

Complex regional pain syndrome after transfemoral coronary balloon angioplasty

Transfemoral koroner balon anjiyoplastisi sonrası kompleks bölgesel ağrı sendromu

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Summary– Presently described is the case of a 55-year-old man who developed localized pain, allodynia, hyperpathia, and swelling over the right ankle joint following coronary balloon angioplasty, which had been performed via right femoral arterial access. Bone scan confirmed the diagnosis of complex regional pain syndrome. Various analgesics were administered, along with physiotherapist-assisted exercise. The present is the first reported case of complex regional pain syndrome presenting as remote postprocedural complication of transfemoral coronary balloon angioplasty.

Özet– Bu yazıda, sağ femoral arter girişiyle gerçekleştirilmiş sağ ayak bileğinde koroner balon anjiyoplastisi sonrası lokalize ağrı, allodini, hiperpati ve şişlik gelişen 55 yaşındaki erkek olgu sunuldu. Kemik taraması kompleks bölgesel ağrı sendromu tanısını doğruladı. Fizyoterapist yardımlı egzersizle birlikte çeşitli ağrı kesiciler verilmiştir. Bu hasta, sağ transfemoral yaklaşımla yapılan koroner balon anjiyoplastisi sonrası işlem komplikasyonu olarak kompleks bölgesel ağrı sendromu ile bildirilen ilk olgudur.

After coronary balloon angioplasty was performed via right femoral arterial access, the 55-year-old patient developed localized pain, allodynia, hyperpathia, and swelling over the right ankle joint. Complex regional pain syndrome was confirmed by bone scan. Treatment consisted of various analgesics and physiotherapist-assisted exercise.

Presently described is the first reported case of complex regional pain syndrome (CRPS) presenting as remote postprocedural complication of transfemoral coronary balloon angioplasty.

CASE REPORT

A 55-year-old man presented with post-infarct angina in June 2015. Coronary angiography revealed a tight lesion in the left anterior descending artery. Coronary angioplasty with drug-eluting stent was successfully performed via transfemoral artery approach. Using a 23-G needle, 10 cc of lignocaine was injected into the

right groin area, and femoral artery puncture was performed via Seldinger technique using an 18-G femoral puncture needle. A 7-F femoral sheath was inserted over 0.035-in guidewire.

In accordance with convention, the femoral sheath was removed after 6 postprocedural hours, and local bleeding was controlled by manual compression. The patient was advised to avoid moving the leg for the next 6 hours. While maintaining steady position of the outward-turned right leg, the patient developed compression of the superficial peroneal nerve at the femoral neck.

The patient complained of severe burning pain over the dorsum of the foot and ankle 1 week after discharge. Distal pulses were normal, and tablets for neuropathic pain were prescribed. Swelling of the ankle joint, without involvement of distal toes, as shown

Abbreviation:

CRPS Complex regional pain syndrome

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Figure 1. Swelling of the right ankle with exfoliation of overlying skin.

in Figure 1, developed over the next few weeks. Pain was relentless, with extreme allodynia over the ankle joint. Localized x-ray, and arterial and venous Doppler studies were performed, with normal outcome. Diagnosis of sympathetic dystrophy was suspected.

Furthermore, triplephase bone scan revealed characteristic delayed uptake of tracer in the distal ends of the tibia, fibula, tarsal and metatarsal bones, as shown in Figure 2. Immediate rehabilitation by physiotherapy and opioid analgesics were prescribed. The patient was asked to follow up with the rheumatologist and physiotherapist.

The present is the first reported case of complex regional pain syndrome as a complication of transfemoral coronary balloon angioplasty. This discovery is offered to the cardiology forum, in an effort to aid in the differential diagnosis of a rare condition during postprocedural follow-up. The present finding suggests the possibility of rheumatological or neurological complication developing in patients after vascular intervention, and emphasizes the need for a vigilant approach, in order to save resources, for proper diagnosis, and for better patient management.

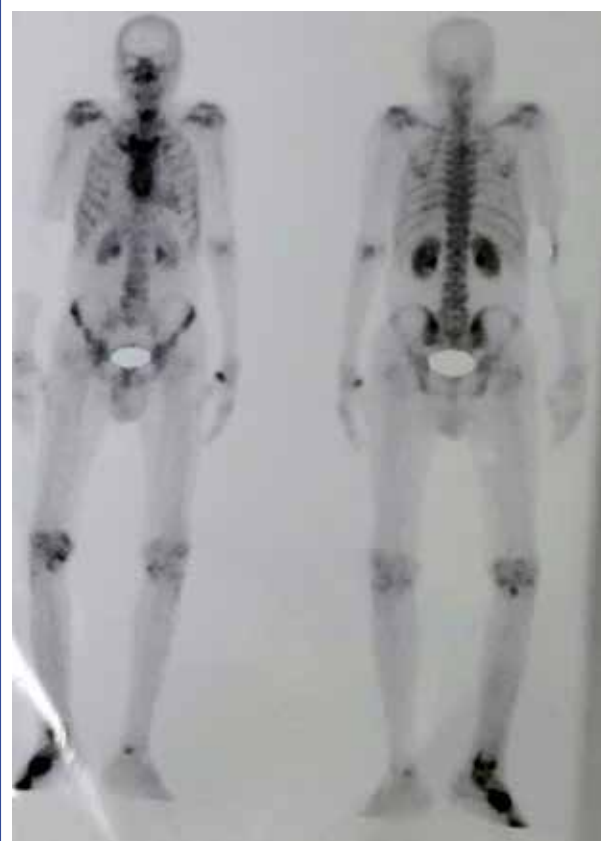


Figure 2. Triple bone scan with delayed uptake in distal ends of the metatarsals, tarsals, and lower ends of the tibia and fibula.

DISCUSSION

Complex regional pain syndrome often develops after tissue injury such as minor limb trauma, fracture, myocardial infarction, or stroke.^[1] CRPS following vascular intervention has been described, primarily in association with transradial approach. The present report is the first to describe CRPS after intervention performed via transfemoral approach, requiring a new etiology. CRPS most commonly affects a single limb, and symptoms are unrelated to the severity of the initial trauma. It is distinguished by type; CRPS type I is a regional pain syndrome that develops following illness or injury in which the nerves were not directly damaged, while CRPS type II follows distinct nerve injury.

Spontaneous pain initially develops within the territory of the affected nerve, but may eventually spread outside the nerve distribution. Pain is the primary clinical feature of CRPS and is usually burning

or electrical in sensation. Allodynia (the perception of a non-painful stimulus as painful), hyperpathia (an exaggerated pain response to a painful stimulus), and spontaneous pain occur. Vasomotor dysfunction, sudomotor abnormalities, or focal edema may occur in isolation or in combination, but must be present for diagnosis. Limb pain syndromes that do not meet these criteria are best classified as “limb pain— not otherwise specified.”

CRPS has classically been divided into 3 clinical phases. Phase I consists of pain and swelling in the distal extremity occurring within weeks to 3 months after the precipitating event. The pain is diffuse, spontaneous, and either burning, throbbing, or aching in perception. The involved extremity is warm and edematous, and the joints are tender. Increased sweating and hair growth develops. In phase II (3–6 months after onset), thin, shiny, cool skin appears. After an additional 3–6 months (phase III), atrophy of the skin and subcutaneous tissue plus flexion contractures complete the clinical picture.^[2] Autonomic testing or bone scans are occasionally useful when diagnosis is uncertain.^[3]

A variety of surgical and medical treatments have been developed, with conflicting reports of efficacy. A brief course of glucocorticoids may be helpful for

CRPS type I or II. Other medical treatments include the use of adrenergic blockers, nonsteroidal anti-inflammatory drugs, calcium channel blockers, phenytoin, opioids, and stellate ganglion blockade can also be used.^[4]

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