HEMATOMA RISK AFTER NEEDLE ELECTROMYOGRAPHY

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ABSTRACT: *Introduction*: Although needle electromyography (EMG) appears to be a relatively safe procedure based primarily on clinical experience, no evidence-based guidelines exist for EMG procedures in patients taking anticoagulant or antiplatelet medications. We sought to determine whether there is an increased risk of hematoma formation after EMG of potentially high-risk muscles in patients taking anticoagulant or antiplatelet agents. *Methods*: After undergoing routine EMG, if any of seven predetermined high-risk muscles were tested, study subjects then underwent ultrasound to evaluate for hematoma formation. *Results*: Patients were divided into three groups based on medication (warfarin, aspirin/clopidogrel, no blood-thinning medication), with at least 100 muscles examined per group. Two small, subclinical hematomas were seen on ultrasound; there was no difference in hematoma risk between groups ($P = 0.43$). *Conclusions*: Our findings suggest that hematoma formation from standard needle EMG is rare even in high-risk muscles, which have been avoided historically in anticoagulated patients.

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There are no absolute contraindications to performing a well-directed and selective needle electromyographic examination (EMG).[1] However, needle EMG is an invasive procedure and, under certain conditions, there is potential for associated iatrogenic complications.[2–9] Coagulopathy, either acquired or medically induced, has historically been considered a relative contraindication to needle EMG.[1,10] A survey of electrodiagnostic practices across the country showed many labs avoided needle EMG in patients on anticoagulation, requiring that such medication be stopped prior to the test, or that certain muscles be avoided in anticoagulated patients.[6] The American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) recommends that superficial muscles be examined first, prior to proceeding to deeper muscles in anticoagulated patients, and that prolonged pressure be applied after removal of the needle.[10]

Recently, we have attempted to develop an evidence base to support the practice of needle EMG in anticoagulated patients, given the potential risk of stopping such medication for stroke or other cardioembolic events.[11,12] In one study looking at the tibialis anterior muscle, 1 patient on warfarin and 1 on aspirin (ASA) developed small subclinical hematomas after needle EMG of the tibialis anterior muscle; no hematomas were detected in a control group.[12] In that study, there were 101 patients taking warfarin, 57 taking ASA and/or clopidogrel, and 51 patients in the control group. In a recent study by Gertken et al. no hematomas were detected on magnetic resonance imaging (MRI) after needle EMG of the paraspinal muscles (431 cases).[11] This finding was in contrast to an earlier study by Caress et al., who detected small, subclinical hematomas retrospectively on MRI of the paraspinal muscles of 5 of 17 patients who had recently undergone needle EMG of those muscles.[13]

The objective of this study was to determine whether there is an increased risk of hematoma formation after routine needle EMG in patients who are taking ASA, clopidogrel, or warfarin when potentially high-risk muscles, such as the paraspinals, are examined. This would provide further objective data to facilitate the development of evidence-based guidelines for EMG procedures in this patient population. Our hypothesis was that patients who take ASA, clopidogrel, or warfarin would have the same risk of developing an ultrasonographically detectable hematoma after routine needle EMG as a control group not taking anticoagulant medication.

METHODS

Subjects. Adult patients referred to the Mayo Clinic EMG laboratory for needle EMG were eligible to participate in the study if they underwent needle EMG of any or all of the muscles of interest (cervical, thoracic, lumbar paraspinals, flexor pollicis longus, flexor digitorum longus, posterior tibialis, iliopsoas). Subjects provided verbal informed consent and signed an authorization in accordance with the Health Insurance Portability and Accountability Act (HIPPA). Study participants were divided into three groups based on their medication status (warfarin, clopidogrel and/or ASA, and control subjects on neither class of medication).

Trial Design. This investigation was conducted using a prospective, case–control design of patients who underwent needle examination of "high-risk" muscles. For the three patient groups (warfarin,

Abbreviations: ASA, aspirin; BMI, body mass index; EMG, electromyography; HIPAA, Health Insurance Portability and Accountability Act; INR, international normalized ratio; MRI, magnetic resonance imaging; NSAID, non-steroidal anti-inflammatory drug

Key words: aspirin, clopidogrel, electromyography, hematoma, ultrasound, warfarin

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Table 1. Patient demographics.

	Control	ASA/clopidogrel	Warfarin
Number of muscles	100	116	107
Number of patients	70	78	58
Age*	48.9 (21–91)	66.8 (27–89)	69.6 (33–85)
Gender	37 M, 33 F	30 M, 48 F	31 M, 27 F
BMI†	28.3 (5.7)	30.5 (7.7)	28.7 (5.8)
Time lapse‡	23.3 (15–45)	25.42 (15–110)	27.4 (15–85)
NSAID use	7 patients	8 patients	0 patients
EMG by staff	58 patients	45 patients	47 patients
EMG by fellow	7 patients	0 patients	3 patients
EMG by resident	13 patients	25 patients	8 patients
75-mm (23-gauge) needle	1 muscle	1 muscle	2 muscles
50-mm (26-gauge) needle	113 muscles	99 muscles	104 muscles
25-mm (30-gauge) needle	2 muscles	0 muscles	1 muscle

M, male; F, female; BMI, body mass index; NSAID, non-steroidal anti-inflammatory drug; EMG, electromyography.
**Mean, in years (range).*
†Mean, in kg/m^2 (standard deviation).
‡Mean time lapse, in minutes, between needle and ultrasound (range).

ASA/clopidogrel, and control), we examined a minimum of 100 muscles per patient group.

Our study was approved by our institutional internal review board. Patients who agreed to participate signed a HIPPA form and completed a questionnaire, including medication use, past medical history, and use of herbal supplements. All potential blood-thinning agents were documented, as was the level of experience of each EMG examiner, the needle size used for the study, and the time elapsed between needle EMG of the muscle of interest and the time of ultrasound examination.

The needle examination was performed by a staff physician, fellow, or supervised resident working in the EMG laboratory that day according to standard procedures. After completion of the needle examination, one of the study investigators performed sonographic examination of the muscles of interest to evaluate for the presence of hematoma. A portable ultrasound machine (Logiq E; GE Healthcare, Milwaukee, Wisconsin) with a 38-mm footprint linear-array transducer (7–13 MHz) was used for the study. The presence and size of any identified hematoma was then objectively quantified and retrospectively correlated with the patient's medication use at the time of the examination. The ultrasonographer was not blinded to the patient's anticoagulation status. For both needle EMG and ultrasonography the technique used was identical to that described in a previous study of hematoma formation in the tibialis anterior muscle.[12]

RESULTS

A total of 205 patients were included in the study. There were 70 patients (100 muscles) in the control group, 78 patients (116 muscles) in the antiplatelet (ASA/clopidogrel) group, and 58 patients (107 muscles) in the anticoagulation (warfarin) group. Group demographics are shown in Table 1, including electromyographer experience, needle size, and time elapsed between needle EMG and ultrasound examination. Use of non-steroidal anti-inflammatory drugs (NSAIDs) or herbal supplements was also recorded. A listing of muscles examined for each group is shown in Table 2.

Patients in the ASA/clopidogrel group were taking variable doses and/or combinations of the two medications. Fifty-six patients were taking ASA, 4 were taking clopidogrel, and 18 were taking both ASA and clopidogrel. In the warfarin group, the international normalized ratio (INR) ranged from 1.6 to 4.0. Forty-eight patients (68 muscles) had an INR of at least 2.0, and 10 patients (13 muscles) had an INR of at least 3.0. Eleven of the patients taking warfarin were also taking ASA, and 1 was taking clopidogrel.

There were only 2 hematomas detected in the 206 patients studied. Of the 70 controls, none had ultrasound evidence of a hematoma. One 78-year-old woman in the ASA/clopidogrel group taking 75 mg/day clopidogrel had a subclinical hematoma in the tibialis posterior muscle that measured 8.8 mm × 1.2 mm. The needle EMG was

Table 2. Muscles examined.

Muscle	Control	ASA/clopidogrel	Coumadin
Total	100	116	107
Cervical paraspinals	17	10	6
Thoracic paraspinals	17	26	18
Lumbar paraspinals	13	21	21
Tibialis posterior	7	11	10
Flexor digitorum longus	10	13	9
Flexor pollicis longus	25	18	31
Iliopsoas	11	17	12

performed by a clinical neurophysiology fellow using a 50-mm (26-gauge) concentric needle. The other hematoma was also subclinical; it was detected in an 84-year-old man in the warfarin group in the flexor pollicis longus muscle and measured 16 mm × 3 mm. The INR at the time of the examination was 2.3. The needle EMG was performed by a staff electromyographer, and a 50-mm (26 gauge) concentric needle electrode was used. The patient reported that he "bruised easily." In both cases, the patients were completely asymptomatic at the time of the ultrasound examination and 24 hours later when followed up by phone, and there was no evidence of swelling or bruising noted at the site of needle entry.

When all three groups were compared using chi-square analysis, there were no significant differences in hematoma rate ($P = 0.43$). The hematoma incidence rate for the warfarin group had a point estimate of 0.93%, or 1 of 107 muscles examined (95% confidence interval 0.2–5.1%). For the ASA/clopidogrel group, the point estimate was 0.85%, or 1 of 116 muscles examined (95% confidence interval 0.2–4.7%), and for the control group the point estimate was 0% (95% confidence interval 0–3.6%). The overall rate of hematoma formation in the entire study population was 0.62%, or 2 of 323 muscles examined (95% confidence interval 0.2–2.2%).

DISCUSSION

Although antiplatelet and anticoagulation therapies theoretically increase the risk of hematoma formation after needle EMG, the actual risk has not been well studied. Until recently, the literature contained only a few case reports and small studies reporting varying incidences of this complication in association with needle EMG. However, due to the lack of evidence-based guidelines, many clinicians avoid performing needle EMG on anticoagulated patients, limiting the scope and therefore the utility of the examination. Other clinicians may require the patient to discontinue anticoagulation prior to performing needle EMG, thereby placing the patient at risk for a thrombotic event. For example, a high-risk patient with atrial fibrillation who is anticoagulated for stroke prophylaxis has a significantly increased (up to threefold) risk of stroke when not on warfarin.[14–16]

Recently, we have attempted to develop an evidence base to support the practice of routine needle EMG in anticoagulated patients. In our previous study of patients on the same medications as our current study, two very small, clinically inapparent hematomas were detected (one in a patient on warfarin and one in a patient on ASA).[12] In that investigation, we only evaluated a single muscle (tibialis anterior), and one critique of that study was that the results were not generalizable to all muscles. The current study protocol was developed as a result, in which we evaluated several other high-risk muscles, including muscles often avoided in anticoagulated patients, such as the paraspinal and iliopsoas muscles. The objective of this study was to provide further data to facilitate clinical decisionmaking when faced with an anticoagulated patient who is referred for EMG.

The overall incidence of subclinical hematoma formation in this study group was 0.62% (2 of 323 muscles). In addition to an estimate of the overall risk of hematoma formation after routine EMG, further breakdown of patient groups allowed us to make an estimate of the risk in patients who are on antiplatelet therapy (point estimate 0.85%) and anticoagulation with warfarin (point estimate 0.93%). When compared with a control group, needle EMG was not associated with a statistically significant increased rate of hematoma formation in patients who were anticoagulated or receiving antiplatelet therapy.

These findings are consistent with our recently published study in which we looked for hematoma formation in the paraspinal muscles after needle EMG. No hematomas were found in that retrospective review of 431 MRIs.[11] This finding is in contrast to an earlier study by Caress et al. showing a high rate of hematoma formation in the paraspinal muscles after needle EMG (all asymptomatic and only one of which was identified on the original MRI report).[13] It is not clear why there is discrepancy between these studies. Possible explanations include differences in needle EMG technique or differences in MRI protocols; however, the Caress study was based on MRI reports from the mid-1990s. The Gertken et al. study was from 2008, and significant advances in imaging occurred in the interim, making the latter seem a less likely explanation. Regardless, our center has now studied hematoma formation following needle EMG in multiple patient populations using multiple techniques, and a very low incidence of hematoma has been found in every instance.

There has been one prior case report of compartment syndrome requiring fasciotomy after needle EMG of a calf muscle. That patient was not taking any blood-thinning medication other than occasional diclofenac.[5] During data collection for our study, the investigators noted the degree of vascularity of the tibialis posterior and flexor digitorum longus muscles apparent on ultrasound. There were frequently multiple veins within or surrounding those muscles. Due to this observation, in our practice we are more cautious in examining the tibialis posterior or flexor digitorum longus in

an anticoagulated patient than we were prior to the implementation of neuromuscular ultrasound. Despite this finding, there was no increase in the incidence of hematoma formation within these muscles.

Small sample size is one limitation to this study. Given the rarity of significant bleeding events after needle EMG, caution may be warranted in making more generalized conclusions from our findings with regard to other practitioners due to the potential for differences in needle technique between examiners. It is theoretically possible that small hematomas may have been missed on ultrasound; however, given the fact that two small hematomas were detected, it seems unlikely that a clinically significant hematoma could have been overlooked. It is possible that a hematoma could have developed after the muscle was scanned, but we assumed that if a hematoma were to develop secondary to the needle EMG procedure, it would begin to accumulate immediately. Any such hematoma should therefore have been visible 15 minutes after needle removal (the minimum time lapse between needle examination and ultrasound examination). A final limitation is that the study investigators were not blinded to anticoagulation status when performing the ultrasound examination.

This study is a further step in developing a body of literature to support the practice of needle EMG in anticoagulated patients. In our laboratory, we now perform needle EMG on any patient with an INR of $\leq$3.0. In patients with an INR $>$3.0 the study is done at the discretion of the individual electromyographer. Despite the findings of this study, we still recommend that electromyographers use caution when performing needle examination in patients who are taking anticoagulants, and the risks and benefits of the procedure should be considered carefully for individual patients.

In conclusion, this study has provided further evidence that hematoma formation is rare following standard needle EMG examination. Although antiplatelet therapy and anticoagulation increase the theoretical risk of a hematoma after needle EMG, we believe that the risks of the examination, when carried out in a reasonable manner, are minimal and may be lower than the risks of discontinuing anticoagulation.[14–16]

REFERENCES

1. Kimura J. Electrodiagnosis in diseases of nerve and muscle: principles and practice. Philadelphia: F.A. Davis; 1989. p 227–248.
2. Al-Shekhlee A, Shapiro BE, Preston DC. Iatrogenic complications and risks of nerve conduction studies and needle electromyography. Muscle Nerve 2003;27:517–526.
3. Baba Y, Hentschel K, Freeman WD, Broderick DF, Wszolek ZK. Large paraspinal and iliopsoas muscle hematomas. Arch Neurol 2005;62:1306.
4. Butler ML, Dewan RW. Subcutaneous hemorrhage in a patient receiving anticoagulant therapy: an unusual EMG complication. Arch Phys Med Rehabil 1984;65:733–734.
5. Farrell CM, Rubin DI, Haidukewych GJ. Acute compartment syndrome of the leg following diagnostic electromyography. Muscle Nerve 2003;27:374–377.
6. Gruis KL, Little AA, Zebarah VA, Albers JW. Survey of electrodiagnostic laboratories regarding hemorrhagic complications from needle electromyography. Muscle Nerve 2006;34:356–358.
7. Reinstein L, Twardzik FG, Mech KF Jr. Pneumothorax: a complication of needle electromyography of the supraspinatus muscle. Arch Phys Med Rehabil 1987;68:561–562.
8. Rosioreanu A, Dickson A, Lypen S, Katz DS. Pseudoaneurysm of the calf after electromyography: sonographic and CT angiographic diagnosis. AJR Am J Roentgenol 2005;185:282–283.
9. Vaienti L, Vourtsis S, Urzola V. Compartment syndrome of the forearm following an electromyographic assessment. J Hand Surg [Br] 2005;30:656–657.
10. Guidelines in electrodiagnostic medicine. Risks in electrodiagnostic medicine. American Association of Electrodiagnostic Medicine. Muscle Nerve 1999;8(suppl):S53–69.
11. Gertken JT, Hunt CH, Montes Chinea NI, Morris JM, Sorenson EJ, Boon AJ. Risk of hematoma following needle electromyography of the paraspinal muscles. Muscle Nerve 2011;44:439–440.
12. Lynch S, Boon A, Smith J, Harper C, Tanaka E. Complications of needle electromyography: hematoma risk and correlation with anticoagulation and antiplatelet therapy. Muscle Nerve 2008;38: 1225–1230.
13. Caress JB, Rutkove SB, Carlin M, Khoshbin S, Preston DC. Paraspinal muscle hematoma after electromyography. Neurology 1996;47: 269–272.
14. Risk factors for stroke and efficacy of antithrombotic therapy in atrial fibrillation. Analysis of pooled data from five randomized controlled trials. Arch Intern Med 1994;154:1449–1457.
15. Gage BF, Waterman AD, Shannon W, Boechler M, Rich MW, Radford MJ. Validation of clinical classification schemes for predicting stroke: results from the National Registry of Atrial Fibrillation. JAMA 2001;285:2864–2870.
16. Petersen P, Boysen G, Godtfredsen J, Andersen ED, Andersen B. Placebo-controlled, randomised trial of warfarin and aspirin for prevention of thromboembolic complications in chronic atrial fibrillation. Lancet 1989;1:175–179.