CASE REPORT

Acute dystonic reaction due to dexketoprofen trometamol

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Abstract

Dexketoprofentrometamol (DKP), is a tromethamine salt of the water-soluble S-enantiomer of ketoprofen. As with all other non-steroidal anti-inflammatory agents, the most common side effect of DKP is gastric complications. In this paper, we report a case of dystonic reaction after intravenous DKP use. A 24-year-old man was admitted to our hospital after suffering a leg burn from boiling oil. He had no drug hypersensitivity. An intravenous preparation containing the active ingredient DKP was injected for analgesia, after which the patient experienced an involuntary flexion response in both upper extremities. With a suspected diagnosis of dystonia, biperiden lactate 5 mg/ml was administered via the intramuscular route and the contractions abated within 30 seconds of the injection.As non-steroidal anti-inflammatory agents are commonly used and prescribed in emergency departments, it should be kept in mind that an acute dystonic reaction can develop against one of these agents, DKP.

Keywords: Acute dystonia, Dexketoprofen, Emergency.

Introduction

Dexketoprofentrometamol (DKP), is a tromethamine salt of the water-soluble S-enantiomer of ketoprofen. It reaches its peak plasma concentration (Cmax) approximately 30 minutes after oral intake. Due to its rapid onset of action, it is very beneficial in the management of acute painful conditions.[1]

DKP is a non-specific cyclooxygenase inhibitor that acts primarily by inhibiting prostaglandin synthesis. Being highly lipophilic, it can cross the blood-brain barrier. By this route, it activates the serotonin (5-hydroxytryptamin) system to increase serotonin release, which exerts an analgesic effect.[2] In Turkey tablets are available in 12.5, 25, and 50 mg forms and a parenteral form containing 50mg/2 ml DKP.

As with all other non-steroidal anti-inflammatory agents, the most common side effect of DKP is gastric complications.[1] In this paper, we report a case of dystonic reaction after intravenous DKP use.

Case Report

A 24-year-old man was admitted to our hospital after suffering a leg burn from boiling oil in May 2014. His past history was unremarkable and he had no drug hypersensitivity. On physical examination, he appeared to be in good health and his vital signs were stable (blood pressure=125/75 mmHg, pulse=76 per minute, respiratory rate=18 per minute, pulse oxygen saturation= 98%). He had a 5x5cm second degree burn on the dorsal surface of his foot. The wound was covered with dressings and tetanus prophylaxis was applied. An intravenous preparation containing the active ingredient DKP was injected for analgesia, after which the patient experienced an involuntary flexion response in both upper extremities. With a suspected diagnosis of dystonia, biperiden lactate 5 mg/ml was administered via the intramuscular route and the contractions abated within 30 seconds of the injection. The patient later stated that he had experienced a similar reaction to the same drug before. However, during that episode he was not administered any drug to antagonize the effect of DKP.

Discussion

Dystonia is a syndrome considered to be secondary to basal ganglion abnormalities and cortico-striate-thalamo-cortical pathway dysfunctions, which are characterized by involuntary, excessive, and prolonged muscle contractions.[3] It may be a primary disorder, but it may also be observed in the form of acute dystonia minutes, hours, or even days after the use of a triggering agent.[4]

Many drugs have been reported to induce dystonic reactions as a result of dopamine D2 receptor blockage; it has been emphasized by various studies that the properties and dose of a drug and an individual's sensitivity to that drug affect the emergence of dystonic reactions.[5]

Serotonin is a neurotransmitter that is involved in taking part in endogenous pain control by inhibiting painful stimuli in the central nervous system.[6] It has been reported that serotoninergic activity suppresses dopaminergic cell activity and synaptic release. It has also been suggested that it suppresses dopamine synthesis in

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the midbrain, striatum, and cortex.[7] This effect is primarily based on the competition of tryptophan, the precursor of serotonin, with tyrosine, the precursor of dopamine, to cross the blood-brain barrier. As a result, an increased serotonin level reduces dopaminergic activity. It has been advocated that these mechanisms are responsible for the dystonia-like extrapyramidal reactions caused by selective serotonin reuptake inhibitors.[8] We suggest that dystonia in our case emerged from a reduced dopaminergic activity led by the serotoninergic mechanisms triggered in turn by DKP. We believe that some genetic factors in our case underlie the emergence of dystonia, which has not been reported in any patient before despite the widespread use of DKP. Hence, the presence of the A1 allele of the D2 dopaminergic receptor gene polymorphism has been indicated as a possible factor for the emergence of certain movement disorders such as dystonia.[4]

According to the Naranjo algorithm[9] that determines the probability of drug side effects, our patient scored seven points, with two points for the emergence of dystonia after taking DKP, one point for recovery from dystonia after administering biperiden lactate, two points for the absence of any alternative explanation for dystonia, one point for a history of a similar reaction to DKP in the past, and one point for the objective confirmation of dystonia by neurological examination. According to that algorithm, 9 points or above are considered a definite drug side effect, 5-8 points a probable drug side effect, 1-4 points a possible drug side effect, and 0 points a suspected drug side effect. In conclusion, we suggest that dystonia was probably secondary to DKP in our case.[9]

As non-steroidal anti-inflammatory agents are commonly used and prescribed in emergency departments, it should be kept in mind that an acute dystonic reaction can develop against one of these agents, DKP.

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