

Transition to Buccal Buprenorphine in a Young Adult with Hereditary Kaposiform Lymphangiomatosis After Extended Use of Oxycodone

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Introduction

Kaposiform lymphangiomatosis (KLA) is a rare genetic disorder characterized by abnormal proliferation of lymphatic vessels, predominantly affecting children and young adults. KLA may be associated with significant morbidity and mortality, imposing considerable social and medical burdens on affected individuals. [1] This details the case of a 20-year-old male with KLA and pain secondary to bone involvement who had been managed with long-acting, high-dose opioid medications but was rotated to buccal buprenorphine.

Methods

IRB review was waived per institutional policy.

Case Presentation

The subject was diagnosed with KLA at age 17 and suffered from chronic hip and back pain due to pelvic lymphatic congestion and T9, T10, and L2 lesions. His pain was managed by his pediatric oncologist with oxycodone ER (20 mg BID) and oxycodone (5 mg occasional use). Given his age and life expectancy, it was requested he transition to adult care under a pain specialist. While treatment with full-agonist opioids provided acceptable pain control at rest, his pain would flair upon exertion, and it also caused significant sedation. He reportedly slept up to 18 hours a day, affecting his ability to attend classes as a college student. After careful evaluation and discussion, it was decided to transition to buprenorphine for pain control while attempting to mitigate long-term opioid risks and side effects. Oxycodone ER was weaned down over a few months, to 30 mg daily with PRN oxycodone available, before he was transitioned to buccal buprenorphine. His use of oxycodone increased to a few times a day most days when weaning oxycodone ER (daily total OME<70 mg). He was started on buccal buprenorphine 150 mcg BID PRN with Tizanidine 2mg TID PRN for withdrawal symptoms. He denied any significant withdrawal as he started the rotation. Initial pain control was acceptable, but buprenorphine was increased to 300 mcg BID PRN after a couple of months when requested. The sedation he had experienced with oxycodone resolved. Pain control improved to the point that he discontinued pregabalin. Sufficient pain control and quality of life have persisted without further dose escalation of buprenorphine.

Discussion

Side effects and risks related to chronic use of opioid medications present challenges independent of pain relief. Buprenorphine is a partial mu-agonist-, weak kappa-antagonist, and delta-agonist. This results in comparable analgesia to schedule II opioids, with slow dissociation, a ceiling effect for respiratory depression, and decreased risk of constipation, immunosuppression, and hypogonadism. [2, 3] It has been shown that patients on full agonist therapy may be safely transitioned to buprenorphine without increased risk of withdrawal or loss of efficacy. [4, 5] There may be theoretically decreased risk of other long-term opioid-related side effects due to the unique pharmacology. These were all considered when rotating to buprenorphine in this patient.

Discussion Continued

Due to poor bioavailability, the preferred formulations for treating chronic pain include buccal, sublingual, or transdermal. Consider buprenorphine in patients where a traditional mu- agonist might be considered for long-term use or to help mitigate opioid-related side effects in those already using chronic opioid therapy.

References

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