

Radial Nerve Injury after Venipuncture

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Venipuncture is the most commonly performed invasive procedure in hospitals daily.¹ Nerves in the antecubital fossa classically lie on a plane just beneath and in close proximity to the veins, making them susceptible to injury during phlebotomy.¹ Cadaveric studies have demonstrated a great deal of variability in the nerves of the antecubital fossa in relationship to veins, suggesting that even a nontraumatic, straightforward venipuncture can directly damage a cutaneous nerve.² Despite this, there remains a relative paucity in the literature reporting cases of peripheral nerve injury secondary to venipuncture. Of the few reported cases, an even smaller percentage of these have been validated with electrodiagnostic evidence of injury. The majority of these studies report injury to the lateral antebrachial cutaneous nerve. To the best of our knowledge, there has been only one reported case in current literature of radial nerve injury.

We report the case of a 51-year-old female who experienced a sharp, painful, burning sensation down her arm at the time of needle insertion into her antecubital fossa during routine venipuncture. She immediately complained of numbness, tingling, and weakness of her right hand. On physical examination, there was evidence of multiple puncture sites at the right antecubital fossa. All compartments of the hand and forearm were soft and nontender. There was no evidence of hematoma, ecchymosis, or erythema. She exhibited weakness in extension of the right wrist and digits. There was decreased sensation to pin prick at the dorsal first web space and over the dorsal thumb. Multiple imaging studies were obtained by the primary service, including an X-ray, venous duplex, and a computed tomography scan. All studies failed to reveal evidence of an acute injury. After 3 days of no improvement with conservative management, nerve conduction studies were performed. Sensory study of the right radial nerve was within normal limits. The distal motor latency and nerve conduction velocity of the right radial nerve was normal but showed significantly reduced amplitude. Electromyography (EMG) showed evidence of

increased insertional activity without evidence of denervation activities in the right extensor indicis (EI), extensor digitorum communis (EDC), and brachioradialis (BR). Motor unit recruitment was reduced in the right EDC and absent in the right EI. These findings confirmed the diagnosis of incomplete right radial nerve injury with possible neurapraxia versus axonotmesis. There was no evidence of denervation or axonal injury. The patient was instructed to obtain repeat studies in 4 to 6 weeks and reassured that full recovery was anticipated. Strengthening and desensitization exercises were performed daily, and a mild improvement in right wrist extension was noted at the time of discharge 10 days after her initial nerve injury.

Clinical manifestations of radial nerve injury are dependent upon the level in which the injury has occurred. The patient in our study demonstrated both motor and sensory deficits affecting the hand and wrist, suggesting an injury proximal to the bifurcation of the nerve occurring in the area of the antecubital fossa. Electrodiagnostic testing remains paramount when attempting to confirm the location of a radial mononeuropathy. In our patient, the decreased amplitude of the radial nerve action potential observed during nerve conduction velocity testing is suggestive of radial neuropathy. This finding is further supported by the EMG findings of increased insertional activity in the radially innervated EI, EDC, and BR, as well as the lack of motor unit recruitment in the EI and the reduced motor unit recruitment in the EDC.

While poorly represented in the literature, peripheral nerve injuries have been described after both routine venipuncture and in the blood donation population. Anecdotal reports of difficult access requiring multiple attempts often precede the nerve injury.² When reviewing the blood donation population specifically, the incidence of nerve injury was found to be between 1 in 21,000 and 1 in 26,000 venipunctures.³ The majority of these injuries are self-limiting and resolve spontaneously. Newman and Waxman found

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that 70, 90, and 96% of venipuncture-related nerve injuries resolve within 1, 2 and 6 months, respectively.³ However, chronic disabling deficits have been reported at an incidence of 1 in 1.5 million phlebotomies.⁴ Horowitz found that 87% of patients who require ongoing care by pain management specialist continue to experience some degree of permanent nerve damage.²

While traumatic venipuncture may lead to hematoma development resulting in extrinsic nerve compression, it was found that this mechanism occurs in only 24% of patients,³ suggesting direct nerve injury from the needle as the more commonly etiology. As a result, it remains the practitioner's responsibility to use caution during this routine procedure. Furthermore, a diagnostic work-up is warranted in the

patient who complains of weakness, burning or any degree of paresthesia during, immediately after, or within the first week following routine venipuncture.

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