

CASE REPORTS

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Ulnar Neuropathy in Medical Patients

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ANESTHETIZED patients who undergo surgical procedures may develop ulnar neuropathy.¹⁻⁶ Although the mechanism of ulnar neuropathy in the perioperative setting is often unclear, improper positioning or padding of the upper extremity during surgery is sometimes implicated^{7,8}; however, there is little direct evidence to implicate improper intraoperative care as a cause of perioperative ulnar neuropathy. Several studies have noted that surgical patients may develop symptoms of this complication in the presence of appropriate care.^{4,6} Recently, we reported that 7 of 1,502 prospectively studied surgical patients (0.5%) developed perioperative ulnar neuropathy.⁹ All seven patients reported an initial onset of symptoms 2-7 days after their procedure, which suggests that one or more factors in the postoperative period may contribute to ulnar neuropathy.

Postoperative patients and patients who are admitted with medical conditions may have similar hospital-care characteristics (e.g., bedrest). Do medical patients who are not undergoing surgery also develop ulnar neuropathy during hospitalization?

This report describes a prospective study of ulnar neuropathy in patients admitted to internal medicine services for nonsurgical conditions, and reports on two of these patients who developed ulnar neuropathy during the first week after admission.

Methods

The study was approved by the Mayo Institutional Review Board. Patients 18 yr and older who were admitted to internal medicine services at the Mayo hospitals in Rochester, Minnesota from June 1997 through August 1997 were asked to participate. Patients who met the enrollment guidelines and gave consent participated in the study. All internal medicine subspecialties were provided at the hospitals during the study period. A total of 990 of 5,119 patients admitted to internal medicine services were enrolled in the study during the 3-month period; 17 of 1,007 patients who met the enrollment guidelines declined to participate in the study. Any or all of the following defined ulnar neuropathy: (1) current symptoms of paresthesia in the ulnar distribution; (2) signs of abnormal two-point discrimination in the volar surface of the distal fifth digit; and (3) weakness of the first dorsal interosseous and abductor digiti minimi muscles. Patients with current signs or symptoms of the condition were not enrolled, and persons with preexisting ulnar neuropathy were excluded.

The methodology used in this study has been described previously.⁹ In brief, specially trained research assistants performed a standardized baseline neurologic examination of the upper extremities on all participants. Each patient was assessed daily until time of discharge or for up to 7 days after admission using a standardized questionnaire and screening neurologic examination designed to detect manifestations of ulnar nerve dysfunction. Patients who were discharged before 7 days were interviewed by telephone 7-9 days after admission with a standardized questionnaire. Postadmission surveillance of at least 7 days was successful in all but 4 of 990 study patients. Data from these four patients have not been included in our analyses.

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Table 1. Patient Characteristics (N = 986)

	% of Patients*
Age (years)	65.0 ± 16.4
Height (cm)†	169.2 ± 40.6
Weight (kg)†	77.4 ± 20.3
Body mass index (kg/m ²)†	28.0 ± 12.2
Gender	
Male	53.0
Female	47.0
Diabetes	
No	81.6
Non-insulin-dependent	8.9
Insulin-dependent	9.4
Smoking status	
Never	46.0
Former	37.9
Current (within 6 months)	16.0
Alcohol use (> 3 drinks/day)	1.8
History of numbness or tingling in hands	30.6
Prior surgery/injury to nerves of the hand	8.9
Family history of nerve problems	8.5
Previous neck problems	22.2
Previous arm or wrist fracture‡	23.6
Previous carpal tunnel surgery	4.6
Length of stay (days)	
Outpatient procedure	2.6
1-2	30.0
3-7	43.7
> 7	23.6

* Where applicable, values are mean ± SD.

† Height and weight information were missing for 9 patients (N = 977).

‡ Information related to previous arm or wrist fracture was missing for 1 patient (N = 985).

A general description of the study population is shown in table 1. One neurologist evaluated three patients who were identified by the research assistants as possessing signs or symptoms suggestive of ulnar neuropathy; the condition was diagnosed in two of these patients. Therefore, 2 of 986 study patients (0.2%)(95% CI 0.02-0.73%) had ulnar neuropathy.

Case 1

A 55-yr-old, 72-kg man with a 6-month history of recurrent disseminated aspergillosis was admitted for intravenous antifungal therapy. Except for chronic, treated hypertension, he was healthy before the onset of the infection. He lost 11 kg in the intervening 6 months, was chronically fatigued, and developed a macular dermatitis on his anterior abdominal wall. The dermatitis was painful, and when in bed he rested primarily in the supine position. On admission he had no signs or symptoms of ulnar neuropathy; however, on the third hospital day he experienced intermittent tingling in the fourth and fifth digits of his right hand. His perception of two-point discrimination on the volar surface of his fifth digit was not changed from the time of his admission examination. By the fifth day, the tingling had become constant, and a burning dysesthesia was present with elbow flexion of greater than

90°. The patient had no motor weakness. These symptoms improved gradually, with complete resolution during the next 6 months.

Case 2

A 67-yr-old, 102-kg man with severe bronchiectasis was admitted for treatment of recurrent *Pseudomonas* pneumonia with intravenous antibiotics and respiratory therapy. His other medical problems included coronary artery disease with stable angina and treated hypertension. Previous painful bilateral hip arthroplasties prevented the patient from sleeping on his side, and when in bed he rested primarily in the supine position. The patient had no signs or symptoms of ulnar neuropathy on admission. On the fourth hospital day, he experienced tingling and aching in the fourth and fifth digits of his left hand. His perception of two-point discrimination on the volar surface of his left fifth digit had decreased from 3 to 7 mm from the time of his admission exam. He developed similar symptoms in the fourth and fifth digits of his right hand on the sixth day; however, his perception of two-point discrimination in the right fifth digit was not changed from the time of his admission exam. He had no motor weakness in either upper extremity. The symptoms on the left side were exacerbated with elbow flexion of greater than 90° if prolonged for more than 30 s. The symptoms in his right hand resolved within 2 weeks, but the symptoms in his left hand persisted for 8 months.

Discussion

Our finding that medical patients can develop symptoms of ulnar neuropathy during hospitalization suggests that factors common to inpatient care of medical and postoperative patients may contribute to this condition. Interestingly, both of these medical patients and most of the postoperative patients in our earlier report⁹ were men 50-75 yr who self-reported that they spent much of their time in the hospital lying in the supine position.

Perioperative ulnar neuropathy is consistently reported to occur most often in men.^{1,3-5} Anatomic differences at the elbow between men and women may contribute to this finding. The tubercle of the coronoid process is approximately 1.5 times larger in men than in women.¹⁰ In addition, there is less adipose tissue over the medial aspect of the elbow of men compared with women who have similar body fat composition.¹¹ These differences may increase the opportunity for external compression of the ulnar nerve near the elbow in men.

Hospitalized patients who rest in the supine position commonly flex their elbows and rest their hands on their upper abdomen or chest.¹² This position may increase pressure on the ulnar nerve by two mechanisms. First, flexion of the elbow, especially to greater than 110°, markedly stretches the cubital tunnel retinaculum and increases pressure in the postcondylar groove of the humerus.¹³ The stretched retinaculum may directly compress the ulnar nerve distal to the postcondylar groove.¹⁴ Second, the humerus is internally rotated and the forearm is pronated when a hand is positioned on the upper abdomen or chest. We speculate that the supine position and placement of the hands on the abdomen or chest may result in external compression of the ulnar nerve and its vascular supply in the postcondylar groove of the humerus or the subtrochanteric groove of the coronoid process of the ulna.

In conclusion, signs or symptoms of ulnar neuropathy developed within 7 days of admission in 2 of 986 patients (0.2%) admitted to internal medicine services. This finding supports previous reports that,

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in some cases, the mechanism of injury associated with postoperative ulnar neuropathy may not occur intraoperatively. Based on the cases reported in this study and in our previous prospective study of perioperative ulnar neuropathy, we speculate that symptoms of ulnar neuropathy in both medical and surgical inpatients may be related to prolonged periods of bedrest in the supine position.

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Reversible Catecholamine-induced Cardiomyopathy by Subcutaneous Injections of Epinephrine Solution in an Anesthetized Patient

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CATECHOLAMINE-INDUCED cardiomyopathy has been recognized for several decades.¹ It is associated with certain pathologic conditions, such as pheochromocytoma

and subarachnoid hemorrhage, in which excessively high concentrations of endogenous catecholamines are regarded as the cause.²⁻⁵ Large doses of exogenously administered norepinephrine in humans and animals also produce characteristic myocardial lesions that include focal myofiber necrosis and degeneration and mononuclear leukocytic infiltration.^{1,6-8} Although previous case reports are confined to these pathologic conditions or catecholamine overdoses, cardiomyopathy that developed acutely after a clinically relevant dose of exogenously administered epinephrine has never been reported in humans.

Clinical pictures of catecholamine cardiomyopathy are divergent and may present dissociation between diffuse impairment of myocardial contractile function *versus* little or no evidence of myocardial damage detected by

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