GIANT CELL TUMOUR OF BONE : AN UNUSUAL METASTATIC PATTERN (A Case Report)

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Introduction

Giant cell tumours (GCT) of bone are rare bone tumours contributing to 5 per cent of all primary bone tumour [1]. They are commoner in women and in the appendicular bones, especially around the knee, occurring usually in the postpubertal individual between the second and fourth decade [2]. In 1940, Jaffe introduced a system of grading GCT into benign, borderline and sarcomatous forms based on stromal architecture and giant cells. However the biologic behaviour of the tumours has not always been in consonance with that suggested by the histologic pattern [3].

The natural history of GCT is one of repeated recurrences and late malignant transformation [4]. This behaviour has made 'optimum management' a much debated issue [5]. Intralesional adjuvant therapy with the use of high speed burr, phenol, liquid nitrogen, electrocautery or polymethyl methacrylate cement (PMMA) have been used to prevent recurrences following a not-so-radical curettage of the tumour. Use of PMMA has been accepted widely as it is tumoricidal in addition to providing structural support and a good contrast to visualize later recurrences [6].

Metastases in GCT are rare but may occur by the haematogenous route to the lungs. Spread to regional nodes, mediastinum, para-aortic nodes is rare [4]. This report is of a young adult male with GCT which showed an unusual clinical course.

Case Report

A 17-year-old male patient presented with history of minor trauma to the left knee followed by pain, swelling and restriction of movements in June 89. Radiograph of left knee had shown an eccentric, well-defined, osteolytic lesion in the lateral condyle of the femur with a thinned-out cortex and a breach in its continuity. Histology of an open biopsy specimen had revealed a GCT with a prominent giant cell component and relatively small stromal component (Fig 1). He underwent definitive surgery elsewhere in the form of curettage, application of phenol to the wall of the cavity, filling-up of the cavity with bone chips (taken from left iliac crest) and a fibular bone strut graft (taken from the same leg). He made an uneventful postoperative recovery.

He remained asymptomatic for 14 months when he reported to us with a progressive soft tissue swelling of the left calf just above the fibular donor site. Radiogram of knee and leg showed a large soft tissue mass with areas of calcification within it on the posterior aspect of the leg extending up to the knee (Fig 2). However, there was no clinical or radiologic evidence of local recurrence at the primary site. A wide local excision along with the excision of the medial head of gastrocnemius was performed. Histology revealed GCT with similar histologic features as noted at the time of the initial diagnosis. He was administered postoperative radiotherapy to the calf and knee (including the primary site) in the dose of 5000 cGy/25 fraction/35 days with no adverse effects. He remained well for another 15 months when he presented with a diffuse swelling and pain over the left knee. There was radiologic evidence of local recurrence and of soft tissue involvement (Fig 3). Histology now revealed malignant GCT with soft tissue involvement and a predominant spindle cell element with only occasional giant cells (Fig 4). He underwent an above-knee amputation.

Within six months after discharge from hospital he reported with large, hard, and painful, left inguinal and left axillary lymphnodes, and a 2 cm diameter hard subcutaneous nodule over the right forearm. The stump was clinically and radiologically normal. Chest radiogram was normal. Cytology from the lymph nodes and forearm nodule revealed metastatic GCT. He

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Fig. 1 : Tumour showing large numbers of osteoclastic giant cells with scanty stromal cells of bland appearance (H&E, 40 X).

Fig. 3 : Radiogram of left knee showing ill-defined osteolytic lesion involving entire lower end of femur with soft tissue extension.

Fig. 2 : Radiogram showing large soft tissue swelling with areas of calcification on the postero-medial aspect of the leg extending upto femoral condyle.

was given palliative radiotherapy to the left groin and axilla to relieve pain. He was subsequently managed symptomatically but died of progressive disease, at home, 4 months later.

Discussion

GCT of bone occurs commonly in the 3rd and 4th decades of life after epiphyseal fusion. Occurrence of GCT prior to epiphyseal fusion is rare and contributes 1.7 per cent of the GCT [7]. Occur-

Fig. 4 : Abundant stromal cells showing marked anaplasia with scanty giant cells (arrow) at the second recurrence (H&E, 100 X).

rence in teenagers is uncommon.

Choosing the correct surgical procedure and addition of intralesional adjuvants is important to prevent recurrences. 'Lets curette now - we can always resect later' is an attitude which leads to unnecessary amputations [3]. Recurrences occur generally after 2-3 years and malignant transformation is noted after 2-3 recurrences [4]. Meticulous surgical technique is important as GCT is known to be seeded readily on raw surfaces [3], as was evident in this case. Such occurrences are however not commonplace. Soft tissue recurrences are known to occur in GCT presenting with pathological fractures [8].

Radiotherapy for GCT is usually reserved for inoperable advanced lesions and those in

inaccessible or difficult anatomic locations (e.g. spine). Radiotherapy carries a low but definite risk of radiation-induced sarcomatous change in 1.5-13 per cent of cases [9]. Dahlin from Mayo's clinic reported a lag of 7-9 years for radiation-induced malignant transformation [10]. It is unlikely that the 'malignant transformation' which occurred in the present case, within 15 months of exposure, was induced by radiation. We presume it represents *de novo* malignant GCT. Secondary malignant GCT exhibits a variety of histological appearances. The most common are fibrosarcoma, malignant fibrous histiocytoma and osteosarcoma. Prognosis of such GCT is uniformly bad [9].

Though attempts have been made to correlate histologic, radiologic and biologic behaviour they have not been uniformly successful in predicting accurately the course of the disease. This lead Cabales [11] to conclude that no GCT was benign and they should be regarded as low or high grade malignant tumours. This fact is amply illustrated in this case which rapidly progressed from a grade I GCT to malignancy in about 2 years time. Though extension to soft tissues and implantation are known, the primary mode of metastatic spread is haematogenous mostly to the lungs. Regional and distant metastases are uncommon [5,7].

This case has represented several unusual features. Beginning at an early age (17 years) it rapidly progressed over a period of 40 months to malignancy and death. Soft tissue metastasis and lymphnode metastasis in the absence of pulmonary

metastasis was unusual.

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