Picture stories

Osteonecrosis in an adolescent boy with acute lymphoblastic leukaemia

***Julius Scott**[1], **Latha Magatha Sneha**[2], **Sughapriya Velagoundanpalayam Chandran**[3], **Prabhu Radan**[4], **Sabitha Challa**[5]

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Introduction

Recent advances in therapeutic strategies improving survival of acute leukaemia, focus is now on the long term effects of the treatment affecting the quality of life in these survivors. Osteonecrosis with a prevalence rate of 1-65% is now being increasingly identified as the important source of morbidity in leukaemia survivors[1]. The multisite involvement and poor correlation between the symptoms and diagnosis, challenges the early clinical diagnosis.

Case report

A 16 year old boy, diagnosed as having T cell acute lymphoblastic leukaemia (ALL) one year back and currently on maintenance chemotherapy, presented to us with pain in the left knee joint for 3 months and was found to be limping for the past 1 month. He had received prednisolone in the induction phase (60mg/m^2 x 4 weeks) and intermittent dexamethasone (10mg/m^2 x 2 weeks) in the first and second delayed intensification phases. His weight was 83 kg and height 177 cm. Initially he was evaluated with a plain x-ray and ultrasound scan of the left knee joint which were inconclusive. Blood investigations revealed normal complete blood counts. As the pain was progressive and restricted his daily activities, magnetic resonance imaging (MRI) of the left knee was done which revealed multiple serpiginous subchondral pattern, suggestive of ischaemia (Figures 1 and 2).

[1]*Professor, Head, Division of Paediatric Haematology and Oncology,* [2]*Assistant Professor,* [3]*Final year MD Paediatrics,* [4]*Associate Professor, Department of Radio Diagnostics,* [5]*Medical Officer, Sri Ramachandra University, India*
**Correspondence:* jxscott@hotmail.com

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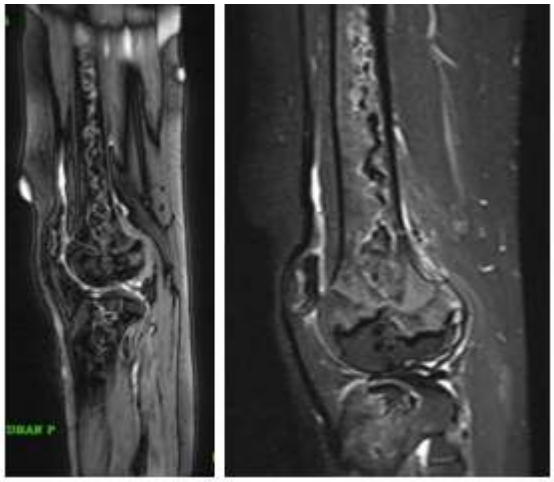

Figure 1: *MRI left knee showing multiple bone infarcts of tibia and femur*

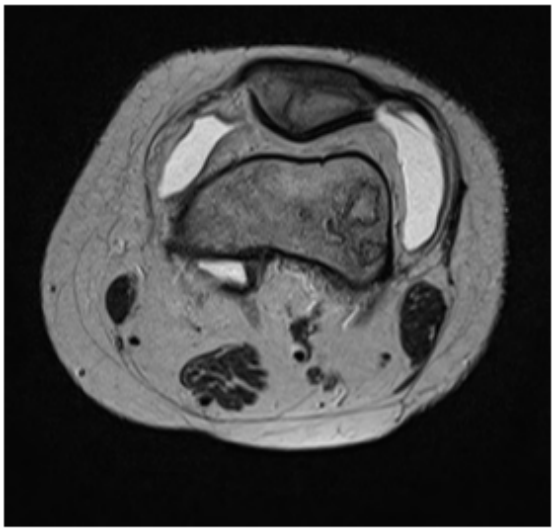

Figure 2: *MRI knee joint axial section showing joint effusion and infarcts of patella and femur*

He was diagnosed to have osteonecrosis of the left knee joint. He was reviewed by an orthopaedician and an endocrinologist who advised non weight bearing physiotherapy, adequate pain relief, weight reduction together with calcium and vitamin D supplementation. He was also given pamidronate. His pain has reduced considerably and he is able to walk with support.

Discussion

Osteonecrosis is a rare complication of ALL treatment in children[2,3]. Presumably there is cell death due to a reduced blood supply to the bone which is subsequently resorbed by osteoclasts causing demineralization, thinning of trabeculae and mechanical failure[3].

Osteonecrosis appears to have a multifactorial origin. Steroid therapy is a key factor contributing to osteonecrosis in children with ALL[4]. Other contributory factors include folate-antagonist methotrexate causing homocysteinaemia leading to venous vascular occlusion and also hypercoagulable state due to corticosteroids and asparaginase interaction which can impair the circulation and subsequently causing osteonecrosis[5]. Adolescents are more prone to develop osteonecrosis than other age groups[6].

Osteonecrosis occurs less often with schedules using intermittent rather than continuous administration of steroids. Steroids are directly toxic to osteocytes, inducing apoptosis, lipid infiltration of marrow and osteocyte hypertrophy, leading to decreased blood-flow[7]. Plain radiographs and bone scintigraphy are of limited value in diagnosing early lesions. MRI is the most sensitive and specific diagnostic tool for early epiphyseal osteonecrosis[8].

Older age and female gender are risk factors for osteonecrosis. Joint replacements are not suitable in young patients in view of their longer life expectancy and active lifestyle increasing the risk of multiple revisions[9]. Surgery should not be performed soon after ALL therapy as osteonecrosis is self-limiting in most ALL children[6]. Clinical rather than radiological screening of osteonecrosis is proposed as there is no guarantee that intervention prevents progress of asymptomatic cases to symptomatic osteonecrosis. Furthermore, many asymptomatic osteonecrosis patients never become symptomatic[10].

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