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Clinical case

Adhesive capsulitis of the hip: Concerning three case reports

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Abstract

Purpose. – To describe the diagnosis and treatment of adhesive capsulitis of the hip (ACH).
Method. – A literature review and consideration of three case reports.
Discussion. – Adhesive capsulitis of the hip is a supposedly rare but probably underestimated condition which predominantly affects middle-aged women. Clinical assessment reveals a painful limitation of joint mobility. The diagnosis is confirmed by arthrography, where the crucial factor is a joint capacity below 12 ml. Osteoarthritis and complex regional pain syndrome type 1 are the two main differential diagnoses. Whether the treatment is pharmacological, physical or surgical depends on the aetiology of the condition. Physiotherapy is essential for limiting residual deficits and functional impairments.
Conclusion. – Adhesive capsulitis of the hip is probably more common than suggested by the limited medical literature. The condition is frequently idiopathic but can be secondary to another joint pathology. The first-line treatment consists of sustained-release corticosteroid intra-articular injections and physical therapy. Arthroscopy and manipulation under anaesthesia may be useful in cases of ACH which are refractory to treatment.

Keywords: Adhesive capsulitis of the hip; Arthrography; Physiotherapy

1. Introduction

Adhesive capsulitis of the hip (ACH) is due to retraction of the fibrous joint capsule of the hip. Despite the fact that ACH was first described by Caroit et al. [2], there have been few publications on the condition since then [3,8,9,12] and its precise aetiology remains unknown. Lequesne et al. described two kinds of ACH: idiopathic ACH (due to diabetes, for example) and secondary ACH (due to osteochondromatosis, for example) [8,12]. The clinical assessment of ACH is similar to that for adhesive capsulitis of the shoulder (ACS) and features a combination of pain and restricted active and passive joint motion.

The treatment can be pharmacological, physical or surgical. All in cases, physiotherapy is essential for limiting deficits and functional impairments.

The purpose of this paper is to review the diagnosis of this underestimated pathology [12] and examine therapeutic strategies. We present three clinical case reports.

1.1. Case report 1

A 34-year-old woman consulted for right coxofemoral pain. She had a medical history of iodine allergy, well stabilized fibromyalgia (2003) and surgery for a haemorrhagic ovarian cyst (2006).

The current set of symptoms had appeared progressively since July 2004, starting with lower back pain. Coxofemoral pain was rhythmically present during gait and was exacerbated by impacts and joint motion. The patient was not able to walk for more than 2 km. A clinical assessment did not reveal any static equilibrium disorders. Segmental examination of the spinal column and a neurological assessment gave strictly normal results. The patient reported intense pain (90 out of 100 on a visual analogue scale [VAS]) on palpation of the right inguinal fold. Passive and active motion of the right hip joint was limited and painful: flexion/extension was 90/15°

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(compared with 110/25° on the left side); external/internal rotation was 15/20° (20/30° on the left side); and abduction/adduction was 15/15° (45/30° on the left side). Functional assessment revealed significant discomfort, with a Lequesne index of 12.5 (out of 26) and a Merle d'Aubigné score of 11 (out of 18) [2,4,5].

The patient had already received class 1 and 2 analgesic treatment, massage, transcutaneous electric nerve stimulation (TENS) for pain relief and physiotherapy with slow manipulation in order to improve joint amplitude.

The laboratory results (blood cell count, inflammation markers, blood phosphate/calcium balance and a Waaler-Rose assay) were normal. Standard radiological examination revealed moderately intense right coxofemoral osteoarthritis, in the absence of any sign of osteonecrosis or rapidly progressing coxarthrosis (Fig. 1). Magnetic resonance imaging (MRI) (Fig. 2, performed to screen for a secondary aetiology) confirmed the presence of small degenerative lesions such as those seen in protrusive coxofemoral osteoarthritis. There was no evidence in favour of primary osteochondromatis, subchondral damage or impaction at the head of the femur.

On the basis of the clinical assessment and a nearly normal radiological and MR examination, ACH was suspected. Hip joint capacity is usually evaluated via intra-articular injection of iodine-containing contrast medium until significant reflux, induced pain or at least a sensation of fullness occurs. Given that this patient had an iodine allergy, lidocaine, (Xylocaine®) was injected into the coxofemoral joint. Only 8 ml could be injected (that is, well below the normal value of 15 ml), confirming the diagnosis of ACH. This diagnosis was also supported by the fact that procedure was extremely painful. At the same time, a corticosteroid (Lederspan®) was injected and reduced the pain from 90 out of 100 to 10 out of 100 on the VAS, without any additional loss of hip joint motion.

Physiotherapy was prescribed in order to increase the amplitude of coxofemoral joint motion. The treatment consisted of slow, progressive manipulation in abduction and rotation, traction, stretching below the pain threshold, overall muscle strengthening (first isometric and then dynamic) and proprioceptive exercises. The therapy was administered five times a week (for about 1 h each time) over two months in this young, motivated patient.

Two additional corticosteroid injections (with a one-month interval between them) were required following an increase in the pain level (VAS: 80 out of 100). The patient continued physiotherapy three times a week for the next 12 months. We

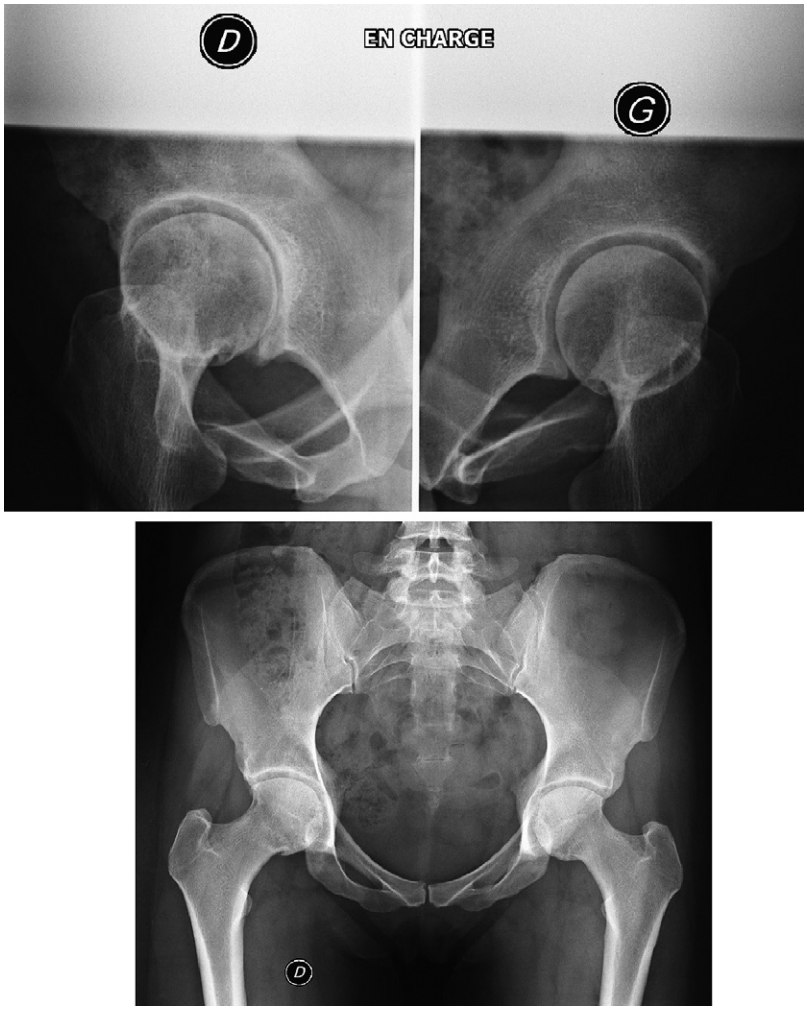


Fig. 1. a: radiographs of the hips. Case 1; b: anteroposterior radiograph of the pelvis, standing. Case 1.

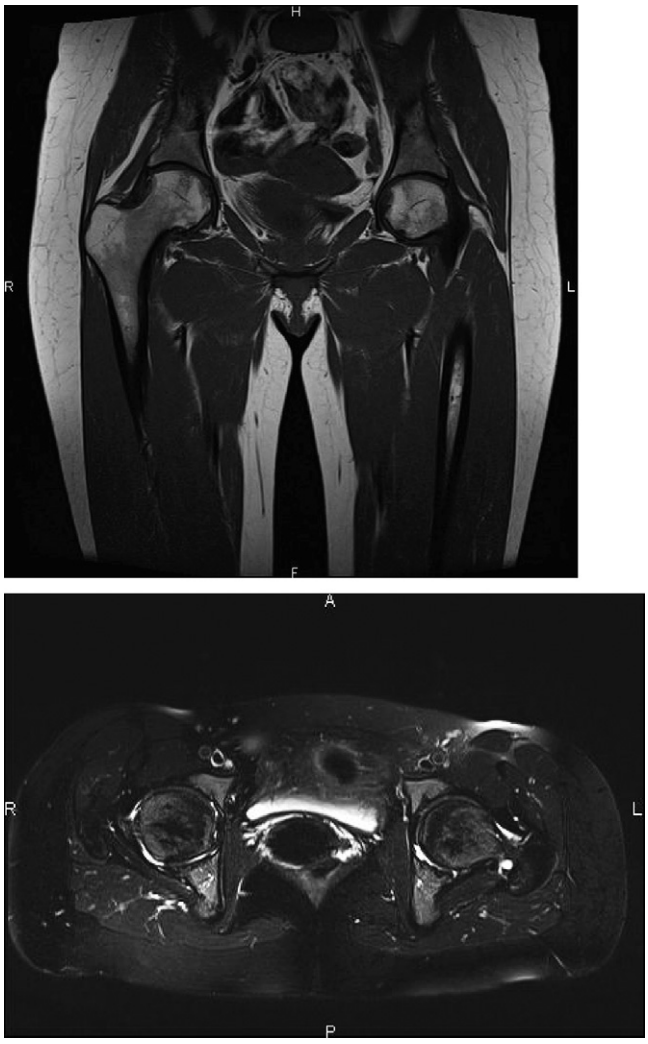


Fig. 2. MRI of the hips. Case 1.

observed an improvement in joint amplitude (abduction: 45°; adduction: 40°; internal and external rotations: 35°), a decrease in pain (to 20 out of 100) and a functional improvement (with a Lequesne index of 6 and a Merle d'Aubigné score of 15).

1.2. Case report 2

A 76-year-old man consulted for progressive camptocormia that had appeared a few months previously (in the absence of a notable triggering factor) and was associated with pain in the left hip. He had a medical history of chronic back pain with radiculalgia.

An examination of the spinal column and the right hip gave completely normal results. Left hip motion was severely restricted, with a loss in extension (−25°), passive flexion (90°) and internal rotation (−20°). The left knee jerk reflex was absent. Functional assessment revealed a Lequesne index of 4 and a Merle d'Aubigné score of 14.

The laboratory test results were normal. Electromyography demonstrated moderate to severe chronic, pluriradicular involvement (bilateral L3, L4, L5 and S1) compatible with lumbar spinal stenosis (confirmed by MRI). Standard radiological examination of the hip revealed slight bilateral coxofemoral osteoarthritis (predominantly in the posterior area) (Fig. 3).

An intra-articular injection of iodine-containing contrast medium evidenced an abnormally low coxofemoral joint capacity (10 ml) (Fig. 4a–c). We observed many osteoarthritis-induced osteochondromatous nodules in the joint recess, suggesting to lower polar coxarthrosis. ACH caused by osteochondromatosis was suspected.

The pain decreased after injection of an anaesthetic and a corticosteroid into the hip joint.

The patient refused surgery but accepted physiotherapy three times a week for 12 months.

Progression of the condition was characterized by a slight decrease in pain (the VAS score fell from 60 out of 100 to 50 out of 100) and a slight improvement in the coxofemoral joint amplitude (flexion: 100°; rotation: 0°). The functional scores did not change.

1.3. Case report 3

A 77-year-old woman consulted for left coxofemoral pain that had appeared one month previously. Left hip motion was limited, with 30° of flexion and no internal/external rotation. Locomotor and neurological tests gave normal results. A functional assessment revealed significant discomfort, with a Lequesne index of 15 and a Merle d'Aubigné score of 13.

The laboratory test results were normal. A standard radiological examination revealed moderate left coxofemoral osteoarthritis, predominantly in the posterior area (Fig. 5).

Given the difference between the clinical and radiological results, we performed intra-articular injection of iodine-containing contrast medium, evidencing an abnormally low coxofemoral joint capacity (9 ml) (Fig. 6). Adhesive capsulitis of the hip resulting from moderate coxofemoral osteoarthritis was suspected.

We performed a synovectomy, with removal of loose intrajoint cartilaginous fragments. Unfortunately, the improvement was only temporary and hip arthroplasty had to be performed one year later, due to disabling osteoarthritis.

2. Discussion

Adhesive capsulitis of the hip is characterized by clinically evidenced pain and restricted joint motion. Lequesne et al. propose classifying ACH into idiopathic and secondary forms:

- idiopathic ACH is uncommon but may follow on from diabetes or prolonged phenobarbital treatment;
- secondary ACH has five different aetiologies, the most common of which is synovial chondromatis. The other aetiologies are osteoid-osteoma, internal and posterior osteoarthritis, labrum glenoidal lesions and ligament lesions [7,12].

The similarities between ACH and ACS are striking.

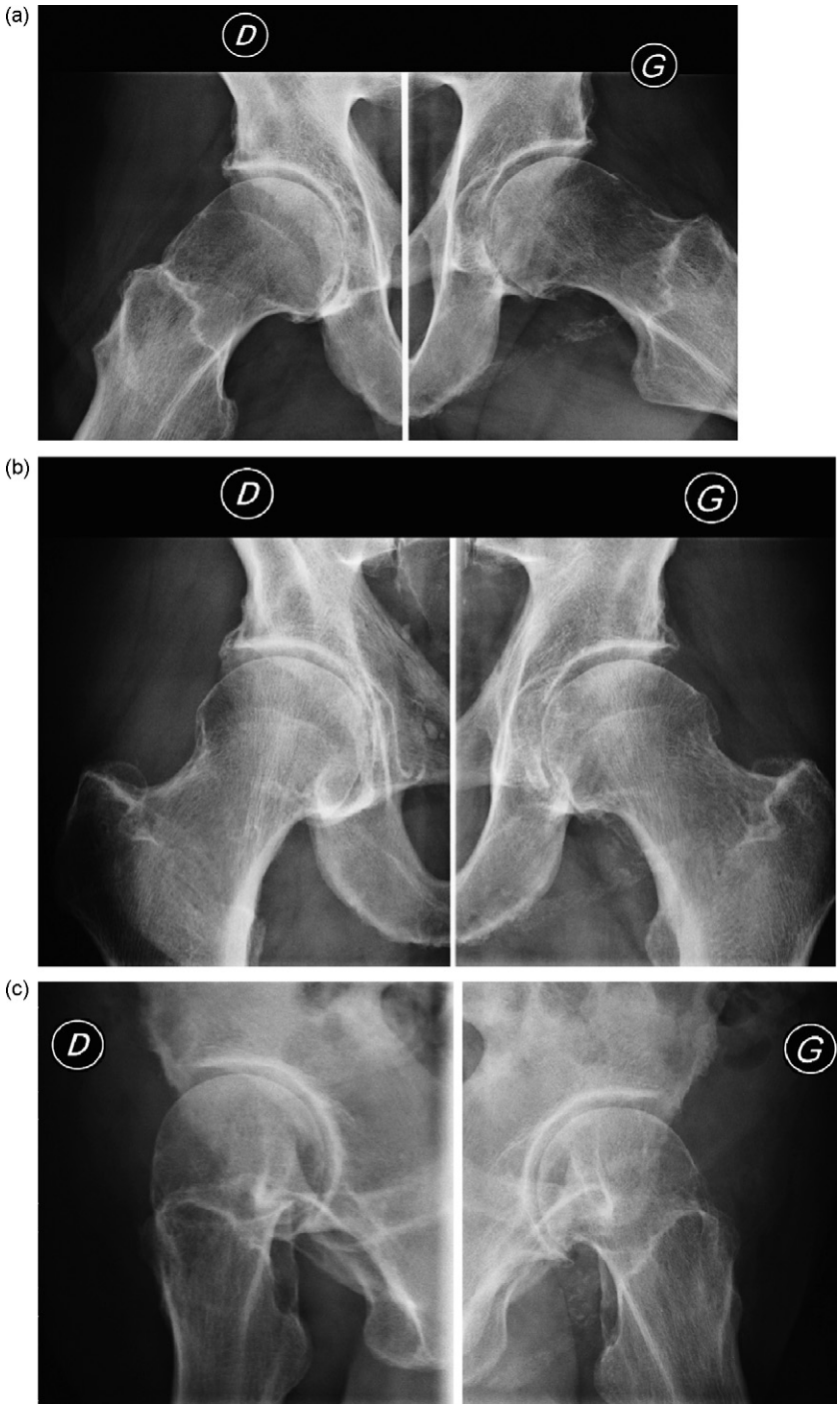


Fig. 3. a–c: anteroposterior and sagittal radiographs of the hip. Case 2.

3. Aetiology, classification and physiopathology

Adhesive capsulitis of the shoulder is a common pathology, affecting between 2% and 5% of the general population. In contrast, the incidence of ACH is unknown but is probably higher than is generally believed. The condition may preferentially affect women between the ages of 35 and 50 [3,7,12]. McGrory and Enddrizzi [9] suggested that ACH is probably often undiagnosed because it results in less deleterious functional repercussions than adhesive capsulitis of the other joints (the shoulder, for example). Griffin et al. made the same observations [5].

Both conditions can be idiopathic or secondary to medical or orthopaedic pathologies.

However, the ACS is the only one of the two that can be caused by neurological pathologies (hemiplegia, parkinsonian syndrome), cardiological pathologies (myocardial infarction) or pulmonary pathologies (pneumonia).

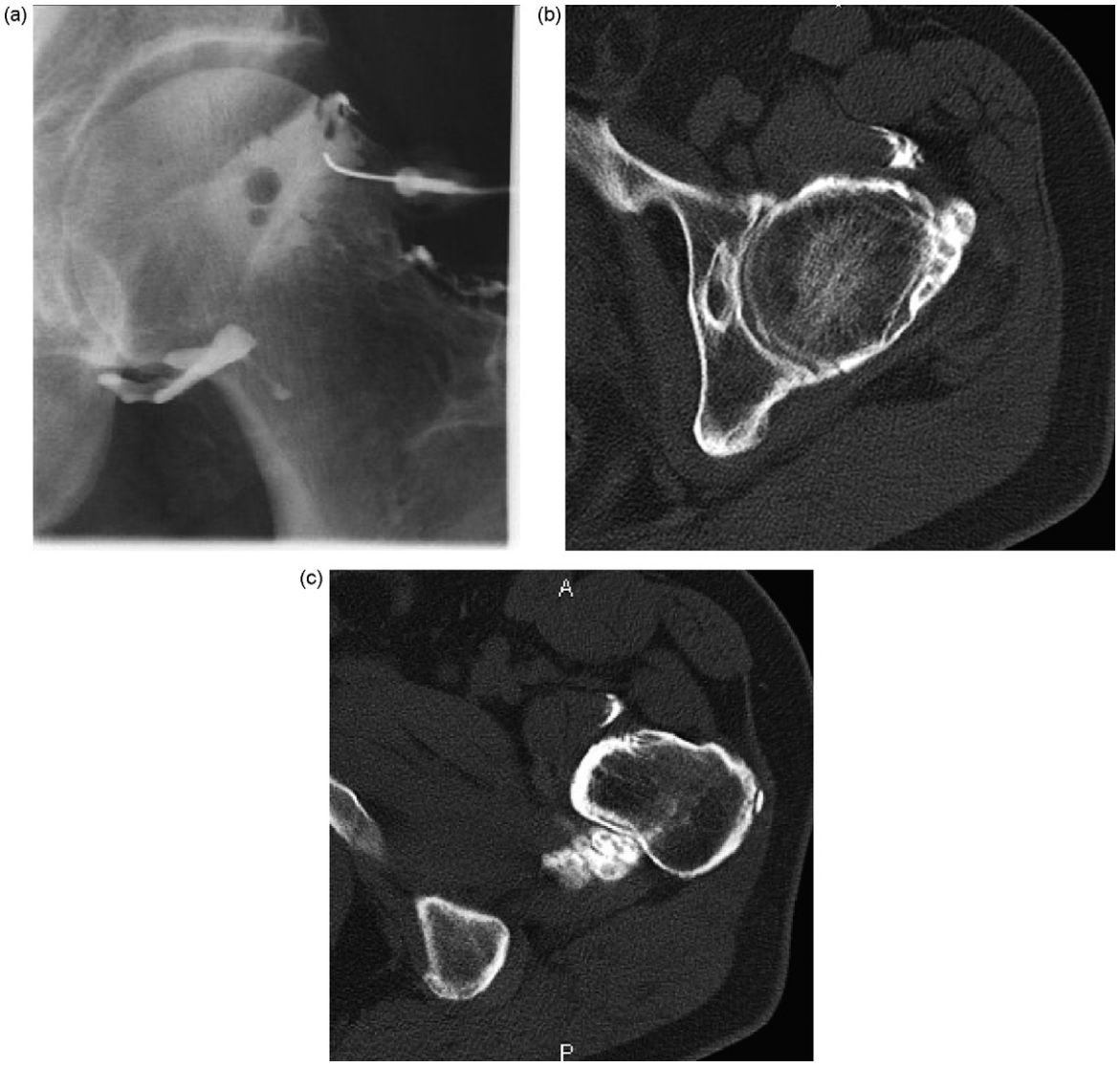


Fig. 4. a–c: hip arthrography. Case 2.

Synovial chondromatosis, however, is peculiar to ACH.

Although several potential explanations for the physiopathology of ACH have been put forward, (capsular fibrosis, abnormal nonenzymatic glyco-oxidation of proteins in diabetes, for example), the underlying cause remains unknown.

4. Clinical assessment

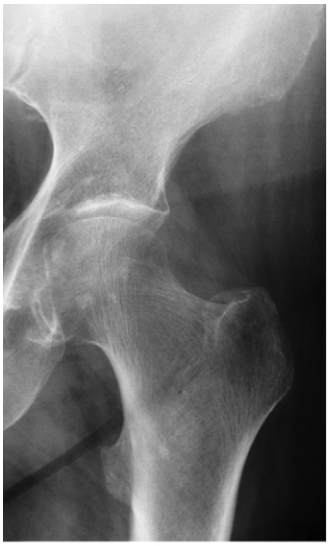
Both ACH and ACS are characterized by progressive and painful loss of both passive and active joint motion. In ACH, this limitation is all three planes (flexion-extension, internal-external rotation and abduction-adduction) [12].

5. How can we diagnose ACH?

Adhesive capsulitis of the hip can be suspected in the absence of any coxofemoral pathologies (such as osteonecrosis or severe osteoarthritis) or if there is a contrast between the most prominent symptoms and a reassuring radiological assessment.

Even though the MRI and computed tomography (CT) criteria for ACS are now well known [4,6,10,11], two groups of clinical researchers have issued different diagnostic guidelines for ACH. Lequesne et al. propose that radiological assessment with intracoxofemoral injection of iodine-containing contrast medium can reveal a decrease in joint volume (below 12 ml), since the normal range is between 12 ml and 18 ml (with an average of 15 ml).

Thomas Byrd and Jones consider that radiological assessment with intracoxofemoral injection of iodine-containing contrast medium only demonstrates the presence of ACH when the joint volume is below 5 ml. When the volume is between 5 ml and 12 ml, these latter authors recommend performing a volume comparison with the contralateral coxofemoral joint. If the difference between the two is at least 25 %, ACH can be suspected.



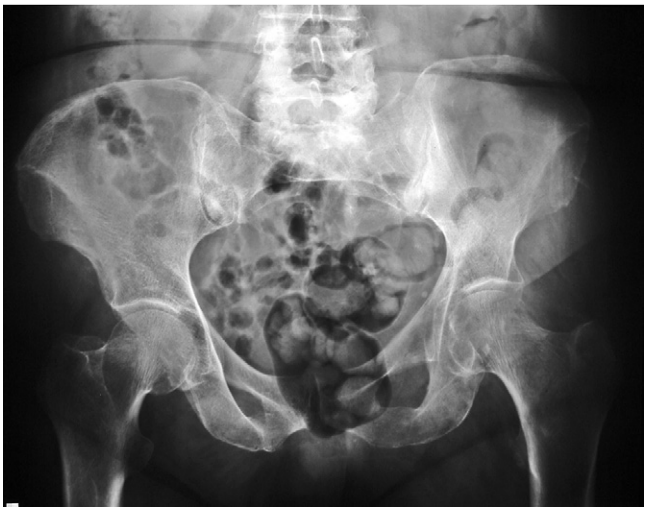


Fig. 5. Radiograph of the left hip. Case 3.

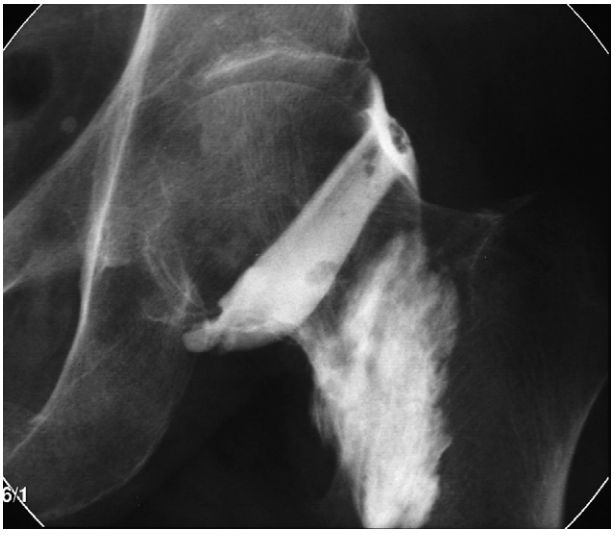


Fig. 6. Hip arthrography. Case. 3.

Thomas Byrd also suggest performing MRI of the hip in order to detect potential bone or cartilage pathologies, following by either a therapeutic hip arthroscopy (that is to treat the causal affection identified by the MRI) or a diagnostic-therapeutic hip arthroscopy (for difficult cases with normal imaging results). Arthroscopy enables confirmation of ACH associated with fibrinous and hemorrhagic fragments in the capsular recess and/or acetabular fossa (validated arthroscopic criteria for ACH) [12].

When we retrospectively reviewed the MRI results for our three patients, we did not find any of the specific signs described by the two groups of researchers mentioned above. In our medical practice, we follow Lequesne's guidelines: decreased coxofemoral joint capacity radiologically confirmed by intra-articular injection of iodine-containing contrast medium.

6. Differential diagnosis

Cases of inflammatory and tuberculous coxofemoral arthritis are easy to diagnose with standard laboratory and radiological assessments. Complex regional pain syndrome type 1 can be suspected in case of painful joint stiffness, notably with severe osteoporosis of the upper femoral epiphysis or signs of associated neurotrophic damage (oedema and vasomotor disturbances). These diagnoses were ruled out in our patients as the latter did not have osteopaenia, oedema and inflammatory signs on radiological assessment (standard radiology, CT, MRI and scintigraphy).

All three patients had mild to moderate posterior osteoarthritis. Adhesive capsulitis of the hip may explain the coxofemoral stiffness in the presence of slight coxofemoral osteoarthritis. The fact that the latter was posterior argues in favour of ACH. Nevertheless, we are unable to say whether the reduced coxofemoral joint capacity was progressing into symptomatic coxofemoral osteoarthritis. Is the restricted motion exclusively due to osteocartilaginous factors or rather ACH combined with severe coxofemoral osteoarthritis? In other words, is ACH under-diagnosed? In the absence of studies, firm conclusions cannot be drawn.

A controlled, prospective study with systematic coxofemoral radiological assessment via intra-articular injection of iodine-containing contrast medium should be performed, in order to evaluate the joint volume in mild to severe osteoarthritis. One possible consequence of such a trial might be earlier physical therapy for mild coxofemoral osteoarthritis where the symptoms are essentially due to ACH.

7. Treatment

Idiopathic ACH usually responds favourably to maintenance treatment. This treatment consists in physiotherapy and radiologically-controlled intra-articular corticosteroid injections performed once or twice a month. According to Lequesne's guidelines, the total number of injections varies between five and 12. Even though nonsteroidal anti-inflammatories and corticosteroid intra-articular injections can relieve pain in cases of idiopathic ACH, physiotherapy is the only way to recover joint amplitude. There is a lack of medical literature

on the subject. Long-term physical therapy may go on for between five and 24 months.

The broad outline of the physiotherapy approach is the same as for the adhesive capsulitis of the shoulder [1]:

- progressive eccentric manual stretching, depending on the pain threshold (to get the best possible muscle extension) and eccentric muscular strengthening (with or without transcutaneous excitomotor stimulation);
- self-physiotherapy for maintaining good mobility;
- proprioceptive exercises for better postural control and avoiding joint over-use;
- hydrotherapy for better movement awareness.

The treatment is prescribed once a day for the first four weeks and then three times a week for at least two months. On average, follow-up lasts for nine months after the onset of symptoms.

Secondary ACH requires immediate capsulotomy or synovectomy to treat the underlying affection.

Refractory idiopathic ACH can also benefit from arthroscopy. According to Thomas Byrd and Jones, the average time between the initial appearance of symptoms and surgery is 12 months (range: four to 21 months), whereas the average time between the beginning of physiotherapy and surgery is 7.4 months (range: two to 18 months) [12]. Thomas Byrd and Jones also recommend a combination of surgery and manipulation under general anaesthesia.

In fact, Mont et al. have reported a case where arthroscopy did not succeed because it was not preceded by manipulation. A lack of manipulation may lead to more severe capsular distension and would thus increase the risk of iatrogenic damage or surgical failure.

If arthroscopy is required (for secondary ACH or refractory idiopathic ACH), the above-mentioned physiotherapeutic approach remains valid and should be started on the second day postsurgery. The use of crutches is recommended for five to seven days, in order to help restore normal gait.

Additional research is needed to better understand the physiopathology of ACH and interpret radiological assessments in this context. Better knowledge would enable us to diagnosis ACH more rapidly and thus initiate appropriate treatment sooner.

8. Conclusion

Adhesive capsulitis of the hip is probably more common that the limited medical literature leads us to believe. It preferentially affects middle-aged women. Clinical assessment is similar to that used for ACS. The condition may be associated with other intra-articular pathologies. The basic treatment consists of a combination of coxofemoral joint sustained-release corticosteroid injection and physiotherapy. A combination of manipulation and hip arthroscopy is indicated in cases of refractory idiopathic ACH and secondary ACH.

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