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Case Report

Bilateral Distal Femoral Flexion Deformity After Total Knee Arthroplasty in a Patient with Rheumatoid Arthritis
在一位患有類風濕性關節炎的病人，進行了兩邊全膝關節置換術後逐漸形成股骨遠端屈曲畸形

Chan Chun-Ming Jimmy*, Lui Tun-Hing

Department of Orthopaedics and Traumatology, North District Hospital, Hong Kong

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ABSTRACT

Rheumatoid arthritis is an autoimmune systemic disease with predominant peripheral polyarthritis, often leading to severe joint destruction. This is a case report of an 81-year-old woman with long-standing severe rheumatoid arthritis requiring multiple orthopaedic operations for joint destruction since 2000. These operated joints improved her functional mobility until recently, when she found that her knees were fixed at around 70° of flexion with limited motion. There was chronic progressive flexion deformity of bilateral distal femurs, which was an extremely rare complication of total knee arthroplasty.

中 文 摘 要

類風濕性關節炎是一種主要侵害週邊關節的自體免疫性疾病，經常造成嚴重的關節破壞。這一病例報告是關於一位患有長期嚴重類風濕性關節炎的八十一歲女士，她由二零零零年起，因為關節破壞而接受了多次的骨科手術，這些手術後的關節改善了她的活動能力。但最近，我們發現她的膝關節有七十度屈曲畸形，兩邊膝關節不能活動，這種慢性逐漸的股骨遠端屈曲畸形是一個頗為罕見的全膝關節置換術後的併發症

Introduction

Patients with severe rheumatoid arthritis (RA) usually develop multiple joint destruction. Arthroplasty is one of the most useful operations in controlling pain and improving their functional status. However, one of the known complications of joint arthroplasty is insufficiency fracture, which is due to normal physiological stress.[1]

Patients with RA have a higher risk of osteoporosis and fractures than the general population for several reasons, including underlying inflammation, impaired walking ability, and long-term glucocorticoid use. Moreover, insufficiency fractures have been reported as the most common type of fracture in patients with RA.[2]

Case Report

A 81-year-old Chinese woman with a negative family history of autoimmune disease was diagnosed with rheumatoid-factor-positive RA since the age of 40 years. She had early-onset synovitis and full-blown erosive arthropathy affecting all the joints of her upper and lower limbs.

She was initially followed up by a rheumatologist and was previously put on steroids and sulphasalazine. Steroids were tapered off from 2006 and leflunomide replaced sulphasalazine in 2003 because of persistent active disease. In 2004, she was diagnosed with active pulmonary tuberculosis, and leflunomide was therefore stopped. Sulphasalazine was prescribed again from 2006. Methotrexate was never used because the patient was a hepatitis B carrier (hepatitis B surface antigen positive). In 2000, she underwent multiple orthopaedic operations, including fusion of the bilateral interphalangeal joints of both thumbs and first metacarpal–phalangeal joint, together with excisional arthroplasty of the second to fifth toes of both feet in 2000. Bilateral total knee replacements (Figure 1) and right total hip replacement were performed in 2001, and bilateral total elbow replacements were done in 2002.

The patient had a biologically active disease with elevated erythrocyte sedimentation rate to around 100 mm/hour. Clinically, she had on and off joint pain and swelling affecting mainly the shoulders, wrists, and ankles. These joints showed radiographic

* Corresponding author. *E-mail:* ccmjimmy@googlemail.com.

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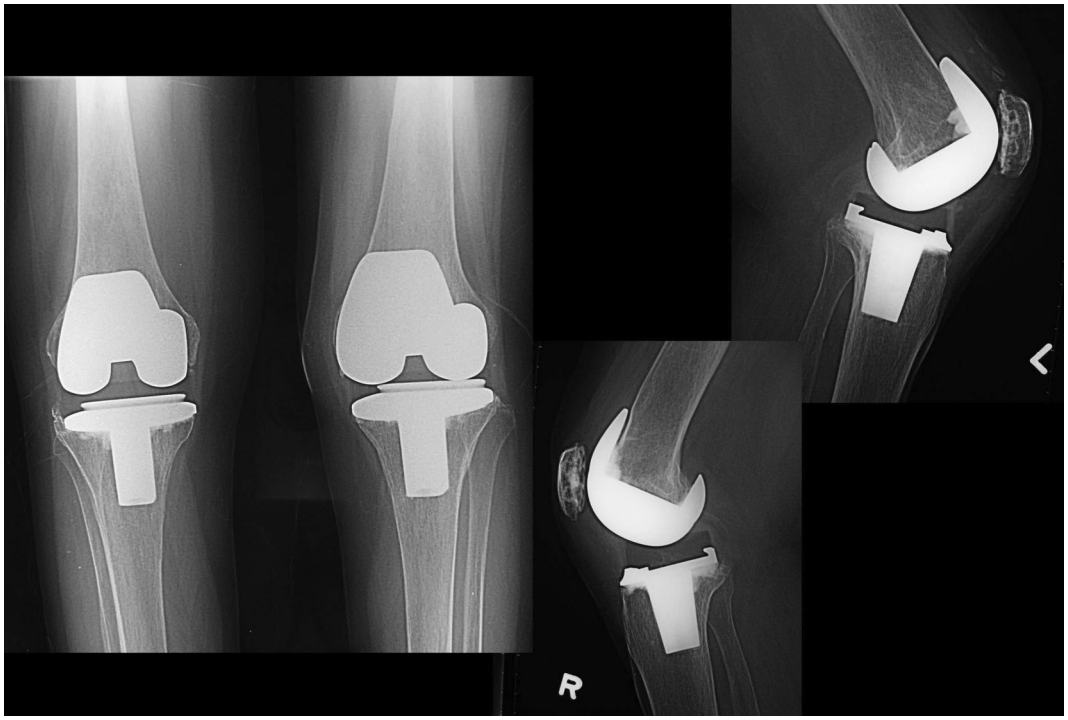

Figure 1. X-rays of the knees 2 years after total knee arthroplasty.

features of severe destruction. However, the patient refused further operations on these joints and defaulted from orthopaedic follow-up from 2003. She walked with a frame indoors until 2008. She complained of progressive bilateral knee pain that limited her movement and walking ability. She therefore had a low morale to mobilise, refused physiotherapy, and became chair-bound. However, she could not recall any injury or acute onset of knee pain in the past 10 years.

She later noticed that her knees were fixed at 70° with limited flexion and extension from late 2009. X-rays showed flexion deformity of bilateral distal femurs, mainly at the metaphysis (Figure 2). Clinically, the patient experienced pain during passive flexion and extension of both knee joints. As a result of the poor general condition of the patient, and as there was no pain at rest, no further treatment or analgesia was given.

Discussion

In general, patients with RA have suboptimal bone quality, especially at the subchondral bone,[3,4] which is due to a major

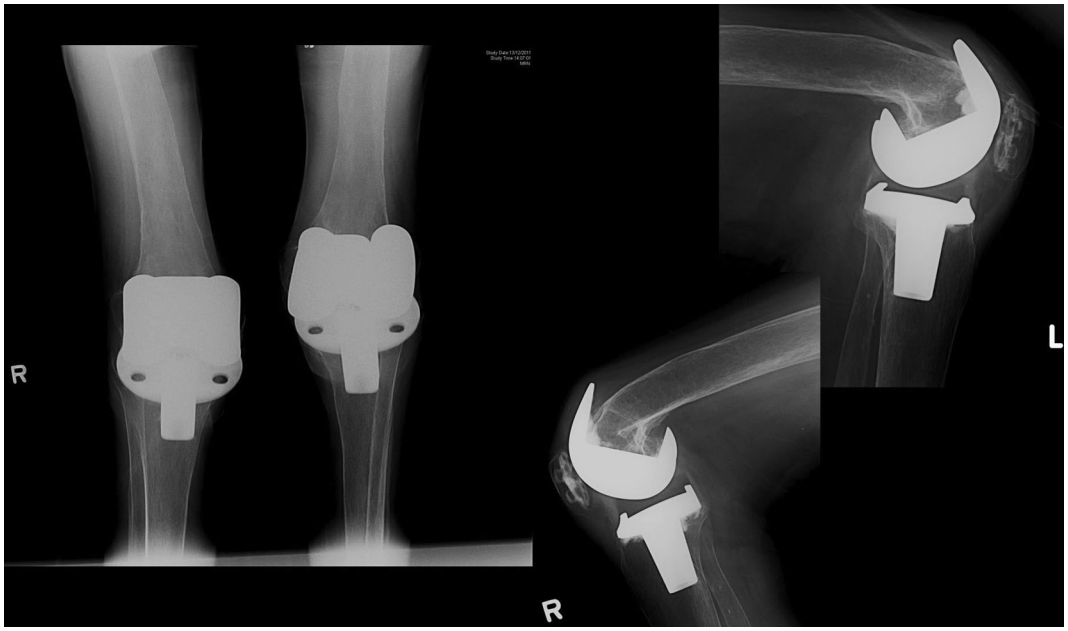

Figure 2. X-rays of the knees showing flexion deformity of the distal femurs.

increase in the rate of bone resorption. On our patient's X-rays, there were obvious cortical thinning and decreased bone density suggestive of osteoporosis. Many factors might account for this, including the use of glucocorticoids, replacement of bone by inflammatory granulation tissue, and prostaglandin released by rheumatoid synovium. All these could have had local deleterious effects on subchondral bone quality because of their direct role in bone resorption,[5,6] and therefore a greater risk of fractures. It is also known that patients with RA, especially those with past steroid use, are more susceptible to insufficiency fractures[2,7] when compared to the normal population, and these insufficiency fractures are more common in the lower limbs, including the femur, tibia, and fibula. However, these fractures can be relatively subtle and difficult to detect because plain X-rays taken in the early period often cannot demonstrate the fractures. They can be relatively asymptomatic, therefore, diagnosis is usually delayed.

Patients with joint arthroplasty are also known to be susceptible to insufficiency fractures if there is pre-existing osteoporosis.[1] Insufficiency fractures of the femoral neck after total knee replacement are frequently reported.[8]

These made our patient, who had RA and bilateral total knee replacements, highly susceptible to insufficiency fractures of the femur. The metaphysis of the long bone has a higher rate of bone turnover than the diaphysis, thus, in patients with RA there might be more severe weakening of the metaphyseal cortex compared to other areas.[4,9] Our patient might have developed insufficiency fractures of bilateral distal femurs at the metaphysis, which later healed in the flexed position, resulting in bilateral flexion deformity.

Because the patient had defaulted from orthopaedic follow-up for many years with no X-rays of her femur, it was difficult to tell when she had developed the fractures.

This flexion deformity of the femur could therefore explain why the patient had marked limitation in extension of the knee joints. Other causes such as acute traumatic fracture or septic arthritis seemed less likely because our patient had no history of trauma or infection.

Moreover, there was also pain elicited during passive flexion, which could have several explanations. The pain could have been due to subtle insufficiency fractures of the femurs as described above, which slowly healed by themselves. Another possibility was impingement of the quadriceps by the anterior fringe of the femoral component of the knee prosthesis. On the X-rays, the flexion deformity of the femur distorted the alignment of the femoral component of the knee prosthesis. The anterior flange of the femoral component no longer aligned smoothly with the femur but impinged onto the quadriceps. This impingement effect might have caused the pain, and it might also have been the cause of the limitation in knee flexion. Another cause for the limited range of movement could have been immobilisation because she had been chair-bound for the past few years, had a low morale to mobilise, and refused physiotherapy for mobilisation and walking.

In conclusion, this is believed to be the first case report of flexion deformity of the distal femur, which was likely to be due to insufficiency fractures, in an RA patient with total knee replacements. Clinicians should maintain a high degree of awareness of the possibility of insufficiency fractures in these patients, especially those who complain of increasing pain in weight-bearing sites, without any history of injury. A plain X-ray may not be adequate to diagnose these fractures, and further investigations such as bone scan, computed tomography, or magnetic resonance imaging may be necessary.

References

1. Yamanaka H, Goto K, Murata Y. Subchondral insufficiency fracture of the femoral head after total knee arthroplasty in a patient with rheumatoid arthritis. *Mod Rheumatol* 2006;**16**:105–8.
2. Nampei A, Hashimoto J, Koyanagi J. Characteristics of fracture and related factors in patients with rheumatoid arthritis. *Mod Rheumatol* 2008;**18**:170–6.
3. Lereim P, Goldie I, Dahlberg E. Hardness of the subchondral bone of the tibial condyles in the normal state and in osteoarthritis and rheumatoid arthritis. *Acta Orthop Scand* 1974;**45**:614–27.
4. Moran EL, Bogoch ER. Animal models of rheumatoid arthritis. In: An TH, Fredman RJ, editors. *Animal models in orthopaedic research*. Boca Raton, FL: CRC Press; 1998. p. 369–90.
5. Grynpas MD, Lieberman I, Bogoch ER, et al. The effect of rheumatoid arthritis on subcondral bone. *Orthop Trans* 1992;**17**:365.
6. Robinson DR, Tashjian AH, Levine L. Prostaglandin stimulated bone resorption by rheumatoid synovia. A possible mechanism for bone destruction in rheumatoid arthritis. *J Clin Invest* 1975;**56**:1181–8.
7. Kay LJ, Holland TM, Platt PN. Stress fractures in rheumatoid arthritis: a case series and case–control study. *Ann Rheum Dis* 2004;**63**:1690–2.
8. McElwaine JP, Sheehan JM. Spontaneous fracture of the femoral neck after total replacement of the knee. *J Bone Joint Surg Br* 1982;**64**:323–5.
9. Takagi T, Tsao PW, Totsuka R, et al. Changes in bone mineral density in rat adjuvant arthritis. *Clin Immunol Immunopathol* 1997;**84**:166–70.