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Case Report
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Unraveling the Enigma: A Rare Case of Avascular Necrosis of Hip Joint in a Young, Marfanoid Patient with Suspected Tuberculosis

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Abstract

Tuberculosis is an infectious disease caused by mycobacterium tuberculosis, it is a contagious infection that usually affects lungs but can spread to other parts of the body as well and one of its complications is Pott disease. Rarely TB can cause osteonecrosis of the femoral head in an immunocompromised individual. Ten to fifteen percent of osteo-articular TB is TB arthritis of the hip joint. Osteonecrosis of the femoral head can be caused by excessive use of corticosteroids, using alcohol in opulence or secondary to infections.

We present to you a case of 35 years old male with marfanoid features and a history of pleural tuberculosis who presented in emergency with complains of bilateral lower limb weakness and low-grade fever.

This case report depicts association of TB with AVN; hence TB should be considered a differential while diagnosing cause of AVN especially in endemic areas where TB is still prevalent.

Categories: Internal Medicine, Infectious Disease, Orthopedics

Keywords: hfref: Heart failure with reduced ejection fraction; Lower limb pain; Marfanoid features; Tuberculosis; Avascular Necrosis (avn)

Introduction

Tuberculosis (TB) is a serious disease caused by an acid-fast bacillus i.e., Mycobacterium Tuberculosis [1]. It primarily affects lung but can affect other parts and organs of the body as well such as kidney and spine [2]. Spinal tuberculosis is also known as Pott disease. It can lead to osteomyelitis, kyphotic deformity and spinal mechanism inability [3]. This is case report of 35-year-old male with marfanoid features and a history of pleural tuberculosis who presented in our emergency with complains of bilateral lower limb weakness.

Case Presentation

A 35 years old male with tall and slender build, disproportionately long arms and fingers, pointed nose and mitral valve regurgitation, ejection fraction of 35%. The habitus was consistent with Marfanoid features (Figure 1,2,3). He was farmer by profession, known case of hepatitis C for which he took treatment and achieved Sustained Virologic Response (SVR), he had history of pleural tuberculosis diagnosed 4 years back for which he took anti-tuberculous therapy for 6 months. Now presented with a history of low-grade fever, undocumented weight loss, bilateral lower limb weakness and mild pleural effusion (Figure 4).

According to the patient he was in a usual state of health 2months back when he gradually developed lower limb weakness associated with pain in lower back and hip joint upon walking which progressed over 2 months to a level that he got bedridden because of weakness and pain. He also complained of low-grade fever, usually nocturnal and was relieved by taking paracetamol. His vitals at the time of admission were Blood pressure 110/70, pulse 105bpm, random blood sugar 187 mg/dl, respiratory rate 22 and temperature was afebrile. Initially an impression of Pott's disease was made, but upon examination there was no tenderness at the back while upon hip joint examination he had tenderness and limited movements especially external and internal rotation. All his baselines and relevant labs were sent (Complete blood count, Erythrocyte sedimentation rate, liver function

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Figure 1: Marfanoid Features showing pointed nose.

Figure 2: Disproportionately long fingers.

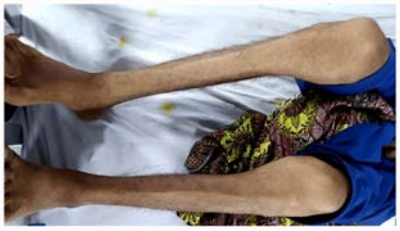

Figure 3: Long, wasted lower limbs.

tests, urea creatinine electrolytes, Prothrombin time/International normalized ration, serum calcium levels, vitamin D levels, C- reactive protein levels and lactate dehydrogenase levels done results of which are mentioned in (Table 1): his Human Immunodeficiency Virus, Anti-nuclear Antibody test and Extractable Nuclear Antigen Antibodies profile were also sent and were negative, however his direct Coombs test was positive). His lumbosacral and hip joint Magnetic Resonant Imaging (MRI) were performed findings of which were consistent with AVN and ruled out any possibility of paravertebral abscess or Pott's disease. (Figure 2,4). His echo showed severe mitral regurgitation with dilated left atrium and ventricle, HFREF (Heart Failure with reduced Ejection Fraction), Ejection Fraction was 25% to 30%, TAPSE (Tricuspid annular plane systolic excursion) was 21mm and PASP (Pulmonary arterial systolic pressure) was 30mm Hg (Figures 5-7). On the basis of history, labs and MRI report a suspicion of TB was made which was the likely cause of AVN hip joint and we planned to order patient's CT scan and joint fluid aspiration while taking orthopedics on board and planned to take cardiologists on

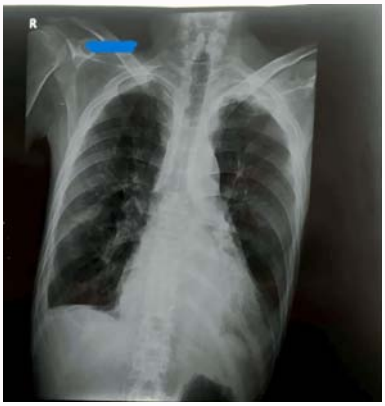

Figure 4: Chest X-ray Posterio-anterior view showing mild pleural effusion.

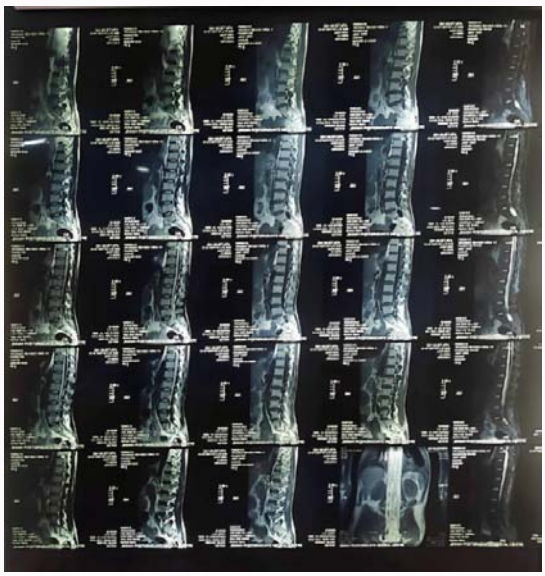

Figure 5: MRI lumbosacral spine and Hip showing avascular necrosis of hip joint.

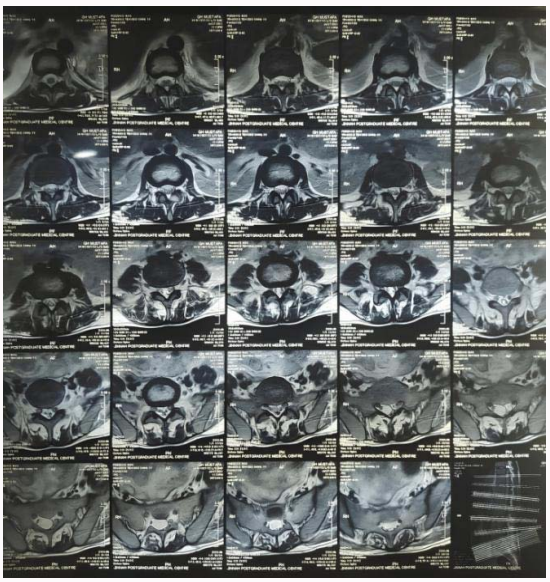

Figure 6: MRI showing Avascular necrosis of hip joint.

Table 1: Showing Reference Range and Patient's Laboratory Findings from April to August 2023.

Variables	Reference Range	Apr-23	May-23	Jun-23	Jul-23	Aug-23
ESR (Erythrocyte Sedimentation Rate)	0-10 mm/1st hr	-	-	-	-	110
RBC (Red Blood Cell)	4.50 – 5.50 ×10^12/L	3.75	3.4	5.34	4.7	-
Hemoglobin	13.0 – 17.0 g/dL	9.6	9.3	14.6	12	8.3
HCT (Hematocrit)	40 – 50 %	31.4	31.7	46.9	39.9	25.6
MCV (Mean Corpuscular Volume)	83.0 – 101.0 fL	83.7	93.2	87.8	84.7	79.5
MCH (Mean Corpuscular Hemoglobin)	27.0 – 32.0 pg	25.6	27.4	27.3	25.5	25.8
MCHC (Mean Corpuscular Hemoglobin Concentration)	31.5 – 35.0 g/dL	30.6	29.3	31.1	30.1	32.4
Platelets Count	150 – 400 ×10^9/L	844	916	334	75	858
Neutrophils	40 – 80 %	80.4	93.6	92.6	59	72
Lymphocytes	20 – 40 %	4.1	3.1	2.1	37	20
Eosinophils	01 – 06 %	14.2	0.2	0.1	1	4
Monocytes	02 – 10 %	1.2	3	5	3	4
Serum Calcium	8.6 – 10.2 mg/dL	-	-	6.4	8.6	-
Serum 25-Hydroxy Vitamin D	> 30 ng/mL	-	-	-	3	-
Serum C3 (Complement 3)	-	-	1.12	-	-	-
Serum C4 (Complement 4)	-	-	0.11	-	-	-
INR (International Normalized Ratio)	-	-	0.98	1.21	1.47	1.15
Total Bilirubin	< 1.0 mg/dL	-	-	0.6	0.39	0.43
Serum Alkaline Phosphatase	< 120 U/L	-	-	63	74	109
Serum ALT (Alanine Aminotransferase)	< 42 U/L	-	80	18	16	10
Serum Uric Acid	2.5 – 7.0 mg/dL	-	-	-	3.7	-
Serum Urea	10 – 50 mg/dL	-	-	18	24	31
Serum Creatinine	0.5 – 1.5 mg/dL	-	1.2	0.8	0.62	0.37
Serum Sodium	136 – 149 mEq/L	-	135	130	131	129

TB: Tuberculosis; ANA: Anti-Nuclear Antibody; ENA: Extractable Nuclear Antigen; AVN: Avascular Necrosis; ESR: Erythrocyte Sedimentation Rate; HIV: Human Immunodeficiency Virus; MRI: Magnetic Resonance Imaging.

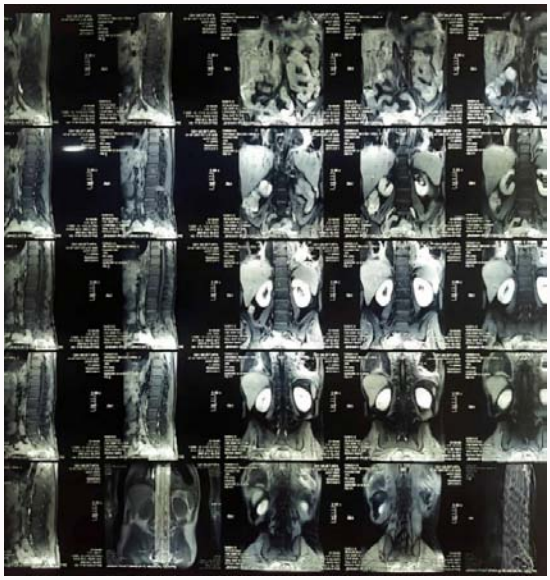

Figure 7: MRI Lumbosacral spine and hip showing synovitis.

board too for his severe MR and Heart failure.

Discussion

Disruption of the proximal femur's blood supply results in Avascular Necrosis (AVN) of the femoral head. The changes in the blood supply could be the consequence of a non-traumatic cause or happen after a traumatic incident [4]. Hematogenous spreads from a primary source, such as the lungs or lymph nodes, are the cause of osteo-articular tuberculosis; contiguous spread from nearby tissue or direct injection are other possible routes. Synovitis is the initial indication of TB arthritis, which eventually develops into periarticular osteopenia, marginal erosion, and joint destruction. Mild, local, and constitutional symptoms are typical for patients [5].

The disease's nature does not involve hip discomfort initially and have few or no X-ray abnormalities. Therefore, it might not be identified until advanced when hip discomfort manifests itself or hip mobility decreases, X-rays reveal femoral head collapse, and hip joint degeneration occurs [6].

It is uncommon for TB arthritis to manifest as femoral head osteonecrosis. Ten to fifteen percent of osteoarticular TB is TB arthritis of the hip joint [7]. Osteonecrosis of the femoral head can be caused by excessive use of corticosteroids, using alcohol in opulence or secondary to infections [8]. Corticosteroids are used frequently for the treatment of different rheumatological conditions. Usage of corticosteroids is associated with several risk factors such as diabetes, glaucoma, osteonecrosis and several kinds of infections [9].

In our case patient who had marfanoid habitus was diagnosed with osteonecrosis of femur that is avascular necrosis of femur

secondary to suspected tuberculosis. The rise in ESR and CRP in this instance, along with the periarticular osteoporosis, marginal erosion, and progressive joint space narrowing resembling the Phemister triad on the X-ray were indicators of a suspected hip infection secondary to tuberculosis.

In immunocompromised patients' tuberculosis should always be considered as the top differential diagnosis for the osteonecrosis of the femoral head [10]. This case reinforces the need for early diagnosis and treatment of Tuberculosis to reduce the possibility of the devastating consequences of complications like paraplegia.

Conclusion

This case report depicts association of Tuberculosis (TB) with Avascular Necrosis (AVN); hence TB should be considered a differential while diagnosing cause of AVN especially in endemic areas where TB is still prevalent. Confirming the diagnosis is helpful for the treatment of patient. Further workup needed to confirm the diagnosis and initiate relevant treatment.

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