Severe Quadriceps Heterotopic Ossification after Knee Revision Arthroplasty in a 42-year-old suffering from Rheumatoid Arthritis: A Case Report

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Abstract
We present a case of severe quadriceps Heterotopic Ossification (HO) following a revision of a Unicompartmental Knee Arthroplasty (UKA) with a Total Knee Arthroplasty, in a patient with Rheumatoid Arthritis and the subsequent treatment and efficacy of Prophylactic Radiotherapy (PRT) as a secondary prevention following surgical resection of clinically apparent HO.

Keywords: Quadriceps Heterotopic Ossification-Unicompartmental Knee Arthroplasty.

Introduction

The overall incidence of HO after TKA is about 15% [1], although this incidence is lower than the most common HO after Total Hip Arthroplasty (THA) which varies between 4% and 42% reported [2], it is thought to be underreported. The most common complaint related to the development of HO is stiffness, in cases of large HO, the stiffness could progressively lead to an increasing limitation of Range of Motion (ROM) or even ankylosis in the affected joint.

HO of the knee is most commonly forming anterior to the distal femoral shaft in the quadriceps expansion [1]. Although The incidence of severe HO with large dimensions affecting the knee and resulting in these severe consequences is extremely rare and confined to a few case reports, these cases are extremely difficult to prevent and have severe clinical limitations to the patient.

Cases of clinically relevant HO can be treated with radiation therapy, surgical excision of the HO, manipulation of the affected knee joint under anesthesia and possibly revision of the arthroplasty components [1]. Patients with Rheumatoid Arthritis showed less HO and is not a clear risk factor for development of clinically relevant HO formation [3].

Case history

A 42-year-old woman with a background of Rheumatoid Arthritis, who had undergone THA (with several Heterotopic Ossification treated successfully with radiotherapy) and UKA, came to our clinic for limitation of knee ROM and disabling pain. After an initial visit that included an X ray of both knees, a clinical evaluation and clinical tests including a blood test performed to rule out underlying infections. It was then decided to revise the UKA with a Cruciate-Retaining Total Knee Arthroplasty (CR-TKA) with a preoperative plan.

The standard medial para-patellar incision with a mini mid-vastus approach was performed. The surgery was performed by the head of our unit who is an arthroplasty surgeon with more than fifteen years of experience. Preoperative knee ROM was 10–90 degrees that was increased postoperatively to 0-110 degrees. The patient underwent postoperative rehabilitation consisting of proprioceptive training, neuromuscular reeducation, reinforcement of the thigh muscles and Continuous Passive Motion (CPM) with a Kinetc device in our clinic, the patient was discharged five days postoperatively. It wasn't possible to continue the physiotherapy protocol in the outpatient clinic due to the Sars-Cov-2 pandemic, resulting in reporting...
a failure to regain full extension or flexion of the knee. At the two month follow up, the patient came to our clinic with a radiography which demonstrated a large calcification in the distal femoral shaft in the quadriceps expansion. At that time ROM was 5-60 degrees. So, it was decided to restart physiotherapy. After three months of physiotherapy, knee ROM wasn’t improving, so it was decided to surgically treat the patient to remove the HO (Fig. 1) that measured 44 mm, after the surgical excision knee ROM was 0-100 degrees. At the nine month post-surgery check-up, the patient has kept a 0-90-degree ROM and radiography shows no calcifications.

Figure 1: Excision of the HO.

Thanks to the experience gained with hip HO, which had successfully been treated by postoperative radiotherapy [4], it was decided that the patient would undergo three fractions of seven Gy per fraction of PRT sessions to the knee. In addition, we prescribed intense physiotherapy immediately post-op which the patient carried out for a further two months.

Discussion

HO of the knee consists of idiopathic bone formation in soft-tissue structures, which can cause nerve entrapment, joint dysfunction, or ankylosis. It is most observed after trauma or surgery and other less common causes like neurological injury or burns. There is a discrepancy between studies regarding gender as a risk factor; some indicate male gender as a risk group and other female [2], individual risk factors could be genetic predisposition and obesity. Other high-risk factors are pre-existing or contralateral HO, Hypertrophic Osteoarthritis, Ankylosing Spondylitis, Diffuse Idiopathic Skeletal Hyperostosis, infection, previous injuries to the knee, Rheumatoid Arthritis, early septic arthritis, preoperative knee deformity, increased lumbar bone marrow density [5]. Excessive manipulation and extensive soft tissue dissection during surgery such as quadriceps tendon splitting, stripping of the anterior femur for measuring purposes, chronic knee effusions, notching of the femur, vigorous soft tissue retraction, hematoma formation, retained bone particles from bone resections, the "press-fit" fixation surgical technique of the tibia. The HO formation mechanism is still poorly known, yet studies show that the main driver is the mesenchymal stem that causes the inflammatory state that is correlated with HO formation, other inflammatory mediators help the HO formation such as prostaglandins and fibroblast growth factors [6]. The main symptoms for clinically significant HO are the following: severe pain and discomfort that may lead to loss of function., limited ambulation, and decreased range of motion that could cause stiffness at the knee joint [1]. Other less common symptoms are: Swelling or warmth of joint area, Fever and Increased spasticity [7]. Most of the HO formation is clinically irrelevant, clinically significant HO of the knee was reported in roughly 20%. Studies show that after Surgical debulking of knee HO was postoperative ROM improved in 82 %. Patients also had improved ambulation in 57 % of cases and improved sitting ability in 93 % of cases [8]. HO formation is firstly seen at 4 weeks postoperatively in plain radiographs either for asymptomatic or symptomatic patients. Studies show that examination of
patients at 1 year demonstrated no effect of HO on range of motion. The extent of HO was noted to stabilize at 1 year, and HO even resolved spontaneously in several patients. The pattern of HO formation in the knee joint follows a distinctly different process from that observed in the hip joint. HO of the knee is most observed initially either in the periarticular soft tissues or along the anterior edge of the distal femur. Deposits of HO are frequently observed in the medial aspect of the knee joint in the area known as the quadriceps expansion (6). There are 5 different classification systems for HO in the knee joint (by Harwin, Dalury, Furia, Rader, and Toyoda), these classifications are not uniform as the clinical aspect, location and size aren’t conclusive with each other. The assessment of TKA severity is unreliable in the absence of a single comprehensive, standardized classification system [9]. Prevention of HO in the knee after TKA is still inconclusive, studies based on HO formatting in the hip after THA showed that selective COX-2 Inhibitors and non-selective NSAIDs like Diclofenac and Indomethacin are equally effective in the prevention of HO but have inferior results to Etoricoxib if prescribed for 10 days after surgery [10]. Perioperative radiation is also a prophylactic measure but it is only effective when performed in a time window of 20 h before and 96 h after surgery (optimal effect 8 h before and 72 h after surgery) [11].

Conclusions

It is crucial to perform a stratification of patients for risk of HO formation after TKA and have a better understanding of the fundamental rule of physiotherapy, of the risks involved in HO formation in severe HO surgery should be performed following appropriate investigations and should only be considered when the HO has fully matured. HO formation post TKA is less frequent than HO formation post THA but it could be due to the fact that it’s remaining a largely underexplored argument compared to HO post THA and further studies are required. This case report can represent a protocol for treatment clinically relevant HO in the knee after TKA, but further research is needed.

References