Sonographic Diagnosis and Treatment of a Median Nerve Epineural Hematoma Caused by Brachial Artery Catheterization

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Percutaneous brachial catheterization has become an alternative method for carotid and vertebral angiography. Brachial artery catheterization carries risk of injury to the adjacent median nerve with secondary epineural hemorrhage, especially in patients receiving anticoagulants. In the case of a mobile cubital brachial artery, it would lead to several attempts of misleading puncture injuries to the adjacent median nerve. Needle injury may result in epineural hemorrhage and then compressed bundles of nerve fibers and may impair their function. Epineural hemorrhage could even occur as a subacute crescendo pattern. Here we report such a rare case. High-resolution sonography was used to diagnose the epineural hemorrhage. Furthermore, sonographically guided percutaneous fine-needle aspiration (FNA) was promptly performed to achieve dramatic symptom relief.

Case Report

A 73-year-old man received warfarin (2 mg) and aspirin (100 mg) daily for 2 years for stroke prevention because of the presence of crescendo fluctuating symptoms suggesting a vertebrobasilar insufficiency. Anticoagulation intensity was ensured by serial prothrombin time measurement (international normalized ratio [INR], >1.5). Carotid and vertebral angiography was then arranged for attempted angioplasty.

Brachial arterial puncture was done with an 18-guage Potts-Cournand needle. Several attempts at arterial puncture were made because of the patient's mobile brachial artery, which led to 3 transient puncture-provoked neuralgic tingling irritations (radiating from the elbow to the second and third fingers). The tingling sensation was consistent with irritation of the adjacent median nerve by the needle. Even though the cerebral angiography was performed successfully, and segmental stenosis of the left vertebral artery was disclosed, the
The patient was discharged 6 hours later, and the vertebral angioplasty was arranged for the next admission.

Over the next 4 days, his daily warfarin dose (2 mg) was maintained, and the puncture site appeared normal. On the fifth day, acute onset of a sensorimotor deficit over the median nerve territory developed, with a crescendo intensity and a local Tinel sign. On manual muscle testing, the right abductor pollicis brevis (APB) was graded Medical Research Council grade 2 (M2), the flexor digitalis profundus was graded M2, and the flexor carpi radialis was graded M3. The problem became steadily worse, which prompted hospitalized urgent investigation. The patient’s prothrombin time was 40 seconds (INR, 2.0).

The nerve conduction studies on the seventh day disclosed that the right median nerve conduction velocity was reduced to 40.5 m/s with a temporal dispersed compound motor action potential of the APB on elbow stimulation compared with 60.0 m/s on the left. The needle electromyography did not show an active denervation pattern at the APB. Electrophysiologic studies suggested segmental demyelination at the median nerve of the elbow.

High-resolution sonography was performed with a 15-MHz linear probe (HDI 5000; Philips Ultrasound, Bothell, WA). In an axial scan, an anechoic hard nodule beneath the epineurium at the puncture site was shown (Fig. 1), which resumed a fusiform pattern along the axis cylinder. In a sagittal scan, an intraneural nerve bundle was constricted and flattened by this hematoma. Furthermore, considerable nerve swelling immediately proximal to this constriction was evident. In a continuous axial scan, obliteration of the normal reticular pattern from the endoneurium was also noted, indicating the axonal swelling. Sonographic findings were compatible with the clinical signs of acute entrapped focal neuropathy. Sonographically guided percutaneous FNA with an 18-gauge needle was then performed (Fig. 2), and 1 mL of dark liquid blood was aspirated, which indicated the diagnosis of an epineural hematoma. The volume of this epineural hematoma was immediately reduced on sonography, and the motor and sensory functions of his right forearm and fingers gradually improved.

Second and third sonographically guided FNAs were performed 7 and 14 days later. The patient’s muscle power was improved (the APB was M4, flexor digitalis profundus M4, and flexor carpi radialis M5). The size of the epineural hematoma was considerably reduced, and retrograde endoneural swelling was markedly improved (Fig. 3). The nerve conduction velocity of the right median nerve was restored to 55 m/s, and compound motor action potential dispersion also disappeared concurrently.

**Discussion**

The percutaneous brachial approach is commonly used for carotid and vertebral angiography. The brachial approach, being a safe method, is especially useful in routine outpatient catheterization as well as in cases without appropriate femoral access. The arterial puncture site has been advised to be as close as possible to the antecubital fossa, where the brachial artery is least mobile. However, a hypermobile cubital brachial artery in some patients has considerably increased the difficulty of blind puncture.

In the cubital fossa, the median nerve is serpentine along the brachial artery. The median nerve is at first lateral to the brachial artery and then crosses to its medial side. Hence, a serpentine median nerve along a mobile brachial artery makes it difficult to achieve successful arterial puncture without nerve injury. Despite the adjacency of the cubital brachial artery and median nerve, the complication rate of median nerve...
injury has been low. The maximal prevalence was 0.6% in brachial catheterization by arterial cut-down and brachial arteriotomy. Nevertheless, concerning the potential median nerve injury in this procedure, the responsible physician must be alert to this complication.

Delayed, subacute onset of clinical median neuropathy with the Tinel sign in situ indicated the focal neuropathy at the puncture site. In addition, given the medical history of anticoagulation with the prolonged prothrombin time (INR, >1.5), epineural hemorrhage of the median nerve should have been suspected. Despite the rarity of epineural hemorrhage, it should be recognized early, because a delayed diagnosis may lead to a malignant Volkmann contracture and a useless upper extremity.

Repetitive puncture needle injuries act as penetrating and stretching vascular injuries at the epineurium, causing subsequent bleeding. The circumscribed epineural hematoma compresses the nerve bundles and impairs their function. The concomitant coagulopathy due to administration of anticoagulants might precipitate the subsequent epineural bleeding and poor absorption of this hematoma.

An intramural hematoma is usually absorbed without leaving a permanent sequela; the phagocytosis of red blood cells usually begins within 24 hours. The process of phagocytosis could be prolonged by anticoagulation; therefore, nerve compression could be prolonged. After acknowledgment of this hazard, early recognition of this complication and immediate therapeutic strategy setting are urgent.

Recently, high-resolution sonography using high-frequency transducers (e.g., 15 MHz) may depict the fine details of a peripheral nerve lesion and may distinguish the epineurium from surrounding vessels and muscles by their different echo textures. In our case, sonography successfully showed this epineural hematoma, which was distinguishable from a brachial arterial pseudoaneurysm sonographically. Furthermore, sonographically guided FNA decompressed this mass effect and helped differentiate a hematoma from a traumatic neuroma. In addition, the associated findings of acute compressive neuropathy could be shown by sonography, including (1) retrograde endoneural swelling, illustrated by an increased cross-sectional area; and (2) proximal endoneural swelling, represented by obliteration of a reticular internal echo.

Retrograde endoneural swelling indicated pathologic damming of axonal flow by the constriction.

The main complication of anticoagulation therapy is bleeding. The major hemorrhagic risks in this case included high-intensity anticoagulation (INR, >2), age older than 65 years, history of stroke, and concomitant administration of antiplatelet drugs. The cumulative prevalence of bleeding during outpatient therapy with warfarin was 34% at 36 months. After acknowledgment of this, brachial catheterization acted as a potential iatrogenic cause. Concerning the severe morbidity of median
nerve palsy, reversal of anticoagulation is necessary for most patients undergoing percutaneous catheterization.10

A few days before the transarterial procedure, the warfarin dose should be reduced until the INR is 1.2 or less.9 However, sudden cessation of anticoagulation carries the risk of acute ischemic stroke. Warfarin should not be abruptly withdrawn but should be continued until replaced by anticoagulation therapy with heparin (e.g., 10,000 U subcutaneously twice per day).10 Heparin may be stopped on the day of brachial catheterization, and anticoagulation therapy may be restarted later.10 For successful anticoagulant adjustment, we suggest that cerebral angiography via the brachial approach should be performed as an inpatient diagnostic procedure.

The serpentine median nerve along our patient’s mobile cubital brachial artery led to misleading puncture injuries. Sonographically guided, accurate brachial arterial puncture is possible, promising increased safety. We suggest that sonographically guided puncture could be performed in selected patients at high risk of bleeding.

In conclusion, we stress the value of high-resolution sonography in the early recognition of an epineural hematoma after brachial catheterization and the therapeutic effect of immediate decompression by sonographically guided FNA. In addition, sonographically guided brachial arterial puncture should be considered in patients receiving anticoagulants. Cautious preprocedural anticoagulant adjustments (INR, ≤1.2) are essential to ensure the safety of brachial catheterization.

References