

Shoulder-Hand Syndrome Post Myocardial Infarction: Case Report

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Citation

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Abstract

Shoulder-Hand Syndrome (SHS) is reported to occur in 10-15% of patients following acute myocardial infarction (MI), and can involve pain and stiffness of the shoulders and hands. This case report describes a 69 year-old lady who presented to the Accident and Emergency Department complaining of severe shoulder pain three weeks after suffering an MI. Recent case reports of SHS post MI are scanty. The case highlights the need for clinicians to be aware of SHS, so that the diagnosis is considered, and treatment and follow-up is arranged as appropriate.

CASE REPORT

A 69 year-old lady presented to the Accident and Emergency department complaining of bilateral arm pain. She mentioned that she had had an MI approximately 3 weeks prior to this presentation. Since then she had been relatively well.

She had first noticed non specific neck pain 3 days before this attendance. There had been no preceding injury or trauma, and the pain settled of its own accord after one day. The following day the patient was aware of generalised left arm pain. By the next day the pain had spread to involve her right arm. When she arrived at the hospital she described severe pain in both shoulders, spreading down the anterior aspect of her arms as far as her elbows. She said that she was unable to move her shoulders to any reasonable extent, and on further questioning she admitted that she had noticed swelling of both hands, although this had now resolved, and that her hands were mottled.

On examination she was haemodynamically stable. There was no cervical spine tenderness. She was able to fully flex her neck, but movement in all other planes was markedly restricted.

She had no tenderness over her shoulders. She could abduct and flex her shoulder joints to 20-30° but was unwilling to move them in other directions.

Movements were normal at her elbows, wrists and hands. Her arms had normal tone, power was 5/5 below the

shoulders, coordination was normal, biceps reflexes were normal, and sensation was normal to fine touch.

Her electrocardiogram (ECG) revealed a rate of 98, sinus rhythm, had a normal axis with no acute changes. Her blood tests were normal, including cardiac enzymes.

A diagnosis of Shoulder-Hand Syndrome was made. The patient was discharged home with non steroidal anti-inflammatories, and physiotherapy was arranged. She was advised to see her General Practitioner if there was no improvement within a few days so that steroid injections could be given if appropriate.

DISCUSSION

Shoulder-Hand Syndrome (SHS) is reported to occur in 10-15% of patients following acute myocardial infarction (MI).¹ The syndrome has three stages. It initially involves pain, tenderness, and limitation of motion at the shoulder girdle, which may be followed by swelling, pain, stiffness, and discolouration of the hands and fingers.² Subsequently pain, swelling and discolouration can resolve, but stiffness and flexion deformity of the fingers become more prominent.² Patients can go on to develop progressive atrophic changes in the hands, severe atrophy of the interosseous muscles, and limitation of movement at the metacarpophalangeal and interphalangeal joints. Flexor tendons can contract, particularly on the ulnar aspect of the hand, creating an appearance similar to that found in Dupuytren's.^{1, 2}

The course of SHS is variable. It can develop at the time of MI, or up to 16 weeks later.³ It can last from 1 month to over 5 years.^{1, 3} One or both shoulders may be involved, and in most cases, one or both hands.¹ Treatment of the syndrome can involve anti-inflammatories, physiotherapy, local heat, hydrocortisone injections, sympathectomy/sympathetic blockade, TENS, acupuncture, biofeedback, psychotherapy, removal of trigger areas, dorsal column stimulators, intraspinal narcotics.^{1,4}

The precise aetiology of SHS is unclear. In 1936 it was suggested that it resulted from nerve fibres being caught in a cardiac scar.³ The inclusion of sympathetic fibres in scar tissue may explain why only a percentage of patients develop the syndrome, why it takes weeks to months to develop, why in one individual it may occur after one attack and not another, and why there is no relationship between the severity of the syndrome and the extent of the initial myocardial infarction.¹

In 1946 the term Reflex Sympathetic Dystrophy (RSD) was coined by Evans.⁴ RSD refers to a syndrome in which there is disturbance of autonomic regulation, hyper- or hypohydrosis, oedema, hyper- or hypoesthesia, allodynia, motor dysfunction with weakness or tremor, joint stiffness, trophic changes of skin, hair and nails, and psychological disturbance.⁴ Features tend to have a glove and stocking distribution.⁴

In 1995, RSD was renamed Complex Regional Pain Syndrome (CRPS).⁴ The pathophysiology may involve chronic maladaptive sensitisation of wide dynamic range neurons in the dorsal column of the spinal cord. When

peripheral skin mechanoreceptors are activated, transmissions occur via large diameter A beta nerve fibres. These are influenced by activity of local sympathetic nerves. Normally these impulses do not activate wide dynamic range neurons. However, when such neurons are sensitised, as in CRPS, their threshold of stimulation falls so that they do respond to sympathetically maintained impulses evoked from nociceptive afferent A beta fibres. This information is converted into noxious impulses which go to the brain.⁴

There are many causes for CRPS, including trauma, radiculopathy, cerebrovascular accidents, cancer, multiple sclerosis, infections.⁴

SUMMARY

Shoulder-Hand syndrome is reported to occur in up to 15% of patients post MI.¹ However, recent case reports are scanty. If the syndrome is as common as suggested it is important that clinicians are aware of its nature. Patients presenting with upper limb pain/stiffness who have a history of ischaemic heart disease should have the diagnosis considered, so that appropriate treatment and follow-up is arranged, and subsequent morbidity is minimised.

References

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