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Complex Regional Pain Syndrome-Acute Myocardial Infarction Unveiling: Case Report

Authors
Neethu Joy[1], Bijumon L[2], G Madhu[3], Suresh Raghavan[4]
[1]Junior Resident, [2]Junior Resident, [3]Lecturer, [4]Professor
Department of General Medicine, Govt.T.D Medical College, Alappuzha,Kerala,India
*Correspondence Author
Neethu Joy
Junior Resident, Dept of General Medicine, Govt.T.D Medical College, Alappuzha, Kerala, India-688005
Email: *neethujipmer@gmail.com*

ABSTRACT
Complex regional pain syndrome(CRPS),formerly known as reflex sympathetic dystrophy, are neuropathic pain conditions usually affecting one limb, after an injury, due to damage or malfunction of peripheral and central nervous systems. Here we present a case of CRPS following an unusual inciting injury and unusual timing of presentation. A high index of clinical suspicion led to the correct diagnosis in this atypical presentation.

INTRODUCTION

CRPS are conditions characterized by continuing regional pain that is disproportionate in time or degree to the usual course and associated with abnormal sensory, motor, sudomotor, vasomotor, trophic findings. Historically CRPS was described by Silas Weir Mitchell during the American civil war. He also introduced the name "causalgia". It frequently begins following a soft tissue injury, a fracture, or surgery. CRPS is divided into type 1 and type 2. Differentiating feature is type 2 is associated with a nerve injury. CRPS is diagnosed by clinical evaluation and there is no diagnostic gold standard.

CASE REPORT

A 76 year old male, manual labourer, heavy smoker, non alcoholic with no significant past history or family history presented with left sided chestpain suggestive of ischemic heart disease.

On examination his vitals were normal. General physical examination was normal.

Systemic examinations were found to be normal with no relevant positive findings. Investigations revealed normal routine blood examination, LFT, RFT, lipid profile, TFT, blood sugar, serum electrolytes, urine routine were normal. Viral markers-negative. HIV, VDRL- nonreactive.

CRP was negative. CPK-MB mild elevation present. Chest x ray was normal. Trop I was positive, ECG suggestive of acute anterior wall MI. Thrombolysis not done due to late presentation. Treatment started with antiplatelets, LMW Heparin, cholesterol lowering agents, ACE Inhibitors.

On the 7 th day he developed sudden onset of severe pain and swelling of right upper limb mainly hand and forearm with minimal involvement of shoulder. Examination of right upper limb showed edema, redness, increased warmth, hyperalgesia and allodynia of hand both dorsal and palmar aspect. Detailed examination of right upper limb showed sensory signs (allodynia, hyperalgesia, hyperpathia) not confined to a particular radical or peripheral nerve, vasomotor signs (hyperemia, increased warmth) present all over the forearm mainly over dorsum with minimal tenderness, sudomotor (edema, increased sweating) diffusely all over the forearm more over dorsal aspect, motor signs (decreased range of motion) mainly in finger joints and wrist joints, consistent with clinical diagnosis of CRPS. No other neurovascular deficit. Movements of joints of hand and wrist were limited. Arterial pulsations were normal. Adsons test was negative. There is no limitation of movement of neck or other body parts. No bluish discolouration of left upper limb. Patient didn't have any intravenous cannula in the right upper limb. Clinically we suspected the possibility of CRPS and further investigated. Other possibilities of inflammatory arthritis, cellulitis, DVT, peripheral neuropathy, thoracic outlet syndrome, thrombophlebitis, cervical spondylosis with radiculopathy excluded by detailed clinical examination and relevant investigations.

Blood Culture including fungal culture-negative.

X ray of right upper limb was normal with no evidence of fractures or other pathology.

MRI of cervical spine showed degenerative changes with no evidence of compressive myelopathy. Nerve conduction study of right upper limb was normal. Doppler study of right upper limb was normal. Bone scan was done and showed increased uptake.

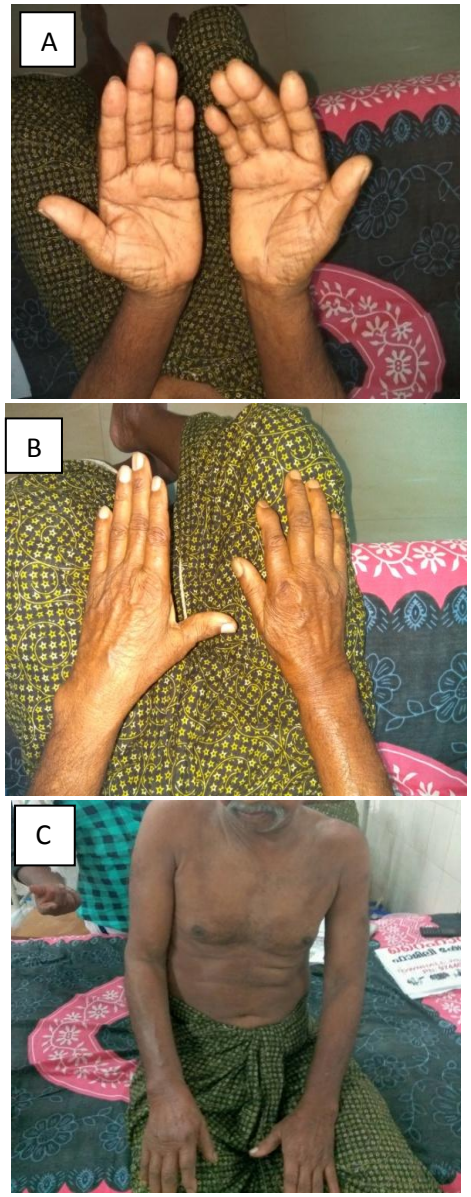

Fig.1 A edema and movement limitation of right hand.

B edema of both hands and wrist (dorsal aspect)

C sparing of arm and shoulder joint

DISCUSSION

The synonyms for CRPS include reflex dystrophy, causalgia, transient osteoporosis, posttraumatic osteoporosis, shoulder hand syndrome, sudeck's atrophy, algodystrophy, reflex sympathetic dystrophy etc. Precipitating factors include trauma, fractures, immobilization, MI, strokes, surgery, burns, malignancies, emotional stress etc. CRPS occurs in women more than men. CRPS usually begins days to weeks after the inciting

event. CRPS develops due to peripheral and central nervous system sensitization by persistent noxious stimuli. Initial changes are mediated by nociceptive (A delta and polymodal c) fibres which transmit to the spinal cord causing a release of excitatory neurotransmitters (glutamate, asparagine) which act on NMDA receptors releasing inflammatory neuropeptides, substance P and CGRP. These lower synaptic excitability of synapses and making them hyperexcitable. Chronic CNS sensitization occurs through afferent processing of the persistent peripheral noxious stimuli.ACE inhibitors blocks the normal metabolism of neuropeptides such as substance P and CGRP, thus having increased risk of developing CRPS.

CRPS is diagnosed by the new IASP- revised CRPS criteria (Budapest criteria) and criteria includes:

1) Continuing pain disproportionate to any inciting event.
2) At least one symptom of sensory (hyperesthesia or allodynia), Vasomotor (Asymmetry of temperature or skin colour changes), Sudomotor (Edema or asymmetry of sweating), Motor (Decreased range of motion, motor dysfunction).
3) At least one sign of sensory (hyperalgesia, allodynia), vasomotor (asymmetry of temperature or skin colour changes), sudomotor (edema, asymmetry of sweating), motor (Decreased range of motion, motor dysfunction).
4) No other diagnosis better explaining the signs and symptoms.

CRPS has three stages. Stage 1(acute stage) is characterized by pain, swelling, colour changes, painful movements etc. Stage 2 (dystrophic phase) is characterized by beginning of atrophy of subcutaneous tissue and intrinsic muscles. Stage 3 (atrophic stage) is characterized by severe atrophy, muscle spasms, progression of osteoporosis leading to pathological fractures etc.

CRPS is a diagnosis of clinical exclusion. No laboratory tests or imaging modality is available to confirm the diagnosis. The characterstic radiologic appearance is soft tissue swelling and patchy osteopenia. A three phase technetium bone scan (TPBS) is helpful in confirming the diagnosis, with poor sensitivity and good specificity. Bone scan performed early in the disease may be abnormal but scans can be normal as the disease progresses.

Treatment of CRPS includes anti inflammatory agents (NSAIDs, corticosteroids), anticonvulsants, narcotic drugs, anti osteoporotic therapy, physical therapy (electro acupuncture, massage, ultrasound), stimulation of inhibitory neurons (spinal cord stimulation), sympathetic blockade and psychological therapy. All therapies work best when instituted early in the course of CRPS.

This patient was started with NSAIDs and he showed dramatic improvement and he is under regular follow up.

CONCLUSION

CRPS is a diagnosis of exclusion. Atypical presentations pose diagnostic challenge. CRPS is reported to occur in upto 15 % of patients post MI. In our case having an atypical early presentation(usually occurring within three months) and the inciting event is acute MI(rare inciting event), a high index of clinical suspicion based on the clinical findings which led to the correct diagnosis of CRPS. Introduction of ACE inhibitors also contributed to the inflammation. This case underlines the importance of proper clinical examination since no other investigations help in the diagnosis. Early diagnosis improves the outcome and morbidity of patient due to early introduction of treatment and follow-up. Delayed diagnosis leads to more recurrence and bad prognosis.