CASE REPORT

Post-traumatic delayed onset pectoralis myospasm secondary to α - γ dysfunction

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SUMMARY

A restrained motor vehicle accident victim suffered from delayed onset left pectoralis myospasms refractory to multiple treatments: behavioural, conservative, physical therapy, opiate, muscle relaxer and incomplete response to invasive pain management spinal blocks. After conduction of a literature review, several authors had noted the mechanism of α - γ loop dysfunction resulting in myospasms, and also case studies which described painful postsurgical myospasms that were treated with neurectomy and/or botulinum toxin A with successful results. The patient in this case underwent an initial lidocaine injection to observe response to treatment, followed by two treatments with botulinum toxin A treatment with subsequent resolution of symptoms. Successful therapy and previous research supports that botulinum toxin A can be an effective treatment for myospasms secondary to trauma-induced α - γ dysfunction, as suggested by the cellular pathophysiology.

BACKGROUND

The case is of a woman involved in a motor vehicle accident who had a delayed onset of myospasms that had been refractory to traditional medical management that were effectively treated with intramuscular botulinum toxin (BT) A after being worked up with an electromyogram (EMG) and trial of lidocaine and mercaine mixture injection. Muscle contraction is a result of many complex interwoven mechanisms and in the event of trauma may cause dysfunction ranging from loss of contraction to continual contraction, all of which may happen at gross, cellular and subcellular levels. The history is that of recurrent pain episodes characterised by a focal point of spasmodic emphasis followed by regional radiant pain. The pain was characterised by a short duration followed by a gradual release ending with sustained tension ultimately followed by another myospasm, suggestive of intrafusal and extrafusal contribution to myospasms. Their contribution to α - γ loop dysfunction, in addition to their regenerative properties following injury as the nidus for the dysfunction is reviewed in this paper.

CASE PRESENTATION

On presentation, a 46-year-old woman had been ineffectively controlled with opiate analgesia for spasmodic left pectoral pain and requested evaluation for diagnosis and management of her chronic condition. As the restrained driver in a motor vehicle accident, her immediately sustained medical

injuries were minor strains to the left forearm, hand and arm, bruises from the seat belt over the lateral pectoralis and across the breast, as well as injuries to her left knee from the lower part of the dash board being displaced. She did not sustain any fractures, cervical radiculopathy or immediate neurovascular injuries. Following the accident she was given analgesics and treated conservatively for a forearm ligament strain.

About 4 months after discontinuing the physical therapy, she awoke with left-sided chest pain, which originated from the centre of her breast, radiating towards her shoulder and back. She was taken to the emergency room, where she underwent a cardiac workup, which was negative and then treated with morphine followed by a 1-week hospitalisation for continued management and observation. During the stay, she had strong spasmodic pain that averaged 6/h followed by intervening episodes characterised by tightness with spasms, which were rated 5–7 on a 10-point scale. For relief she was started on morphine and then was given an opiate drip and deferred lying on the affected side. During spasm episodes stretching of the pectoralis, palpation of the chest wall and continued use of the arm exacerbated the stabbing, pulling and throbbing pain, which was followed by a gradual release of the muscle.

After discharge she visited multiple specialists and visited a referral centre for evaluation, all of which failed to produce a diagnosis or effective course of management. Pertinent workup included: electrolytes, thyroid stimulating hormone, complete blood count, ECG, stress test, muscle biopsy, MRI head, chest, abdomen and neck, EMG wrist and arm, EEG and coronary angiography; and alternative medical treatment included: chiropractic manipulation, acupuncture and behavioural stress management.

INVESTIGATIONS

The patient initially underwent an EMG of the pectoralis muscle, which demonstrated elevated activity at rest, as well as isolated areas of increased activity that corresponded to palpable spasmodic tissue as well as her area of concern. Following EMG she underwent an initial trial of 50% 1:1 mix of lidocaine with 0.25% mercaine intramuscular injections.

DIFFERENTIAL DIAGNOSIS

► Electrolytes: hypokalaemia, hypomagnesaemia, hypocalcaemia

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- ▶ Myopathy: infectious, eosinophilic, inclusion body, ossificans, idiopathic, traumatic, sarcoidosis, medication induced, steroid induced, hypothyroid induced, inflammatory, type IV McArdle's
- ▶ Neurology: neurodegenerative disease, mononeuritis multiplex, polymyalgia rheumatica, amyotrophic lateral sclerosis, complex regional pain syndrome
- ▶ Cardiac: myocardial infarction, unstable angina, vasculitis
- ▶ Other: fibromyalgia, toxin

TREATMENT

Intramuscular BTA injection

As the initial therapy with the lidocaine/mercaïne mix had been effective, she then received the initial botulinum 100 units in 1:1 mix with preservative free saline therapy in an area circumferential to the inner pectoralis muscle. This was followed, 4 months later, by repeat injections to further areas of spasmodic activity.

OUTCOME AND FOLLOW-UP

Three to 4 weeks following the initial botulinum therapy, the patient was without pain or spasmodic activity in the treated area, as well as without acute or subacute complications. The second treatment was again without acute complications and with resolution of pain and spasmodic activity. On follow-up, the major symptom was some mild weakness with certain daily tasks that involved the left pectoralis muscle and some pressure in the area with repetitive use of the muscle.

DISCUSSION

Complications of nerve tissue regeneration included: degeneration, poor reinnervation and misconnections during reinnervations.^{1 2} In the Alvarez *et al.*,¹ study on reorganisation of Ia afferents, after peripheral nerves were injured, on regeneration they were capable of responding to an electrically induced stimulus, but when stretched nearly all of the neurons failed to depolarise. This suggested that electrical and mechanical pathways differed for causing a central discharge. To assess the extent of regeneration and activity of muscle spindle fibres, researchers¹ used the marker vesicular glutamate transporter isoform 1 (VGLUT-1). In addition to the active and functional presence in the periphery VGLUT-1 demonstrated the ability to ectopically depolarise surrounding neurons with glutamate.³

With research, Amir *et al.*⁴ further supported the phenomenon of after hyperpolarisation potentials (AHPs) and its ability to prevent neuron depolarisation secondary to repetitive stimulation attempts. This supports that during a continuous muscle contraction the nerve may fail to respond to electrical stimuli during repolarisation secondary to the AHP, thereby preventing propagation. Although nerve propagation would be inhibited, damaged muscle tissue would persist to contract secondary to irresponsiveness of the stretch-evoked pathway, which would normally inhibit further contraction via Ib fibres from the Golgi tendon organ.^{5 6} Being such, as neural mechanisms fail to inhibit continuous contraction, cessation becomes dependent on depletion of local factors: glutamate and electrolytes.

The aforementioned provides pathophysiological support to recurrent spontaneous muscle spasms following traumatic muscle injuries, which when left untreated, are subject to depletion of local factors for contraction cessation. In addition to others, Mast⁷ and Layeeque *et al.*⁸ have further described both surgical and non-surgical treatments for myospasms, respectively. Mast⁷ described bilateral poststernotomy pectoral myospasms, which ultimately were treated with uncomplicated

bilateral pectoral neurectomies. Layeeque *et al.*⁸ completed a non-randomised retrospective study to assess the efficacy of BT serotype A on the treatment of the myospasm after breast reconstruction with subpectoral tissue expansion. Although this was a non-random study, the data demonstrated a profound advantage to the use of BT for control of the spasms and improvement in hospital stay, as well as decreased use of morphine.

BT was chosen in this case secondary to its mild side-effect profile, a long half-life, irreversible inhibition at the presynaptic cleft and minimally invasive administration. Furthermore this prevents the damaged neural mechanisms, the cross-synaptic depolarisation as well as direct depolarisation, from causing muscle contraction leaving only local mechanisms, such as calcium influx from the sarcomere, as a means of muscle contraction.

Review of similarly published studies:

Layeeque: described the efficacy of botox on the treatment of myospasm after breast reconstruction with subpectoral tissue expansion. Non-randomised study demonstrated profound advantage to botox use with resolution of spasms.

Mast: described a patient, status poststernotomy for osteomyelitis, who subsequently developed bilateral pectoral myospasms, treated with neurectomies. Similar in presentation of muscle belly injury and resulting spasmodic pain.

Konishi: described antagonistic presentation of γ loop dysfunction in quadriceps femoris after anterior cruciate ligament reconstruction. Antagonistic, as it is a distal tendon disruption, producing weakness instead of spasmodic activity.

Learning points

- ▶ α - γ Loop dysinnervation after trauma may result in intermittent unrestricted muscle stimulation at the level of the intrafusal and extrafusal muscle fibres, resulting in myospasm.
- ▶ These mechanisms are short-lived secondary to depletion of essential components and increased after hyperpolarization potentials (AHPs) after tetany.
- ▶ As essential components are being depleted and AHPs increased after tetany, the muscle spindle unit will cease to contract at which point the Ib contribution will be equal to or greater than the Ia, causing muscle relaxation.
- ▶ Botulinum toxin A is an option for long-term management of α - γ dysfunction-induced myospasms.
- ▶ Diagnosis of spasm is made with electromyogram with an initial trial to therapy assessed with intramuscular lidocaine/mercaïne injection.

Contributors DLB provided sources and involved with patient correspondence. KM was involved with information synthesis, formatting of material, provided sources for orthopaedic references and correspondence. PB was involved with data synthesis, composition of the paper, ensuring accuracy of information provided and correspondence with publication.

Competing interests None.

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