Partially Decalcified Allogenic Bone Graft - In Fibrous Dysplasis Treatment

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Abstract

Background: Fibrous dysplasia of the bone is an uncommon, congenital skeletal disorder, and it not inherited. Spontaneous resolution of fibrous dysplasia does not occur. There is no cure for fibrous dysplasia, and existing guidelines for the treatment are not universally accepted. Treatment for fibrous dysplasia generally consist from clinical observation in non-symptomatic cases to surgical intervention in symptomatic or severe cases.

Method: This study evaluated the fibrous dysplasia patients in a series of ten cases, treated by curettage and filling of the cavity using partially decalcified allogenic bone grafts, prepared by partial decalcification with 0.6N Hydrochloric acid (HCl) of human bones.

Result: In five cases graft was fully incorporated and healing was considered complete and in another five cases, healing was partial. There was no failure of graft in any case.

Conclusion: Study shows that the results of treatment of fibrous dysplasia by decalcified allogenic bone grafts are favourable.

Keywords: Fibrous dysplasia, Allogenic bone grafts, Partially decalcified allogenic bone, Bone graft, Cystic lesion

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Introduction

Fibrous dysplasia is a benign intra-medullary, fibro-osseous lesion, which can present in one bone (monostotic) or multiple bone (polyostotic), and can be associated with cutaneous hyperpigmentation, endocrinopathies and precocious puberty in females[1]. The true incidence and prevalence of fibrous dysplasia are difficult to estimate, particularly for the more prevalent monostotic form, because many patients are asymptomatic and are often diagnosed incidentally after radiographic evalution for other reasons. These lesions are not rare. It comprises about one percent of primary bone tumour and represent approximately five to seven percent of benign bone tumours[2,3,4]. Clinical presentations usually occur at adolescence or late childhood. Majority of lesions being detected by the age of thirty years. More severe form can arise in infancy[1]. Fibrous dysplasia has no gender predilection, occurs in equal proportion in male and female[5]. Common sites of skeletal involvement are long bones, ribs craniofacial bones and pelvis.

Fibrous dysplasia is postulated to occur as a result of developmental failure in the remodelling of primitive bone to mature lamellar bone and a failure of the bone to realign in response to mechanical stress. Failure of maturation leaves a mass of immature isolated trabeculae enmeshed in dysplastic fibrous tissue that are turning over constantly but ever (or very slowly) completing the remodelling process. In addition, the immature matrix does not mineralize normally. The combination of a lack of stress alignment and insufficient mineralization results in substantial loss of mechanical stress, leading to the development of pain, deformity and pathological fractures[3].

Exact aetiology of fibrous dysplasia is unknown but studies indicate that genetic factors may be responsible, and the disease has been linked with a mutation in the $G_s\alpha$ gene, located at chromosome 20q13.2-13.3[6,7]. Malignant transformation is very infrequent, and is usually precipitated by radition therapy[5,9].

Monostotic lesions are more frequent and lesion enlarge in proportion to skeletal

growth[9]. Monostotic lesions mature after skeletal growth ceases. Polyostotic lesions often continue to enlarge after skeletal maturity, with progressive deformity and an increase in pathological fracture.

There is no cure for fibrous dysplasia, and spontaneous resolution of the lesion does not occur. The age of the patient and location, size and biological behaviour of the lesion, all influence the selection of therapeutic intervention[10,11,12]. Small and asymptomatic lesions do not progress, and do not cause deformity or functional impairment, should simply be monitored[1]. Large size lesions may require curettage of lesion and filling of bone defect using bone graft or other bone substitute with or without fixation to prevent pathological fracture and/or to eradicate symptomatic lesion. Usually the autogenous bone graft is used to fill up these large defect but sometime these surgical procedure requires abundant quantity of bone which the recipient's body cannot supply without significant donor area morbidity and risks. Deep frozen and freeze dried allograft are the other alternate material but bot easily available in developing countries. Allogenic bone graft has been the most obvious alternate to autogenous graft. It has been widely used for structural reconstruction or filling up the bone cavity. Partially decalcified allogenic bone appears to be the most appropriate substitute to autogenous bone graft because it has adequate mechanical strength, is biocompatible, biodegradable, bioactive and ideal natural scaffold for induction and growth of the new bone.

Methods

Graft preparation: Freshly obtained bones were partially decalcified in 0.6 N Hydrochloric acid (HCl) solution. The HCl solution being changed every day. This process was carried out for five to seven days. The bone was washed thoroughly in distilled water and stored in 70% to 90% Ethanol at 4^0C temperature in a domestic refrigerator. The bone was used within one to six months. Before implantation, bone was washed with distilled water and bone was cut into match stick shaped small pieces. The cavity was then thoroughly packed with grafts pieces. Post operatively, limbs were protected in a plaster cast or in a suitable splints.

Result

A quantitative assessment of healing was attempted depending upon the osseous replacement of the original cavity that was grafted. Healing was considered complete if the obliteration of the original cavity was more than 75% of its total volume. If obliteration was 25% to 75%, healing was considered as partial. Less than 25% of obliteration of original cavity was regarded as graft failure.

Ten cases of fibrous dysplasia evaluated retrospectively, surgically treated with curettage and bone grafting using partially decalcified allogenic bone and were followed for an average of 79 months (range 53 to 104 months). The average age of the patients at diagnosis was 19 years (range, 4 to 38 years). Maximum numbers of patients were of second decade of their life. Of these, six were male and four were female. All of the cases had monostotic lesion and no one had pathological fracture. In three cases, the lesion were located in humerus, femur involved in three cases and tibia involved in three patients. Only in one case the lesion was located in the first metatarsal, which is not a common site for fibrous dysplasia (Table 1). All the patients were followed up at regular interval. Earliest radiological sign of incorporation was visible at around three month. Out of ten, in five cases graft was fully incorporated and the healing was considered complete, and in five cases the healing was partial. There was no graft failure in any patients.

Complications, like graft rejection, local recurrence, infection or fracture was not there in any cases. Only one patient had persistent discharging sinus, for about 18 months, from tibia and that was healed later on with antibiotics and supportive treatment. The graft was incorporated partially in subsequent time.

Table 1: Summary data of patients

S. No.	Age at presentation (yrs)	Gender (M/F)	Site Involved	Pathological Fracture	Healing	Follow up Duration (months)
1	20	M	Tibia shaft	Nil	Partial	104
2	24	F	Tibia shaft	Nil	Partial	104
3	38	F	First metatarsal	Nil	Complete	99
4	30	F	Proximal humerus	Nil	Complete	94
5	14	F	Distal tibia	Nil	Complete	91
6	14	M	Proximal humerus	Nil	Partial	70
7	4	M	Proximal femur	Nil	Complete	66
8	16	M	Proximal femur	Nil	Complete	64
9	8	M	Humerus shaft	Nil	Partial	53
10	26	M	Femur shaft	Nil	partial	53

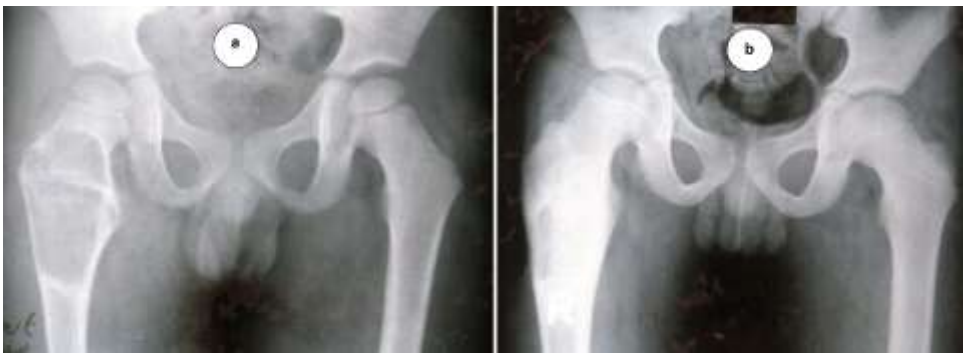

Fig. 1: (a) Preoperative anterioposterior radiograph of a four year old child, showing the fibrous dysplastic lesion in cervico-trochenteric region of femur, (b) radiograph at 4-1/2 years post-surgery, showing complete healing

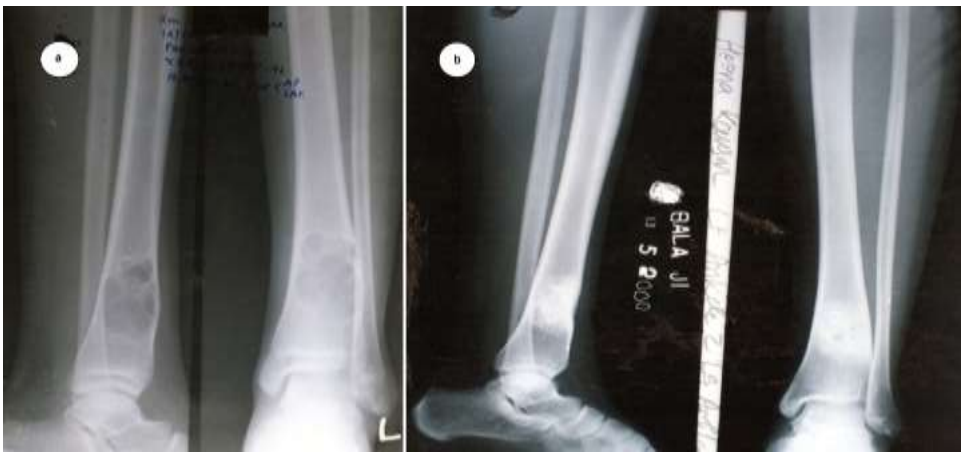

Fig. 2: (a) An anterioposterior and lateral radiograph of a 14 year old patient, showing the fibrous dysplastic lesion of distal tibia, (b) Radiograph made at 4 years postoperatively, showing a complete healing of the lesion with full incorporation of bone graft

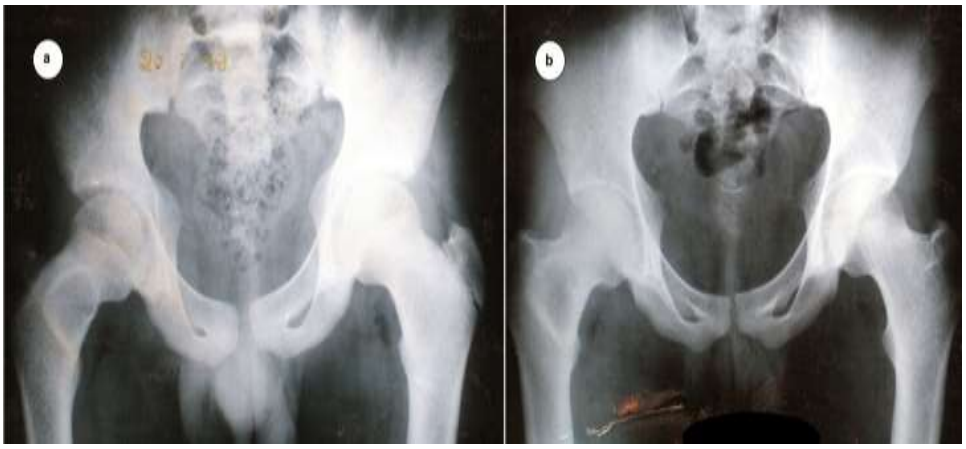

Fig. 3: (a) Anterioposterior radiograph of pelvis, showing the fibrous dysplastic lesion of the neck region of femur in a 16 year old patient, (b) Radiograph at 5 years post-surgery, showing complete healing of the lesion

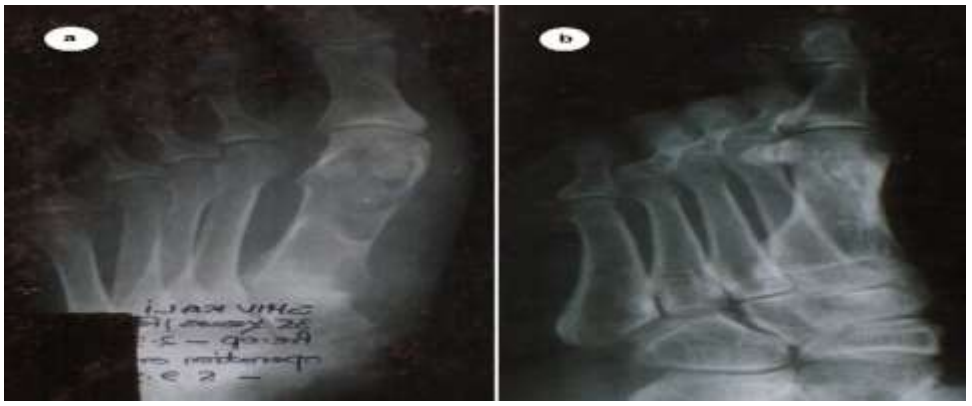

Fig. 4: (a) Radiograph of foot, showing the fibrous dysplastic lesion of first metatarsal, (b) Radiograph at 8 years postoperatively, showing complete healing of the lesion

Discussion

A solitary, small fibrous dysplastic lesion can in many instances be left entirely alone after the diagnosis has been established. Large sized and polyostotic lesions requires surgical interventions and treated by curettage of the lesion and followed by bone grafting. Usually autogenous bone graft is the preferred graft used. Since autogenous bone graft are not available in large amount and their removal caused morbidity at donor site, allograft bone continue to play an important role in orthopaedics reconstruction procedures.

Allogenic bone graft has been the most obvious alternative to autogenous material. It has been widely used for structural reconstruction or filling up of cavitary lesions. There are few reports describing the successful use of decalcified bone allografts in clinical conditions. Kakiuchi et al[13] demonstrated 160 patients, including 73 benign bone tumours, successfully treated by surface decalcified human bone matrix.

Tuli S. M. et al[14] showed the favourable results in case of large osteoperiosteal gaps treated by using allogenic decalcified bone grafts.

Simple curettage is associated with a high risk of recurrence, as is curettage with use of autegenous cancellous bone graft[3]. As internal repair and remodelling begin, the graft of the normal bone is replaced gradually by dysplastic bone and in many instances, the cavity eventually reverts to its preoperative status. Guille et al[15] reported a large series of 22 patients. In their study, complete resorption of all autogenous cancellous bone graft was observed radiologically and none of the lesion were eradicated or decreased in size. Harris et al[16] demonstrated the series of ten cases, all of whom were treated with curettage and autogenous bone grafting, five of them had poor results.

Cortical autogenous bone, used to replace curetted cavities or inserted through dysplastic lesion to strengthen them against fracture, persist much longer then do

canellous graft. In normal repair of cortical bone graft, only the osteonal portion (approximately 50% of the graft) is replaced by dysplastic host bone, whereas the interstitial lamellae (the remaining 50%) are not replaced and persist. Because cortical allogenic have the least and slowest internal replacement by host bone, more of the graft persist for longer. This makes the fibrous dysplasia one of the few for which allogenic bone grafts are biologically preferable to autogenous graft[3].

Vascularised bone grafts also provide a safe and reliable means of ensuring good continuity of bone with little risk of recurrence and failure. There is a limited role for such grafts in the benign lesion of fibrous dysplasia because of the surgical morbidity of harvesting the graft and the surgical experience and time required.

Conclusion

In summary, as this study shows that the results of treatment of fibrous dysplasia by decalcified allogenic bone grafts are favourable, hence decalcified allogenic bone graft may also be used as a promising scaffold for fibrous dysplasia treatment.

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