Transition from Injectable to Percutaneous Local Anesthetic Therapy in a Patient with Post-Herpetic Neuralgia

To THE EDITOR

In 1980, a 35-year-old woman with a history of a systemic vasculitis being treated with prednisone 30 mg per day was referred to the Charity Hospital of New Orleans / Louisiana State University Department of Anesthesiology for treatment of pain associated with an outbreak of acute herpes zoster in the left mid-cervical dermatomes. Blisters were sufficiently confluent that the epidermis of the entire affected area had become a continuous area of weeping exudate. Her severe pain was not being relieved with acetaminophen plus codeine. Each of a series of 5 stellate ganglion blocks with 15mL 0.25% bupivicaine afforded relief lasting several days. Within several weeks the skin had healed with only minimal scarring; however, pain in the affected area did not abate sufficiently to permit treatment with non-prescription analgesic medications (1).

As long-term repetition of stellate ganglion blocks was not feasible in this patient, for the next 14 years, periods of pain exacerbation (approximately 4 to 8 episodes per year) were treated with fanlike injections of 20 - 40 mL 0.25% bupivacaine into the subcutaneous tissue within the affected dermatomes using a 3.75 cm 27 gauge needle.

In 1994, local anesthetic injections were discontinued in favor of self-administered topical percutaneous local anesthetic therapy by self-application of 9% lidocaine (base) in a petrolatum/paraffin vehicle (bLIPP) (2).

Three years into this phase of local anesthetic palliative pain management, 3% tetracaine (base) in petrolatum/paraffin was substituted for 9% lidocaine but within 4 months, this resulted in the development of erythema, induration, and pain in the area to which the ointment was being applied. Sensitivity to repeated administration of a para-aminobenzoic acid-type local anesthetic was diagnosed and topical percutaneous local anesthetic therapy with 9% bLIPP recommended.

Over the next 16 years the patient continued to

obtain good relief from 9% bLipp, sometimes supplemented with non-prescription pain medications. In 2010, an attempt was made to switch the patient to a (then) relatively new commercial 5% (base) lidocaine patch (Lidoderm TM). Perhaps because of a slower lidocaine release rate from this patch, the patient asked her local anesthetic treatment be switched back to 9% bLIPP and home application of this treatment has provided gratifying relief during the next 5 years. Once the supply of lidocaine (base) crystals from the LSU Department of Anesthesiology's became depleted, the BLIPP recipe was communicated to a local compounding pharmacist who afterwards has provided it to her until the present time. The patient has never experienced any symptoms of cutaneous irritation of systemic local anesthetic toxicity from this form of therapy.

The authors wish to communicate to pain specialists a couple of critical points based on the decades long treatment of this patient and a literature review. First, soon after the realization that topical formulations of the base (uncharged) form of some local anesthetic drugs could effectively anesthetize the dermis a 2.5% (base) lidocaine / 2.5% prilocaine cream (EMLA) was marketed to reduce the pain of venipuncture in children (3). Other non-commercial formulations have been developed that have been proved sufficiently powerful to eliminate intra-procedural pain of skin grafting (4). Because EMLA dries and becomes ineffective if not constantly covered with a non-water-permeable dressing and because inclusion of prilocaine entails some risk of methemoglobinemia, it may not be suitable for other than short-term use to reduce pain of medical procedures invading the dermis (5) [approximate maximum depth of anesthesia 5mm (6)]. Availability of a topical percutaneous lidocaine-releasing patch (Lido-derm) permitted effective of the cutaneous component of post-herpetic neuralgia in some patients (7). For patients requiring delivery of higher hourly amounts of lidocaine, a custom formulation such as used in our patient may be necessary to provide relief.

Additionally, the maximum rate of lidocaine that can be safely administered to an individual patient can be difficult to predict. There is one case report of a patient wearing a 5% Lidoderm patch experiencing symptoms of non-life-threatening, but clinically significant, systemic local anesthetic toxicity after falling asleep on a heating pad (8). Patients with decreased ability to metabolize lidocaine (liver failure, congestive heart failure (9) should be expected to tolerate lower doses than healthy patients. Since maximum blood lidocaine levels usually plateau within a day following application of a topical percutaneous lidocaine formulation (2), if it is learned that an individual patient obtains valuable relief from a percutaneous lidocaine formulation, but concern about systemic toxicity exists based on either the area of application or drug concentration (i.e. release rate) or the presence of a medication that would slow metabolism, one method of estimating safety (under at least baseline conditions) could be to admit the patient to a hospital with a toxicology laboratory and obtain serial blood lidocaine levels at several-hour intervals after application.

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