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International Journal of Physical Medicine and Rehabilitation

Case Report

Shoulder Avascular Necrosis, Arthritis, Tendinopathy and Polyneuropathy, Long Term Complications of HIV: Case Report

Naglaa Hussein[1], MD, PhD, Mark Thomas[2], MD, MD, Mohammad Dwary [3]

[1]Department of Physical Medicine, Rheumatology and Rehabilitation, Alexandria University, Alexandria, Egypt;[2]Department of Physical Medicine and Rehabilitation, Albert Einstein College of Medicine, New York, United States;[3]Department of Physical Medicine and Rehabilitation, Alexandria Medical pc, Bronx, New York

INTRODUCTION

Long term complications of Human Immune Deficiency Virus (HIV) might be variables affecting joints as well as nerves presented as variable forms of arthritis and polyneuropathy. Avascular necrosis also has been reported. All these complications reported in the literature with variable incidence rate.

CASE STUDY

54-Y-old female with past medical history of hypertension, Human Immune Deficiency Syndrome (HIV) presented to pain management clinic complaining of bilateral hip pain x3 years, bilateral shoulder pain x1 year and Low back pain for many years associated with numbness of both feet. She is being treated from HIV with HAART therapy with undetectable viral load for many years and normal CD4 count. She denies history of alcohol abuse or use of any drugs, also no history of steroid intake. Clinical exam; Hip: positive tenderness over both groins and both greater trochanteric bursae. ROM: Right/Left: Flexion: 70/100, external rotation: 10/15, Internal rotation: 5/5. Positive FABER test bilaterally with groin tenderness, leg length discrepancy on left side, with limping gait. Shoulder exam: tenderness over gleno-humeral and acromioclavicular joint bilaterally. ROM: Right/Left: Flexion 120/140, Abduction 100 bilaterally, external rotation full, Internal rotation 20 bilaterally. Positive Neer and Hawkins tests Bilaterally. Back exam: Alignment: lordosis, positive tenderness over lower lumbar vertebrae and both sacroiliac joints, paravertebral muscle spasm. limited lumbar spine ROM. Neurological exam: glove and stocking hypoesthesia, Deep tendon jerks showed upper extremity jerks G1, unelicited patellar jerks bilaterally and ankle jerk G1 bilaterally. Manual muscle testing revealed upper extremities muscles: G5/5 except for both thenar muscles G4/5, Lower extremities: ankle muscles G4/5, both Quadriceps G 4/5, Hip flexors right G3/5 and left G 4/5. Laboratory findings revealed: Normal CD4 count, Normal CD4% and CD4/CD8, HIV-1 RNA PCR ultra <20 D (normal), HIV 1-RNA (log-10) <1.30, Normal hemoglobin, Glycosylated hemoglobin 5.6. Normal Sedimentation rate and C-reactive protein, normal liver and kidney functions. Rheumatological testing all negative including: Anti CCp, rheumatoid factor, Antinuclear antibodies, anti-Double stranded DNA. Shoulders X- ray: severe degenerative osteoarthritis of humeral head and narrowing of gleno-humeral and acromio-clavicular joints. Shoulder MRI: avascular necrosis of humeral head, together with degenerative arthritis of gleno -humeral joints with effusion, degenerative arthritis of acromio-clavicular joints, as well as rotator cuff tendinosis. Hips X ray: degenerative arthritis, worse at the left side. Dorso-lumbar spine X-ray: Degenerative arthritis. The patient declined nerve conduction study.

DISCUSSION

This case represents long standing complication of HIV and or anti-retroviral therapy. The patient has undetectable viral load for years and normal CD4 count, but still suffered from avascular necrosis of both shoulders together with shoulders, hips and dorso-lumbar arthritis. Rotator cuff tendinosis as well as peripheral polyneuropathy. These could not be explained by any autoimmune rheumatological disease in light of negative laboratory findings. In addition, her age is not old enough to explain this severe degenerative arthritis. There is no history of alcohol abuse or long-term steroid use that can explain avascular necrosis. Her glycosylated hemoglobin was 5.6 consider within normal although could be representing a prediabetes status, that can explain to less extent the rotator cuff tendinosis and may be the polyneuropathy as it was reported that these complication can happen in patient with prediabetes but the presence of all these combinations; avascular necrosis , hips, shoulders and spine arthritis as well as polyneuropathy can be better explained by long term HIV complication and or both HIV as well as prediabetes state contribute to the pathology.

Revising literature regarding the incidence of avascular necrosis among HIV patients. HIV-infected individuals have an increased risk of Avascular Bone Necrosis (AVN). Antiretroviral

Correspondence to: Naglaa Hussein, Department of Physical Medicine, Rheumatology and Rehabilitation, Alexandria University, Alexandria, Egypt, E-mail: naglaa.hussein@alexandriamedical.net

Received date: March 29, 2021**; Accepted date:** April 05, 2021**; Published date:** April 12, 2021

Citation: Hussein N, Thomas M, Dwary M (2021) Shoulder Avascular Necrosis, Arthritis, Tendinopathy and Polyneuropathy, Long Term Complications of HIV: Case Report. Int J Phys Med Rehabil. 9:600.

Therapy (ART) and particularly Protease Inhibitors (PI) have been implicated as a risk factor, Among HIV-positive persons, AVN incidence is up to 100 times higher than in the general population, and, rarely, AVN occurs at multiple sites [1].

Wang et al. suggested that the increased incidence of osteonecrosis in HIV/AIDS may be due to an increased frequency of risk factors previously associated with osteonecrosis such as hyperlipidemia, corticosteroid use, alcohol abuse, and hypercoagulability. Use of protease inhibitors was not independently associated with osteonecrosis. Use of megesterol acetate may be a new osteonecrosis risk factor [2].

In this particular case, we have not found any of those suggested risk factor that can explain avascular necrosis of shoulders rather than long term HIV infection and anti-retroviral therapy.

Mehta et al. [3] reported that the incidence rate of AVN is higher in HIV-infected patients than in the general population. They had suggested that Although the exact etiology of AVN remains unclear, the literature has shown a relationship between AVN and exposure to Highly Active Antiretroviral Therapy (HAART). In addition, AVN has been reported before the era of HAART, thus suggesting the involvement of other causative factors as well. They reported three case reports. Affected sites of AVN included the hip and shoulders. The incidence of AVN within their patient population was higher than the general population. Although the introduction of HAART has improved patient longevity, but lead to longer exposure to antiretroviral (ARV) therapy. Thus, it is likely that treatment-related complications may become more apparent in the HIV-infected population. This may be the case with AVN [3].

Adizie et al. reported that articular syndromes that have been described in association with HIV include HIV-associated arthropathy, Seronegative Spondylo-Arthropathies (SPA) (reactive arthritis, Psoriatic Arthritis (PsA) and undifferentiated SPA), Rheumatoid Arthritis (RA) and painful articular syndrome.

HIV-associated arthritis can occur at any stage of HIV illness. It presents as an asymmetric oligo arthritis, symmetrical polyarthritis or as a monoarthritis. The asymmetric, oligo arthritis variant is the most common form, has a male preponderance, and predominately affects the knees and ankle [4].

Mody et al. [5] reported that HIV may increase the risk of certain rheumatic conditions in HIV patients through several mechanisms. Researchers have found the HIV p24 antigen and HIV DNA in the synovial fluid of specific joints affected in individuals with HIV-associated arthropathy. Related viruses are known to cause arthritis in animals and humans. HIV also causes dysregulation of the immune system that can facilitate autoimmunity. The virus causes not only immunodeficiency, but also sometimes other transient serum abnormalities, such as positive IgG rheumatoid factor and anti-CCP antibodies [5].

As regard our patient, the patient suffered from bilateral symmetrical non-erosive sero-negative arthritis of the hips and shoulders. That could be explained in light of HIV associated arthropathy as mentioned previously.

Mota et al. studied the relation between HIV infection and reactive arthritis. They reported that HIV changed the profile of reactive arthritis and intensity of articular and extra-articular manifestations. HIV seems to play a role in the main reactive arthritis immunopathogenesis mechanisms either acting as direct arthritogenic agent or causing an immune dysfunction in the CD4 T lymphocytes T CD 8 relationship leading to the deregulation in the production of cytokines or in advanced immunosuppression stages predisposing to infection by other arthritogenic pathogens [6].

Could this case be a picture of reactive arthritis associated with HIV as explained by the above- mentioned mechanism. It is possible but the patient has no history of extra-articular manifestation, but it still be a possibility.

In this case there was glove and stocking hypoesthesia with depressed deep tendon jerks and lower extremities muscle weakness, this represents peripheral polyneuropathy sensory-motor, that could be also explained partly by the long-standing HIV status. The presence of prediabetes that was discovered by us while checking all laboratory tests, could be an accentuating factor for the occurrence of this polyneuropathy. However, the patient glycosylated hemoglobin was 5.6, the laboratory reference considers this within normal level and considered 5.7 is the start of abnormality. We still have to consider it as a possible perpetuating factor to the occurrence of peripheral polyneuropathy.

Revising Literature regarding the incidence of polyneuropathy among HIV patients, we found that Schütz et al. reported that Distal Symmetric Polyneuropathy (DSP) related to human immunodeficiency virus (HIV) is one of the most common neurologic complications of HIV, possibly affecting as many as 50% of all individuals infected with HIV. They have suggested that there are two potentially neurotoxic mechanisms play a crucial role in the pathogenesis of HIV DSP: neurotoxicity resulting from the virus and its products; as well as adverse neurotoxic effects of medications used in the treatment of HIV [7].

Scott R et al. suggested that Signs of peripheral neuropathy remain despite virologic/immunologic control but frequently occurs without symptoms [8].

CONCLUSION

Shoulder avascular necrosis, arthritis, tendinopathy and polyneuropathy could be among long term complications of HIV although controlled status of the disease.

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