Case Report

Femoral head disintegration in a paraplegic patient: loose bodies in neuropathic joints

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A case report of potential loose body formation in a hip joint of a paraplegic patient is presented. The case is used to discuss why loose bodies do not persist in neuropathic joints, although they might be expected to be common when this type of joint pathology exists. *Spinal Cord* (2000) 38, 112–116

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Introduction

A patient with paraplegia due to a thoracic spinal injury presented with spontaneous right femoral head disintegration and a decubitus ulcer. An arthrotomy of the hip was done while treating the ulcer, and multiple potential loose bodies were encountered.

There are only two possible sources for loose bodies in diarthrodial joints, the cartilage covered ends of bones and the soft tissues lining the joint capsule. Loose bodies in joints may arise from fragments (termed nidi) of pre-existing joint structures, chondral, osseous or both, through trauma or joint surface disintegration through disease, or may form from intrasynovial proliferation of new chondral or osteochondral nodules, synovial osteochondromatosis, which bud into the joint space from attachments to the synovium, and by frictional forces, are sheared from the synovium as loose bodies (also termed nidi of a different type). In neuropathic or Charcot joints considerable fragmentation of the pre-existing joint tissue, both the bone and overlying articular cartilage, may take place, often with multiple loose pieces of bone and cartilage tissue becoming loose in the joint space. Yet it is remarkable that, despite so many potential loose bodies forming in cases of neuropathic joints, true persistent loose bodies showing increased radiographic calcification, rounding, and growth in size over time (termed layering) appear to be extremely rare in these joints. This report presents a typical case of a neuropathic joint with the formation of multiple potential loose bodies due to bone fragmentation and discusses probable reasons why loose fragments do not persist as loose bodies in joints with this disease process.

Case report

The patient was a 36-year-old male who was admitted to the hospital with a fever, bleeding and drainage from a right ischial decubitus ulcer. The patient had been rendered paraplegic at the T11–T12 level 9 years previously by a gunshot wound of the chest and spine. Radiographs and repeat physical examinations demonstrated full passive motion of both lower extremities without heterotopic bone around the hips. There were some transient pressure sores of several toes without osteomyelitis.

Two and a half years before his admission, he had been involved in a motor vehicle accident during which his vehicle fell 20 feet from an elevated expressway. A spiral fracture of the right distal femur and an intertrochanteric fracture of the left proximal femur were treated with open reduction and internal fixation by means of plates and screws. The two fractures healed without deformities. There was no documentation of a right hip injury other than multiple skin lacerations on both legs. Radiographs at that time showed a normal right hip joint.

On a radiograph of the abdomen taken 9 months prior to the present admission, absence of the right femoral head with superior and lateral displacement of the femur with respect to the acetabulum was noted. On the admitting radiographs 9 months later the right femoral head was described in a radiology report as having been surgically excised; however, he had had no surgery around the right hip. Thus the femoral head had spontaneously undergone disintegration.

When admitted, he gave a history of a fire at his house a year before with the destruction of all of his furniture. The patient thereafter sat on the bare floor for extended periods of time. Subsequently he developed bilateral ischial decubitus ulcers. While the ulcer over the left ischium healed, the right ischial

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ulcer continued to remain open and became purulent just before his admission.

Radiographs demonstrated the lack of a right femoral head, displacement of the femoral shaft, no osteolytic changes, and a healed left hip fracture (Figure 1). However, some irregular calcification could be appreciated within the joint adjacent to the right acetabulum. A Tc⁹⁹ diphosphonate bone scan showed no increased bone activity around the right hip. The preoperative diagnosis was 'acute digestion of the misshapened femoral head most probably due to infection or neuropathic joint'. He became afebrile on broad spectrum intravenous antibiotics. After consultation between the orthopaedic surgery and plastic surgery services, it was decided to debride the hip joint, later performing a flap to close the ischial ulcer. The ulcer was a clean 4 × 5 cm decubitus which communicated with the right ischial tuberosity but not with the right hip joint.

At surgery a lateral incision centered over the greater trochanter of the right femur allowed an exposure of the external rotator muscles which were divided to expose the intact joint capsule. When the joint was surgically opened, there was no evidence of pus or serosanguinous joint fluid. Instead, multiple rounded osteochondral fragments of various sizes and estimated as approximately 20 in number were found within the joint space. The majority of these were free of any synovial attachment, but a few had pedicles. The synovium itself showed no evidence of proliferating cartilaginous nodules such as is sometimes seen in secondary synovial osteochondromatosis. The surgical wound was closed primarily and never drained purulence. Cultures from the joint were negative. Three months later after local wound care of the decubitus ulcer, he underwent an excision of a pseudobursa, a partial excision of the ischium, and a gluteus maximus flap rotation which was successful.

Figure 1 Radiograph of the pelvis taken shortly before right hip arthrotomy. There is a healed fracture of the left proximal femur with a sliding screw *in situ*. No right femoral head is present, some fragments of bone are within the joint, and the femur is displaced superiorly

There was no osteomyelitis of the bone at that time. He was free of any infection at follow-up over the ensuing 3 years.

The surgical specimen from the hip joint is shown in Figure 2. It consisted of osteochondral fragments of various sizes up to a centimeter in largest dimension. Yellow bone was apparent on the surfaces of a number of these rounded loose bodies. One pedunculated group can be seen near the center of the photograph. A radiograph of the same tissue (Figure 3) revealed fragmented trabecular bone in their centers. Multiple fracture lines were present in some

Figure 2 Gross photograph of all the fragments present within the right hip joint. Many rounded loose bodies are seen with odd shapes and varied sizes. Most seem to have a white cartilaginous surface. One small nest of osteochondral nodules may be reactive

Figure 3 Radiograph of same tissue pieces (Polaroid Type 55 film-Faxitron) showing all of the loose bodies consist of cancellous bone fragments that demonstrate many individual fracture lines. Six or seven of the smaller specimens are quite rounded and appear to be early loose bodies

Figure 4 Photomicrograph of one of the rounded loose bodies revealing a nidus of broken bone and attached considerably resorbed articular cartilage (center). However, on the surface are typical changes of cartilage formation of the type seen in loose bodies. Some of this new cartilage has calcified too. This is certainly a loose body histologically. (H&E $\times 70$)

Figure 5 Histologic preparation of one of the larger bodies showing broken bone trabeculae, an avascular center, and some reactive surface changes of fibrosis not unlike early loose bodies. (H&E $\times 70$)

pieces. Only the smaller specimens showed calcification that might be due to cartilage degeneration and layering (findings in older loose bodies). The radiograph thus revealed that the nidi of the majority of the loose bodies was primarily fragmented bone.

However, histologic sections demonstrated considerable new cartilage formation around these bone fragments (Figure 4). This explained the gross appearance of white cartilage. Cartilage calcification was present in the larger specimens but was not

extensive. The yellow bony portions of the loose bodies observed grossly were shown microscopically to consist of fragmented necrotic bone without much loose body layering (Figure 5). All of the bone tissue was necrotic, a finding consistent with free loose bodies. The interpretation was that there were loose bodies, multiple in number, a few of which were growing in size, which had formed as a result of unrecognized asymptomatic fragmentation of the femoral head in a neuropathic joint. Examination of soft tissues showed no evidence of inflammatory cells which might be expected in a pyarthrosis.

Discussion

When Charcot first made an association between certain neurologic disorders which cause an absence of protective sensation and certain fractures in his patients that often progressed to significant deformities, he postulated a trophic influence of the spinal cord itself in the production of the joint changes.¹ This theory was immediately opposed by a number of prominent German physicians including Virchow and Volkmann. 'Volkmann took the position that the spectacular joint changes were mechanical in origin – mechanical in the sense that they originated in a multiplicity of subclinical traumata which went unperceived because of the insensitivity of the affected joints.'¹ This became known as the German theory of neuropathic joints. Charcot and his French theory followers described an injury to the spinal cord as an etiologic factor in the production of trophic [neurotrophic] joints.

While there is still some support for a trophic disturbance before fracturing occurs,^{2,3} the majority of physicians today subscribe to a mechanical causation of the changes that occur in the susceptible patients.⁴ This has been supported by the elegant experimental studies in cats by Eloesser,⁵ who cut the posterior roots in the lower spine of the animals thus ablating the sensory function in these limbs. Some of the cats developed neuropathic or Charcot joints. Certainly it has been observed that after periods of immobilization of the leg because of some unrelated injury, the patient who is at high risk for the development of a neuropathic joint may do it at that time. Probably the disuse osteoporosis which occurs then renders the bones less strong so the neuropathic fracture is more prone to happen at that time just as stress fractures may occur in normal individuals who develop disuse osteoporosis.

Neuropathic joints can develop secondary to a number of different disease conditions that affect peripheral sensation.^{4,6} These can be classified as congenital or acquired.⁷ In the congenital group are spina bifida and congenital indifference to pain. Those with acquired disease are more numerous. They include neurosyphilis, syringomyelia, diabetes mellitus, leprosy, and traumatic injuries to the brain or the spinal cord. Those patients with diabetes are more

numerous than those with other ailments, so the majority of observed cases of neuropathic joints today are foot and ankle cases in diabetics.^{8,9}

The mechanism that leads to the classic destroyed joint that is described as a neuropathic joint is fracturing and refracturing over a fairly short period of time, so that often the bone ends are pounded into small fragments or a mixture of large and small fragments. The injury does not have to be in a weight-bearing joint. Wrists, elbows, and shoulders can be affected in some diseases which affect the protective sensation of these joints. And neuropathic spinal fractures have been documented in many patients at a spinal level where sensation is absent. In children with spina bifida or congenital indifference to pain, growth plate fractures can be observed, and these can cause secondary deformities due to disturbed bone growth.¹⁰ Not only the bone ends of a joint may fragment, but the soft tissue restraints of the joint, the capsule and ligaments, are torn and stretched so significant deformity such as a Lisfranc dislocation may occur in a foot which only shows limited bone fracturing. There is synovial proliferation, and joint effusions with and without hemorrhage can occur sporadically.

The authors have watched mostly in diabetics a number of cases from a point where the first fractures occurred through a progression of further fracturing which was then arrested by early treatment. Other cases have been studied from series of radiographs of single patients.^{11,12} A fairly early specimen from a knee which was arthrodesed due to syringomyelia was processed as large mount slides; this showed the typical changes of cancellous bone fractures.^{13,14} The most detailed pathology study was from Erdheim's laboratory by Moritz.⁷

The original fractures that occur are not unusual in themselves. But in a normal patient who develops a major joint fracture, pain and swelling prevent further weight-bearing and movement of the joint. These protective features caused by early symptomatology prevent further injury to the bones in normal typical fracture cases. But the patient with a neuropathic joint does not have these symptoms, so the joint is stressed again and again often causing further fractures and ligamentous soft tissue injury. A femoral or humeral head in a joint which is moved despite the occurrence of significant fracturing will be pounded by muscular or weight-bearing forces into even smaller fragments. Such fragments do not have to be of the same size. Some may be larger than others. A piece of bone with the insertion of a tendon may avulse and be spared from some of the recurrent trauma which affects other parts of the joint complex.

Often the patient does not realize that fracturing has even occurred. Thus some injuries are recognized by physicians long after they originally took place. But also some fracturing can take place over short periods of time.¹⁵ Because of the phenomenon of refracturing and progression of fracturing to fragmentation, the

amount of joint damage is far out of proportion to the patient's symptoms. While the diagnosis can sometimes be suspected from radiographs alone, the confirmation is a physical examination that demonstrates a lack of protective sensation at the particular involved joint.

There have been many descriptions of the features of neuropathic joints.^{6,16,17} The original tissues tear and fragment. A healing response begins as it does in the normal fracture or sprain. But one injury follows another. As bone callus forms to repair the earlier fractures, that callus itself is subjected to abnormal stresses, and it can fragment too. The differentiation of callus from heterotopic bone may be impossible. Thus there is original tissue and repair tissue all being battered again and again by abnormal joint stresses.

All of this brings us to the point of the present paper, loose osteochondral bodies in neuropathic joints. Fragmentation of joint surfaces and osteochondral fractures are the source of most loose bodies.^{18,19} One would imagine that loose bodies should be a commonplace occurrence in neuropathic joints. But we have found from a review of the literature that they are excessively rare. Some authors have described their presence in this disease process, but they do not demonstrate this in their publications.^{17,20,21} Thus there is the possibility that the fragments in those studies were not loose bodies. Sometimes the joints do contain much fragmented bone, but true loose bodies have characteristic radiologic features.¹⁸ We were unable to identify typical radiographic older loose bodies with their rounded and densely calcified appearance in any published paper that describes cases of neuropathic joints. An extensive search was made in atlases and texts as well as journal articles. One ununited and probably avascular portion of a tibia plateau was noted with rounding that suggested a moveable fragment, but it had not displaced from its bed, so probably was yet attached.²² Much of the abnormal bone seen on radiographs in these patients is new reactive bone in the damaged synovial and capsular tissues, but none appears to be loose. Pieces of old or reactive bone can migrate when there is capsular tearing, but this is in tissue planes and would be expected to resorb over time.²³

The resorption of bone fragments in early neuropathic joints has been documented many times.⁷ Sometimes resorption of all broken bone can occur in as short a time as 1–3 months.¹⁴ Histologic studies show many tiny fragments of bone and cartilage within the synovium of recently formed neuropathic joints.^{11,24}

In the present case resorption of bone due to sepsis was a consideration in a paraplegic patient with a decubitus ulcer.²⁵ However, a bone scan was negative, no evidence of a pyarthrosis or a sinus tract was found at surgery, cultures were negative, and the specimen did not show any sepsis. Also when he had the fracture of the femur after the fall, the right femoral head was intact on the radiographs

taken at the time of that injury and on follow-up studies.

That rounded fragments that could produce true loose bodies can occur in a neuropathic joint is proved by the findings in the present case. So the question is why these early fragments do not persist as loose bodies? Our answer to this is the abnormal synovial lining of such joints. We think the reactive joint surface catches the potential loose body nidi and vascularizes them.^{16,26} They are then either resorbed or rarely may persist as an intrasynovial ossicle for a time. Certainly the cartilage from the original joint surface is also resorbed in this very active healing process. This is the same process that can resorb a true loose body which can reattach to a synovial membrane even after many decades of being loose.²⁶ In neuropathic joints we speculate that the newly formed loose body nidi do not stay loose, but become reattached early because the joint lining is hyperactive and seems to have the ability to trap the potential loose bodies. Unfortunately, specimens in this disease are of great rarity. What we are saying is that potential loose bodies disappear, so true persistent loose bodies do not exist in neuropathic joints, except possibly in rare circumstances.

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