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CASE REPORT

Avascular Necrosis of Bilateral Femoral Head in a Patient with Sickle Cell Disease: A Case Report

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ABSTRACT
Avascular necrosis (AVN) popularly called osteonecrosis, a serious orthopaedic complication or pathology caused by the interruption of blood flow to the bones resulting in tissue death. It occurs very frequently in sickle cell disease (SCD) as this disease is caused by genetic mutations that produce abnormal haemoglobin leading to vascular occlusion and ischemia. This is a case presentation of a 21-year-old female who has SCD and is presented with complaints of back pain radiating to knee, difficulty in standing, and stiffness of shoulders. Imaging revealed symptoms of AVN in both femoral heads and osteonecrosis of the left iliac wing during evaluation. Laboratory tests confirm anemia and elevated inflammatory markers, while MRI early diagnosed AVN. The patient received a multidisciplinary approach with pharmacological treatment like Hydroxyurea for treating sickling; NSAIDs and opioids for pain relief; and enoxaparin to prevent clotting, recommended physiotherapy to maintain joint mobility, and surgery was considered after some time. This case exemplifies the challenges associated with managing AVN within an SCD population and underscores the need for early diagnosis and a holistic treatment strategy to avoid disease progression and boost patient outcomes.
Keywords: *Osteonecrosis, Vaso-occlusion,* bone infarction, Ischemia, Sickle Cell Disease, Hydroxyurea

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INTRODUCTION

Osteonecrosis, referred to as avascular necrosis (AVN), occurs when the blood supply to the joints and head of a long bone becomes disrupted, leading to death of the tissue involved. This condition is common among orthopaedic disorders in sickle cell disease patients. Eventually, these altered forms of bone structures will cause destruction of bones with pain and loss of function because they collapse. Some bones have minimal blood supply; hence even slight vascular or perfusion compromise can lead to avascular necrosis in them. The femoral head is the most commonly involved bone by the above process. [1] The local AVN is generally because of trauma or micro trauma. In contrast, in systemic AVN, epiphyseal necrosis or bone infarction occurs and is mostly multifocal. AVN is an adverse condition which eventually leaves the hip joint osteoarthritic especially in younger adults. It is most common among middle-aged individuals, with about 75% of cases occurring between the ages of 30 and 60, resulting in a significant economic impact due to the productive age group affected. Additionally, 30%-70% of cases involve both hips [2]. Osteonecrosis of the hip and shoulder joints can be caused by various factors, including alcohol abuse, steroid use, chemotherapy, immunosuppressive drugs, and sickle cell anemia. In the early stages, patients may not experience any symptoms, but as the disease progresses, most develop joint pain. Initially, the pain occurs when weight is placed on the affected joint but may later be present even at rest. The pain typically progresses gradually and can vary in intensity from mild to severe. As the

condition worsens and the bone and joint surface deteriorate, the pain may intensify, limiting joint movement and causing stiffness. MRI is extremely effective for the early detection of osteonecrosis, offering 99% specificity and sensitivity. MRI can also be used to quantify the size of the lesion by digitizing the affected area of the femoral head with abnormal bone texture. Osteonecrosis is particularly common in patients with sickle cell disease (SCD) due to the disease's promotion of sickle cells and bone marrow hyperplasia [3].

Sickle cell disease has a genetic basis for abnormal haemoglobin production that leads to the vascular occlusion, for which ischemia and tissue infarction occur thereafter. The effect of micro vascular occlusion due to sickle cell disease is especially striking in the bones and joints, with several sites progressively affected over time. AVN commonly occurs in the femoral head, and sometimes it occurs in the humeral head, temporomandibular joint, or even vertebrae. Patients suffering from SCD and AVN experience severe chronic pain and are likely to depend on opioid analgesics for pain relief, thus leading to increased cases of acute medical care. This would dramatically decrease the quality of life (QoL) and further increase already high healthcare costs dedicated to managing SCD [4]. The treatment of avascular necrosis mainly includes pain management and maintenance of joint functions. Analgesics, such as NSAIDs or anti-inflammatory drugs, are considered as pharmacological options. Non-pharmacological approaches include physical therapy (range-of-motion exercises, strengthening exercises, functional training) or surgical interventions (like osteotomy, core decompression, or joint replacement) [5].

CASE REPORT

On February 9, 2024, a twenty-one-year-old female patient presented to a tertiary care hospital with primary complaints of back pain persisting for 6 months, radiating to her lower knee joint, difficulty standing for the past 6 months, and stiff shoulder pain with restricted movement for the last 2 months. She has a medical history of sickle cell disease, which often initially presents with symptoms like fatigue, pallor, and abnormal haemoglobin levels in lab tests. She is not currently taking any prescribed medications. Her social history revealed no tobacco or alcohol use.

On examination, the patient's vital signs were normal, but she appeared pale. Laboratory results showed haemoglobin at 7.7 g/dL, RBC count at 3.70 x10^6 cells/cmm, WBC count at 11,200 cells/cmm, platelets at 244,000/cmm, ESR at 72 mm/hr, MCH at 20.4 pg, MCHC at 32 g/dL, MCV at 63.9 fl, and PCV at 24.1%. Both liver and renal function tests were normal. A sickling test was performed and returned positive. A compressive myelopathy test was conducted to rule out rare neurological complications of SCD, and a rheumatoid test to rule out rheumatoid arthritis, which was negative. The D-dimer test was elevated at 3759.5 ng/ml (normal range: 0-500 ng/ml).X-rays of thigh and knee suggested multiple enlarged lymph nodes noted in right lower region of femur and Subcutaneous tissue over bilateral knee joint upper thickened and diffuse synovial thickening. MRI of cervical spine and pelvic region conclude that loss of cervical lordosis indenting the anterior thecal sac at C5 - C6 and mild bilateral hip joint effusion, AVN of bilateral femoral head and osteonecrosis of left Iliac wing. For the suspected manifestation, the patient was hospitalised for 8 days. He was being treated with the following medications: Inj. Tramadol (100mg/2ml), Cap. Hydroxyurea (500mg) once a day, Tab.Sodium bicarbonate(300)mg, Inj. Pantoprazole (40 mg/dl) once a day, Inj. ondansetron(4 mg/dl) once a day, Tab. FA (Folic Acid) once a day, Inj. Ceftriaxone (1 g) from third to seventh day, Inj. Enoxaparin (40 mg) from third to sixth day, Tab. Ibuprofen(400 mg) once a day. Hydroxyurea capsule and sodium bicarbonate were given for sickle cell disease. Hydroxyurea is used in SCD to increase the production of fetal haemoglobin (HbF), which helps reduce the sickling of red blood cells. This is crucial for decreasing the frequency of vaso-occlusive crises (painful episodes caused by blocked blood vessels) and improving blood flow to tissues. Sickle cell patients are prone to acidosis due to chronic haemolysis (breakdown of red blood cells), leading to the release of intracellular acids therefore sodium bicarbonate provide alkaline medium. Tramadol injection which is opioid analgesic and ibuprofen tablet which belong to class of NSAIDS were given for pain management. For acid reflux, injection pantoprazole and nausea was treated with injection ondansetron. Tab folic acid and multivitamin injection was prescribed for deficiency of vitamins. Ceftriaxone injection was given to prevent further infection as dead or damaged bone tissue (due to AVN) can become susceptible to bacterial infection. Injection Enoxaparin is prescribed to reduce the risk of blood clots due to the immobility and hypercoagulable state in SCD, and to improve blood circulation, which may help prevent further ischemic damage to the bones.

Table 1; Current treatment chart of patient during hospitalization

Drug	Dose	Route	Frequency	1	2	3	4	5	6	7	8
Inj.Tramadol	50mg/ml	IV	SOS	√	√	√	√	√	√	√	√
T. Sodamint	300mg	PO	2-2-2-2	√		√		√	√	√	√
Hydroxyurea	500 mg	PO	OD	√	√	√	√	√	√	√	√
Inj. Pantoprazole	40mg	IV	12hr	√	√	√	√	√	√	√	√
Inj. Ondansetron	4mg	IV	8hr	√	√	√	√	√	√	√	√
Inj.NS	1 pint	IV	12hr	√	√	√	√	√	√	√	√
T.FA	5mg	PO	OD	√	√	√	√	√	√	√	√
Inj.Optineuron	1A	IV	12 hr	√	√	√	√	√	√	√	√
Inj.Ceftriaxone	1g	IV	8hr			√	√	√	√	√	
Inj.Enoxaparin	40 mg	SC	OD			√	√	√	√		
T. Ibuprofen	400 mg	PO	OD					√	√	√	

DISCUSSION

AVN is a complication of sickle cell disease that occurs when bone tissue dies due to a lack of blood supply. It's the second most common chronic complication of SCD. Here, we presented the case of a 21 years old female patient who had similar complaints as that Avascular necrosis i.e. back pain (radiating to lower knee joint), difficulty in standing, rigid shoulder pain and difficult in movement. Patient as medical history of sickle cell disease. On performing X-ray we noticed multiple enlarged lymph nodes noted in right lower region of femur and Subcutaneous tissue over bilateral knee joint upper thickened and diffuse synovial thickening. MRI concluded that the patient had mild bilateral hip joint effusion, AVN of bilateral femoral head and osteonecrosis of left Iliac wing. The aim of treatment is to stop the pain and maintain a mobile joint. Treatments include resting the joint, physiotherapy, the use of pain relief, joint replacements and bone grafts. Pharmacological treatments include analgesics (NSAIDs, opioids), Hydroxyurea to reduce sickling, and anticoagulants like enoxaparin to prevent clotting. Non-pharmacological interventions involve physical therapy to maintain mobility and strength. Preventive measures, including optimising hydration and managing vaso-occlusive crises, are critical for minimizing progression. Surgical options such as core decompression, osteotomy, or total joint replacement may be considered in the future if symptoms progress.

Here the patient was prescribed Hydroxyurea and sodium bicarbonate for managing sickle cell disease. Tramadol and ibuprofen were administered for pain relief, while enoxaparin was given to prevent blood clots. Ceftriaxone was included to prevent infection, and a multivitamin was added to address any vitamin deficiencies. There was no clinical pharmacist intervention found in the given treatment. No drug-drug, drug food and drug disease interaction were found in the case.

CONCLUSION

The presented case report illustrates the complexity of managing AVN in a 21-year-old female SCD patient, who experienced back, knee, and shoulder pain for months prior to treatment. AVN is fairly common in SCD, with up to 50% of patients developing it by age 35. Her management included pharmacological treatments to manage pain, address SCD-related complications, and prevent further ischemic damage. Hydroxyurea was crucial for reducing the frequency of vaso-occlusive crises, and anticoagulant therapy (enoxaparin) was used to prevent clotting. Surgical options were not pursued in this case, but they are available for patients with advanced disease. The patient's treatment highlights the need for a multidisciplinary approach to AVN management in SCD, addressing both the underlying disease and the secondary complications to improve long-term outcomes.

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