Injection nerve palsy: What’s to blame?

Sir,

We read with interest the article “Injection nerve palsy” by Kakati et al. and appreciate their meticulous study on nerve injection injuries (NII). Critically, several mechanisms of NII that may play a role in the nerve damage were not fully explored in their article. It has previously been shown that injection pressure is a factor in the degree of NII with high pressures (>11 psi) causing more damage than those with low pressures (<11 psi). This measurement is affected both by the gauge of the needle used and the size of the barrel used, with a smaller gauge and larger barrel leading to a higher pressure of drug delivery. Additionally, the needle tip can play a role in the degree of damage. While tapered needles generally do not cause as much damage, in the case of beveled needles it is critical to insert the needle parallel to the nerve fibers as this leads to reduced chances of nerve damage. Also, the authors have only briefly described the importance of chemical injury in the pathogenesis of NII. It is worthwhile to mention here that in many instances the peripheral neuritis and nerve damage have been seen to occur even with the use of correct technique of injection. Chemical neurotoxicity of the agent injected has been blamed in such cases, as has also been suggested by a number of animal studies. This concept was explored by Gentili et al. who injected different compounds into nerve fascicles of rats and found that penicillin G, diazepam, and chlorpromazine were the most neurotoxic, damaging even the extrafascicular nerve fibers, while minimal damage was caused by injections of iron-dextran, meperidine and cephalothin. Also, the quantity of drug injected dictated the degree of injury. In a recent study by Senes et al., the authors created a list of drugs and their comparative neurotoxicities. They found that hydroxybenzoate, alcohol and diacetin were the most neurotoxic, sodium cefuroxime and phenobarbital were moderately neurotoxic, while naloxone (with hydroxybenzoate), vitamin K (with glycocholic acid and lecithin), and glycocholic acid and lecithin when used individually were comparatively least neurotoxic. Others have gone on to show the various mechanisms by which nerves can be damaged by the agent. Steroids cause direct toxicity on peripheral nerve fibers and cause ischemic changes in the nerve. Penicillin has been shown to cause granuloma formation when given into sciatic nerves in dogs. Lidocaine, procaine and tetracaine split the myelin lamellae and cause more severe damage than bupivacaine. It is not unreasonable to think that the differing neurotoxicities of drugs as well as how these drugs are delivered could have effects on patient recovery both, in cases that resolve on their own and those that do or not improve with surgery. Though Kakati et al. have mentioned that there were no data available regarding the nature of drugs their patients received or the precise way in which they received them, it would be interesting to know what role these factors play if at all.

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References