University Journal of Surgery and Surgical Specialities

ISSN 2455-2860 2018, Vol. 4(1)

Curettage and Bone Grafting for Monostotic fibrous dysplasia - A case report

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Abstract : INTRODUCTION Fibrous dysplasia is a common benign tumour comprising of 5-7 percent of benign bone tumours - most commonly affecting long bones. Bone pain is the commonest mode of presentation of monoostotic fibrous dysplasia. Herewith presenting a case of mono-ostotic fibrous dysplasia treated with curettage and bone graft with a combination of autograft and allograft.MATERIALS AND METHODS A 17 year old male presented with left hip pain of 1 year duration which restricted activities of daily living. Examination revealed normal range of movements. X rays revealed the presence of bony cyst - fibrous dysplasia. Skeletal survey ruled out other bone involvement. Mirels criteria had a score of 10 suggesting the need for prophylactic fixation to prevent pathological fracture. Through lateral approach the cyst was approached. A cortical window was created and cystic contents removed and sent for biopsy. Complete curettage of the lesion done. The cavity was packed with autograft harvested from ipsilateral iliac crest and allograft from 2 femoral heads.RESULTS The biopsy report confirmed the diagnosis of fibrous dysplasia. The patient had uneventful post operative period. Upon 1 year follow up the patient has returned to previous activity levels and improvement in pain according to visual analogue scale. Pathological fracture has also been prevented and the lesion has not progressed in size.CONCLUSION Curettage and bone grafting is a simple and effective procedure for the treatment of mono-ostotic fibrous dysplasia. The amount of graft needed to fill the cavity after curettage may be large and may need allograft in addition to autogenous cancellous bone

Keyword : Monostotic fibrous dysplasia, impending pathological fracture, auto-allograft

Introduction: Fibrous dysplasia is a common benign skeletal lesion comprising of 5-7% of benign bone tumours [1, 2].which may affect one bone (monostotic) or multiple bones (polyostotic). Commonly involved regions are long bones, ribs and craniofacial bones with equal sex predilection. Fibrous dysplasia is caused by activating mutation in the gene that encodes the subunit of stimulatory G protein (Gs) located at 20q13.2-13.3[3]. Current treatment options include bisphosphonates [4] and curettage and bone grafting [5].Curettage and bone grafting using a combination of autograft and allograft to prevent pathological fracture and improve function is presented here.

Case report: A 17 year old boy student presented to the OPD ith pain in the left thigh for 1 year duration. Pain is of dull aching type, increasing at night. Patient is unable to stand or walk. Pain is increased to the level of restricting activities of daily living. Patient unable to squat or sit cross legged. On examination left thigh diffuse tenderness of the upper 1/3rd thigh. Range of movements of the hip was restricted. Flexion was 35^0. Abduction was 20^0. Adduction was 10^0. External rotation was 10^0 Pre operative Harris hip score was 40.9 which was poor. Mirel score was 10 suggesting the need for intervention There was no evidence of any skin involvement. Lab investigations were undertaken and revealed a normal haemogram, normal renal parameters and normal calcium, phosphate and alkaline phosphatase levels. X ray revealed expansile osteolytic lesion over the proximal 1/3rd of the femur with endosteal scalloping and no periosteal reaction {fig -1}. Whole body radiographs were taken to identify any other similar lesions present. No other bony lesions were present. Hence the diagnosis of monostotic fibrous dysplasia was made.

OPERATIVE MANAGEMENT:

Under spinal anaesthesia, patient in left lateral position, through lateral approach to the left proximal femur, skin subcutaneous tissue was opened in layers. A window was made over the lesion located under fluoroscopic guidance. Osteolytic lesion completely curetted out. Autograft harvested from ipsilateral iliac crest.

Allograft harvested from 2 femoral heads, which were double autoclaved, morcellized and mixed with the autograft. {fig -2}.

The cavity completely packed with the combination of autograft and allograft {fig -3,4,5,6}.

Wound closed in layers over drains.

Post operative period uneventful.

Intravenous antibiotics were administered for 3 days.

Analgesics were administered for 7 days and then sos.

Non weight bearing for 6 weeks to allow osseointegration

Gradual weight bearing as tolerated was started subsequently

Biopsy:
Histopathological examination confirmed the diagnosis.
FOLLOW UP: Patient was followed up monthly for a period of 6 months and bimonthly there after. Patient has resumed normal weight bearing. Lesions remained stable in size and there was an improvement in pain scores.
VISUAL ANALOG SCORE FOR PAIN

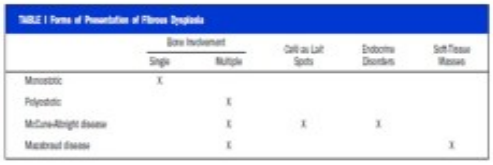
Pre op	9
2 weeks post op	9
1 month post op	8
3 months post op	4
6 months post op	2
12 months post op	1

Range of Movement and Harris Hip scores:

S. No.	Pre op	1 year follow up
Flexion	35	110
Abduction	20	30
Adduction	10	15
External Rotation	10	25
Internal Rotation	10	15

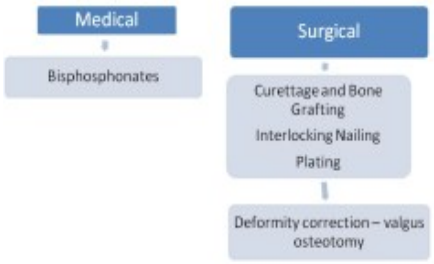
Post operative Harris hip score was 97, graded as excellent Patient able to squat and sit cross legged. Patient returned to normal activity and is able to ride a bus to school.

DISCUSSION:
Lichtenstein[8] is credited with having coined the term fibrous dysplasia in 1938.
Fibrous dysplasia may occur due to a failure in remodelling of primitive bone into mature lamellar bone, which negatively affects the mechanical properties of the affected bone [7]
Fibrous dysplasia is of different types and rarely presents in syndromic constellations.



PRESENTING SYMPTOMS [6]
Bone Pain
• Deformity
* Classical is Shepperd Crook deformity
• Fatigue Fracture
* Classical is Parrot beak deformity
Pathologic Fracture **GROSS PATHOLOGY** [7]**:**
Yellowish white tissue with a distinctive gritty feel, lesion can be easily peeled away from the encircling shell of reactive bone by blunt dissection bleed briskly when cut controlled by rapid and complete curettage back to normal bone

HISTOPATHOLOGY [7]:
Delicate trabeculae of immature bone, with no osteoblastic rimming, enmeshed within a bland fibrous stroma of dysplastic spindleshaped cells without any cellular features of malignancy-“alphabet soup”
pattern
DIFFERENTIAL DIAGNOSIS [7]:
Simple bone cysts
Nonossifying fibromas
osteofibrous dysplasia, adamantinoma,
low-grade intramedullary osteosarcoma and
Paget disease.
CURRENT TREATMENT OPTIONS KEY DIFFERENCES BETWEEN MONOSTOTIC AND POLYOSTOTIC TYPES:



Polyostotic lesions tend be larger Monostotic lesions tend to stop growing at skeletal maturity – hence any intervention should be planned only after skeletal maturity
CONCLUSION: Curettage and bone grafting is a simple and effective procedure for the treatment of mono-ostotic fibrous dysplasia. The amount of graft needed to fill the cavity after curettage may be large and may need allograft in addition to autogenous cancellous bone.
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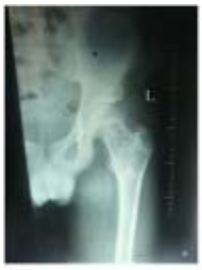
IMAGES



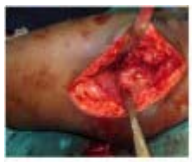
Pre op X ray

Intra operative

Allograft from 2 femoral heads

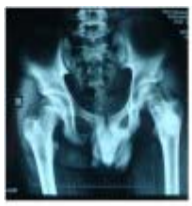
Auto graft mixed with allograft

graft placed in cavity



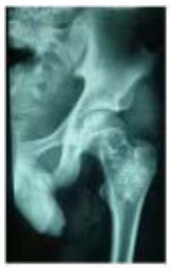
At the end of grafting

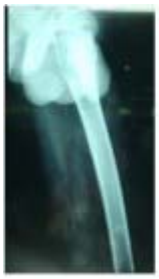
Post operative X ray



6 months follow up

12 months follow up
X rays





Clinical pictures