FIBULAR STRUT GRAFTING FOR FIBROUS DYSPLASIA OF THE FEMORAL NECK

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When fibrous dysplasia affects the femoral neck, normal bone is replaced by fibro-osseous dysplastic bone that is both mechanically weakened and biomechanically abnormal. Surgical management is recommended for persistent pain, progressive deformity, or impending fracture. Surgical options include curettage and cancellous bone grafting, osteotomy and nail-plate fixation, intramedullary rodding, and cortical bone grafting.

We present the case of a patient with a painful, dysplastic lesion of the femoral neck who underwent cortical bone grafting using dual fibular strut grafts. To ensure long-term graft incorporation, the fibular cortical grafts bridged the lesion in the femoral neck and were securely anchored to the normal bone of the lateral femoral cortex and head of the femur. No supplemental internal fixation was required. The biological basis for success of the fibular strut grafting procedure is that creeping substitution of the cortical graft necrotic bone does not replace the interstitial lamellae, which persist to lend structural support. Fibular strut grafting is an excellent procedure for fibrous dysplasia of the femoral neck. (*J Natl Med Assoc.* 1992;84:893-897.)

Key words • fibrous dysplasia • femur • bone grafting

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The first description of fibrous dysplasia is attributed to Weil in 1922.[1] In 1937, the disease became a well-recognized entity when Albright et al[2] reported a syndrome characterized by precocious puberty, areas of skin pigmentation, endocrine abnormalities, and fibro-osseous lesions of bone. That same year, McCune and Bruch[3] reported a child with similar clinical findings. In 1938, Lichtenstein[4] coined the term fibrous dysplasia, and with Jaffe[5] in 1942, separated the disease into its principal clinical forms—polyostotic and monostotic. The histological features of the fibro-osseous lesions have been well-described.[6-12] Normal bone is replaced by irregular trabeculae of woven bone intermixed with mature collagenous tissue. The etiology of fibrous dysplasia is unknown, but it appears to be an abnormality of bone-forming mesenchyme.[12,13]

Fibrous dysplasia most commonly affects the proximal femur. The polyostotic form often causes a characteristic progressive varus deformity due to the forces of the gluteal musculature and weightbearing on bone with compromised structural integrity. Clinical manifestations of the femoral varus deformity can include a limp, disabling pain, shortening of the extremity, or a fatigue fracture. Monostotic fibrous dysplasia of the femoral neck rarely results in a severe varus deformity, but it can cause hip pain and/or a fatigue fracture in adolescence or early adulthood.[14] The case reported here presents the successful management of a patient with symptomatic fibrous dysplasia of the femoral neck using fibular strut grafting.

CASE REPORT

A 36-year-old female with a 20-year history of Albright's syndrome presented with a 6-month history of progressive left hip pain. The patient underwent curettage with bone grafting for fibrous dysplasia of the femoral neck 17 years prior to admission. Plain roentgenograms

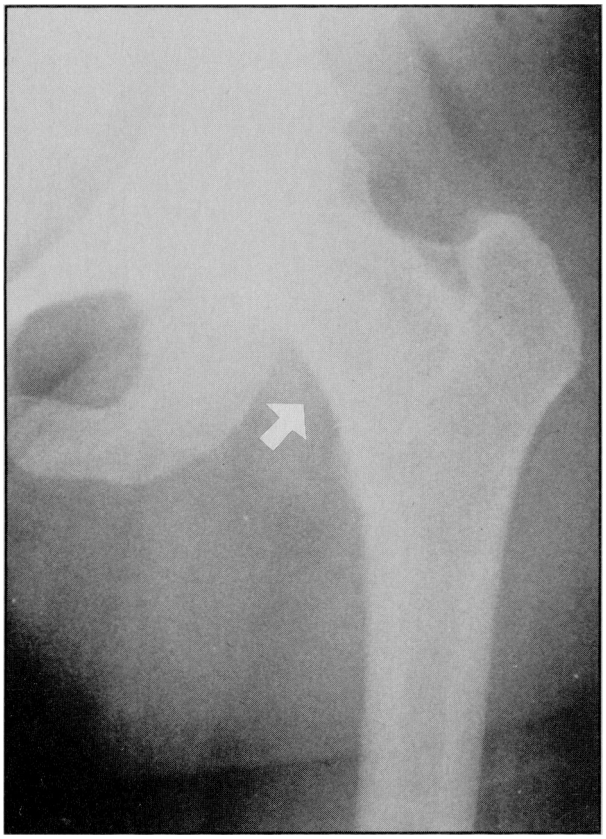

Figure 1. Anteroposterior roentgenogram of the left hip showing the "ground glass" appearance of the clearly defined, fibro-osseous femoral neck lesion.

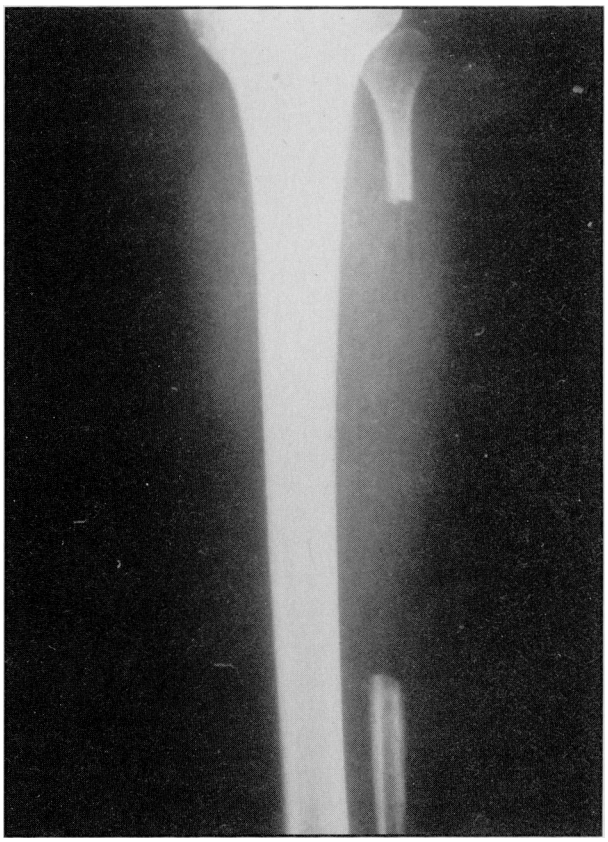

Figure 2. A 14-cm segment of autogenous cortical bone was harvested from the patient's fibula. The graft was then divided into 8-cm and 6-cm segments for insertion of dual cortical strut grafts across the dysplastic lesion in the femoral neck.

and computed tomography of the left hip revealed a lesion consistent with recurrent femoral neck fibrous dysplasia (Figure 1). A technetium scan showed focal uptake in the left proximal femur. The patient underwent autologous fibular strut grafting of the femoral neck using the procedure described by Enneking and Gearen[14] (Figure 2). A biopsy of the femoral neck revealed the irregular osseous trabeculae and collagenous tissue characteristic of fibrous dysplasia (Figure 3). The patient had an uncomplicated postoperative hospital course and was discharged home after completing physical therapy. The patient had significant relief of her left hip pain postoperatively. At her 1-year follow-up examination, the roentgenograms showed excellent incorporation of the femoral neck cortical autografts (Figure 4).

SURGICAL TECHNIQUE

After induction of general anesthesia, the patient was placed on the fracture table. A lateral incision was made over the left fibula to obtain the cortical autograft. A 14-cm segment of the fibula was harvested using an oscillating saw. The fibular graft diameter was measured to ensure accurate size and depth of the reamed femoral neck tunnels.

The left femoral greater trochanter and diaphysis were exposed using the straight lateral approach to the hip. Using the image intensifier, a guide wire was inserted into the subchondral bone of the femoral head. The image intensifier confirmed proper placement of the wire in the inferior femoral neck. Cannulated and flexible reamers were placed over the guide wire to create a tunnel for fibular strut graft insertion. The guide wire and reamers were removed after the tunnel was reamed. An 8-cm section of the fibular graft was inserted into the tunnel. A second guide wire was placed in the fibular neck superior and parallel to the first guide wire. After reaming was performed using the cannulated and flexible reamers, a second 6-cm fibular strut

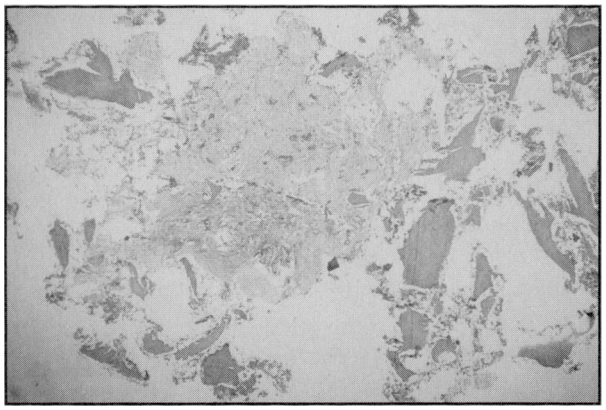

Figure 3. A biopsy of the femoral neck lesion demonstrates the dense collagenous tissue and irregular trabeculae characteristic of fibrous dysplasia.

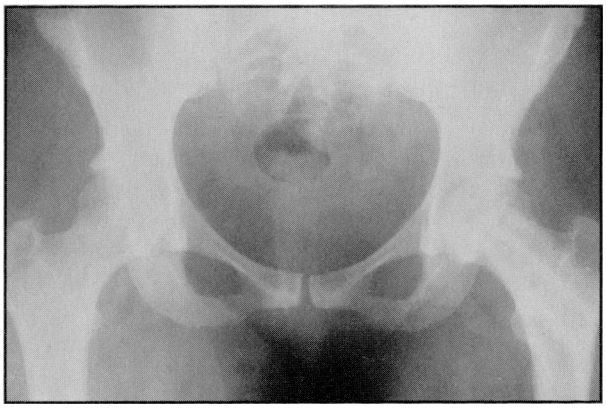

Figure 4. Follow-up roentgenogram of the left hip shows excellent incorporation of the fibular cortical autografts.

graft was placed superior to the first graft. Both fibular strut grafts were firmly impacted into the femoral head. The region of fibrous dysplasia was not resected. After satisfactory position of the fibular grafts was confirmed using the image intensifier, the wound was closed.

DISCUSSION

Although fibrous dysplasia is considered a benign condition, lesions involving the proximal femur can cause substantial morbidity. The bone is biomechanically abnormal and mechanically weakened due to the fibro-osseous replacement of normal bone. Nonoperative treatment is usually recommended for dysplastic lesions involving the upper extremity. However, progressive deformity and fatigue fractures invariably ensue following nonoperative management of the lower extremity. Surgical treatment is recommended for fibrous dysplasia of the femoral neck under limited circumstances including progressive or severe extremity deformity, impending or recurrent fractures, and persistent pain.[13,15] Surgical treatment options include curettage and cancellous bone grafting, cortical bone grafting, intramedullary rodding, osteotomy and nail-plate fixation, and resection of the proximal femur.

Several studies have documented unsatisfactory results following curettage and cancellous bone grafting for lower extremity fibrous dysplasia.[6,14-16] A probable cause of failure is graft resorption with replacement by fibrous dysplastic bone. Unlike an amputation, an intralesional curettage does not necessarily remove all dysplastic bone.[13,16] Stephenson et al[13] reported unsatisfactory results in 25 of 31 patients (81%) following curettage and bone grafting.

Open reduction and internal fixation has been recommended for fibrous dysplasia of the proximal femur. In the study by Stephenson et al,[13] 19 of 22 (86%) patients had satisfactory results following fixation with compression plates, sliding hip screws, and intramedullary rods. Breck[1] described a patient successfully treated using total femoral plating and a Jewett nail. However, a nail-plate apparatus does not confer biomechanical stability to extensive dysplastic lesions involving the proximal femur. A region of stress concentration exists distal to the plate, which can cause a fatigue fracture.[15] The strength of nail-plate fixation is inadequate to resist the pull of the gluteal musculature and the stress of weightbearing, both of which tend to deform the biomechanically weakened bone.

Intramedullary devices have frequently been used as a mode of fixation for dysplastic lesions of the femur. In 1962, Harris et al[6] reported the use of intramedullary devices in three patients with proximal femoral lesions. A coxa vara deformity developed in the one patient with femoral neck fibrous dysplasia following Kuntscher rod fixation. The femoral head is not often a site of fibrous dysplasia.[6] Therefore, theoretically, a fixation device that provides firm purchase in both the normal femoral head and uninvolved femoral diaphysis is an attractive surgical option.

The Zickel nail consists of three components: a triflanged nail, a set screw, and a specially shaped intramedullary rod.[17,18] As Freeman et al[15] have noted, the triflanged nail gains purchase in the normal head and traverses the dysplastic lesions in the femoral neck. The Zickel nail resists the muscular forces and weightbearing stresses that tend to cause varus angulation of the neck. In contrast to nail-plate fixation, the

intramedullary rod obtains rigid fixation in the distal femur without significant stress concentration.[15] In 1977, Connolly[19] first reported the successful use of the Zickel nail in the management of a patient with polyostotic fibrous dysplasia and bilateral varus deformities of the femoral neck and subtrochanteric region. Freeman et al[15] recently reviewed the surgical management of four patients (six femora) with polyostotic fibrous dysplasia involving the proximal femur. All four patients were able to return to normal activities following multiple osteotomies and Zickel nail fixation.[15] These results indicate that Zickel nail fixation is the treatment of choice for a symptomatic severe varus deformity in polyostotic fibrous dysplasia.

In 1986, Enneking and Gearen[14] reported satisfactory results in 15 patients using cortical bone grafting for fibrous dysplasia of the proximal femur. Five of the patients had polyostotic dysplastic lesions and 10 had monostotic dysplatic lesions. None of the patients had a severe varus deformity of the proximal femur requiring an osteotomy and intramedullary fixation. The number and source of the cortical strut grafts differed among the patients. Dual autogenous tibial grafts, dual autogenous fibular grafts, single autogenous fibular grafts, and dual allogenic fibular grafts were all used for the cortical bone-grafting procedures. The acquisition of autogenous bone has several drawbacks including weakened site of donor bone, higher postoperative morbidity, a second surgical incision to harvest donor bone, and limited availability and supply of donor bone.[20] Springfield[21] prefers the use of allograft cortical bone because it is readily obtainable and a theoretical decreased rate of resorption is seen when compared with autogenous cortical bone. In Enneking and Gearen's[14] investigation, 14 of the 15 patients underwent autogenous cortical bone grafting. No complications related to bone graft harvesting were encountered. At an average follow-up of 6 years, all 15 patients had relief of pain and no progression of the femoral neck deformity.

In the case reported here, the patient underwent insertion of dual autogenous fibular strut grafts. At a preliminary follow-up of 1 year, the patient had substantial relief of her hip pain. For this patient, the graft is providing excellent structural support without the need for internal fixation. The dual fibular grafts bridge the fibrous dysplastic lesion in the femoral neck. The fibular strut grafts are securely anchored to the normal bone of the lateral femoral cortex and head of the femur. The region of fibrous dysplasia in the patient's femoral neck was neither osteotomized nor resected. Malawer and Shmookler[22] have observed that resection of the dysplastic lesion is seldom required in cortical bone grafting. In Enneking and Gearen's[14] series, 12 of the 15 patients had a fatigue fracture when first examined. Although our patient's left hip roentgenogram did not reveal a definite fracture line, Enneking and Gearen have noted that the fracture line is often difficult to detect roentgenographically. A fatigue fracture, undetectable by the roentgenographic studies used, could have been the source for our patient's initial complaint of hip pain.

The patient's curettage with bone-graft procedure, performed 17 years prior to fibular strut grafting, was complicated by local recurrence of disease. A biopsy specimen from the femoral neck revealed recurrent fibrous dysplasia. The intralesional curettage probably failed to remove all of the fibrous dysplastic bone. The femoral neck bone graft was resorbed and replaced by recurrent fibrous dysplasia. The regenerated fibro-osseous lesions became symptomatic, which caused the patient's complaint of hip pain.

In a patient with a severe varus deformity of the proximal femur, cortical bone grafting is probably not feasible when the dysplastic bone totally encases the graft. The bone is usually normal in the femoral head and subtrochanteric cortex in the majority of patients with proximal femoral fibrous dysplasia.[14] The fibro-osseous lesions usually involve only the femoral neck and intertrochanteric areas. For long-term graft incorporation, the cortical bone graft should be firmly impacted into the normal bone of the femoral head and securely anchored to the lateral cortex of the femur.[14] Our case report indicates that cortical bone grafting is an excellent procedure for fibrous dysplasia of the femoral neck when the proximal femur is relatively undeformed. At her 1-year follow-up examination, the patient denied hip pain and was able to resume her normal functional status. Although the patient has satisfactory results at current examination, long-term follow-up is necessary to determine the ultimate fate of the fibular bone-graft procedure.

BIOLOGY OF CORTICAL BONE GRAFTS

The biological process of cortical bone-graft incorporation has been described previously.[14,20,23-25] In cortical bone, creeping substitution of the graft necrotic bone does not replace the interstitial lamellae, which persist to lend structural support. The bone in the haversian system is resorbed by the osteoclasts prior to the osteoblastic apposition of new bone. Burchardt et al[24,25] used a dog fibular model and reported that

following transplantation, the graft loses 60% of its original strength at 6 weeks, with a return to original strength at 52 weeks. It has been postulated that a human cortical bone graft regains its original strength at 2 years following transplantation.[21]

CONCLUSIONS

For carefully selected patients, cortical bone grafting is an excellent procedure for fibrous dysplasia of the femoral neck. Curettage with bone grafting is not effective because it has a high rate of local recurrence. Cortical bone grafting is indicated for a symptomatic dysplastic lesion without a severe varus deformity of the proximal femur. Cortical bone grafts provide strong structural support to bone biomechanically weakened by fibrous dysplasia.

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