J Anesth (2004) 18:310–312
DOI 10.1007/s00540-004-0266-0

Journal of
Anesthesia

A case of complex regional pain syndrome type II after transradial coronary intervention

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Abstract
The transradial approach for coronary catheterization is now a routine technique without serious complications at the puncture site. We report a case of complex regional pain syndrome type II (CRPS type II) in the hand after the transradial coronary intervention, which may alert medical personnel that the technique may cause serious regional pain with disability. A 61-year-old woman underwent coronary intervention via the right radial artery for the treatment of unstable angina. After the operation she complained of severe pain in the right hand, consistently felt along the median nerve distribution. The nerve conduction study suggested carpal tunnel syndrome. We made a diagnosis of CRPS type II, and the patient received stellate ganglion blockade, cervical epidural blockade, and administration of amitriptyline and loxoprofen. The symptoms gradually improved and her activities of daily living markedly improved. The median nerve appeared to be damaged by local compression and potential ischemia. Careful attention should be paid to avoid CRPS type II, associated with excess compression.

Key words Carpal tunnel syndrome · Median nerve · Nerve compression · Nerve injury

Introduction

The transradial approach has become a routine technique for coronary catheterization, together with advances in the miniaturization of devices for coronary catheterization. It has been reported to result in fewer complications, such as massive bleeding or nerve injury, at the puncture site [1,2]. We report a case of complex regional pain syndrome type II (CRPS type II) in the hand after transradial coronary intervention (TRI).

Address correspondence to: N. Sasano
Received: April 19, 2004 / Accepted: July 27, 2004

Case report

A 61-year-old woman underwent coronary intervention (CI) via the right radial artery to treat unstable angina. She had no history of diabetes mellitus. A 6 French guiding catheter, with an outer sheath diameter of 2.6 mm and length of 16 cm, was inserted uneventfully. CI was completed successfully, and no paresthesia was observed during insertion or removal of the catheter. Approximately 1 h after CI, she started to complain of severe continuous pain in her right hand and tightness of the hemostatic pad and bandage on the wrist. She was treated with intravenous pentazocine, which failed to alleviate the pain. Because of heparinization, hemostasis was not obtained until 7 h later, when the hemostatic pad and bandage were removed and the severe pain subsided. Although she still complained of tingling pain, she was discharged the following day. Because the pain did not improve over the next week, neurological consultation was requested. After a nerve conduction study, however, the pain became intense and intolerable, and she presented to the emergency department that night. Carbamazepine administration failed to relieve the pain, and she presented to our pain clinic the following day.

The patient was guarding her right hand by covering it with the other hand so that nobody could touch it. Physical examination revealed tingling and burning pain and severe allodynia in two-thirds of the palm on the radial side; the first, second and third digits; and the radial side of the fourth digit, which was consistent with the median nerve distribution. The hand was slightly swollen and cyanotic. Radial artery pulsation was palpable, and discoloration at the puncture site was observed. Thermography demonstrated that the temperature in the right hand was approximately 1°C higher than that in the left. The results of the nerve conduction study performed in the previous hospital revealed bilateral delayed sensory nerve conducting

velocities distal to the wrist in the median nerves and intact radial and ulnar nerves, a finding that is consistent with bilateral carpal tunnel syndrome (CTS) [3], in which the median nerve is compressed in the carpal tunnel. Careful questioning revealed that she had had slight pain in both hands for 3 years, suggesting that she had had CTS. We made a diagnosis of CRPS II based on IASP diagnostic criteria, and the patient was treated with cervical epidural blockade, right stellate ganglion blockade, and administration of amitriptyline and loxoprofen. The symptoms gradually improved, and 2 months later, allodynia in the palm faded away and she was able to use chopsticks with her right hand. At present, that is, 6 months after treatment, she still complains of pain in the hand with severe allodynia in the finger tips; however, her activities of daily living (ADL), such as cooking, shopping, and driving, have markedly improved.

Discussion

TRI is now a safe and routine technique as well as radial artery cannulation for monitoring during operation or in the intensive care unit. Usually, no or minor neurological symptoms in the hand are associated with TRI [4,5], in spite of the high incidence of radial artery occlusion after cannulation [1–5]. Papadimos and Hofmann reported a rare case of CRPS type I following TRI [6]. They found that the radial artery was occluded for a distance of 12 cm proximal to the puncture site, possibly due to the prolonged hemostatic compression. To our knowledge, no CRPS type II or nerve injury has been reported in association with radial artery puncture. On the contrary, a number of reports support the safety and usefulness of the procedure [7,8], whereas it has been shown that venipunctures may cause CRPS type II [9].

Ischemia of the median nerve seems to be the most likely cause for the development of CRPS type II in the present case. There appear to be several factors responsible for the median nerve ischemia. First, the tightness of the hemostatic pad and bandage seems to have increased the carpal tunnel pressure or may have directly compressed the median nerve for as long as 7 h, and presumably caused a considerable decrease in epineurial blood flow of the median nerve. Michelsen and Posner showed that epineurial blood flow is compromised at a pressure threshold of 20–30 mmHg in the carpal tunnel, that compression for 2 h at 50 mmHg not only decreases epineurial blood flow but also results in epineurial edema and a block in axonal transport, and that compression at even higher levels (80 mmHg) results in complete intraneurial ischemia and endoneurial edema [10]. Second, hematoma formation in the carpal tunnel after TRI could increase the interstitial pressure, which was already high because of the preexisting CTS, resulting in further deterioration of microcirculation to the median nerve. Mack et al. demonstrated acute CTS caused by a sudden increase in carpal tunnel pressure due to hemorrhage after wrist trauma and emphasized prompt carpal tunnel release [11]. Hemorrhage associated with anticoagulant therapy was also reported to cause acute CTS [10,12]. Third, any radial artery obstruction after TRI may have precipitated the median nerve ischemia because the radial artery supplies the blood flow to the median nerve. Some reports showed pain or numbness in the hand due to radial artery obstruction [13] or radial artery harvesting for coronary artery bypass graft (CABG) [14], probably because of impairment of the blood supply from the radial artery to the median nerve. The aforementioned rare case of CRPS type I after TRI reported by Papadimos and Hofmann [6] may have been caused by ischemia of the median nerve, because the radial artery was compressed for 20 h and occluded for a distance of 12 cm, even though the patient was diagnosed with CRPS type I caused by radial artery catheterization trauma.

In the present case, electrical stimulation of the damaged median nerve may have induced neuropeptide release [15] to develop CRPS. Also, psychological factors seemed to have played an important role to magnify the symptoms. The pain was iatrogenic, and the neurological examination, which the patient underwent in that hope that it would help relieve the pain, ironically intensified the pain. Furthermore, disability in the dominant hand created serious problems in terms of ADL. These led the patient to become angry and to distrust the medical staff and the treatment, which in turn aggravated the symptoms.

In this case, psychological support as well as nerve blockade and drug administration reduced the patient's pain and improved her ADL. As psychological support, we made a substantial effort to establish rapport, spent enough time on listening to her complaints, set a goal on functional restoration for daily activity rather than complete pain relief, advised her that anger worsens the pain, and so on. A possible alternative treatment that could have been effective was prompt carpal tunnel release. Mack et al. emphasized emergent carpal tunnel release within 8 h of the onset of symptoms to avoid delayed or incomplete recovery [11].

In summary, we report a case of CRPS type II after TRI, presumably due to median nerve compression and ischemia. Cardiologists should be aware that TRI may cause serious pain and disability in the hand, and careful attention should be paid to avoid CRPS type II associated with excessive compression.

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