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| Abstract | <p>Groin pain is a common condition in athletes and accounts for up to 6 % of all athletes' injuries. It affects athletes involved in running, jumping and kicking, and it is particularly common in professional soccer players. Groin pain is a serious condition because it can impair athletic performance and cause young athletes to change the level of sport and retire early from sport activity. The diagnosis of groin pain is difficult because of the anatomical complexity of the groin area, the large number of potential sources of groin pain and the coexisting pathologies which can affect the patient. Management is a challenge for physicians because treatment strategies are still debated, because of the little availability of evidence-based protocols and because many therapies are based on poor evidence clinical studies (level IV studies). In this chapter, the most common causes of groin pain that affect athletes and their management are discussed.</p> | |
| Keywords (separated by " - ") | Adductor tendinopathy - Femoroacetabular impingement - Groin pain - Groin pain disruption - Hip arthroscopy - Osteitis pubis | |

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29.1 Introduction

Groin pain is well known among both athletes and physicians. Groin injuries account for about 6 % of all athletes' injuries, and the incidence increases up to 13 % in specific sports such as soccer [13]. The incidence ranges from 12 % to 16 % of all injuries per season in a recent prospective study of hip and groin injuries in professional soccer players, with a mean absence from competitions of 15 days [61]. This is probably due to typical soccer movements like jumps, dribbling and rapid twisting which cause high stress to the pubic symphysis and muscular imbalance. Kicking and running on uneven surfaces, male gender and preseasonal training are considered risk factors for developing groin pain [26].

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Diagnosis and management are major challenges for physicians because the aetiopathogenesis is not clear yet. Diagnosis is difficult because of the anatomical complexity of the groin area, the biomechanics of the pubic symphysis region and the large number of potential sources of groin pain (Table 29.1). A recent review reported that 30–90 % of patients are affected by different co-existing groin pathologies [56]. This condition justifies the term 'groin pain syndrome' (GPS). The nomenclature is also confusing. Many athletes with a diagnosis of 'sports hernia' or 'athletic pubalgia' have a spectrum of related pathologic conditions resulting from musculotendinous injuries and subsequent instability of the pubic symphysis without any finding of inguinal hernia at physical examination. For this reason, the term 'groin pain disruption' introduced by Gilmore is becoming more popular [19].

Management of groin pain is difficult, and patients usually undergo prolonged rest and many different treatments. The management of groin pain is multidisciplinary and consists of rehabilitation, physical therapies or surgery for patients who do not respond to conservative treatments [55].

29.2 Groin Anatomy

Knowledge of groin anatomy is of great importance in understanding the causes of groin pain. The groin region consists of ligaments, tendons, muscles and fascia which all insert to the pubic

t1.1 **Table 29.1** Differential diagnosis of groin pain

| t1.2 | Intra-articular pathologies | Extra-articular pathologies | Non-musculoskeletal disorders |
|-------|---------------------------------------------|--------------------------------------------------------|------------------------------------|
| t1.3 | Femoroacetabular impingement (FAI) syndrome | Insertional adductor and rectus abdominis tendinopathy | <i>Intra-abdominal pathologies</i> |
| t1.4 | Acetabular labral tears | Groin pain disruption | Appendicitis |
| t1.5 | Chondral lesions | Osteitis pubis | Diverticulitis/diverticulosis |
| t1.6 | Femoral neck stress fractures | Greater trochanter pain syndrome | Lymphadenitis |
| t1.7 | Osteoarthritis | Lumbar radiculopathy | Inflammatory bowel disease |
| t1.8 | Transitory synovitis | Pubic ramus stress fracture | Inguinal hernia |
| t1.9 | Osteonecrosis of the femoral head | Apophyseal avulsion fractures | |
| t1.10 | Osteochondritis dissecans | Sacroiliac joint disorders | <i>Genitourinary</i> |
| t1.11 | Legg-Calvè-Perthes disease | Nerve entrapment | Adnexal torsion |
| t1.12 | Epiphysiolysis of the femoral head | Snapping hip syndrome | Nephrolithiasis |
| t1.13 | Septic arthritis | | Orchitis |
| t1.14 | Oncologic process | | Ovarian cysts |
| t1.15 | | | Pelvic inflammatory disease |
| t1.16 | | | Urinary tract infections |
| t1.17 | | | Endometriosis |
| t1.18 | | | Prostatitis |
| t1.19 | | | Testicular cancer |
| t1.20 | | | |

50 bone and symphysis. The inguinal region consists of
 51 the inferior part of large flat muscular sheets
 52 (obliquus externus, internus and transversus abdominis),
 53 the rectus abdominis, the pyramidalis, the
 54 inguinal canal, the symphysis pubis and the femoral
 55 triangle. Central to the groin area is the inguinal
 56 ligament. The inguinal ligament is an important
 57 connective tissue structure which supports soft tissues
 58 in the groin as well as the external abdominal
 59 oblique muscle. It arises from the inferior aponeurosis
 60 of the external abdominal oblique and runs
 61 obliquely across the pelvis. On its superior and lateral
 62 end, it connects to the anterior iliac spine of the
 63 ilium and extends to the pubic tubercle of the pubis
 64 bone on its inferior and medial end. The inguinal
 65 ligament supports the muscles that run inferior to its
 66 fibres, including the iliopsoas and pectineus muscles
 67 of the hip. It also supports the nerves and blood vessels
 68 of the leg as they pass through the groin, including
 69 the femoral artery, femoral vein and femoral
 70 nerve. The support provided by the inguinal ligament
 71 is important to maintain the flexibility of the hip
 72 region while allowing vital blood and nerve supply
 73 to the leg. A small opening in the muscles and
 74 connective tissues of the abdomen, the superficial
 75 inguinal ring, is located just superior to the inguinal
 76 ligament. This opening is part of the inguinal canal

and permits the spermatic cord in males and the
 round ligament of the uterus in females to exit the
 abdominopelvic cavity and pass through the external
 tissues of the pelvis. The pubic symphysis is an
 amphiarthrodial joint with limited mobility (it can
 be moved roughly 2 mm with 3° of rotation) but
 with good capacity of load absorption, thanks to the
 presence of hyaline and fibrous cartilage and connective
 tissue on its surface. The abdominal and paravertebral
 muscles act synergistically to stabilize the symphysis
 pubis during movements, particularly during static or
 dynamic single-leg stance [4]. The adductor muscles
 act as antagonists and exert opposing traction and
 rotation on the pubic symphysis. The femoral triangle
 is located in the upper inner thigh, and several
 structures pass through it: the femoral nerve, the
 femoral vessels and the sartorius, the iliopsoas,
 the pectineus and the adductor longus muscles.
 The adductor muscles also comprise the adductor
 brevis, the adductor magnus and the gracilis.
 Many peripheral nerves cross or innervate the
 anatomic structures of the inguinal region. These
 include the ilioinguinal nerve (T8–L1); the obturator
 nerve (L2–L4); the medial and intermediate cutaneous
 nerve of the thigh (L2–L3), with sensory function;
 and the femoral nerve (L2–L4) [4].

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29.3 Insertional Adductors and Rectus Abdominis Tendinopathy

Insertional tendinopathy of the adductors and rectus abdominis is a frequent cause of groin pain in athletes. It involves the adductor muscles and/or the rectoabdominal muscles [57]. The incidence of insertional adductors tendinopathy is about 2.5–3 % in athletes and is more frequent in soccer, basketball, hockey and rugby players and long-distance runners (Paajanen 2011). More than 70 % of patients are males. Even if insertional adductor tendinopathy can develop independently, in most of the cases, it occurs in association with osteitis pubis. Hiti et al. suggested that groin pain and pubic osteitis are the most common causes of chronic groin pain in athletes [21].

The aetiopathogenesis is multifactorial. It is related to functional overuse and repeated micro-traumas caused by torsion and traction of abdominal and adductor tendon insertions. The overloading of the pubic symphysis and insertional tendons could be induced by the strength imbalance between the hypertonic adductor muscle and hypotonic large flat muscular sheets of the abdomen [42]. Other authors suggest that this condition could also be induced by the hypertonia of the quadriceps femoris muscle [57]. Some intrinsic and extrinsic factors may predispose athletes to develop insertional adductor tendinopathy. The muscular imbalance is the main intrinsic factor, while reduced flexibility of the posterior chain muscles and/or iliopsoas-lumbar hyperlordosis; sacroiliac, sacrolumbar and hip arthropathy; and marked asymmetry and/or dissymmetry of lower limbs are other risk factors. Incorrect athletic training, unsuitable footwear and unfavourable conditions of the playground are considered as extrinsic factors [57].

29.3.1 Clinical Examination and Diagnosis

The main symptom is groin or lower abdominal pain, with radiation to the medial aspect of the

thigh, abdomen and, in some cases, perianal area. The symptoms are unilateral at the beginning and occur after sport; but this condition is progressive in nature, limiting or stopping the sporting activities. In advanced stages, the pathology could progress bilaterally and could affect social life and everyday activities such as climbing stairs and getting up from a bed or a chair. Sometimes sneezing, coughing, defecating and sexual activity can reproduce the symptoms [48].

Diagnosis is based on clinical examination and supported by imaging. Painful points such as tendon insertions of adductor, rectoabdominal and iliopsoas muscles, the pubic symphysis and iliac spines are evaluated. Pain can also be reproduced with adduction or contraction of the abdominal, the iliopsoas, the rectus femoris and the adductor muscles against resistance and with passive stretching of the adductors and iliopsoas muscle. The mobility of the hips on all planes should be assessed. Specific tests show the shortening of the anterior chain (test of Thomas), the posterior chain (hamstring muscles) and sacroiliac joint (test of Patrick and test of Gaenslen). Finally, a peripheral neurological examination should be conducted.

Plain radiographs, ultrasound scan and MRI are useful to confirm the diagnosis. Plain radiographs are useful to exclude different causes of groin pain, including femoroacetabular impingement (FAI), hip osteoarthritis or fractures. Flamingo stress views are used to assess pelvic stability, which is measured as the amount of vertical displacement observed at the symphysis [18]. Flamingo stress views are obtained with the patient bearing weight alternately on each leg. If a displacement greater than 2 mm at the symphysis pubis is observed, a macro-instability of the symphysis pubis can be diagnosed. Other indirect signs of pelvic instability can be observed at plain radiography, such as spurs of the cortical bone, subchondral cysts and associated widening of the sacroiliac joint (Fig. 29.1). Ultrasound evaluation allows to assess musculotendinous structures, soft tissues and insertional area of tendons and ligaments. MRI usually shows bone marrow oedema, insertional tendinopathy of the adductors and rectus abdominis [45] and



Fig. 29.1 Flamingo stress views show pelvic instability with displacement at the symphysis pubis greater than 2 mm. Note the subchondral cyst (*black arrow*) which is an indirect sign of degenerative changes

196 symphysis capsular disruption; central disc
 197 protrusion may also be present, in particular in
 198 soccer players [7].

199 **29.3.2 Treatment**

200 The management is multidisciplinary. Treatment
 201 includes rest and focused rehabilitation.
 202 Rehabilitation phases can be divided in acute,
 203 subacute and return to sport [57]. The first goal
 204 during acute phase is pain reduction. For this pur-
 205 pose, pharmacological, instrumental, physical
 206 and manual therapies are recommended. Laser
 207 therapy and extracorporeal shock wave therapy
 208 are useful to relieve pain. Rehabilitation measures
 209 consist of postural balance techniques through
 210 global and site-specific stretching, the use of
 211 mechanical and proprioceptive orthotic insoles
 212 and global postural re-education [60]. In the early
 213 stages, physical therapy involves isometric
 214 strengthening of the abdominal muscles and
 215 adductor muscles in the gym or in a therapeutic
 216 swimming pool. In the subacute phase, muscle
 217 strengthening is increased by the introduction of
 218 concentric and eccentric exercises and by cardio-
 219 vascular reconditioning. Core stability exercises
 220 are useful in this phase [39]. Finally, running is
 221 gradually introduced, at first on a treadmill. The
 222 return-to-sport phase of rehabilitation consists of
 223 aerobic running with increasing speed.

224 If conservative measures have failed for at least 3
 225 months, surgical intervention may be necessary

[57]. Adductor longus tenotomy is commonly 226
 performed in order to reduce the stress of the hyper- 227
 tonic adductor muscle on the pubic symphysis and 228
 to reduce the muscular imbalance of the adductor- 229
 abdomino. Many authors reported good results and 230
 high rate of return to sport after adductor longus 231
 tenotomy. In a large series of professional soccer 232
 players, Mei-Dan et al. reported good or excellent 233
 results in 80 % of patients with a mean return to 234
 sport in 11 weeks (range, 4–36 weeks) [37]. 235
 Robertson et al. reported improvements in 91 % of 236
 patients (99/109) in particular in patients with the 237
 worst preoperative symptoms [49]. More recently 238
 good results have been reported with bilateral mini- 239
 invasive adductor tenotomy for athletes suffering 240
 from unilateral adductor longus tendinopathy refrac- 241
 tory to nonoperative management [35]. At the time 242
 of the latest follow-up, 76 % of patients returned to 243
 their pre-injury level of sport or higher levels, with a 244
 median time to return to sport of 18 weeks. However, 245
 3 of 29 patients ceased to participate in sport. 246
 Concern following adductor tenotomy is the poten- 247
 tial for loss of hip adductor strength, although this 248
 does not seem to influence participation in high- 249
 level sports (Akermark et al. 1992; [37]). 250

251 **29.4 Femoroacetabular**
 252 **Impingement Syndrome**

Femoroacetabular impingement (FAI) syn- 253
 drome is a common cause of pain and discomfort 254
 in young active non-dysplastic patients [16]. 255



Fig. 29.2 FAI of a 31-year-old soccer player. Large chondral lesions are evident at plain radiographs

256 Two types of impingement have been described,
 257 namely, cam impingement and pincer impingement.
 258 Cam impingement is caused by an abnormal
 259 morphology of the femoral head with increasing
 260 radius into the acetabulum during forceful
 261 motion, especially flexion. Pincer impingement
 262 is the result of an altered anatomy of the acetabulum,
 263 as coxa profunda or abnormal retroversion or
 264 anteversion of the acetabular rim, which
 265 cause pathological contact between the acetabular
 266 rim and the femoral head-neck junction [16].
 267 Dynamic pincer impingement can occur in normal
 268 hips if the required range of movement is large
 269 or translated, as in dancers, gymnasts and
 270 hockey players [5]. Cam-type impingement is
 271 more common in young and athletic males,
 272 while the pincer-type impingement is more
 273 common in middle-aged females [16]. However,
 274 a minority of patients present pure FAI (14 %);
 275 most patients have a combination of both forms
 276 (86 %), the so-called mixed pincer and cam
 277 impingement.

278 FAI alters the biomechanics of the hip result-
 279 ing in painful and limited range of motion,
 280 mostly in flexion and internal rotation. In cam
 281 impingement, the nonspherical portion of the
 282 femoral head adducting against the acetabular
 283 rim leads to deep chondral lesions and extensive
 284 labral tears. In pincer-type impingement, the
 285 first structure to fail is the acetabular labrum,
 286 leading to ossification of the rim and additional

287 deepening of the acetabulum and worsening of
 288 the coverage. In pincer impingement, chondral
 289 lesions are smaller than in cam type and often
 290 limited to a small rim area [16]. However, FAI is
 291 not symptomatic in all cases. FAI in healthy
 292 young adults may be asymptomatic up to 35 %
 293 of cases [30].

29.4.1 Diagnosis 294

295 Groin pain and limited hip motion are the clinical
 296 key symptoms and signs of FAI. A decreased
 297 ROM, in particular of internal rotation, is the
 298 most common sign in case of FAI. People with
 299 asymptomatic FAI also demonstrate reduced
 300 ROM compared with people with no evidence of
 301 FAI [11]. Many specific clinical tests have been
 302 developed to support clinical diagnosis of FAI. A
 303 recent systematic review showed that hip-specific
 304 tests have high sensitivity but poor specificity and
 305 that only the FADIR test (flexion, adduction and
 306 internal rotation test) and the flexion-internal
 307 rotation test are valuable screening tests for FAI
 308 and acetabular labral tears [47].

309 Standard anteroposterior pelvic and lateral
 310 cross-table radiographs supported the first clinical
 311 suspicion of FAI disease (Fig. 29.2). MRI
 312 arthrography with gadolinium is important for
 313 assessing the status and extent of labral and
 314 cartilage lesions (Fig. 29.3) [22].

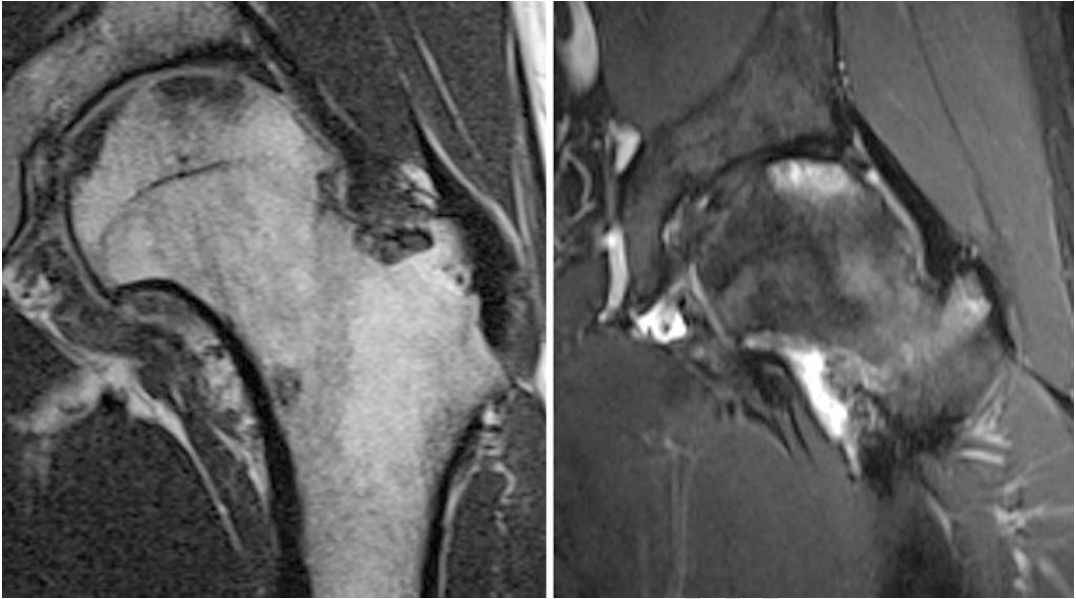


Fig. 29.3 T1- and T2-weighted MRI with gadolinium of the same patient showing big osteochondral lesions and labral tear

29.4.2 Treatment

315 The management of FAI is still controversial.
 316 The appropriate timing for surgery is also
 317 debated, even though recent studies showed that
 318 delayed surgery may lead to progression of the
 319 disease to the point where joint preservation is no
 320 longer indicated [34]. Some authors suggest that
 321 patients should undergo surgery within 6 months
 322 of symptom onset [3].

323 Surgery is indicated to relieve symptoms, to
 324 treat concomitant degenerative joint disease and
 325 to encourage return to sport. According to present
 326 data, arthroscopy, open surgery and arthroscopic
 327 followed by mini-open surgery are comparable
 328 for functional results, biomechanics and return to
 329 sport [46]. Hip dislocation and open osteochon-
 330 droplasty were considered the gold standard treat-
 331 ment, with good to excellent results in 70–80 % of
 332 patents [16, 34, 41]. These authors suggest that
 333 complex bony abnormalities including extra-
 334 articular impingement, major deformities and
 335 global pincer FAI are better treated by open tech-
 336 niques, which also allow femoral osteotomies or
 337 acetabular reorientations when they seem appro-
 338 priate. Heterotopic ossification is the most fre-
 339 quent complication after open surgery [46].
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Recent studies reported good results after less
 341 invasive arthroscopic treatment in terms of time
 342 to recovery and return-to-sport activity, allowing
 343 93 % of patients to return to their pre-injury sport
 344 [46]. In a study on more than 600 patients, the
 345 quality of life scores improved in 76.6 % of cases
 346 after hip arthroscopy at 3 years follow-up [36].
 347 However, arthroscopy is more technically
 348 demanding. A need for a revision hip arthroscopy
 349 has been reported for persistent symptoms, fur-
 350 ther debridement, lysis of adhesions and advanced
 351 osteoarthritis. A recent study showed that, in
 352 patients who undergo revision hip arthroscopy
 353 for persistence of groin pain, findings of FAI are
 354 still evident at imaging and revision surgery [20].
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356 Finally, it is difficult to state whether surgery
 357 modifies the evolution of osteoarthritis in young
 358 patients and contributes to prevent the development
 359 of osteoarthritis. Open dislocation and debridement
 360 show a higher rate of conversion to total hip arthro-
 361 plasty, particularly in patients with pre-existing
 362 severe osteoarthritis and cartilage lesions [46].
 363 Concomitant cartilage lesions and degenerative
 364 changes result in lower clinical and functional
 365 scores, short-term pain relief, no evidence of long-
 366 term satisfactory outcomes and higher rate of con-
 367 version to total hip arthroplasty [46].

t2.1 **Table 29.2** Stages of osteitis pubis according to Rodriguez et al.

| t2.2 | Side of pain | Side of pain | Characteristics of pain |
|-------|--------------|----------------------|---------------------------------------------------------------------------------------------------------------|
| t2.3 | Stage 1 | Unilateral symptoms | Inguinal, with radiation to adductors. Usually involve the dominant leg |
| t2.4 | | | Mechanical, settles after rest, returns after training |
| t2.5 | Stage 2 | Bilateral symptoms | Inguinal pain involving the adductor muscles |
| t2.6 | | | Increases after training |
| t2.7 | Stage 3 | Bilateral symptoms | Groin, adductor region, suprapubic, abdominal muscles |
| t2.8 | | | During training, kicking, sprinting, turning. Cannot achieve training goals, forced to withdraw |
| t2.9 | Stage 4 | Generalized symptoms | Generalized, pelvic girdle and radiation to lumbar region |
| t2.10 | | | Walking, getting up, straining at stool, simple activities of daily living. Pain with defecation and sneezing |
| t2.11 | | | |
| t2.12 | | | |
| t2.13 | | | |

368 **29.5 Osteitis Pubis**

369 Osteitis pubis is a painful degenerative condi-
 370 tion of the pubic symphysis, surrounding soft
 371 tissues and tendons. It was first described by
 372 Beer in 1924 [53], and it is currently considered
 373 as one of the most debilitating syndromes to
 374 affect athletes [50]. The incidence in the general
 375 athletic population has been reported as
 376 0.5–7 %. It mostly affects basketball players,
 377 distance runners and athletes participating in
 378 kicking sports, such as soccer or football, where
 379 the incidence is up to 13 % in patients with
 380 groin pain [9]. It affects more often males, while
 381 it has been reported in females in about 5 % of
 382 cases [58].

383 Even though the pathogenesis is still debated,
 384 currently the new concepts of ‘sports-related
 385 chronic groin injury’ and ‘groin disruption
 386 injury’ describe a condition of chronic groin pain
 387 associated with pubic instability [58]. Muscle
 388 imbalance between the abdominal and hip adduc-
 389 tor muscles is considered the most important
 390 pathogenetic factor in the development of this
 391 condition [14]. The abdominal muscles act syner-
 392 gistically with the posterior paravertebral mus-
 393 cles to stabilize the symphysis, allowing
 394 single-leg stance while maintaining balance and
 395 contributing to the power and precision of the
 396 kicking leg. The adductors are antagonists to the
 397 abdominal muscles. Imbalances between abdom-
 398 inal and adductor muscle groups disrupt the equi-
 399 librium of forces around the symphysis pubis,
 400 predisposing the athlete to a subacute periostitis
 401 caused by chronic microtrauma.

29.5.1 Clinical Presentation and Diagnosis

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Athletes with osteitis pubis commonly present
 with anterior and medial groin pain and, in some
 cases, may have pain centred directly over the
 pubic symphysis. Pain may also be felt in the
 adductor region, lower abdominal muscles, peri-
 neal region, inguinal region or scrotum. The pain
 is usually aggravated with running, cutting, hip
 adduction and flexion against resistance and
 loading of the rectus abdominis [6].

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The diagnosis is clinical, based on clinical his-
 tory, physical examination and functional assess-
 ment. Physical examination findings include
 tenderness to palpation of the pubic symphysis
 and pain with resisted strength testing of the
 adductor and lower abdominal muscle groups.

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Imaging studies include plain radiography,
 MRI and CT scan, and they are advocated to
 exclude concomitant pathologies. MRI can show
 bone marrow oedema, and MRI findings were
 graded according to subchondral bone oedema,
 fluid in the pubic symphysis and periarticular
 oedema [29]. Osteitis pubis has been classified
 according to the severity of symptoms by
 Rodriguez et al. (Table 29.2) [50].

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29.5.2 Treatment

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Management is conservative first, while surgery
 is indicated in unresponsive patients. Many stud-
 ies proposed different treatment options depend-
 ing on the severity of symptoms. However, a

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433 recent systematic review showed that no level 1
434 studies are reported in literature. Current treat-
435 ment of osteitis pubis is based on level 4 evidence
436 studies, making it difficult to compare the effi-
437 cacy of different treatment protocols [9].

438 A progressive rehabilitation programme pro-
439 duces good results [24]. Patients are moved
440 through the protocol stages after they are able to
441 perform exercises without pain and have achieved
442 adequate levels of movement and core stability
443 grading. The first stage is to focus on pain control
444 and improve lumbo-pelvic stability. Gentle pro-
445 longed stretching, except for the adductors and
446 ischiopubic muscles, is started. Cycling on an
447 exercise bike is introduced as cardiovascular
448 training. In the second stage, Swiss balls and
449 other aids are indicated to perform resistance and
450 strength contraction exercises of the pelvic floor,
451 transversus abdominis and multifidus muscles.
452 Gluteal strengthening is started. In the third and
453 final stage, eccentric work on the sliding board is
454 started. Running time is gradually increased, and
455 changes of pace and direction are introduced. To
456 reproduce the sport requirements, athletes start
457 training on the field performing exercises mim-
458 icking their sport. Kicking is allowed only at the
459 end of this stage. Eccentric abdominal wall
460 strengthening exercises are started. Good results
461 have been reported with this progressive rehabili-
462 tation programme, and most of the athletes diag-
463 nosed with stages III and IV returned to sport
464 within 3 months (10–13 weeks).

465 Surgery is indicated in 5–10 % of cases which
466 do not respond to conservative treatment [62].
467 Different surgical techniques have been
468 described, such as curettage of the pubic sym-
469 physis, polypropylene mesh placement into the
470 preperitoneal retropubic space and pubic sym-
471 physis stabilization. Satisfying results have been
472 reported in a systematic review with these tech-
473 niques (72 % of return to sport for curettage of
474 the pubic symphysis, 92 % for polypropylene
475 mesh placement into the preperitoneal retropubic
476 space and 100 % for pubic symphysis stabiliza-
477 tion) [9]. However, it is difficult to state which is
478 the best treatment strategy for osteitis pubis
479 because of lack of level 1 randomized controlled
480 trials. Osteitis pubis has a negative impact on the

481 career of an athlete, who may be obliged to stop
482 their sporting activities. Prevention programmes
483 based on specific sports-related demands should
484 be tailored to the needs of each individual athlete.
485 A correct diagnosis and an early treatment are
486 fundamental for the management because
487 patients diagnosed earlier experience fewer
488 symptoms and faster return to play [64].

29.6 Sportsman Hernia/Athletic Pubalgia

489 Sportsman hernia is a syndrome characterized by
490 chronic groin pain in athletes that is associated
491 with a small direct inguinal hernia [32]. The term
492 athletic pubalgia is currently used to describe the
493 disruption and/or separation of the more medial
494 common aponeurosis from the pubis, usually with
495 insertional tendinopathy of the adductors and rec-
496 tus abdominis muscles. In advanced phases, it can
497 be associated with osteitis pubis and FAI. Currently,
498 there is little consensus concerning this condition
499 about aetiopathogenesis and even the nomencla-
500 ture. While many authors distinguish between the
501 terms 'sports hernia' and 'athletic pubalgia', others
502 consider them different aspects of the same
503 pathology considering the close relationship
504 between structures implicated in the development
505 of this condition [12, 31]. Different terms have
506 also been used to describe this condition, includ-
507 ing sports hernia, athletic pubalgia, Gilmore's
508 groin [19], footballers' groin injury complex,
509 pubic inguinal pain syndrome (PIPS) and syn-
510 drome of muscle imbalance of the groin [40]. As
511 recent studies showed that this pathology rarely
512 arises as a single condition but multiple coexisting
513 pathologies are often present, such as posterior
514 inguinal canal wall deficiency and intra- and extra-
515 articular pathologies, the term 'groin pain disrup-
516 tion' (GPD) has become more popular [17].

517 GPD is more frequent in males, but an increas-
518 ing number of female patients are being diag-
519 nosed [38]. The aetiology is debatable. Currently,
520 many authors believe that the underlying aetiolo-
521 gy is muscular imbalance and pelvic instability
522 [31, 40, 38], although isolated traumatic tears of
523 the conjoint tendon are occasionally diagnosed.
524
525

526 **29.6.1 Clinical Presentation**
527 **and Diagnosis**

528 Athletes typically complain of gradually increas-
529 ing activity-related lower abdominal and proximal
530 adductor-related pain. The pain is typically
531 located over the lower lateral edge of the rectus
532 abdominis muscle and may radiate towards the
533 testis, suprapubic region or adductor longus ori-
534 gin. The onset is usually insidious, but in some
535 cases it can involve an initial sudden ‘tearing’
536 sensation. GPD pain is often aggravated by sud-
537 den acceleration, twisting and turning, cutting
538 and kicking, sit-ups, coughing or sneezing [2].

539 Diagnosis is dependent upon concordance
540 between the patient history, physical examination
541 and imaging investigation. Palpation for a posi-
542 tive inguinal cough impulse is usually either neg-
543 ative or equivocal. Valsalva manoeuvres such as
544 coughing and sneezing can occasionally repro-
545 duce symptoms. Fifty percent of the hernias
546 became more apparent with Valsalva manoeuvre,
547 and imaging obtained during Valsalva manoeu-
548 vre aids in the detection and characterization of
549 suspected abdominal wall hernias [23].

550 **29.6.2 Management**

551 The first approach to GPD is traditionally nonsurgi-
552 cal. However, there are issues unique to the athlete
553 regarding timing, sports seasons and level of athlete.
554 Physical therapy is focused on core stabilization,
555 postural retraining and normalization of the
556 dynamic relationship of the hip and pelvic muscles
557 [31]. After a period of rest, a gradual pain-free pro-
558 gression to sports may be possible. However, there
559 are very little data about the effectiveness of nonsur-
560 gical treatment. Physical therapy and laparoscopic
561 surgical repair have been compared in a recent RCT
562 (Paajanen et al. 2011). The authors found that only
563 50 % returned to sport in the nonsurgical group at
564 1-year follow-up. Surgery is indicated after 3–6
565 months of failed conservative treatments and when
566 the athlete is limited in season and unable to partici-
567 pate. A number of different surgical techniques
568 have been described, including repair of the external
569 oblique, transversus abdominis and transversalis

fascia, repairs with mesh reinforcement, laparo- 570
scopic repairs, mini-open repairs and broad pelvic 571
floor repairs with or without adductor releases and 572
neurectomies [31]. Surgical repair of the sportsman 573
hernia is associated with good functional outcomes, 574
and 80–100 % return-to-sport rates have been 575
reported ([28]; Paajanen et al. 2011). After surgery, 576
a 3-month programme of post-operative physiother- 577
apy is indicated to maintain pelvic stability and 578
restore function. 579

29.7 Greater Trochanter Pain 580
Syndrome 581

Lateral hip pain is a debilitating condition charac- 582
terized by pain located at or around the greater 583
trochanter. This is the site of confluence of three 584
bursae, the hip abductor-lateral thigh muscles and 585
the iliotibial band (ITB). Described for years as 586
trochanteric bursitis, advanced imaging and surgi- 587
cal findings evidenced disorders involving partial 588
tear or avulsion of the anterior aspect of the glu- 589
teus medius and minimus tendons, external snap- 590
ping hip and insertional tendinopathies with no 591
real bursal involvement [10]. For these reasons, 592
the term ‘greater trochanteric pain syndrome’ 593
(GTPS) is now used to better define this clinical 594
condition [54]. GTPS is more frequent in women 595
(F-M=4:1) aged 40–60 years and affects from 596
10 % to 25 % of the general population and up to 597
35 % in patients who have leg length discrepan- 598
cies and low back pain [63]. Abnormal force vec- 599
tors acting across the hip, leading to abnormal hip 600
biomechanics, are predisposing factors and also 601
age, gender, ipsilateral ITB pain, knee osteoarthri- 602
tis, obesity, low back pain and specific sporting 603
activities. The higher prevalence in women could 604
be related to the configuration of their pelvis. 605
Though common in sedentary patients, runners 606
are also particularly predisposed [2]. 607

29.7.1 Clinical Presentation 608
and Diagnosis 609

Diagnosis of GTPS is clinical. Patients usually 610
report pain anterior or posterior to the greater 611

612 trochanter from several months. Local tenderness
613 over the greater trochanteric area can be noted at
614 palpation and positive single-leg stance and
615 resisted external rotation tests [33]. Plain radiog-
616 raphy excludes concomitant hip or knee joint dis-
617 ease and can detect insertional calcific deposits at
618 the greater trochanter. Ultrasound scans can be
619 indicated to assess abductor tendon thickening,
620 tendinopathy and partial- or full-thickness tears
621 [27]. When there is suspicion of involvement of
622 the gluteus muscle tendons, MRI is effective to
623 recognize partial- and full-thickness tears, tendon
624 calcification and muscle fatty atrophy.

625 29.7.2 Management

626 Conservative measures including relative rest,
627 anti-inflammatory medication, ice, stretching and
628 strengthening, physical therapy, shock wave ther-
629 apy, ultrasound and local corticosteroid injection
630 are commonly used [10]. However, often symp-
631 toms linger and recurrence occurs, and symptom-
632 atic athletes need to modify training for prolonged
633 periods. Corticosteroid injections have been used
634 for many years, but recurrence of symptoms and
635 incomplete relief have been commonly recorded
636 [10]. An RCT evaluated three treatment proce-
637 dures, home training, corticosteroid injection and
638 shock wave therapy [51]. Corticosteroid injec-
639 tions were found to be effective in the short term,
640 with declining effectiveness over a few months.
641 Repetitive low-energy radial SWT without local
642 anaesthesia did not result in early pain relief, but
643 provides a beneficial effect over several months,
644 with a success rate of 68 % at 4 months and 74 %
645 at 15 months. Home training exercises included
646 progressive exercise including piriformis stretch,
647 iliotibial band stretch standing, straight leg raise,
648 wall squat with ball and gluteal strengthening.
649 Their effects were evident after 4 months, with a
650 41 % success rate, increasing to 80 % at 15
651 months. Home training exercises were more
652 effective in the longer term. In a case control
653 study, Furia et al. found that 76.5 % of patients
654 who participated in regular sporting activities and
655 were treated with SWT were able to return to
656 sports at their pre-injury levels, compared to the

657 66.7 % in the control group [15]. At the final fol- 657
658 low-up, the number of patients with excellent and 658
659 good results was significantly higher after SWT. 659

660 Several surgical procedures have been 660
661 described for patients' refractory to conservative 661
662 treatments. Brooker reported on five patients 662
663 treated with fenestration or T-shaped incision of 663
664 the iliotibial band [8]. At 1 year, patients were sat- 664
665 isfied and had near-normal function, with a Harris 665
666 Hip Score of 88 compared with a baseline score of 666
667 46. Slawski and Howard performed a simple lon- 667
668 gitudinal incision of the iliotibial band (ITB) and 668
669 bursectomy [52]. All the patients were satisfied. 669
670 Kagan firstly described rotator cuff tears of the 670
671 hip [25]. He reported good outcomes after open 671
672 repair and suture reattachment of the gluteus 672
673 medius at a median follow-up of 45 months. 673
674 Recently, Voss et al. also reported successful 674
675 short-term outcomes on 10 patients who under- 675
676 went endoscopic repair of gluteus medius tears 676
677 [59]. However, despite the good results reported 677
678 after open and arthroscopic procedures, all the 678
679 studies are small and retrospective case series 679
680 reporting success rates difficult to compare. 680

681 29.8 Summary

682 Evaluation and treatment of groin pain in athletes 682
683 is challenging. It is important to remember that 683
684 'groin pain' means 'pain in the groin area' and is 684
685 not a diagnosis. The groin anatomy is complex, 685
686 and pain is often caused by the association of dif- 686
687 ferent conditions. Frequently, groin pain is a 687
688 component of a more extensive pattern of 'groin 688
689 pain disruption' which involves several concur- 689
690 rent pathologies. These may include not only 690
691 intra-articular and extra-articular pathologies 691
692 around the hip but also lumbar spine diseases, 692
693 nerve entrapments and intra-abdominal and geni- 693
694 tourinary pathologies. Muscular imbalance and 694
695 pelvic instability seem to be the common denomi- 695
696 nator for many conditions causing athlete's groin 696
697 pain. Correct diagnosis is mandatory for appropri- 697
698 ate management. As the differential diagnosis 698
699 for chronic groin pain is wide, thorough clinical 699
700 examination is paramount. Symptoms may over- 700
701 lap, and no high-specificity tests are available. 701

702 Many different treatment protocols and strategies
 703 have been proposed to manage groin pain.
 704 Conservative management is indicated to stabi-
 705 lize the pelvis and pubic symphysis. Core stabili-
 706 tity exercises and muscle stretching and
 707 strengthening exercises of the abdominal, adduc-
 708 tor, flexor and extensor hip muscles are effective
 709 for this purpose. Better results have been reported
 710 after surgical treatment for FAI and sportsman
 711 hernia. Surgery is also indicted for patients who
 712 do not respond to conservative management.
 713 However, given the complexity of the pathology,
 714 proper treatment should be multidisciplinary.

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