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Current Use and Trends in the Management of Tardive Dyskinesia: Role of VMAT2 Inhibitors

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ABSTRACT: Objectives: Tardive Dyskinesia (TD) is a debilitating condition that requires prompt care and intervention. Studies demonstrated the probable role of Vesicular Monoamine Transport 2 (VMAT2) in the pathogenesis of TD and use of VMAT2inhibitors in managing TD. Our aim is to provide available data on the management of TD and to determine the efficacy ofvarious VMAT2 inhibitors for independent use. Also, to identify their use in combination and assess if there, any change inoutcome with early intervention.

METHODS: We did a pivotal search of the scientific literature by querying PubMed and Google Scholar for studies on treatment modalities of TD including atypical antipsychotics and VMAT2 inhibitors. Also, references from publications were accessed for review.

RESULTS: Early detection and prevention are of paramount importance in managing TD. Cessate the antipsychotic been using andother dopamine blocking agents that probably implicated in causing the symptoms. Certain studies showed the use of new atypical antipsychotics like Paliperidone, Quetiapine in small titrated dose resolving the symptoms. According to the available data, using benzodiazepines, botulinum toxin injections and VMAT2 inhibitors like Valbenazine, Tetrabenazine, andDeutetrabenazine also managed TD with efficacy. Valbenazine has breakthrough global approval in resolving the symptoms. Although other VMAT2 inhibitors were used for TD earlier and showed to be effective in managing TD, larger trials are required showing their safety and reliability in efficacy.

CONCLUSIONS: VMAT2 inhibitors were tested as efficacious in managing TD. Valbenazine and recently deutetrabenazine has been approved by US Food and Drug Administration (FDA) to treat TD. However, Tetrabenazine is yet to be approved by FDA. More clinical trials are required exploring their efficacy by comparing them or using them in combination. Our review also suggests timely detection and earlier intervening, especially if witnessed in children and adolescents would differ the outcome of TD.

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Guanfacine and Impulsivity – Review of Literature

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ABSTRACT: Prefrontal cortex (PFC) represents one of the most evolved regions of primate brain that is thought to regulate human specific features such as cognition, emotion and behavior (Arnsten and Jin, 2012). PFC is a site of action of guanfacine, an agonist of alpha 2 adrenergic receptors. Compared to clonidine, another alpha adrenergic drug, guanfacine is more selective for α2A adrenergic receptor subtype (van Zwieten et al., 1994; Uhlen at al., 1995) and is weaker in producing hypotension andsedation (Jurado at al., 1998) resulting in better tolerability of the medication. Studies have shown that endogenous noradrenergic stimulation of alpha2A receptors is essential for PFC regulation of behavior, thought and emotion as blockade ofα2A receptors in the monkey dorsolateral PFC significantly impairs working memory (Li and Mei, 1994) and behavioral inhibition (Ma et al., 2003; Ma et al., 2003). So far FDA has approved guanfacine in treatment of attention deficit hyperactivity disorder in children but the medication is used off label for treatment of oppositional defiant disorder, conduct disorder, pervasive developmental disorders, motor tics and Tourette's syndrome as well. Impulsivity as used in clinical terms is very broadly defined and encompasses personality traits as well as cognitive functions such as emotion regulation and behavioral inhibition. Numerous studies have shown effectiveness of extended release guanfacine in reducing impulsiveness in children with ADHD and recently in autism spectrum disorder (Scahill et al., 2015), however limited data is available on use of guanfacine in treatment of impulse control and aggression in adults.

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Who Am I: Delusion of Misidentification of Self

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