OPEN

Complex Regional Pain Syndrome and Acute Carpal Tunnel Syndrome Following Radial Artery Cannulation

A Neurological Perspective and Review of the Literature

Reynaldo P. Lazaro, MD

Abstract: Complex regional pain syndrome (CRPS) associated with acute carpal tunnel syndrome (aCTS) was developed in a 38-year-old right-handed man following radial artery cannulation (RAC) during the course of lumbar spine surgery. Inciting events and risk factors that might have led to these complications included: multiple arterial punctures and subsequent hematoma formation, radial artery spasm compounded by aggressive hemostasis, anatomical changes in the wrists related to repetitive manual activities in the workplace, and possible protracted hyperextension of the wrists during perioperative and operative procedure. Although CRPS is considered a rare complication of RAC, the condition is disabling and debilitating, especially when associated with aCTS.

(*Medicine* 94(3):e422)

INTRODUCTION

CRPS is characterized by a constellation of signs and symptoms including burning and throbbing pain in the affected extremity associated with allodynia, sensitivity to extremes of temperature, and various signs of sympathetic hyperactivity.[1] Abnormalities of movements such as dystonia and tremor[2,3] are also known to occur along with various trophic changes affecting the skin, joints, and bone. Severity of the symptoms is variable. When the inciting event is a musculoskeletal injury, CRPS is classified as type 1. When it is due to peripheral nerve injury, it is classified as type 2. The apparent rarity and disabling nature of CRPS associated with aCTS following RAC prompted us to review the subject from the neurologic standpoint.

CASE PRESENTATION

This 38-year-old, right-handed man, a manual laborer by profession, underwent a successful lumbar interbody fusion for degenerative disc disease with an anterior retroperitoneal approach. Over the course of his 7-hour surgery, he was placed in a prone position for 2 h and a supine position for 5 h under general anesthesia. It was unclear how his wrists were positioned during the surgery following RAC. Prior to surgery, several attempts to cannulate the radial artery, first on the right, then on the left, were followed by extensive hematoma formation at the volar aspect of the wrists and forearms.

A few hours after surgery, the patient began complaining of numbness and paresthesias in the thumb, 2nd and 3rd fingers, and lateral half of the 4th finger along with burning pain in the wrist and forearm bilaterally. There were no signs of infection. The hematoma resolved within 3 weeks following surgery. Following a peripheral electrodiagnostic study performed by a neurologist at a local hospital, a diagnosis of carpal tunnel syndrome (CTS) was made. A trial of steroid injection for possible pronator syndrome was without benefits. He requested an opinion from a second surgeon after he was advised to undergo surgical decompression for CTS.

The patient's medical history was remarkable for hypertension controlled with combination of Lisinopril and Hydrochlorothiazide and polycystic kidneys. There was no history of diabetes, hyporthyroidism, or amyloidosis. He never suffered from anxiety or depression before or after surgery. His body mass index was not measured, but he was a muscular man and weighed 200 pounds. He developed deep vein thrombosis a week after surgery that required Warfarin therapy.

Examination performed a month after hospital discharge showed sensory deficits in the median nerve (MN) distribution and bilateral weakness of the abductor pollicis brevis (APB) muscle (4/5, Medical Research Council Scale). The skin over the thenar eminence was also hypoalgesic. The proximal muscles innervated by the MN and by the ulnar and radial nerves were 5/5. Allodynia along with mild swelling of the fingers and patchy erythema in the hands was noted. Resolving hematoma in the volar aspect of the wrists was discernible. Peripheral nerve conduction studies were performed using standard techniques for transcutaneous stimulation and recording with surface electrodes. The MN motor latencies at the wrists were prolonged bilaterally (right = 5.1 ms, left = 4.7, $n \leq 4.0$). The forearm motor velocities were normal. Sensory nerve action potential (SNAP) recorded antidromically over the 2nd finger showed an amplitude of 6.6 μV ($n \geq 10$) and a latency, recorded at the onset of the response, of 3.5 ms on the left ($n \leq 2.9$). The SNAP on the right was absent. Monopolar needle electromyogram (EMG) showed signs of active denervation (sharp positive waves and fibrillation potentials) in the APB muscle on the right, whereas the forearm muscles were normal on both sides. Ulnar and radial nerve conduction studies were normal. Three-phase bone scan and computerized axial tomography imaging of the wrists performed a month after onset of symptoms, blood chemistries, and hemogram were normal.

Owing to the patient's persistent pain despite his use of Oxycodone and various over-the-counter drugs, Pregabalin 600 mg a day was added to pain control regimen. He was

Editor: Raymond L. Rosales.
Received: October 17, 2014; revised: December 3, 2014; accepted: December 4, 2014.
From the Neurology and Electroneuromyography Clinic, Oneonta, New York, USA.
Correspondence: Reynaldo P. Lazaro, Neurology and Electroneuromyography Clinic, 41-45 Dietz Street, Oneonta, NY 13820, USA (e-mail: aacmbbilling@gmail.com).
The author has no funding and conflicts of interest to disclose.

ISSN: 0025-7974
DOI: 10.1097/MD.0000000000000422

encouraged to actively exercise his hands to prevent contractures of the joints and maintain muscle tone. Over the course of one and a half years following the onset of symptoms, his hand grip and dexterity slowly improved enabling him to perform sedentary work on a part-time basis despite his pain and frequent alternating episodes of cool and warm sensation in the hands. The improvement in hand grip correlated with the presence of signs of reinnervation (polyphasic and large amplitude motor units) in EMG of the APB muscle and minimal improvement in nerve conduction velocities. In view of clinical and electrophysiologic improvement, persistence of CRPS and possibility of exacerbating it surgically, and presence of prominent musculoskeletal tenderness in the wrists, the second surgeon consulted for CTS elected to withhold surgical decompression. Likewise, the need for sympathetic nerve block was withheld.

DISCUSSION

The literature on arterial cannulation is voluminous. RAC is frequently utilized to monitor hemodynamic changes during major surgical procedures and in critically ill patients. It also allows for frequent blood samples for arterial gas measurement. The radial artery is the most common site for transcutaneous coronary angiogram or intervention because of its accessibility, safety, good collateral circulation, and low incidence of complications,[4–6] but some complications such as thrombosis and occlusion, pseudoaneurysm, forearm compartment syndrome, infection, and skin necrosis still occur.[7–10] Repeated arterial punctures may result in extensive hematoma formation and arterial spasm[11] jeopardizing the carpal tunnel (CT) segment of the median nerve. The thick muscular coat and abundance of alpha adrenoreceptors[12] make it prone to develop spasm when traumatized, resulting in ischemia of the MN that is already compromised by build-up of pressure in the CT due to hemorrhage. Attempts to control bleeding following cannulation through local hemostasis only complicate matters. Fortunately, the likelihood of serious ischemia is minimized by the presence of palmar arterial arch.

RAC requires hyperextension of the wrist to facilitate catheter placement.[13] It also reduces tortuosity of the artery. At times, this position may have to be maintained for protracted period thereby increasing the likelihood of traction injury to the median nerve. Returning the wrist to neutral position during surgery can minimize injury. Mechanically and anatomically the neutral position has been shown to be the best position associated with least median nerve compression.[14] However, the length of time that the patient's wrists were held in hyperextended position was not available in the operative report. Physiologically, it has been demonstrated that MN conduction block can develop following protracted hyperextension of the wrist.[13] A similar effect on MN can occur following several minutes of palmar flexion of the wrist.[15] Thus, undue traction on the median nerve in individuals engaged in manual labor can easily facilitate the onset of CTS especially when complicated by hemorrhage.

Acute exacerbation of CTS has been reported following RAC.[16] The median nerve can also be injured during brachial artery cardiac catheterization.[17] The patient, who never experienced symptoms of CTS prior to RAC but was involved in many years of manual labor, developed extensive hematoma formation in both wrists as a result of repeated punctures of the radial artery. Median nerve compression following RAC[18] as well as other focal neuropathies induced by anticoagulant therapy[19–22] and those that occur in some hematologic disorders[23–25] are treated either conservatively or surgically depending on the treating physician's experience and orientation. The patient could have developed injury to the palmar cutaneous branch of the median nerve in addition to injury to the main trunk. This nerve passes through the anterolateral aspect of the wrist above and outside the CT and provides sensory innervation to the skin over the thenar eminence.[26] Although not corroborated electrophysiologically, this will explain the sensory deficit over the thenar eminence bilaterally.

Review of the literature discloses that both types of CRPS have been reported to follow RAC,[27–33] but in general, this complication is considered rare. It has been reported after radial artery harvesting for coronary bypass,[34] after venipuncture,[35] and in some cases of CTS characterized by prominent musculoskeletal pain.[36] Deciding what type of CRPS our patient developed is a moot point. Propensity toward CRPS was likely, with or without median nerve injury, due to multiple punctures that resulted in disruption of adrenoreceptors in the radial artery during RAC. Recognition of signs and symptoms remains the mainstay of diagnosis of CRPS. Symptoms and objective clinical findings, most of which are mediated by sympathetic nerve hyperactivity, are easily recognizable. Laboratory procedures such as thermography, 3-phase bone scan, and imaging studies provide useful corroborative information, but they do not explain the phenomenology and complex interaction between the central and peripheral nervous system in this condition. Moreover, they have poor sensitivity but reasonable specificity.[37]

Procedural variables in RAC[4–6] such as experience of the physician, preprocedural preparations, anatomical variations, caliber and manipulation of the needle-catheter, use of spasmolytic drugs, adequate anticoagulation, and use of high pressure hemostasis are worthy of considerations in the prevention of non-neurological and peripheral neuromuscular complications. From the neurologic standpoint, given the rarity of the latter, one would question the necessity for routine electrophysiologic or sonographic examination of the median nerve prior to RAC. In patients with occupational risk factors, with or without history of CTS, such necessity may be justified. Similarly, patients with concurrent medical conditions such as diabetes, rheumatoid arthritis, and hypothyroidism, and those with peripheral nerve disorder secondary to amyloidosis or genetic disorders, who are at risk for CTS, may require EMG or sonographic screening prior to RAC. With this in mind, the economic aspect of healthcare and medicolegal relevance of such approach become important issues worth pondering in the cost consciousness of our current health care system.

CONCLUSION

CRPS following RAC is rare but a potential threat to develop when the alpha-adrenoreceptor-rich artery is subjected to multiple punctures. Extensive hematoma formation can complicate matters further because of potential injury to the median nerve unduly stretched by hyperextension during RAC. Such injury may occur in individuals engaged in manual labor or in those with pre-existing peripheral neuropathy secondary to metabolic or systemic disorders because of chronic changes in the physioanatomical dimension of the carpal tunnel.

REFERENCES

1. de Mos M, Sturkenboom M, Huygen F. Current understandings on complex regional pain syndrome. *Pain Pract.* 2009;9:86–99.

2. Schwartzman RJ, Kerrigan J. The movement disorder of reflex sympathetic dystrophy. *Neurology.* 1990;40:57–61.
3. Van Hilten JJ, van de Beek WJT, Vein AA, et al. Clinical aspects of multifocal or generalized tonic dystonia in reflex sympathetic dystrophy. *Neurology.* 2001;56:1762–1765.
4. Caputo RP, Tremmel JA, Rao S, et al. Transradial arterial access for coronary and peripheral procedures: executive summary by the transradial committee of the SCAI. *Catheter Cardiovasc Interv.* 2011;78:823–835.
5. Scheer BV, Perel A, Pfeiffer UJ. Complications and risk factors of peripheral arterial catheters used for haemodynamic monitoring in anaesthesia and intensive care medicine. *Crit Care.* 2002;6:199–204.
6. Slogoff S, Keats AS, Arlund C. On the safety of radial artery cannulation. *Anesthesiology.* 1983;59:42–47.
7. Bhat T, Teli S, Akhtar M, et al. Access-site complications and their management during transradial cardiac catherization. *Expert Rev Cardiovasc Ther.* 2012;10:627–634.
8. Franklin C. The technique of radial artery cannulation: Tips for maximizing results while minimizing the risk of complications. *J Crit Illn.* 1195;10:424–432.
9. Qvist J, Peterfreund RA, Perlmutter GS. Transient compartment syndrome of the forearm after attempted radial artery cannulation. *Anesth Anal.* 1996;83:183–185.
10. Wallach SG. Cannulation of the radial artery: diagnosis and treatment algorithm. *Am J Crit Cre.* 2004;13:215–319.
11. Ruiz-Salmeron RJ, Mora R, Velez-Gimon M, et al. Radial artery spasm in transradial cardiac catherization: assessment of factors related to its occurrence and of its consequences during follow-up. *Rev Esp Cardiol.* 2005;58:504–511.
12. He GW, Yang CQ. Characteristics of adrenoreceptors in the human radial artery: clinical implications. *Thorac Cardiovasc Surg.* 1998;115:1136–1141.
13. Chowet AL, Lopez JR, Brock-Utne JG, et al. Wrist hyperextension leads to median nerve conduction block: implications for intra-arterial catheter placement. *Anesthesiology.* 2004;100:287–291.
14. Kuo MH, Leong CP, Cheng YF, et al. Static wrist position associated with least median nerve compression: sonographic evaluation. *AM J Phys Med Rehabili.* 2001;80:256–260.
15. Schwartz MS, Gordon JA, Swash M. Slowed nerve conduction with wrist flexion in carpal tunnel syndrome. *Ann Neurol.* 1980;8:69–71.
16. Martin SD, Sharrock NE, Mineo R, et al. Acute exacerbation of carpal tunnel syndrome after radial artery cannulation. *J Hand Surg Am.* 1993;18:455–458.
17. Kennedy AM, Grocott M, Schwartz MS, et al. Median nerve injury: an underrecognized complication of brachial artery catherization. *J Neurol Neurosurg Psychiatry.* 1997;63:542–546.
18. Marshall G, Edelstein G, Hirshman CA. Median nerve compression following radial artery puncture. *Anesth Analg.* 1980;59:953–954.
19. Kokosis G, Blueschke G, Blanton M, et al. Acute carpal tunnel syndrome secondary to iatrogenic hemorrhage: the value of the CT scan. *Hand.* 2011;6:206–208.
20. Lazaro RP, Brinker RA, Weiss JJ, et al. Femoral and obturator neuropathy secondary to retroperitoneal hemorrhage: the value of the CT scan. *Comput Tomogr.* 1981;5:221–224.
21. Patten BM. Neuropathy induced by hemorrhage. *Arch Neurol.* 1969;21:221–224.
22. Wilbourn A. Iatrogenic nerve injuries. *Neurol Clin.* 1998;16:55–82.
23. Kilpatrick J, Leyden M, Sullivan J, et al. Acute median nerve compression by haemorrhage from acute myelomonocytic leukaemia. *Med J Aust.* 1985;142:51–52.
24. Molitor PJ, Wimperis JZ. Acute carpal tunnel syndrome in haemophiliacs. *Br J Clin Pract.* 1990;44:675–676.
25. Parthenis DG, Karagkevrekis CB, Waldram MA. Von Willebrand's disease presenting as acute carpal tunnel syndrome. *J Hand Surg Br.* 1998;23:114.
26. Stewart JD. Focal peripheral neuropathies. New York: Raven Press; 1993.
27. Cho EJ, Yang JH, Song YB. Type II complex regional pain syndrome of the hand resulting from repeated arterial punctures during transradial coronary intervention. *Cather Cardiovasc Intervent.* 2013;82:E465–E468.
28. Jeong MY, Yu JS, Chung WB. Usefulness of thermography in diagnosis of complex regional pain syndrome Type 1 after transradial coronary intervention. *J Invasive Cardiol.* 2013;25:E183–E185.
29. Lai CJ, Chou CL, Liu TJ, et al. Complex regional pain syndrome after transradial cardiac catheterization. *J Chin Med Assoc.* 2006;69:179–183.
30. Longas Valien J, Sanchez Tirado JA, Aisa Hernandez O, et al. Complex regional pain type 2 after radial artery catherization: a case report. *Rev Esp Anestesiol Reanim.* 2006;53:509–512.
31. Papadimos TJ, Hofmann JP. Radial artery thrombosis, palmar arch systolic blood velocities, and chronic regional pain syndrome 1 following transradial cardiac catheterization. *Catheter Cardiovasc Interv.* 2002;57:537–540.
32. Sasano N, Tsuda T, Sasano H, et al. A case of complex regional pain syndrome type II after transradial coronary intervention. *J Anesth.* 2004;18:310–312.
33. Silviu B, Mark WJ, Reuben I, et al. Complex regional pain syndrome type 1 following radial artery catheterization. *Int J Cardiol.* 2005;101:167–168.
34. Schmid C, Tjan TD, Scheid HH. Severe complex regional rain syndrome type II after radial artery harvesting. *Ann Thorac Surg.* 2002;74:1250–1251.
35. Horowitz SH. Venipuncture-induced neuropathic pain: the clinical syndrome, with comparisons to experimental nerve injury models. *Pain.* 2001;94:225–229.
36. Lazaro RP. Neuropathic symptoms and musculoskeletal pain in carpal tunnel syndrome: prognostic and therapeutic implications. *Surg Neurol.* 1997;47:115–119.
37. Schurmann M, Zaspel J, Lohr P, et al. Imaging in early post-traumatic complex regional pain syndrome: a comparison of diagnostic methods. *Clin J Pain.* 2007;23:449–457.