Assessment and management of chronic pain in patients with stable total hip arthroplasty

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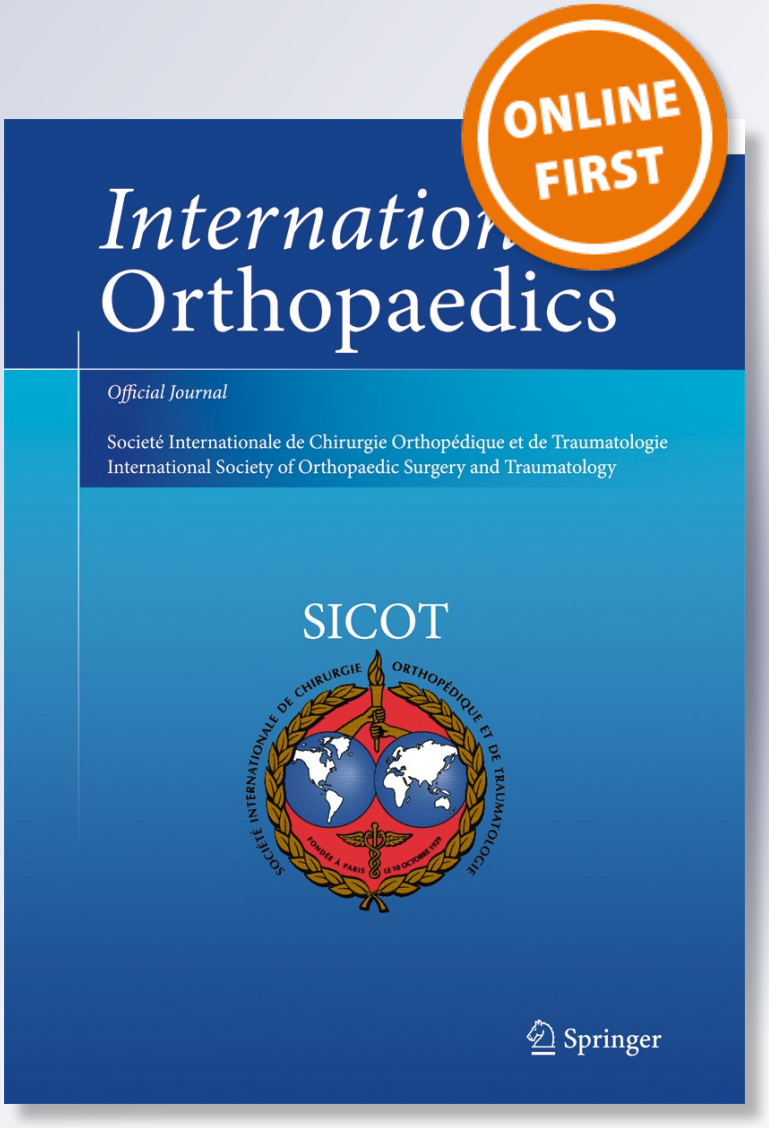

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REVIEW ARTICLE

Assessment and management of chronic pain in patients with stable total hip arthroplasty

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Abstract Total hip arthroplasty (THA) is one of the most successful operations that can restore function and relieve pain. Although a majority of the patients achieve significant pain relief after THA, there are a number of patients that develop chronic pain for unknown reasons. A literature search was performed looking for chronic pain after total hip arthroplasty and stable THA. Major causes of chronic pain include aseptic loosening or infection. However, there is a subset of patients with a stable THA that present with chronic pain which can have several aetiologies. These include soft tissue, bony, neurological, vascular and psychological causes. Essential for successful treatment is the ability to make the correct diagnosis. Thus therapy may be either non-operative or operative. In addition, diagnosis and management often may require multidisciplinary approaches to successfully alleviate chronic pain in these patients with a stable prosthesis.

Introduction

Total hip arthroplasty (THA) is extremely successful in restoring function and improving the quality of life in patients with osteoarthritis related groin pain [1]. Modern minimally-invasive approaches help to reduce the postoperative pain and accelerate the rehabilitation [2]. Although a majority of the patients achieve significant pain relief after THA, there are a number of patients with radiographically stable THA implants that develop chronic pain for unknown reasons. In a recent nationwide survey of 1,231 THA patients in Denmark, up to 28.1 % had chronic ipsilateral hip pain at more than one year following the procedure and 12.1 % of these patients had moderate to significant limitations of their daily activities secondary to the chronic hip pain [3]. This article gives an overview of the possible predictors and causes of chronic hip and groin pain in patients with a stable THA.

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" [4]. Acute pain signals a specific nociceptive incident and is self-limited. In contrast, chronic pain is defined as pain that lasts longer than three to six months and is associated with a loss in the alert and protection function. This differs from acute pain in that localisation of the cause can be difficult in chronic pain patients. Investigation of patients that are experiencing very debilitating pain with a stable THA implant may not reveal any tissue damage around the prosthesis. Furthermore, chronic pain may develop from an acute painful condition, but continues beyond the normal time expected for the healing process or persists or recurs for different reasons.

Somatic predictors

In the literature, concurrent evidence has been reported regarding somatic predictors for developing chronic hip pain. Certain healthy populations have an increased risk of developing hip pain even without surgery. Women had

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double the risk of developing hip pain when compared to men. Other risk factors are age, high body mass index (BMI), whole-body vibration and physically demanding work [5]. The same risk factors as above have also been found in patients who developed chronic pain after THA with stable implants. These studies further confirm that women have the greatest risk of developing postoperative pain after surgery. Furthermore, patients with high preoperative pain levels as well as the elderly, obese and depressive individuals had persistent increased pain levels two to five years postoperatively [6]. In terms of revision THA operations, female gender, higher BMI, depression and age are independently significantly associated with each of the following outcomes: higher moderate to severe pain, use of non-steroidal anti-inflammatory drugs (NSAIDs), moderate to severe activity limitation and dependence on walking aids [7].

Psychological factors

A study by Rolfson et al. [8] reports that certain psychological factors may predispose women to have a worse outcome than men after THA as measured by pain, satisfaction and health-related quality of life (HRQoL) scores. Rolfson et al. evaluated the association between anxiety and patient-perceived outcomes with respect to pain and satisfaction after THA. This study demonstrated that a factor previously poorly understood in hip surgery, pre-operative anxiety/depression, might contribute to persistent discomfort and poorer outcomes after THA. In fact, the pre-operative EuroQol System (EQ-5D) dimension of anxiety/depression was among the strongest predictors of pain relief and satisfaction after THA. Furthermore, in contrast to these psychological factors, the authors report that the pre-operative severity of the radiographs and patient function was not related to postoperative outcome [8].

Personal attitude and expectations regarding surgery are assumed to influence postoperative analgesia and pain relief. Gandhi et al. found that the patient expectation of greater pain relief from surgery independently predicted greater improvements in the WOMAC (Western Ontario McMaster University Osteoarthritis Index) pain score one year after surgery [9]. This finding agrees with several reports in the literature [10, 11]. Furthermore, the perioperative setting such as the quality of the admission process or medical and nursing care related to the patient's surgery seem to also play a major role for better health-related quality of life after THA. In a study by Baumann et al. the immediate postoperative patient satisfaction with perioperative care was a good predictor of self-perceived health status after surgery. In addition, these patients with good self-perceived health status preoperatively also have high postoperative HRQoL. However, patients reporting poor preoperative HRQoL showed no improvement in HRQoL after surgery [12].

Exogenic predicting factors

Apart from somatic and psychological predictors for the development of chronic hip pain after THA, there are various exogenic factors that may influence the outcomes. A systematic review by Macfarlane et al. with the search terms "THA, THR, hip operation and analgesia/anesthesia" concluded that the use of regional analgesia significantly reduced postoperative pain after THA. However, this review did not evaluate chronic pain development after THA [13]. Another factor is the administration of neuroactive drugs (Ketamine) may have a positive effect on the postoperative pain level after stabile primary hip implant. Patients who received an intravenous Ketamine infusion at the time of the incision and 24 hours postoperatively needed significantly less morphine in the perioperative time period. In addition, Ketamine also showed a morphine-sparing effect after THA, even when morphine was combined with multimodal systemic analgesia. Furthermore, Ketamine also facilitated rehabilitation and reduced persistent postoperative chronic pain up to six months after surgery [14].

Basic science experiments have also shown that noxious stimuli related to surgical damage of the soft tissues may produce a hyperexcitable and hypersensitive state in the neurons of the central nervous system [15]. Clinical manifestation of hypersensitivity is poorly understood, however, persistent pain in the setting of a stable implant may be attributed to the consequence of cellular hyperexcitability that can be secondary to inflammation or nerve damage [3]. Current animal models of neuropathic pain have found that multiple inflammatory mediators released from the damaged tissue may excite the primary sensory neurons in the peripheral nervous system and also cause a persistent increase in their excitability [16]. At the cellular level, formation of new channels, both up-regulating and down-regulating of certain receptors and altering the local and distance inhibition are some of the biological features that contribute to the excitability of neuron upregulation that may be a factor in chronic pain after THA [17].

Management of patients with chronic hip pain after a stable THA can present a challenging problem for the orthopaedic surgeon. Successful treatment requires making accurate diagnosis of the aetiology of pain. Aseptic loosening and infection are two important factors that will cause chronic hip pain after a THA and these must be ruled out before considering other less common causes. The differentiation between various origins, causes and time of onset of chronic pain in a stable THA must be elicited to facilitate in making the correct diagnosis. Persisting pain similar to the preoperative pain reported by the patient after a stable THA

may raise questions if the indications for surgery were correct and must be differentiated from new and dissimilar hip/groin pain that arises during the postoperative period. There may be other cofounding variables including psychological factors, workers compensation, retirement payments, and legal situations relating to the operation that may contribute to the chronic pain. Looking more closely at the possible causes for new onset of chronic pain in a stable THA, it is necessary to differentiate between soft tissue, bony, neurological or vascular aetiologies.

Soft tissue damage/irritation

Soft tissue damage or irritation (tendinopathies or bursitis) is a common cause of chronic pain after THA. The generic term for this type of pain is the greater trochanteric pain syndrome (GTPS). The GTPS describes pain in the hip region that typically involves the area from the lateral thigh to the knee. Various entities or spectrum of disease are classified under the GTPS such as trochanteric bursitis, tendinopathy of the gluteus medius and minimus tendon, rupture of the gluteal muscles or tendons, as well as snapping hip syndrome. Risk factors include female gender, rheumatoid arthritis, overweight, low back syndrome, fibromyalgia, and hyperuricemia [18–20]. The GTPS is triggered by chronic microtrauma, overload, muscle dysfunction, or acute trauma [21–23]. Clinically, the pain can be elicited over the greater trochanter with palpation. In addition, active abduction and rotation movements of the hip may also provoke pain. A positive Trendelenburg's sign may also be detected in patients with GTPS [24].

In addition to the clinical examination, the diagnosis can be confirmed by MRI scans or by ultrasound examination. It is essential to evaluate for fatty degeneration of the gluteal muscle and the extent of the gluteal tendon rupture, as well as the irritation of the bursa to make the diagnosis of GTPS [25–28]. The first line of management should be conservative with anti-inflammatories and avoidance of activities. Other nonsurgical treatment options include physical therapy, shock wave therapy and local infiltrations [29–31]. In the case of gluteal tendon rupture, conservative management does not have much of a role. Ruptures of the gluteal tendon should be surgically repaired, or in chronic cases, a bursectomy and resection of prominent osseous bumps causing soft tissue irritation can be performed [29]. It is essential to repair the tendon during the acute period before fatty infiltration and retraction to improve the success rate and outcome. In the case of snapping hip syndrome, a Z-plasty of the iliotibial band is recommended if conservative measures are unsuccessful [32]. For all these surgical therapies endoscopic methods are proving increasingly popular [32–34].

Impingement of the iliopsoas is a rather uncommon cause of soft tissue irritation and therefore a contributor of groin pain after THA. It can cause a bursitis of the iliopectineal bursa and occurs in up to 4.3 % of patients. O'Sullivan et al. and Nunley et al. both reported the mean time from primary hip surgery to presentation of symptoms from iliopsoas tendonitis was characteristically several months [35, 36]. A typical clinical finding is an insidious onset of groin pain during active hip flexion, passive hip extension, and external rotation. A prominent anterior edge of the acetabular component, which may exceed the margins of the native acetabulum or be malpositioned, can exemplify an anatomical cause for the irritation of the iliopsoas bursa and tendon. Additional causes of pain related to the acetabular component include fixation screws protruding through the ilium and into the iliacus muscle or extruded cement debris around the anterior rim of the cup. Nunley et al. demonstrated that selective steroid and anaesthetic injections of the iliopsoas bursa give adequate pain relief in the majority of patients with iliopsoas bursitis. Selective injections with steroids and an anaesthetic should be part of the non-operative treatment both as a diagnostic and therapeutic modality before surgical release of the iliopsoas tendon or component revision surgery is considered [36]. Only in cases of insufficient pain relief through conservative therapy is revision surgery indicated. In contrast, Dora et al. stated that the tenotomy of the iliopsoas and revision of the acetabular component are both successful surgical options. They found that the iliopsoas tenotomy provided the same pain relief and functional results as revision of the acetabular component but avoided the risks of the latter procedure [37].

Bony aspects

There are various types of mechanical impingement in THA that can cause pain in an otherwise stable THA. Three types of impingement exist: bone-to-bone, component-to-component and component-to-bone. The bone-to-bone impingement depends mainly on the offset of the prosthesis. Whereas for the component-to-component impingement, the orientation of the implants and the prosthesis design is the main contributor. Lewinnek et al. established the so-called safe zone for the positioning of the cup. When the cup position is placed in the safe zone, the risk for dislocation is significantly reduced. This safe zone is defined as 5–25° of anteversion and 30–50° of inclination [38], given that the impingement is not only dependent on the positioning of the cup, but also on the orientation and design of the stem. Therefore, the orientation of the cup to the stem to each other is crucial as the orientation of one component determines the orientation of the other component. A reasonable recommendation to balance stability while minimising the risk of mechanical impingement is to use modular systems (Fig. 1). This allows a dynamic examination of the impingement

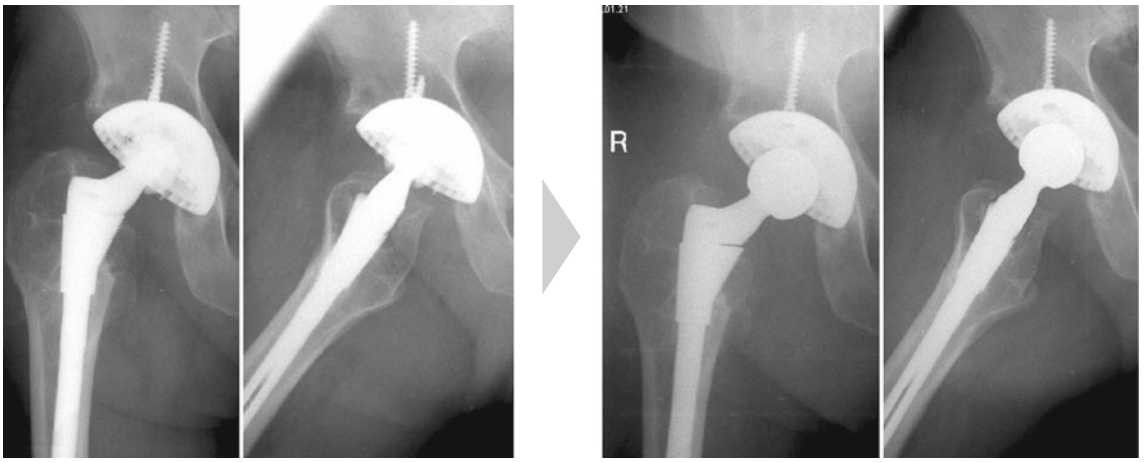

Fig. 1 Increasing the offset with a modular system because of mechanical impingement of THA

range of motion in the operative setting before final component positions are taken. Attention should be turned to the space between the lesser trochanter and the ischium and the space between the greater trochanter and the ilium [39–41]. Also, computer navigation systems can help to prevent incorrect positioning of the implants [42].

A common cause (up to 90 %) of chronic pain in stable THAs are heterotopic ossifications (HO). They are primarily located posterior to the prosthesis. HO are assumed to develop out of osteoblastic progenitor cells that are scattered out of the bone marrow during intramedually broaching. Furthermore, progenitor cells that are located in the soft tissue (inducible osteogenic precursor cells [IOPC]) can be activated through inflammatory stimuli. An HO usually gains clinical relevance when it reaches the Brooker classification III to IV by causing pain and restricted ROM (Fig. 2) [43–45]. Standard prophylaxis can be accomplished with medication (NSAID) or by radiation. However, neither of these procedures seems to have a superior outcome [46]. Clinically relevant ossifications develop in 20–45 % of all THA without prophylaxis compared to less than 5 % under an appropriate prophylaxis. Risk factors for HO include male gender, elderly, post-traumatic or hypertrophic osteoarthritis, rheumatoid arthritis, ankylosing spondylitis and Paget's disease [47–50]. Treatment of HO-related pain after THA is necessary when the patient experiences any

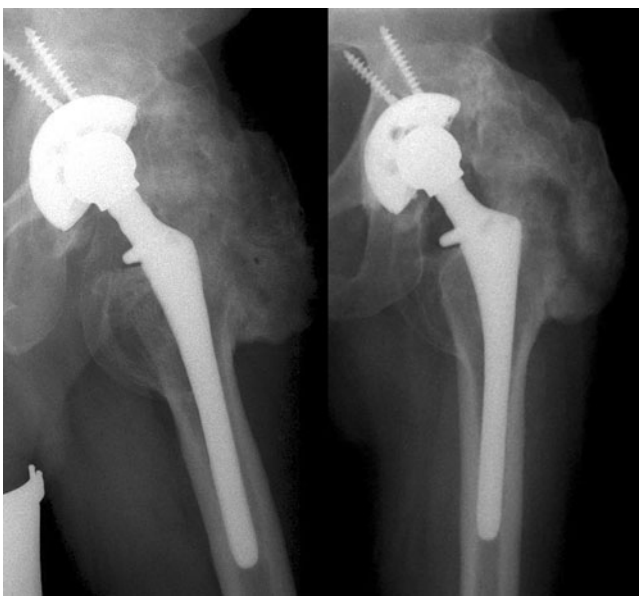

Fig. 2 Heterotopic ossifications after THA (Brooker classification IV)

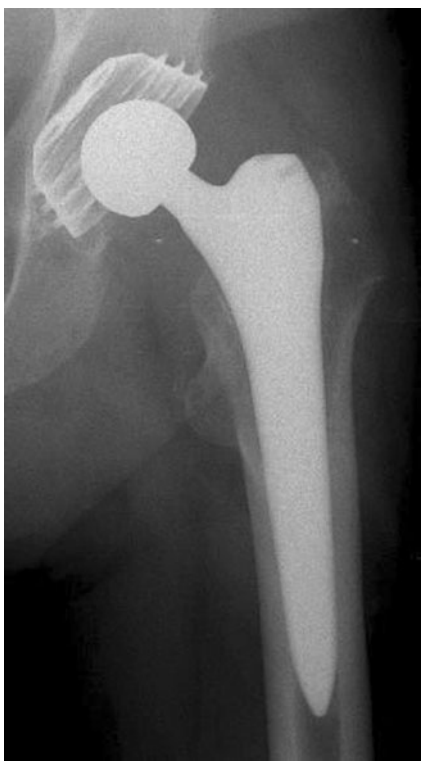

Fig. 3 Stress shielding of the proximal femur

discomfort or restriction of movement. The nonoperative treatments include physiotherapy and analgesia. If the HO have not reached the end of growth treatment with NSAID or radiation can be tried to stop the growth of the HO. But this is no use for pre-existing HO. If the nonoperative treatments fail then surgical resection of the ossification is the only therapeutic option [51]. However, it should be delayed until the heterotrophic ossification reaches maturity and end of growth which usually takes up to one year. To evaluate the activity of the heterotrophic ossification, a three-phase bone scintigraphy or alkaline phosphatase level (ALP) is recommended [52].

Another controversial factor in the development of chronic pain after stable THA is the influence of stress shielding. Stress shielding is defined as a reduction of bone density caused by a biomechanical load on the bone by an implant. The mismatch of the implant's and the bone's stiffness lead to a nonphysiological transmission of power. Cementless hip prostheses are typically affected. This is especially seen in the distal anchoring hip systems when the proximal femur shows radiolucent bone loss, because osteolysis typically takes place in the region of minor load (Fig. 3). In particular, longer and more extensively porous coated stems are affected. After stress shielding was first described it was suggested that this loss of bone substance would lead to increased pain, loosening and fractures. In contrast to this initial hypothesis, recent studies have shown that this is not the case. More problematic is the loss of substance of the proximal bone to provide anchoring in a revision prosthesis [53–59].

Another aetiology of bony causes of chronic pain in a stable THA are fatigue fractures. These fatigue fractures are typically found in the area of the acetabulum. They can either occur directly during surgery, especially in cementless press-fitted cups, but can also develop years after surgery. The latter fatigue fractures are often caused by local osteolysis. The appropriate treatment depends critically on the extent of the fracture and in particular on the stability of the acetabular component [60–64].

Neurological or vascular causes

Injury or irritation of nerves can be another cause of hip pain with a stable THA. Possible aetiologies include intraoperative nerve injury or nerve irritation due to scar entrapment or foreign body. Intraoperative nerve injury occurs through direct injury to the nerve either via pressure of a hook/retractor, over-stretching, or complete nerve transection. Nerve irritation can be caused by haematomas, prosthetic components, screws, scar tissue or bone cement. Furthermore, any pain of the groin or thigh region can have its primary cause in the lumbar spine, especially when the pain radiates in a dermatomal pattern. Therefore it is important to rule out spine-related pain to the hip or groin in the preoperative setting. Other recent onset nerve root irritations can also lead to pain that is very similar to those of a coxalgia [65–68].

Vascular injury or irritation can also cause chronic pain in a stable THA. Vascular-related pain can occur after total hip arthroplasty and especially after revision THA, caused by ischemia. This may occur through direct violation of the vessels or by intraoperative traction on the leg (stretching of the vessels) when there is a pre-existing vascular stenosis. In addition, displacements of the vessels or injuries caused by scar contraction or other aetiologies can result in the formation of arteriovenous fistulas and aneurysms [69–71]. In these cases, the diagnosis can be confirmed with the assistance of Doppler ultrasonography or angiography in addition to clinical examination. These patients can present with a palpable groin mass, a bruit may be heard on a stethoscope, and there may be a sudden drop in the patient's haematocrit. Treatments include consultation with a vascular team and either open bypass or endovascular coiling of the fistula or aneurysm [69–72].

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