition, in cases with cerebellar involvement, cannabis may further impair central compensatory pathways.

Despite these possible problems, the potential for therapeutic application of cannabis in neurologic disease remains a subject of considerable interest. If our experience is any indication, cannabis use by patients with neurologic disease may already be widespread as an alternative to traditional medicine. The long-term implications of this therapy are as yet unknown. □

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