

Case Report

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Rare but Treatment-emergent Extrapyramidal Symptoms-related Adverse Events after Administration of Long-acting Injectable Paliperidone Palmitate

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Paliperidone palmitate, the long-acting injectable form of paliperidone (PLAI), is approved for once-monthly intramuscular injection [1]. Several studies suggest that the loading dose regimen for PLAI does not lead to higher rates of extrapyramidal symptoms (EPS)-related treatment-emergent adverse events (TEAEs) than the oral formulation [2]. However, reports of post-injection delirium/sedation syndrome (PDSS) by olanzapine LAI raise safety issues of LAI. We report a case, which demonstrates that EPS-related TEAEs can be quite refractory to a number of pharmacological interventions and persist for more than 3 months following PLAI administration.

A 53-year-old man was admitted with first episode schizophrenia with persecutory delusions and auditory hallucinations. The patient was started on amisulpride 200 mg, which was titrated up to 800 mg per day. By the 25th day of admission, before he was discharged, the treatment with 800 mg amisulpride proved successful. However, due to poor drug compliance he was switched to PLAI. The initiating dose of PLAI is 150 mgEq given on treatment day 1 followed by 100 mgEq on day 8. Amisulpride dose is 400 mg given on treatment day 1 and tapered by 0 mg on day 8. He developed acute dystonia of the orofacial muscles, akathisia, and swallowing problems. The dystonia and akathisia reached their peak intensity on the 7th day after the 2nd dose of PLAI, when he was transported to the emergency room by his family. A brain MRI, neurological examination, and an electrolyte and toxicology screen yielded negative results. Subsequently, the patient underwent acute hospitalization; he received an intramuscular injection of 4 mg lorazepam once and an oral dose of propranolol 60

mg, benztropine 2 mg, and alprazolam 0.75 mg per day for one week. A week later, his condition appeared to improve when he was orally administered 4 mg of benztropine and 2 mg of diazepam. After 2 weeks, the dystonia subsided, with a significantly lesser improvement in akathisia even after 3 months of treatment.

A possible explanation could be that the PLAI rapidly spread in the blood stream, causing an overdose, which manifested in the form of dystonia and akathisia. This mechanism could be similar to that of the PDSS seen after treatment with olanzapine LAI [3]. Early recognition and adequate management are mandatory because EPS-related TEAEs have been associated with poor compliance and aggravation of psychotic symptoms, aggressiveness, and suicidality. Clinicians should be aware of the potential of PLAI to produce dystonia and akathisia. These distressing symptoms may persist for several months despite discontinuation of the drug, and the akathisia may be relatively refractory to standard interventions. In addition, slow distribution from deep tissues may lead to accumulation, making management of side effects challenging and possibly requiring discontinuation of treatment.

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