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NEUROGENIC OSTEOARTHROPATHY OF THE SHOULDER

Radiology

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ABSTRACT

Neurogenic osteoarthropathy or Charcot joint of the shoulder joint is an uncommon disease characterized by painless destruction of joint due to loss of sensory perception of the joint. Appropriate clinical assessment, diagnostic workup and treatment are essential in the management of Charcot joint. Patient education on activity modification is crucial step in management to slow the impairment associated with a Charcot joint.
This report highlighted a case of neurogenic osteoarthropathy of the shoulder joint secondary to syringomyelia. The clinical features, pathophysiology, differential diagnosis, diagnostic workup and management options are discussed.

KEYWORDS

Neurogenic osteoarthropathy, Charcot joint, syringomyelia, Shoulder

INTRODUCTION

Neurogenic osteoarthropathy is a chronic, destructive condition of the joint due to loss of proprioception, pain and temperature perception [1]. Diabetes mellitus, syringomyelia and syphilis are the most common etiologies of this disease. Other occasional associations are myelomeningocele, leprosy, peripheral nerve injury, spinal cord injury, amyloidosis, familial congenital insensitivity to pain and dysautonomia. Shoulder joint involvement in Charcot arthropathy is uncommon and has been reported in 6% of patients with neuropathic arthropathy.

Neurogenic arthropathy should be considered in cases of unexplained joint swelling which is disproportionate to the pain, with a limited range of active motion and an exaggerated range of passive movements of the affected joint. We are presenting a case of charcot arthropathy of the shoulder secondary to syringomyelia with classical clinical and radiological findings.

Case report

A 36-year-old male patient with known case of syringomyelia was referred for the further treatment in Gujarat cancer and research institute, Ahmedabad. The patient was having painless restriction of right shoulder joint movement. Patient have negative history of trauma, diabetes mellitus and syphilis. Patient have no history of fever. His blood pressure was 120/76 mmHg, pulse 82 beats/min and respiratory rate 14/min. All the hematological investigations were within normal limits.

His haemoglobin (Hb) was 14.8g/dl, WBC 7.7 x 10^3/uL, total plateletcount 283 x10^3 /cm, blood urea 8.85 mg/dl and serum creatinine 0.59 mg/dl.

Patient had history of sudden headache and upper backache during drilling work for which he was operated 12 years ago. Thereafter patient developed complain of decreased movement with difficulty in elevation of right shoulder since 3 year.

Physical examination revealed diffuse swelling about the right shoulder. On palpation there was no joint tenderness or local rise of temperature. There was increased girth and exaggerated passive movements possible at the right shoulder joint in all directions compared to opposite side. There was generalized muscle wasting in the right upper limb.

There was reduced sensation of the entire right upper extremity. The triceps, biceps and brachioradialis reflex were absent.

On X-ray (Fig-1) of right shoulder joint there was amputed appearance of upper shaft of right humerus with resorption of head of humerus. There was loose bony fragment noted within articular area.

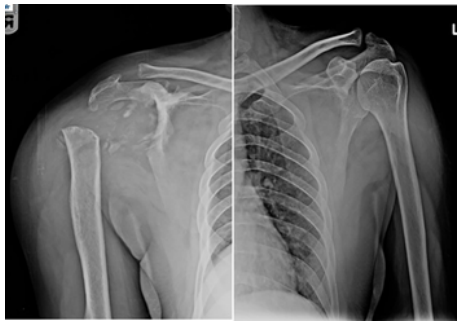

Fig- 1: X-ray right & left shoulder AP view: Widened joint space with amputed appearance of upper shaft of right humerus with resorption of head of humerus.

There was loose bony fragment noted within articular area. Left

CT imaging of this patient shows amputed apperance of right humerus with widening of the shoulder joint and multiple bony fragment with synovial effusion (fig-2). Patient was further worked up with magnetic resonance imaging (MRI) of the cervical and thoracic spine which shows syrinx formation extending from cervico-medullary junction to inferior end plate of D3 vertebral body (Fig 3). Biopsy of this region shows reactive synovial cells (fig-4) which is seen commonly in reactive arthropathy.

He was managed with limb elevation, shoulder abduction brace and non- steroidal anti-inflammatory and was advised regarding activity modification. At present, the patient is doing well with conservative mode of therapy and is performing his daily activities and self care.

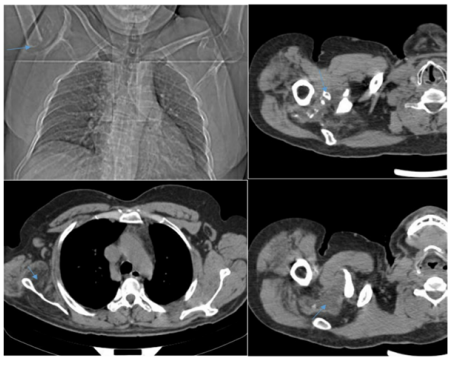

Fig-2 (A) Topogram shows amputated appearance of right shoulder joint. (B)NCCT Thorax image shows bony fragments in shoulder joint (blue arrow). (C) CT Thorax image shows disuse atrophy of right rotator cuff muscles (arrow); normal left rotator cuff muscles. (D) CT thorax image shows reactive synovial thickening

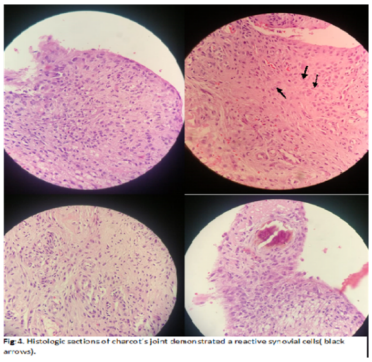

Fig 1. Histologic sections of charcot's joint demonstrated a reactive synovial cells(black arrows).

DISCUSSION:

Neuropathic arthropathy is a destructive articular disease that occurs secondary to a loss or impairment in joint proprioception. Subsequently, the involved joint undergoes premature and excessive traumatic degenerative changes that lead to severe destruction and instability.

Neurological signs such as altered gait pattern, loss of deep reflexes and pain insensitivity should alert the physician to the possibility of underlying condition. Evaluation of the involved joint will reveal relatively painless instability, enlargement and crepitus, all of which may be extreme (bag of bones). (2)In the early phases, recurrent, painless effusions may be the only sign. Neuropathic joint may develop over a period of weeks, months, or years, depending on the cause, location, and severity of the underlying disease. (3)Laboratory findings may be negative for the joint disease but may be positive when applicable to the causative disease, such as syphilis and diabetes.

Currently, there is general agreement on the neuro-traumatic theory where by destructive joint changes occur as a sequelae to ineffective protective neurological mechanism. (4)This mechanism is thought to be owing to an initial increase in intra-osseous blood flow that is neurologically initiated, stimulating osteoclastic resorption.

Consequently, as bone is resorbed (atrophic phase) fracture and joint damage occur. With weight bearing, hypertrophic changes such as osteophytes, sclerosis and loose bodies ensue (hypertrophic phase) (3)Two essential radiographic appearances of a joint occur according to the degree of bony proliferation or resorption, hypertrophic and atrophic. Hypertrophic pattern is characterized by 6Ds: joint Destruction, increased sub-chondral density, debris, dislocation, disorganization and bone destruction. This is most commonly found and more pronounced in the weight bearing joints of the lumbar spine, hip, knee and ankle.

While atrophic patterns how a distinct lack of hypertrophic features. It may occur as a secondary change in a previously hypertrophic joint, or arise in otherwise normal joint. This type is seen most commonly in non-weight bearing joints of upper extremity, especially the shoulder, elbow, wrist as well as hip and foot. The appearance may simulate a sharp surgical transverse amputation of the articular end of the bone. At other times, the bone will taper gradually towards the joint space and has been likened to a licked candy stick. Atrophic variety of shoulder most commonly associated with syringomyelia.

The differential diagnoses to be considered are septic arthritis, tuberculous and microbial infection, primary and metastatic malignant tumor of the bone or soft tissue, synovial chondromatosis, idiopathic osteolysis, nephropathy, Gorham disease and Winchester syndrome.

Neuropathic osteoarthropathy of the shoulder usually progresses slowly, but rapid progression can occur months or even weeks. Diabetic patients are usually affected with the joints of the foot and ankle, larger joints such as the knee is usually involved in patients with syphilis.

Shoulder and the elbow joints are affected in cases of syringomyelia. Syringomyelia is a disorder characterized by a fluid containing cavity (syrinx) in the medullary canal of the spinal cord. The most common sites are the lower cervical and upper thoracic segments, and the cavity may propagate proximally. The etiology for syrinx can be congenital, trauma, degenerative, infection, tumor or vascular related [1]. MRI is considered the gold standard investigation for its visualization.

Syringomyelia is a potential cause of Charcot osteoarthropathy of the shoulder. It may present as instability or a frank dislocation of the shoulder. A neuropathic joint associated with syringomyelia may develop early or late in the course of disease.

The management for neuropathic arthropathy is conservative. Preventing further trauma to the affected joint with proper splinting is the key to treatment. Splinting and aspiration of large effusions will reduce further ligamentous laxity. Studies have reported unsatisfactory results of hemi-arthroplasty for the resurfacing operations. In our case, conservative management of the affected shoulder joint provided an acceptable level of function with tolerable pain. The goal of management is to avoid repetitive trauma to the joint so as to prevent its further damage, which can be best done with activity modification and the use of braces.

CONCLUSION

Neurogenic osteoarthropathy is an uncommon disease. But proper knowledge of the condition and thorough clinical evaluation in the form of proper history, physical examination including the neurological system, appropriate radiological tests and pathological evaluation when needed will help in the diagnosis. Once the diagnosis of a neuropathic joint has been made, its etiology should be found out with appropriate blood investigations, joint aspiration and MRI of the spine. Syringomyelia in cervical cord, amputated appearance of humerus with widening of joint space and bony fragments within joint is characteristic finding of atrophic variety of neurogenic osteoarthropathy.

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