Eur J Orthop Surg Traumatol
DOI 10.1007/s00590-010-0590-z

CASE REPORT

Complex regional pain syndrome after hip replacement in a diabetic patient

Munier Hossain · J. G. Andrew

Received: 21 October 2009 / Accepted: 5 January 2010

Abstract
Objective We present a case report of complex regional pain syndrome (CRPS) type I following total hip replacement in a patient with insulin-dependent diabetes mellitus.
Discussion CRPS type 1 as a primary complication following successful hip replacement surgery has not been reported before. We would suggest that CRPS may be a possible cause of persistent pain after successful total hip replacement and should be considered as a differential diagnosis in the assessment of such patients, especially when investigation into persistent pain following hip arthroplasty remains inconclusive.

Keywords Complex regional pain syndrome · Hip replacement surgery · Complications of hip replacement surgery · Hip arthroplasty

Introduction

Unremitting pain after total hip replacement (THR) is disappointing and requires extensive investigation to establish the cause of pain. Complex regional pain syndrome (CRPS) after hip replacement surgery is very rare. There is little discussion of CRPS type 1 as a complication following THR in the published literature. We came across only one case report in the English language [1]. But the reported patient developed CRPS following an episode of hip dislocation and not following primary hip replacement. We describe a case of Complex regional pain syndrome type 1 as a primary complication after successful primary hip replacement surgery in an insulin-dependent diabetic patient.

M. Hossain · J. G. Andrew
Department of Trauma and Orthopaedic Surgery,
Ysbyty Gwynedd, Bangor LL57 2PW, UK

M. Hossain (✉)
101 Taliesin Court, Cardiff CF10 5NJ, UK
e-mail: munierh@doctors.org.uk

Case report

A 54-year-old retired Caucasian lady shop assistant presented with osteoarthritis of the right hip (Fig. 1). She previously enjoyed walking and cycling. On presentation, she was mobilising with difficulty with the help of two elbow crutches. On visual analogue scale (VAS), her average hip pain was 7/10 (range 4–9). Hip pain significantly disturbed her sleep (VAS 9/10) and affected her general quality of life in spite of taking maximum analgesia (VAS 9/10). She had 10° of fixed flexion deformity and marked stiffness of the affected hip. Her pre-operative Harris hip score was 22.475 (pain 10/44, function 6/47, deformity ¾ and motion 3.4/5). She also had insulin-dependent diabetes mellitus. HbA1C value prior to surgery was 8.4%. Surgery was performed uneventfully by the senior author using the standard posterior approach. Sciatic nerve was protected with posterior capsular and muscle flaps and was confirmed intact at the end of surgery. Immediate post-operative x-ray was satisfactory (Fig. 2). She was mobilised early, reported immediate pain relief and was discharged home 5 days after surgery.

Two weeks after surgery, she started having burning pain and paraesthesia in right foot. There was no history of trauma. The pain gradually increased in severity in spite of taking regular adequate analgesia and affected her mobility. On VAS, her right foot pain was 8/10. Pain was

Published online: 10 January 2010

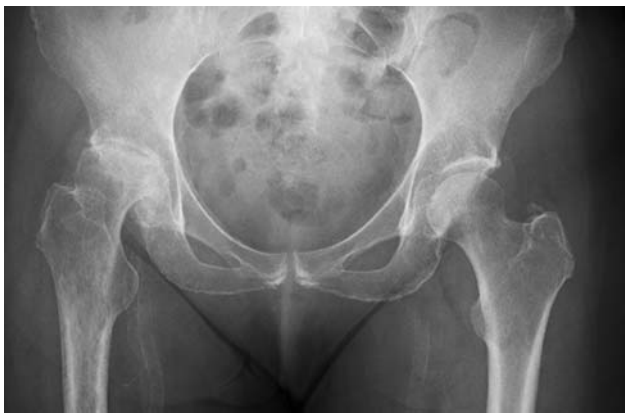

Fig. 1 Radiograph showing osteoarthritis of the affected hip

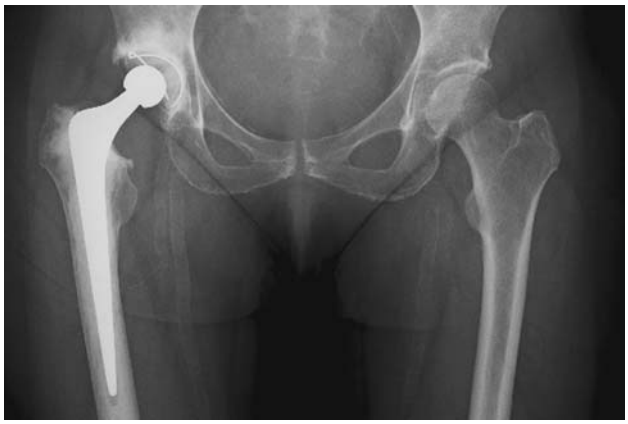

Fig. 2 Radiograph following hip replacement

non-mechanical in type and worse at night. There was no local erythema. Her right ankle was moderately stiff, and there was allodynia and swelling of the right foot. Paraesthesia and allodynia was present around dorsum, lateral and medial border of the foot and not present in a glove and stocking distribution. There was no foot drop or undue bruising of the thigh. Her muscle power and neurological status was unchanged. Back pain had not worsened since surgery. There was no evidence of deep venous thrombosis or deep infection. Her glycaemic control was not different from usual. The infection parameters were within normal limits (CRP < 10). X-ray showed patchy osteopenia of the right foot.

After excluding other possible causes including infection, sciatic nerve palsy, diabetic neuropathy and aggravation of low back pain, we felt that her clinical and radiological features were suggestive of complex regional pain syndrome. This was confirmed on three phase bone scan that showed reduced vascular flow and blood pool phase isotope uptake affecting the right foot and ankle with some peri-articular uptake on the phase three static scans suggestive of vasospastic phase of CRPS. The left foot showed increased tracer uptake on the static phase suggestive of degeneration. Bone scan did not reveal any abnormality of the hip or thigh. Her symptoms subsequently improved on gabapentin. Her neurological status remained the same. At latest follow-up 12 months after surgery, she was mobilising without any walking aid. Harris hip score on latest follow-up was 89.225 (pain 44/44, function 37/44, deformity 4/4 and motion 4.22/5). On VAS, her right foot pain was 0/10. She had stopped taking gabapentin.

Discussion

Investigation into the cause of painful hip following THR is complex and not always conclusive [2]. However, the diagnosis of CRPS type I should be kept in mind, especially when the presentation appears atypical or investigation remains inconclusive.

Complex regional pain syndrome (CRPS) is the most recent descriptive term for a disorder that has had a number of synonyms over the years. The change in terminology also shows the evolution of our understanding of this complex process. It is characterised by pain, sensory-motor and autonomic symptoms. According to the criteria set out by the International Association for the Study of Pain (IASP), the diagnosis of CRPS is primarily clinical and is a diagnosis of exclusion [3]. Any kind of noxious stimulus can be a causative factor. Diagnosis is straightforward in the florid state but can often be difficult if characteristic signs are not present. CRPS presents as spontaneous pain or hyperalgesia/hyperaesthesia disproportionate to the inciting event. Pain is present at rest in most of the patients and is associated with hyperalgesia in almost all the sufferers. A third of patients also have brush-evoked pain or allodynia. Trophic changes are present in 50% of patients and begin with early increase in hair and nail growth and revert to late symptoms of reduced hair and nail growth and skin atrophy. Autonomic disturbances are common and initially present with erythematous and hot swollen limb followed by late changes in cold blue skin. Type I CRPS is not associated with any specific nerve injury. Type II CRPS is associated with a specific nerve injury. Characteristic spotty osteoporosis in plain radiography is only seen in less than half of the patients after 4–8 weeks. Three phase bone scan is reported to be more sensitive especially when clinical examination and radiographs are normal; showing increased tracer uptake in late phases due to increased bone metabolism [1]. Aetiology is still unclear [4]. It is not clear why some patients develop CRPS and others do not. An assertion that affected individuals have predisposing personality remains unproven, but there is evidence of HLA association [5].

At early stage, this condition is more responsive to treatment [6]. The cornerstone of treatment is physiotherapy and symptomatic treatment for neuropathic pain. Narcotic opiates are not recommended. Both calcitonin and bisphosphonates have been found beneficial in randomised trials. Gabapentin has been shown to help with neuropathic pain. Lumbar sympathetic block is also advocated in patients showing increased uptake on bone scan.

Although the reported patient had unstable diabetes, it was agreed that the severity of her symptoms warranted a THR. The decision to proceed with THR was made after the extensive discussion of the risks and benefits with the patient and her family. Her diabetes was reasonably controlled in the perioperative period, and neurological status had remained the same. Her subsequent presentation with severe burning type pain worse at night probably due to touch of bedcovers raised the suspicion of CRPS. CRPS remains a diagnosis of exclusion, and one had to exclude several other possibilities. CRPS type II due to sciatic nerve injury would have been possible. But her surgery was uneventful, there was no significant intra or post-operative bleeding, and there was no foot drop to suggest the possibility of sciatic nerve palsy. The nerve had been confirmed intact post-procedure. Leg length lengthening was minimal and could not have stretched the sciatic nerve (3.5 mm). The wound was healthy, and infection parameters were within normal limits. X-ray showed characteristic osteopenia. Radiological appearances could be due to disuse osteoporosis. Since she was mobilising with bilateral elbow crutches, one would then expect somewhat similar features in the opposite foot. Bone scan suggested only degenerative changes in the opposite foot, but no evidence of osteopenia. This pain might also be caused by aggravation of low back pain. Her back pain had not aggravated post-operatively and was distinct in site and character from the foot pain. Diabetic neuropathy could be a distinct possibility. There are suggestions that patients with diabetes are in general at increased risk of CRPS [7]. Acute onset diabetic neuropathy is known to be associated with CRPS [8]. But her glycaemic control was no worse in the perioperative period, and neurological state had also not changed. The site and type of pain was also distinct and not related to a peripheral nerve distribution and not in a glove and stocking pattern either. Early diagnosis and initiation of treatment ensured a favourable outcome.

We would suggest that CRPS may be a possible cause of persistent pain after successful total hip replacement and should be included in the assessment of such patients especially when investigation into persistent pain following hip arthroplasty remains inconclusive.

Conclusion

Complex regional pain syndrome can complicate an otherwise successful THR and present as unremitting pain. Constant non-mechanical type pain unrelated to physical activity in the absence of infection or loosening should arouse the suspicion of chronic regional pain syndrome. Diagnosis is mainly clinical, but bone scan may be helpful. It is important to keep this diagnosis in mind while managing a patient with painful THR as early diagnosis, and treatment is important for a successful outcome.

Conflict of interest statement None.

References

1. Mittal R, Khetarpal R, Malhotra R, Kumar R (1997) The role of Tc-99 bone imaging in the management of pain after complicated total hip replacement. Clin Nucl Med 22:593–595
2. Bozic KJ, Rubash HE (2004) The painful total hip replacement. Clin Orthop Relat Res 420:18–25
3. Birklein F (2005) Complex regional pain syndrome. J Neurol 252:131
4. Dowd GSE, Hussein R, Khanduja V, Ordman AJ (2007) Complex regional pain syndrome with special emphasis on the knee. J Bone Joint Surg 89-B:285
5. van de Beek WJ, van Hilten JJ, Roep BO (2004) HLA-DQ1 associated with reflex sympathetic dystrophy. Neurology 55:57
6. Schwartzman RJ, Alexander GM, Grothusen J (2006) Pathophysiology of complex regional pain syndrome. Expert Rev Neurother 6:669
7. Acquaviva P, Schiano A, Harnden P, Cros D, Serratrice G (1982) [Algodystrophy: predisposition and pathogenic factors. Results of a multicentric survey concerning 765 cases]. Rev Rhum Mal Osteoartic 49:761–766
8. Schapira D, Barron SA, Nahir M, Scharf Y (1988) Reflex sympathetic dystrophy syndrome coincident with acute diabetic neuropathy. J Rheumatol 15:120–122