



## CASE REPORT

# Permanent pain and brachial plexus injury after coronary bypass grafting: Case report and reviewing the literature

*Koroner bypass sonrası oluşan kalıcı ağrı ve brakial pleksus hasarı: Olgu sunumu ve literatür gözden geçirme*

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## Summary

Neurological complications of cardiac surgery is known for almost a century. Brachial plexus injury after coronary artery bypass grafting is not a rare complication, but the frequency of reporting is less because these are temporary and often symptoms requiring treatment. In a few cases peripheral neuropathy findings are permanent and causes of disability. Diagnosis is based on symptoms, imaging and electrophysiological studies and it is important that both treatments for both medical and legal liability. Here in 63-year-old male patient was diagnosed brachial plexus injury lasting neuropathic pain the left upper limb after uneventful coronary artery bypass surgery presented and causes and consequences were discussed with literature.

Keywords: Brachial plexus injury; coronary bypass; permanent pain.

## Özet

Kardiak cerrahinin nörolojik komplikasyonları neredeyse bir yüzyıldır bilinmektedir. Koroner arter bypass greftlemesi sonrası brakial pleksus yaralanmaları nadir bir komplikasyon değildir, fakat sıklıkla bulgular geçici olduğu ve tedavi gerektirmediğinden tanınma ve raporlanma sıklığı azdır. Çok nadir olguda periferik nöropati bulguları kalıcıdır ve özür lülüğe neden olur. Tanı semptomlar, görüntüleme ve elektrofizyolojik çalışmalara dayanır ve bu hem tedavi hem de medikal ve yasal sorumluluk için önemlidir. Burada sorunsuz koroner arter bypass cerrahisi sonrası sol üst ekstremitesinde kalıcı nöropatik ağrı ve brakial pleksus hasarı saptanan 63 yaşında erkek hasta sunuldu ve literatür eşliğinde sebep ve sonuçları gözden geçirildi.

Anahtar sözcükler: Brakial pleksus hasarı; koroner bypass; kalıcı ağrı.

## Introduction

Neurological complications of heart surgery have been recognised since the early description by Fox et al. in 1954.<sup>[1, 2]</sup> Peripheral nerve injuries occurring postoperatively due to patient malpositioning have been described in the literature for nearly a century, and they still constitute a frequent cause for malpractice claims. These injuries have been reported to occur in patients in the supine position for heart surgery and in the prone position after prolonged spinal surgery.<sup>[3]</sup>

During general anesthesia the patient is at risk for injury. Peripheral nerve injuries can occur in stretching,

compression, or laceration. These injuries include a range of morbidity from transient and clinically minor injury, through to severe permanent injury.<sup>[4]</sup> The prognosis is good; recovery is the rule in most cases after an adequate follow-up period. Nevertheless, in very rare cases, the peripheral neuropathies will persist and cause disability. Here in we present a patient complicated by permanent neuropathic pain and paralysis of the left upper extremity after an eventful coronary artery bypass surgery.

## Case Report

A 63 year old male patient was admitted our neurology clinic because of severe pain, permanent pares-

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**Figure 1. (a–c)** The patient's left arm weakness.

**Table 1.** The patient's electromyographic findings

Nerve	DL (ms)	DA (mV)	PA (mV)	NCV (m/s)
L median	3.78 (NV<4)	3.41 (NV>5)	3.36	35.8 (NV>50)
L ulnar	DL-None	DA-None	PA-None	NCV-None
L radial	3.98	4.29	2.86	48.3

L: Left; DL: Distal latency; DA: Distal amplitude; PA: Proximal amplitude; NCV: Nerve conduction velocity; NV: Normal value.

thetia and paralysis of the left upper extremity after an eventful coronary artery bypass surgery. Coronary angiography performed in 2013, showed a tripl vessel disease. No neurological abnormalities were preoperatively observed. Patient had diabetes mellitus (DM) and hypertension (HT) from 2005 and used his medications. He underwent coronary artery bypass grafting (CABG) under general anaesthesia in a private hospital. According to information obtained from the patient, he was monitored in the intensive care unit (ICU) one day and his post-operative hemodynamics was stable, but he complained of left arm weakness. Patient reported that he was unable to move the left shoulder, elbow, wrist and fingers. He had paraesthesia and reduced sensation through out the left upper limb. After hospital discharge the patient was referred for physical therapy. Six months later his left arm weakness (Figure 1a–c) and sensory loss persisted, and his pain was intolerably degree especially in the nights, therefore admitted to our clinic. In that time, chest X-rays(preoperative, postoperative and current), MRI of the neck and left brachial plexus showed no obvious pathology. An electromyography was performed on 7 months after operation. The sensory nerve conduction study showed no response in the left ulnar nevre and the superficial radial nevre, the median nerve DSAP conduction velocity was slow. The motor nerve conduction study showed no response in the left the ulnar and the radial nerves and decreased amplitude and slow velocity in the right median nevre (wrist, elbow level) (Table 1,

**Table 2.** Sensory nerve conduction

Nerve	DL (ms)	A (mV)	NCV (m/s)
L median	3.14 (NV<3.4)	17.9 (NV>15)	46.2
L ulnar	None (NV<3.5)	(NV>10)	
L radial	1.98 (NV<3.5)	None (NV>5)	55.6

L: Left; DL: Distal latency; NV: Normal value; A: Amplitude; NCV: Nerve conduction velocity.

2). Needle electromyography showed no activity in the left adductor digitiminimi, giant, polyphasic motor unit potentials (MUP) with reduced recruitment in the left abductor pollicis brevis extancor indicis proprius, flexor carpi ulnaris and polyphasic and reduced MUP in left triceps and deltoid. Pathological spontaneous activities were present in all of the analyzed muscles except deltoid. These findings were consistent with subacute, severe partial axonal injury middle and lower trunk of brachial plexus.

Pregabalin 300 mg/day was started gradually increased for neuropathic pain. Patient's pain decreased, although still pain continued, duloxetine 60 mg/day was added. The patient was transferred to the rehabilitation department, and physical therapy was continued.

## Discussion

Neurological complications of heart surgery have been recognised since the early description by Fox et al. in 1954.<sup>[1,2]</sup> Peripheral nerve injuries occurring

postoperatively due to patient malpositioning have been described in the literature for nearly a century, probably with a strong bias toward underreporting, and they still constitute a frequent cause for malpractice claims.<sup>[3]</sup> Brachial plexus injury after CABG is not an infrequent complication but the exact incidence is difficult to determine, because of underreporting of nerve injury complications. Retrospective studies have found that the incidence of permanent nerve damage after a surgical procedure and anaesthesia is 0.03% to 1.4%. Commonly injured nerves include the ulnar nerve (28%), brachial plexus (20%), lumbosacral root (16%), and spinal cord (13%).<sup>[4]</sup> In a study of 312 patients by Shaw et al. detailed evaluation of the patients showed that 6,7% (21 patients) of the total number of patients had brachial plexopathy.<sup>[1]</sup>

Several mechanisms have been proposed as the cause of brachial plexus injury during CABG. During general anesthesia, especially after the use of muscle relaxants, which reduce or abolish defensive muscle tone, the patient is at risk for injury. Peripheral nerve injuries can occur in stretching, compression, or laceration. In the perioperative setting, laceration to the nerve is the least likely to occur with compression and stretching being the most likely.<sup>[3]</sup>

During harvesting of the internal mammary artery (IMA), asymmetrical traction of the sternal halves appears to be associated with a higher risk of brachial plexopathy.<sup>[5]</sup> Vander Salm et al. have documented that median sternotomy can cause first rib fractures and the fractured ribs can cause brachial plexus pathologies.<sup>[6]</sup> Posterior fractures of the first rib can easily remain undiagnosed by routine radiography but our patient's first rib fracture wasn't observed on chest X-rays.

Advanced age has been linked to brachial plexus neuropathy, but diabetes mellitus, sex, height, weight, history of smoking and presence of carotid bruit do not appear to be significant risk factors. The duration of cardiopulmonary bypass (CPB), aortic cross-clamp times, total anesthesia times, hematocrit during CPB or type of oxygenator used have also not been associated with increased frequency of brachial plexus neuropathies after CABG surgery.<sup>[7]</sup> Warner et al. identified male sex,

low and high body mass index, and longer duration of hospital stays as independent predictors for the development of persistent postoperative ulnar neuropathy in noncardiac patients.<sup>[8]</sup> Such an association has not been identified with brachial plexus neuropathies after CABG surgery.<sup>[7]</sup> Although we did not have clear information about intraoperative positioning of our patient, he had concomitant DM and HT.

It is important to distinguish brachial plexopathy from ulnar neuropathy, the main differential diagnosis, as the underlying mechanisms appear to be different as well as the prognosis. In the study by Vahl et al. eight of 27 patients who developed brachial plexus injury had persistent symptoms three months postoperatively.<sup>[9]</sup> Ulnar neuropathy's prognosis tends to be less favourable than brachial plexus injury.<sup>[5]</sup>

Electrophysiologic studies can detect changes in nerve function during the perioperative period, but these changes are very sensitive and often do not reliably predict postoperative neuropathic symptoms. Large, prospective trials demonstrating the importance of electrophysiologic studies in the early diagnosis and prevention of brachial plexus neuropathy are lacking. Measurement of motor and sensory conduction velocities, SSEP's, and electromyography are some of the common modalities used.<sup>[7]</sup> Unfortunately, the true value of electrophysiologic monitoring is not fully understood.

These injuries include a range of morbidity from transient and clinically minor injury, through to severe permanent injury.<sup>[4]</sup> The prognosis is good; however, duration of recovery can vary from hours to months. Recovery is the rule in most cases after an adequate follow-up period. Brachial plexus injury following cardiac surgery usually results in sensory deficits, while injuries following noncardiac surgeries usually result in motor deficits.<sup>[3]</sup> Although the majority of cases are transient, there are cases where the injury is permanent and may have severe implications as in our patient. In a prospective study of 335 patients by Tomlinson et al. 16 patients developed brachial plexus injury with only one patient (0.3%) still symptomatic at the time of discharge.<sup>[10]</sup> Vahl et al. in a prospective study of 1000 patients,

showed that 0.8% patients had symptoms persisting for more than three months.<sup>[9]</sup> Unlike general, our patient had very serious neuropathic pain, which we can take control with the use of two drugs, and permanent weakness of left arm, one year after operation.

In conclusion nerve injuries after anesthesia are well recognized complications. Patients undergoing open heart surgery must be closely followed up for peripheral nerve injury during the postoperative period. While causative factors in postoperative brachial plexus injury can be multifactorial, optimum positioning of surgical patients is helpful to reduce the risk of patient harm.<sup>[4]</sup> Prevention of brachial plexus injury in patients undergoing surgery entails careful positioning and padding of the arms, chest, and neck.<sup>[3]</sup> Although usually temporary and recovered without sequelae, sometimes may cause undesirable permanent results. Therefore use of preventive strategies can reduce the frequency and severity of lesions.

***Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.***

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