Milwaukee Shoulder Syndrome: A Case Report

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Abstract

Milwaukee shoulder syndrome is a progressively destructive shoulder arthropathy mostly seen in mostly women over 70 years age. It is associated with severe pain and generalized restriction of joint movements, usually associated with rotator cuff tear. A 85 years old female presented to our hospital with chief complaint of pain and decreased range of movements in both shoulder joints. Investigations were done including FNAC and Biopsy which could not identify any evident cause. Therefore diagnosis was made on clinico-radiological bases and patient was managed. Surprisingly patient had relief with plain NSAID’s compared to opioids.

Keywords: Milwaukee; Shoulder syndrome; Diagnosis; Osteoarthritic

Introduction

Milwaukee shoulder syndrome was described by McCarty et al. in 1981 and consists of the association of complete tear of the rotator cuff, osteoarthritic changes, non-inflammatory joint effusion containing calcium hydroxyapatite and calcium pyrophosphate dehydrate crystals, hyperplasia of the synovium, destruction of cartilage and subchondral bone and multiple osteochondral loose bodies [1]. It usually starts with a history of trauma leading to rotator cuff injury following which there is deposition of calcium hydroxyapatite crystals. Milwaukee disease usually involves shoulder but neck, lumbar spine, hips, wrists, hands, elbows, knees and feet involvement have also been reported in literature [2]. Milwaukee shoulder syndrome is very rare with only a handful of cases reported in literature so far.

Case Report

A 85 years old female presented to our OPD with chief complaints of pain and swelling in Left shoulder since 2 years increased since 3 weeks with severe restriction of movements. There was no significant history of any trauma. There was no history of any other system involvement. Pt was being managed outside with only painkillers and was suspected for malignancy so was referred to our hospital.

On examination patient had swelling in bilateral shoulder region with severe tenderness, skin was shiny, normal in color with no sinus or scars. There was no local rise of temperature. Swelling was diffuse, hard, tender, non-mobile, fixed to bone, involving humerus and clavicle, non-pulsatile, no crepitus, no egg shell cracking sound was heard either, non-fluctuant without any signs of inflammation. X-ray’s of both shoulder joints and Chest were done which showed severe destruction of bilateral shoulder joints with erosion of bilateral glenoid, humeral head, (Table 2) clavicles and acromion, more severe on the right side (Figures 1 and 2).

Blood work was done

Outside MRI Lt Shoulder s/o?: Plasmacytoma Lt shoulder (Table 1). Pt was taken up for percutaneous needle biopsy from Rt shoulder. Frank blood with bony chips were aspirated which were sent for gram stain, culture and histopathology. Gram stain did not show any bacteria, culture sensitivity did not have any growth. Histopathology showed no evidence of Tuberculosis or any malignancy. Pt was again taken up for FNAC which showed only blood and no malignant cells.

Repeat MRI B/L shoulder with contrast was done

Lt. Shoulder: was suggestive of destructive of the left humeral head and glenoid process of scapula with mild synovial enhancement with no significant synovial effusion. Rt. Shoulder: was suggestive of resorption and destruction of Right Humeral head and glenoid process of scapula with mild synovial enhancement and synovial effusion. MRI Screening of Spine for did not reveal an underlying neurological disorder such as Hypoesthesia, Paraparesis, Monoparesis.

Table 1: X-ray’s of both shoulder joints and Chest were done which showed severe destruction of bilateral shoulder joints with erosion of bilateral glenoid, humeral head, clavicles and acromion, more severe on the Right side.

<table>
<thead>
<tr>
<th>Range of Motion</th>
<th>Right Shoulder</th>
<th>Left Shoulder</th>
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<tbody>
<tr>
<td>Flexion</td>
<td>30°</td>
<td>50°</td>
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<tr>
<td>Extension</td>
<td>10°</td>
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<td>Abduction</td>
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<tr>
<td>Internal Rotation</td>
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<td>40°</td>
</tr>
<tr>
<td>External Rotation</td>
<td>20°</td>
<td>30°</td>
</tr>
</tbody>
</table>

Table 2: Outside MRI Lt Shoulder s/o: ? Plasmacytoma Lt shoulder.

Figure 1: Pain AP view of Rt Shoulder and Lt Shoulder showing destruction of humeral head with destruction of lateral third of clavicle and acromion process with involvement of glenoid rim.

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syringomyelia. Diagnosis by exclusion was made on clinico-radiological basis after ruling out all other common causes and patient was treated conservatively with NSAIDs and physiotherapy to which patient responded well. MRI of shoulder joint shows hyperintensity in bilateral shoulder joints with destruction of bilateral humeral head with glenoid, clavicle and acromion process with mild synovial enhancement.

Discussion

Milwaukee syndrome is a well-defined clinical entity characterized by destructive arthropathy associated with calcium hydroxyapatite and calcium pyrophosphate dehydrates crystals depositions. The true etiology of the disease is still unclear but it is believed that intra-articular calcium hydroxyapatite crystal deposition induces the release of lysosomal enzymes which attack the periarticular tissues. The presence of activated collagenase and neutral protease capable of disrupting articular cartilage and enzymes has been demonstrated in cultured synovial lining cells [1]. Milwaukee shoulder syndrome can be unilateral or bilateral, but in unilateral type the dominant side is involved more frequently [3]. But in our case it presented as bilateral with more destruction of the non-dominant side. The peculiarity of our case was that patient had more relief with simple NSAID's compared to opioids as stated in literature.

References