Akinetic mutism caused by nicotine withdrawal

Sir,

A 50-year-old right-handed, hypertensive, nondiabetic male was admitted with left hemiplegia and coma. Patient used to chew heavy amounts of tobacco throughout the day for the last 20 years. The CT scan of head showed right thalamic bleed with intraventricular extension. Patient was managed conservatively. The fifth day onwards the patient started opening his eyes spontaneously, but not vocalizing at all. Spontaneous motor activity was lacking but he could localize the pain with the right upper limb. His repeat CT scan showed resolving hematoma with no midline shift or hydrocephalus. No recovery occurred after that. A metabolic disorder or systemic infection was suspected. Relevant investigations ruled out the same. Post-stroke depression was another differential but the patient did not show any recovery even after three weeks of antidepressant treatment. Empirical course of broad-spectrum antibiotics and dopa-agonists did not do any good. Now a remote possibility of nicotine withdrawal was considered. As transdermal nicotine patch was not available, patient was given the same brand of tobacco to chew to which he was addicted. The patient responded dramatically and on the second day the patient became alert and started speaking. On the fourth day the patient started walking and was discharged from the hospital.

Akinetic mutism is a condition of silent alert-appearance immobility without recognizable content. This condition can be due to lesions which interfere with reticular-cortical pathways (i.e., lesions involving bilateral frontal lobe or communicating hydrocephalus). Dopamine-blockers, central nervous system infections, anoxia and withdrawal from certain drugs are other causes. Akinetic mutism has never been reported following nicotine withdrawal. Nicotine is the main addictive component of tobacco. \(^1\) Signs of nicotine withdrawal in neurologically normal tobacco addicts include depressed mood, slowed cognition and sleep disruption. \(^2\) As opposed to milder symptoms, altered sensorium may be more likely to occur in patients with underlying structural neurocognitive deficits. \(^3\) Nicotine stimulates dopamine transmission, which mediates its reinforcing effects. \(^4\) Withdrawal from tobacco diminishes activity of dopamine projections, which is responsible for the symptoms caused by tobacco abstinence. It is claimed that nicotine patches are effective in alleviating nicotine withdrawal symptoms. However, in recent studies withdrawal symptoms were found to be significantly lower with tobacco than with nicotine replacement therapies. \(^5\) Our patient responded to tobacco supplementation, though it is not an established form of therapy.

We cannot prove that our patient was akinetic because of nicotine withdrawal, as akinetic mutism might have been related to disruption of the thalamocortical circuit and the observed improvement after tobacco supplementation might have been coincidental. But, bilateral lesions involving this circuitry are required to cause akinetic mutism, of which there was no radiological evidence. The recognition of akinetic mutism as a manifestation of nicotine withdrawal could allow applying the appropriate therapy, in certain complex undiagnosed cases. However, in the light of experimental evidence that nicotine may increase the thrombogenic potential of the cerebral endothelium, the efficacy and safety of any form of nicotine supplementation therapy needs to be established.

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References


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