Understanding and Treating Tardive Dyskinesia

April 7, 1982

Tardive Dyskinesia


Tardive Dyskinesia: A Neurological Disorder

Tardive Dyskinesia is a serious neurological disorder that can occur as a result of prolonged use of certain medications, particularly antipsychotics. The symptoms of tardive dyskinesia can vary widely, but common signs include persistent movement of the mouth, tongue, lips, jaw, or neck, as well as abnormal movements of the arms, legs, or other body parts. The condition can be disabling and can significantly affect a person's quality of life.

Understanding the Pathophysiology of Tardive Dyskinesia

Tardive dyskinesia is a movement disorder that occurs as a result of long-term exposure to antipsychotic medications. The pathophysiology of tardive dyskinesia involves the dysfunction of the basal ganglia, a group of structures in the brain that are involved in the regulation of movement. Antipsychotics can alter the balance of neurotransmitters in the brain, leading to the development of tardive dyskinesia.

Signs and Symptoms of Tardive Dyskinesia

The signs and symptoms of tardive dyskinesia can be difficult to diagnose, as they can vary widely from person to person. Common symptoms include persistent movement of the mouth, tongue, lips, jaw, or neck, as well as abnormal movements of the arms, legs, or other body parts. Other symptoms may include speech problems, such as slurred speech or difficulty in starting speech.

Diagnosis and Treatment of Tardive Dyskinesia

The diagnosis of tardive dyskinesia is typically based on the patient's medical history and a thorough examination by a healthcare provider. Treatment options for tardive dyskinesia may include discontinuing or reducing the dose of the antipsychotic medication, as well as using medications or other treatments to manage the symptoms of the disorder.

Prevention of Tardive Dyskinesia

Preventing tardive dyskinesia is important, as the condition can be difficult to treat once it has developed. Preventive measures may include careful monitoring of patients who are receiving antipsychotic medications, as well as close communication between healthcare providers and patients to ensure that the benefits of the medication outweigh the risks.

Tardive Dyskinesia: A Multidisciplinary Approach

Tardive dyskinesia is a complex disorder that requires a multidisciplinary approach to management. This approach may involve working with healthcare providers from a variety of fields, including psychiatrists, neurologists, and physical therapists, to help manage the symptoms of the disorder and improve the quality of life for those affected.

Conclusion

Tardive dyskinesia is a serious and disabling condition that can significantly affect a person's quality of life. Early recognition and prompt treatment are crucial to managing the symptoms of this disorder. Healthcare providers and patients must work together to ensure that the best possible care is provided for those affected by tardive dyskinesia.
Tardive dyskinesias (TDs) are involuntary movements of the tongue, lips, face, trunk, and extremities that occur in patients treated with long-term dopaminergic antagonist medications. Although they are associated with the use of neuroleptics, TDs apparently existed before the development of these agents. People with schizophrenia and other neuropsychiatric disorders are especially vulnerable to the development of TDs after exposure to conventional neuroleptics, anticholinergics, toxins, substances of abuse, and other agents. TDs are most common in patients with schizophrenia, schizoaffective disorder, or bipolar disorder who have been treated with antipsychotic medication for long periods, but they occasionally occur in other patients as well. For example, people with fetal alcohol syndrome, other developmental disabilities, and other brain disorders are vulnerable to the development of TDs, even after receiving only 1 dose of the causative agent. TD has been associated with polymorphisms of both the dopamine receptor D2 (DRD2) gene, \[1\] TaqI A and TaqI B and associated haplotypes, [2] and of the dopamine receptor D3 (DRD3) gene, [1, 3] the dopamine transporter (DAT) gene, and the manganese superoxide dismutase (MnSOD) gene. Dysfunction of the dopamine transporter has been hypothesized to play a role in the development of TDs. However, Lafuente et al. did not find evidence of involvement of a polymorphism with a variable number of tandem repeats (VNTR) in the DAT gene (SLC6A3) in dyskinesias induced by antipsychotics [4]. Thus, further research is needed to investigate the role of the dopamine transporter in the development and maintenance of TD. Galecki et al. reported the association of a polymorphism of the manganese superoxide dismutase (MnSOD) gene and TD. [5] TDs may be differentiated from acute movement disorders that commonly occur in the same patient groups. The acute movement disorders that occur as exacerbations of effects of cocaine and other dopamine antagonists include akathisia, acute dystonia, and other hyperkinetic dyskinesias. Acute effects of dopamine antagonists on the sufferer include performance (cognition) abnormalities, hallucinations, personality changes, and mood changes [6]. The occurrence of acute movement disorders on exposure to dopamine antagonists is increased in male patients and older patients. Use of cholinergic dopamine antagonists, prolonged exposure to dopamine antagonists, and prior occurrence of acute movement disorders on exposure to dopamine antagonists are also associated with an increased risk for the occurrence of acute movement disorder effects. Withdrawal dyskinesias may also occur in individuals with dopamine antagonists in decreased or withdrawn. They occur following treatment with dopamine antagonists and include a spectrum of hyperkinesias occurring during or after prolonged treatment with dopamine antagonists. Get this Book now! to know how Medical Marijuana and CBD oil totally cure Tardive Dyskinesia.