

CASE REPORT

Myogenic fibrosis of the flexor tendons after amphetamine drug abuse

Stefan Tserovski^{1,*†}, Simona Georgieva¹, Desislava Bogdanova², and Boris Matev¹

¹Department of Orthopedics and Traumatology, Medical University of Sofia, Sofia 1000, Bulgaria and

²Department of Neurology, 'Sveti Naum' University Hospital, Sofia 1797, Bulgaria

*Correspondence address. Department of Orthopedics and Traumatology, Medical University of Sofia, Sofia 1000, Bulgaria. Tel: +359-88-530-5666; E-mail: cerowski_stefan@yahoo.com

Abstract

Myogenic fibrosis of the flexor tendons in a 23-year-old patient was caused by intense drug abuse. He was presented in the clinic with spastic flexor tendon contracture of his right hand. The patient was treated by tendon elongation with a satisfactory result. The treatment of flexor tendon contracture in those cases is very difficult and needs to be done in a complex way.

INTRODUCTION

Amphetamine abuse increases faster than that of any other drug, including cocaine. In all cases of finger flexion contracture of young patients, local or generalized lesion of the CNS or PNS, potential drug abuse must be considered.

CASE PRESENTATION

A 23-year-old right-hand dominant man presented with a 6-month history of limited extension of the third, fourth and fifth finger of his right hand. Initially he was unable to fully extend his fingers. The condition progressed in the next 6 months, as the fingers eventually came to a permanent flexion contracture. He had history of drug (amphetamine) abuse and he was treated in a rehabilitation center 3 months before any hand symptoms have occurred. He underwent preoperative physiotherapy for 1 month without any result. He is unemployed, with no family history of diabetes, Dupuytren's disease or spastic conditions.

We did not find in the literature such as spastic flexor tendon contracture due to amphetamine abuse. That's why it was interesting for us to explore the pathogenesis of this condition.

METHODS

Patient physical examination revealed: flexion contracture of 90°–100° at the metacarpophalangeal and proximal interphalangeal joints and an extension contracture of 30° of the III, IV and V finger of the right hand. The range of motion was classified by the neutral null method: MCP (90-0-0), PIP (100-0-0) and DIP (0-0-30). Physiotherapy was applied, without any result. At the second examination we observed that the patient has adopted permanent finger contractures.

Clinically he presented pain and lack of active extension of the MCP and PIP of the III, IV and V finger (10/10 VAS SKALA). The palmar muscles were weakened. Passive extension was impossible as well as extremely painful when tried. No nodules or bridges on the palmar side of the hand were present. Spasticity

[†]Main author, S.T.S., MD

Received: November 21, 2018. Accepted: January 10, 2019

Published by Oxford University Press and JSCR Publishing Ltd. All rights reserved. © The Author(s) 2019.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

of the superficial tendon flexors III, IV and V as well as a Boutunieri-like contracture is presented.

MRI and EMG findings respond to myogenic disorder as a radicular disorder.

Flexor tendon lengthening of the superficial digitorum muscle by tenotomy at the musculotendineus junction was performed during the surgical procedure. This method has some advantages: no weakening of the tendon is observed as are no sutures in the tendon itself, and it allows early rehabilitation in association with dynamic extension splinting. Tendon elongation of the flexor digitorum profundus was also applied. Additionally a passive release of the MCP and PIP joints was performed (Figs 1 and 2). Increasing in the range of motion of the MCP and PIP joints was achieved. Flexion contracture in both of the joints was reduced by 30–40°.

Postoperative protocol: Patient was instructed to promote active and passive movements using a dynamic splint for extension. Therapy was combined with Botox application in the spastic area of his right forearm. This additional actions improved the active and passive range of motion of the fingers significantly.

DISCUSSION

Drug abuse could cause a lot of medical conditions to the entire human body. Such a specific case after amphetamine use we



Figure 1: Preoperative.



Figure 2: Postoperative.

were unable to find in the literature. With this report we would like to stress the harm after using drugs and also to point out that a local muscle group could be affected.

Amphetamines are a powerful stimulant of the CNS, drug abuse is related to several negative effects on the CNS and PNS. Monomeric cells in the brain are damaged [1] and this leads to affected striatum dopaminergic neurotransmission [2]. Chronic drug abuse leads to impaired cognitive functions, paranoia, depression, psychosis neuroleptic malign syndrome, rhabdomyolysis, Parkinson-hyperpyrexia syndrome and acute dystonic reaction [3].

Drug abuse increases stroke risk as a result of the direct effect on the cerebral circulation as increased blood pressure, vasculitis and vasoconstriction [4].

Contractures of the limbs are complications of central and peripheral neuron lesions (upper and lower motor neuron) with a late paralysis of the muscles. Diseases, affecting peripheral nerve system are most commonly myopathy and rarely peripheral neuropathy. Small cortical infarction in the precentral gyrus could lead to isolated weakness of a certain finger group: radial or ulnar and therefore imitate lesion of the peripheral nerve system pseudo-peripheral paralysis [5].

Amphetamine and other drugs (heroin, mepeidine, cocaine, pentazocine, pritramide) could be myotoxic [6]. We admit that the cause of the patients pathological condition is muscular ischemia. Pathology in the forearm and hand varies in accordance with the severity of the vascular deficit on muscles and nerves and this gives the final clinical picture.

Fibrotic myopathy is commonly seen side effect of opiate injections [7, 8]. Fibrotic myopathy and muscle weakness affected injection area and other areas of the body. Myogenic contracture as a result of parenteral narcotic use is rarely present; they usually develop after trauma, infection, degenerative changes and spasticity. A case of compression neuropathy was described: fibrosis muscle myopathy after injection of pentazocine [7]. Kumar et al. [8] reported of severe myogenic contractures of the knee joints in a 32-year-old woman after pentazocine abuse. Data for the volume of the substance was missing as well as frequency of injections, which could be related to fibrotic changes in the muscle tissue, neither the reversibility of the condition in time was proved [8].

We assumed that our patient was having a myogenic fibrosis of the extensor muscles of the forearm, followed by an overbalance of the flexor extrinsic muscles. The neurologic status showed weakness of the m. extensor digitorum communis. EMG findings responds to myogenic disorder in the hypothenar muscles as well as chronic neurogenic (radicular) disorder of C8-Th spinal nerves with normal peripheral nerves. Laboratory results for increased muscle enzymes were missing (KFK).

Differential diagnosis; we discussed neuropathy in compressive or entrapment neuropathy or double crush syndrome [9] with combination of cubital canal syndrome and cervical radiculopathy with contracture, which was not confirmed by standard nerve conduction study. Central 'pseudo-peripheral' ulnar paralysis was not accepted after the negative MRI of the brain. We initially considered the state as a rigid Dupuytren contracture, but the absence of connective tissue knots and cords on the palmar side of the hand rejected this diagnosis. We also discussed the possibility to classify the patient's condition as Volkmann's ischemic contracture, but it does not demonstrate the classic damage in the intrinsic muscles with the following clawhand deformities.

The treatment of flexor tendon contracture in those cases is very difficult and needs to be done in a complex way.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

1. McCann UD, Ricaurte GA. Amphetamine neurotoxicity: accomplishments and remaining challenges. *Neurosci Biobehav Rev* 2004;**27**:821–6.
2. Guilarte TR. Is methamphetamine abuse a risk factor in parkinsonism? *Neurotoxicology* 2001;**22**:725–31.
3. Asser A, Taba P. Psychostimulants and movement disorders. *Front Neurol* 2015;**6**:75. doi:10.3389/fneur.2015.00075.
4. Westover AN, McBride S, Haley RW. Stroke in young adults who abuse amphetamines or cocaine: a population-based study of hospitalized patients. *Arch Gen Psychiatry* 2007;**64**:495–502.
5. Lhermitte J. De la valeur sémiologique des troubles de la sensibilité à disposition radicaire dans les lésions de l'encephale. *Sem Med* 1909;**24**:277.
6. Pasnoor M, Barohn RJ, Dimachkie MM. Toxic myopathies. *Neurol Clin* 2014;**32**:647–viii.
7. Kim LY. Compression neuropathy of the radial nerve due to pentazocine-induced fibrous myopathy. *Arch Phys Med Rehabil* 1987;**68**:49–50.
8. Kumar D, Gupta A, Sharma VP, Yadav G, Singh A, Verma AK. Pentazocine-induced contractures: dilemma in management. *Indian J Pharmacol* 2015;**47**:451–3.
9. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. *Lancet* 1973;**2**:359–62.