

# Osteochondroma Involving the Hip

31

Daniel E. Porter and Fei Li

# Introduction

The pediatric orthopaedic surgeon has an important role in surveying the progress of children with Multiple Osteochondromatosis (MO) for developing hip pathology, and for managing those with solitary exostoses causing pain or dysfunction around the hip. This chapter aims to provide guidance for the surgeon in terms of frequency and scope of regular review, investigation, management and prognosis of the common hip disorders encountered in these conditions.

## Pathophysiology

#### Molecular Pathogenesis

The genetic basis for MO is the occurrence of mutations in two members of the exostosin (EXT) gene family, EXT1 and EXT2 [1–4]. Each of these genes code for a sub-unit of the polymerization machinery for the glycosaminoglycan (GAG) heparan sulphate (HS). Although MO is genetically heterogeneous [5], there is evidence of a genotype-phenotype correla-

F. Li

S. Alshryda et al. (eds.), *The Pediatric and Adolescent Hip*, https://doi.org/10.1007/978-3-030-12003-0\_31

tion [6, 7]. A puzzling aspect of the disease is that although HS is ubiquitously expressed, the manifestation of MO is specific and primarily restricted to the long bones. This has led to many theories on the molecular pathogenesis of MO, often linked to the complex role of HS in regulating cell response to morphogenic factors which determine the normal limits of bone growth (e.g. hedgehogs and BMPs) particularly within the specialized microenvironment of the growth plate [8-11]. A possible second mutation or cytogenetic loss of the wildtype allele (loss of heterozygosity, LoH) of EXT1 or EXT2 has also been suggested, leading to clonal expansion of EXT-null cells [12]. The cartilage cap of the osteochondroma has been shown to contain much reduced HS levels compared to normal, which supports the LoH model [13].

The MO phenotype is variable (Figs. 31.1 and 31.2) but is generally more severe in males and in individuals with the EXT1 mutation [6, 7, 14].

"The MO phenotype is variable but is generally more severe in males and in individuals with the EXT1 mutation".

# Heparan Sulphate and Osteochondromas

Although the link between GAG biosynthesis and exostoses was made in the mid 1990s,

D. E. Porter (🖂)

Tsinghua University, Beijing, China

e-mail: daniel-porter@mail.tsinghua.edu.cn

First Hospital of Tsinghua University, Beijing, China

<sup>©</sup> Springer Nature Switzerland AG 2019



**Fig. 31.1** MO in a boy aged 6 years. Note characteristic features of osteochondromas in the periacetabular region and around the trochanters. The femoral neck is widened and there is coxa valga of the right femoral neck. Indices of dysplasia are also abnormal in the right hip



**Fig. 31.2** MO in a girl aged 12 years. Here, the only features of the condition are tiny osteochondromas in the left periacetabular region and below the lesser trochanter. There may be mild coxa valga

understanding the mechanism of osteochondroma development remains a major challenge. In the Golgi apparatus, EXT1 and EXT2 catalyse HS polymerization, and additionally influence other components of the HS biosynthetic pathway [8–11]. GAG chains such as HS are found attached to core proteins as proteoglycans (PGs). PGs are ubiquitous residents of most organs and tissues. Their presence at the cell surface and within the extracellular matrix (ECM) is critical for their varied roles in cell growth, morphogenesis and cell-cell/cell-matrix interactions. Much effort has focused on elucidating the identity and behavior of the cells which give rise to osteochondromas. The two mechanisms introduced above: (1) reduction of functional EXT activity in cells within the growth plate enabling a subset of cells to evade factors which normally limit bone growth and (2) LoH and clonal expansion, are both supported by convincing experimental data [12, 15].

Solitary osteochondromas may occur after radiation [16] or trauma, such as in traumatic hip dislocation [17] and slipped capital femoral epiphysis [18]. Radiation may increase the chance of two somatic mutations creating an osteochondroma in the radiation-field. Posttraumatic osteochondromas may not have a genetic cause, but simply represent displaced epiphyseal cartilage following injury.

# Pathogenesis of Hip Abnormalities in Solitary Osteochondromas

In keeping with osteochondromas elsewhere, the lesion forms on the metaphyseal side of the growth plate. However subsequent morphological changes may orientate the osteochondroma towards the head rather than the neck, leading to impingement on the acetabular rim [19]. Osteochondromas adjacent to the triradiate cartilage are rare. Those adjacent to the apophyses of the greater and lesser trochanters, and those of the ischium, pubis and ilium are more common. Concurrent exostoses located on the ischium and lesser trochanter can cause impingement during adduction and internal rotation of the hip.

Solitary exostoses of the proximal femur have been associated with coxa valga for three patients in two case reports [20, 21]. All patients were young adults—their deformities reflecting earlier growth. Their association with deformity is moderately strong evidence in favour of a local exostosis *causative* effect [22].

The cellular activity of the cartilage cap is related to its depth. Just as the physis exhibits reduced cellular activity and diminished thickness after the pubertal growth-spurt, so most osteochondromas will completely ossify at the end of skeletal growth. These undergo further remodeling, and in some cases regress or disappear. Very rarely, an osteochondroma may undergo malignant change. The axial skeleton, including the proximal femur and pelvis, is the usual location for malignant change in an osteochondroma [23].

"Very occasionally an osteochondroma may undergo malignant change. The axial skeleton, including the proximal femur and pelvis, is the usual location for malignant change in an osteochondroma".

#### Pathogenesis of Hip Abnormalities in MO

The deformities associated with MO may be divided into two types:

- Direct pressure effects: Subluxation of the hip may occur due to the direct extrusive force of an intra-articular exostosis [24, 25] or may be due to extra-articular exostoses (usually at the ischium and proximal femur) causing an abnormal hinge-effect on the femoral head [26]. Associated with this, there may be a reduced ischio-femoral interval and quadratus femoris muscle abnormalities (see "Imaging section").
- 2. Characteristic deformities. These are coxa valga and, to a lesser extent, hip dysplasia. The causes of these are uncertain. Porter et al [27] found that volume of exostoses around the proximal femur was associated with coxa valga, and volume around the acetabulum was associated with dysplastic indices; however, these findings have not been confirmed [24]. There is therefore only weak evidence that exostoses themselves cause coxa valga and dysplasia in MO. A 'field-change' effect could also be responsible or contribute (Porter & Li 2017). Leg-length discrepancy is sometimes found in association with hip deformity in MO but causation is not easily established [20]; other manifestations of MO in the lower limbs may be responsible. Labral tears may occur due to abnormal forces imposed by an uncovered lateral portion of the femoral head [27], or due to direct impingement of periacetabular exostoses [19].

# **Natural History**

# Solitary Osteochondromas Involving the Hip

These may develop at any time during childhood. Once the triradiate and other growth-plate cartilages have fused, osteochondromas can no longer form. The natural history is highly variable due to unpredictable growth potential; the osteochondroma may grow slower, at the same rate, or faster than the surrounding bone. In almost all cases the osteochondroma will stop growing at the end of skeletal growth. An osteochondroma which continues to enlarge thereafter is one with the potential for malignant change [28].

Osteochondromas close to or within the hip joint may detach and become a loose body [29, 30].

# Multiple Osteochondromatosis Involving the Hip

Longitudinal studies in MO are uncommon; only those of Wang et al. [26, 31] and Duque Orozco et al. [26] followed children with MO for more than 2 years. However, cross-sectional studies suggest that in infancy there may be no identifiable anatomical hip abnormality. Weiner and Hoyt [32] believed that natural infantile coxa valga and hip anteversion failed to resolve in early childhood in MO. The second half of the first decade of life usually sees a characteristic coxa valga develop. Ozaki et al. [33] noted a neck-shaft angle of over 150° in >50% of children. Wang et al. [31] found that an abnormally horizontal proximal femoral physeal plane (Hilgenreiner's epiphyseal angle) was associated with coxa valga development, but not with future hip dysplasia. There is evidence that the severity of coxa valga and hip dysplasia improves in the second decade of life [26, 33]. However the longitudinal component of one of these studies suggested no significant change in measured indices of dysplasia and coxa valga over 5 years [26]. Reimers migration index is more frequently abnormal than centre-edge angle; however in only 5% of children is there measurable subluxation (migration index >30%) [31].



**Fig. 31.3** 43 year old woman with MO and osteoarthritis of the right hip. (**a**) Osteoarthritis and hip dysplasia. (**b**) Plain radiograph after right cemented hip arthroplasty

Leg length discrepancy is found in association with hip deformity in MO but which lower limb segment is responsible may be difficult to establish [20]. Hip joint osteoarthritis is a recognized late-association in MO. This may be due to acetabular dysplasia, subluxation or direct mechanical effect. Hip replacement may be indicated in adulthood [34] (Fig. 31.3).

### Epidemiology

The osteochondroma (benign cartilaginous exostosis) is the most common benign bone tumour in most series, representing 40% in some archives [35, 36]. Approximately 0.5% of children undergo excision of a palpable or symptomatic isolated (sporadic) osteochondroma during childhood [37]. There appears to be a similar incidence in Caucasians, oriental and black populations. Multiple osteochondromas, alternatively called Hereditary Multiple Exostoses, HME or Multiple Hereditary Exostoses (MHE) is one of the most common inherited orthopaedic disorders, estimated to affect at least 1:50,000 people [36].

# "The osteochondroma (benign cartilaginous exostosis) is the most common benign bone tumour in most series".

New mutations represent only a small percentage of the total, since the condition does not affect life-expectancy or fertility, and individuals can maintain an active lifestyle. In keeping with other point-mutation conditions, there is some evidence that older men have a higher chance of fathering children with MO. There is also some evidence for 'anticipation'; that is, affected children can have a more severe phenotype than their parent. The condition is characterized by dysregulated chondrocyte proliferation, and the formation of cartilage-capped bony nodules (exostoses) which are histologically indistinguishable from the more common solitary/sporadic type. They are located on the external surface of long bone metaphyses, adjacent to the epiphyseal plate. These benign tumours are associated with an increased risk of malignant transformation to chondrosarcoma (0.5-3%) [36, 37].

# "New mutations represent only a small percentage of the total, since the condition does not affect life-expectancy or fertility, and individuals can maintain an active lifestyle".

Although solitary osteochondromas around the hip are usually isolated lesions, their frequency in the general population makes them important to the surgeon. On the other hand, although MO is rare, its effect on the hip joint can be profound and difficult to control. Although osteochondromas and associated deformities can remain throughout life, they generally stop growing at the end of skeletal maturity. Therefore the burden of intervention and medical review falls mainly on the pediatric orthopedic surgeon.

Solitary osteochondromas around the hip may be classified as extra-articular or intraarticular. Both may cause pain or dysfunction due to: impingement and limited range of movement, bursitis/tendinitis, and femoroacetabular subluxation. Exostoses affecting the actual hip joint are uncommon; however considering proximal femur and pelvis together, this area is responsible for greater than 20% of surgical procedures for symptomatic osteochondromas [38].

Many studies on MO have reported the incidence, site and age at which malignant change occurs. Excluding two studies from the Mayo Clinic with an incidence of malignant transformation of 18% and 25% [28, 39], a literature review identifies an average life-time risk of 4%. The risk is higher in males (greater than 5%) [36, 40–52]. Pooling the data from these studies: out of 852 patients who developed a primary malignant bone tumour in MO, only 4 were under 21 years of age [53]. Overall, the pelvis and proximal femur was the site of chondrosarcoma in 56% of patients. In a recent international survey of MO individuals, 11/21 cases of chondrosarcoma occurred in the pelvis or proximal femur - 7 patients were under age 30, the youngest age 19 [23]. Case reports confirm adolescent chondrosarcoma developing from osteochondromas of the pelvis or proximal femur in children and adolescents. These occur in both the solitary form [54] and in MO (Fig. 31.4) but they are rare. The Rissoli Institute's review of 752 tumours of the pelvis in children under age 14 revealed no cases of chondrosarcoma, but 48 benign osteochondromas [55].



**Fig. 31.4** Girl age 17 years with MO and a low-grade chondrosarcoma of the left ilium. (a) Coronal MRI showing thickened cartilage cap >2 cm. (b) Bone scintigraphy showing high uptake within the tumour

"Pooling the data from these studies: out of 852 patients who developed a primary malignant bone tumour in MO, only 4 were under 21 years of age".

# **Clinical Presentation**

Patients will likely have presented with excresence(s) around the knee, ankle or elsewhere. MO involving the hip may present with pain, abnormal gait, limp or swelling.

#### **Essential Clinical Tests:**

- · Gait inspection
- Palpation of painful area for tenderness or local swelling
- Range and comfort of hip movement in all planes
- Check for femoroacetabular impingement—anterior and posterior impingement tests
- Leg length discrepancy assessment

#### Imaging

# Radiographic Abnormalities in Multiple Osteochondromatosis

Several characteristic abnormalities specific to MO are found on X-ray [24, 31, 56]. These include:

- An irregular outline to the medial femoral neck. It is unclear whether there are more exostoses present on the medial aspect of the femoral neck than the lateral aspect. The neck is usually wider than normal. [26, 27].
- Osteochondromas located at the femoral neck or peri-acetabular region.
- Coxa valga and femoral anteversion [32, 33] (Fig. 31.5). The precise combination of anteversion and valgus has not been established; the description 'valgoversion' has been applied by Weiner & Hoyt [32].

- Acetabular dysplasia [57]. One of the earliest reports of MO included an observation of hip dysplasia [58]. Abnormal centre-edge angle and acetabular index are common findings; however, other dysplastic features probably occur only in a minority of patients [27, 59].
- Leg Length Discrepancy. The frequency of this is not well established.

#### **Radiographic Measurements**

Porter et al. [27] identified several key radiographic measurements which, compared with controls, are frequently abnormal in MO. These abnormalities may be seen in typical radiographs: (Fig. 31.1). They include:

- Neck-shaft diameter ratio (mean, 1.6 in MO versus 1.23 in controls)
- Neck-shaft angle (median, 156°)
- Shenton's line was broken in 60% (also [26, 56])
- Centre-edge angle was abnormal in 29% (median, 23°)
- Sharp's acetabular angle was abnormal in 42% (median, 44°)

Other authors have reported abnormal measurements involving Hilgengreiner's epiphyseal angle [31] and Reimers migration percentage [26].

# The Case for Radiographic Surveillance

Some authors have suggested regular radiographic review of MO hips through childhood in the presence of borderline subluxated hips, and for those with marked coxa valga and/or acetabular dysplasia. We believe these should be closely monitored to determine the need for future surgery [24, 60].

Children with MO can also develop deformities of the upper and lower limbs which change steadily over the course of one year [22]. The main areas in which deformities can develop and lead to complications which are difficult to treat are: distal forearm ulnar shortening, ankle valgus and talar-shift, hip joint dysplasia, impingement and subluxation. An annual appointment with a clinician who



**Fig. 31.5** Girl (5 years old) with MO. (**a**) Long-leg radiographs. The right leg is 1 cm shorter than the left. There are confluent osteochondromas around the right lesser trochanter with subluxation of the right hip. (**b**) Axial CT scan at the level of the femoral neck, confirming location

of osteochondromas and persistent femoral anteversion on the right side. (c) Axial CT scan at the level of the acetabulum, confirming subluxation of the right femoral head

understands the potential for developing complications is recommended. It is also reassuring for parents to have an annual appointment.

In addition, for very young children, intraarticular exostoses may not be painful despite the development of substantial underlying hip pathology [61]. Therefore, we strongly recommend a routine radiographic pelvic survey at the time of MO diagnosis so that there can be early detection of osteochondromas and/or hip dysplasia [57] and a management plan instituted. Unfortunately, there are no data to recommend a frequency of radiographic surveillance [60]. A pragmatic approach would allow further hip x-rays if there is pain, restriction of movement or leg-length discrepancy.

# "We strongly recommend a routine radiographic pelvic survey at the time of MO diagnosis so that there can be early detection of osteochondromas and/or hip dysplasia".

Since there is a risk of malignant change in MO in adulthood, and the pelvis/proximal femur is involved in greater than 50% of tumour cases, then we also recommend a pelvic radiograph at the end of pediatric follow-up to iden-



**Fig. 31.6** MO in a girl aged 16 years. (a) Plain radiograph of the left hip exhibits exostoses at the lateral head-neck junction which limits abduction. There is a large exostosis at the medial femoral neck which limits adduction and

tify whether there are exostoses of significance located in the pelvis or proximal femur which might require further characterization or review during adulthood.

#### **Cross-Sectional Imaging**

On CT scan and MRI, the ischio-femoral interval can be measured. The minimum ischiofemoral space (MIFS) has been measured in MO and is found to be associated with impingement and oedema of the quadratus femoris muscle [62]. A normal MIFS has been reported to be approximately 20 mm. An interval of less than 10 mm

internal rotation. Hip subluxation is obvious. (b) Axial MRI at the level of the acetabulum showing subluxation of the left femoral head. (c) Coronal MRI at the level of the acetabulum showing subluxation of the left femoral head

on MRI has been suggested to be associated with diagnosis of extraarticular impingement [63]. Children and adolescents with MO and hip symptoms have labral pathology identified on MRI in nearly half of cases [62].

With respect to the identification of malignant change, taking a cartilage cap thickness of 2 cm or less as a cut-off for benign osteochondroma, sensitivity and sensitivity was 100% and 98% for MR imaging and 100% and 95% for CT imaging in a large series of over 100 tumours, with 17 located in the pelvis. Care must be taken to ensure that the cap is not measured on a tangential section, or else an artificially thick cap may be recorded [64]. "For the identification of malignant change, taking a cartilage cap thickness of 2cm or less as a cut-off for benign osteochondroma, sensitivity and sensitivity was 100% and 98% for MR imaging and 100% and 95% for CT imaging".

### **Bone Scintigraphy**

Historically, bone scintigraphy has been used to help discriminate osteochondromas from sarcomas. Those lesions with malignant cartilage tended to exhibit enhanced uptake on bone scan (Fig. 31.4) [65, 66]. Bone scanning, however, has been largely replaced by MRI, the current investigation of choice to help distinguish benign from malignant lesions.

#### Non-operative Management

Careful attention to indications for surgery and timing of surgery are key, especially as operative intervention elsewhere in the lower limb, aside from the hip may be envisaged. Symptom control with analgesia is appropriate. Physiotherapy may be an important modality, but should not be pursued aggressively in terms of range of movement, as it may exacerbate symptoms and joint damage due to extra-articular FAI.

#### Essential Non-operative Management Methods

- Regular annual clinical review—observe mild hip dysplasia in young children
- Symptomatic treatment of pain

#### **Non-operative Pitfalls**

- Ensure significant hip subluxation is not missed with regular radiographic surveillance, at least for symptomatic lesions
- For cases of extra-articular hip impingement, avoid aggressive physiotherapy which exacerbates symptoms by "improving" range of motion

# **Operative Management**

# Management Based on Clinical Presentation

Due to the risk of painful impingement and/or subluxation, an annual clinical and radiographic review for children with MO involving the hip is warranted. The hip cannot be viewed in isolation when considering the reasons for regular review of children with MO. However, the conditions around the hip which require observation and perhaps intervention can be classified as follows:

- 1. Painful and urgent:
  - (a) Femoral head subluxation due to intraarticular osteochondroma (solitary and MO)

<u>Background:</u> Femoral head subluxation may be due to osteochondromas located in the acetabular floor. These originate from the tri-radiate cartilage. Exostoses of varied size may develop. Those of sufficient size to cause subluxation are rare [24, 26].

<u>Clinical features:</u> There may be pain, crepitus, and reduced range of movement [67].

<u>Investigation</u>: CT is the investigation of choice to identify the location and size of the lesion(s) [67].

<u>Rationale:</u> There is a risk of irreversible articular cartilage damage in unidentified and untreated cases.

<u>Intervention</u>: All cases of true actual subluxation require urgent surgery to excise the intra-articular exostosis. Options for operative treatment include:

- Arthroscopy [67].
- Safe surgical hip dislocation of Ganz [68, 69].
- Anterolateral approach with subluxation rather than dislocation [70].
- Anterior approach with dislocation [69].
- Anterior approach without dislocation [71].
- (b) Femoral Head subluxation due to extraarticular osteochondroma (Solitary and MO)

Background: Subluxation is due to femoroacetabular impingement. The location of the offending osteochondromas may be:

• Juxta-articular. Exostoses originate from the margins of the acetabulum or the femoral head-neck junction causing femoro-acetabular impingement (FAI) [19, 72]. This is usually cam-type impingement [73]. Labral tears may also occur in the adult and pediatric population [19, 62].

• Extra-capsular (Figs. 31.5–31.7). Large exostoses are often located on the lesser trochanter or ischium, and may be extensive enough to



Fig. 31.7 Solitary osteochondroma of the left lesser trochanter in a girl aged 11 years. (a) Plain radiograph. (b) Axial MRI demonstrating both antero-medial and posterior components and reduced ischio-femoral interval. (c)

Coronal MRI showing hip joint located. (d) Post-excision radiograph. Surgery performed for mechanical hip pain and limited hip adduction and rotation (image courtesy of Mr. M Gaston, Royal Infirmary of Edinburgh)

cause subluxation via a hinge-type mechanism which levers the hip out of the joint. 'Kissing' lesions are a recognized feature of MO. Excision of those impinging exostoses can relieve pain [74].

*Clinical features*: Pain on hip movement, typically at end range of motion. Reduced range of movement, especially adduction, extension and external rotation, is commonly found.

<u>Investigation:</u> CT or MRI will confirm subluxation and position of osteochondromas for excision.

Rationale for treatment: Risk of irreversible articular cartilage damage in untreated cases. <u>Intervention:</u> Osteochondroma excision. Descriptions include:

- Arthroscopy [61]
- Safe dislocation (digastric approach) for posterior exostoses [21, 75–78]
- Anterior approach without dislocation [25]
- Lateral approach may be suitable for low anterior or posterior extracapsular lesions.
- Ludloff approach for lesions at or below lesser trochanter [79]
- Femoral triangle approach may be suitable for adolescents and adults [80]

#### (c)Neoplastic change

<u>Background:</u> This is extremely rare in children, and indeed, before the third decade of life. The pelvic girdle or proximal femur is involved in around 50% of cases.

<u>Clinical features:</u> Painful, enlarging bony swelling, usually older adolescent.

Investigation: For prevention, all pediatric patients who are approaching the end of their annual clinical review in adolescence should have a plain X-ray of the pelvis to exclude or identify exostoses requiring adult follow-up. For investigation of suspicious lesions first gain plain radiographs and compare with previous images. MRI is the imaging investigation of choice to identify malignant change (Fig. 31.4).

<u>Rationale:</u> The low grade chondrosarcoma typically associated with MO is curable, but requires early diagnosis and specialist staging and treatment.

<u>Intervention:</u> For suspicious lesions, refer to orthopaedic oncologist for further investigation and staging. Principles of treatment are those for bone tumours in general.

"For prevention, all pediatric patients who are approaching the end of their annual clinical review in adolescence should have a plain X-ray of the pelvis to exclude or identify exostoses requiring adult follow-up".

- 2. Painful and non-urgent
  - (a) Impingement affecting range of movement without subluxation (Solitary and MO):

<u>Background:</u> Osteochondromas causing impingement on hip movement are usually painful [21]. The ischiofemoral interval may be reduced on cross-sectional imaging [62]. Pain may be due to pathology within the quadratus femoris muscle (Fig. 31.7) [63].

<u>Clinical features:</u> Reduced endurance activity, mechanical hip pain, reduced range of movement. Impingement in any direction is possible [81, 82].

<u>Investigations:</u> Plain radiographs, CT, and/or MRI.

Rationale: At the extremes of movement, a 'dynamic subluxation' is possible due to hinging at time of impingement.

Intervention: Osteochondroma excision (Fig. 31.7). Descriptions included in Sect. 1(b) above. Results of surgery suggest that removing femoral head and neck exostoses in these children can improve range of movement (such as hip flexion by an average of 30°) and can reduce or abolish hip pain. The 'safe' surgical dislocation method has risks such as AVN, fracture and osteotomy separation/nonunion [77]. In children greater than 12 years old, head-neck osteochondroplasty may involve the physis if needed due to minimal effects on the remaining growth after that age [73, 78].

(b) Exostosis within the pelvis or proximal femur causing irritation due to bursitis, tendinitis or snapping tendon: <u>Background:</u> Friction of a muscle or tendon over a pedunculated exostosis may cause a bursitis or tendon dysfunction.

<u>Clinical Features:</u> An osteochondroma may cause vague mechanical pain. Squatting and rotation of the hip may cause a painful psoas 'clunk' heard deep within the hip. The iliotibial band may also be responsible for a snapping sensation felt lateral to the hip joint [83]. Locking may rarely occur.

<u>Investigations:</u> Plain radiographs are essential [83]. MRI will identify the extent of a reactive bursa (Fig. 31.8). The cartilage cap has similar signal characteristics to bursal fluid on T2 weighted images [84].

Intervention: Patients should be managed expectantly, as symptoms often improve spontaneously [26]. Osteochondroma excision should be reserved for the treatment of persistent pain.

(c) Coxa valga and/or acetabular dysplasia associated with hip pain:

<u>Background:</u> Dysplasia is present in less than half of patients with MO and does not have a recognized association with solitary exostoses.

<u>Clinical features:</u> Mechanical hip pain located at the groin, thigh or knee.

<u>Investigation</u>: Plain radiographs to measure acetabular index and centre-edge angle.



**Fig. 31.8** Girl (13 years old) with a solitary exostosis on the right anterior iliac bone, deep to psoas major muscle. A reactive bursa is seen on axial T2-weighted MRI scan



**Fig. 31.9** Result of bilateral derotation varising extension osteotomies in a 16 year old girl, one year after surgery. Surgery performed for episodic hip pain, limited walking distance (1 km), fixed flexion deformity, limited adduction and external rotation. Symptoms and range of movement improved after surgery

<u>Rationale:</u> Abnormal joint forces cause associated with proximal femoral/acetabular dysplasia can lead to chondral pathology and an increased the risk of early arthritis (Fig. 31.3).

Intervention: Makhdom et al. [60] believed that early excision of hip osteochondromas might prevent acetabular dysplasia in MO patients. However, removal of intra-articular exostoses by Jellicoe et al. [69] did not cause resolution of hip dysplasia after two years. In children and young adults with head extrusion without acetabular dysplasia, a derotational intertrochanteric varus osteotomy may be suitable (Fig. 31.9) [60, 85]. Where the acetabulum is shallow, increasing head coverage with an acetabular osteotomy may improve symptoms and prognosis [86].

- 3. Painless and non-urgent:
  - (a) Impingement affecting range of movement without subluxation (Solitary MO): See sect. 2 (a) above.
  - (b) Coxa valga and/or acetabular dysplasia without hip pain:

<u>Background:</u> There is some evidence that the indices of coxa valga and dyspla-

sia (neck-shaft angle, Reimers migration index, centre-edge angle, acetabular index) may improve in the second decade of life [26, 33].

<u>Investigation</u>: Plain radiographs to measure acetabular index and centre-edge angle.

Intervention: If the radiographic indices of dysplasia are not severe, a watchful approach may be warranted for younger patients [26].

(c) Leg-length discrepancy:

Background: In MO, leg-length discrepancy (LLD) is more common than in the background population (Fig. 31.5). However, the cause of the LLD could result from the effects of exostoses anywhere from the hip to the ankle. Where there is a significant LLD of greater than 2 cm, surgery can be considered.

<u>Clinical features:</u> There is a short leg gait with potential varus or valgus deformities at the hip, knee or ankle.

<u>Investigation</u>: Long-leg measurement radiographs (Fig. 31.5).

<u>Rationale:</u> There is a risk of back-pain in adulthood in untreated LLD of over 2 cm.

Intervention: We suggest treatment according to standard principles. Wang et al. [31] suggested the horizontality of the proximal femoral physeal line—Hilgengreiner's epiphyseal angle (HEA)-could predict those children who would have a severe coxa valga at skeletal maturity, and might benefit from 'guided growth'. Removal of associated osteochondromas may have a beneficial effect [22]. Where LLD is associated with coxa valga, decision-making can include use of a varus-producing osteotomy with the expectation of reduced femoral length following surgery (Fig. 31.9). Either closing- or opening-wedge osteotomies may be selected to equalize length.

 (d) Enlarging osteochondromas with or without associated coxa valga or acetabular dysplasia:

<u>Background:</u> There is some uncertainty in deciding whether early removal of large juxta-epiphyseal or otherwise juxta-articular osteochondromas is necessary in preschool children with MO. Large osteochondromas are often confluent with the perichondrial ring, so that en-bloc excision of these may damage the growth-plate, resulting in further growth disturbance. On the other hand, a 'safety-first' approach may result in an incomplete excision and rapid recurrence. Both complications have been previously described in the literature [60].

Clinical features:

- Solitary osteochondroma: The hip is a deep joint, and so finding a *palpable* osteochondroma around the proximal femur in children is unusual. There may, however, be a diminished range of hip movement secondary to impingement.
- MO produces the same clinical features as solitary osteochondromas. However, exostoses are much more numerous. In a severely affected individual, several confluent osteochondromas may produce an almost circumferential bulky enlargement of the femoral neck, which makes it difficult for the clinician to localize the problematic area.

<u>Rationale:</u> In younger children, consider careful follow-up. In older children, although very rare, consider the possibility of low grade chondrosarcoma.

*Intervention*: Surgery may not be necessary unless there is persistent pain or rapid enlargement. For surgical approaches, see 1(b) above.

#### **Essential Surgical Techniques**

- Pragmatic use of various hip approaches depending on location, and extent of exostoses including:
  - Hip arthroscopy
  - Surgical hip dislocation
  - Anterior, anterolateral or medial hip approaches
- Varus-producing derotational proximal femoral osteotomy for persistent coxa valga and hip migration in older children.

#### **Operative Pitfalls**

- Metaphyseal cancellous bone is expanded and highly vascular in the intertrochanteric region. Expect to encounter significant osseous bleeding during osteotomy.
- Risk of avascular necrosis for excisions involving the femoral neck or peritrochanteric region.
- Iatrogenic fracture post exostectomy (especially femoral neck).

#### **Classic Papers**

**Porter DE, Simpson AHRW. The neoplastic pathogenesis of solitary and Multiple Osteochondromas. J Pathol. 1999;188:119–25.** An important review of pathogenesis which proposed MO be classified as a familial neoplastic trait.

Schmale GA, Conrad EU, Raskind WH. The natural history of hereditary multiple exostoses. J Bone Joint Surg [Am]. 1994;76-A:986–92. One of the best epidemiological studies of MO.

Shin, SJ, Kwak HS, Cho TJ, Park MS, Yoo WJ, Chung CY, Choi IH. Application of Ganz surgical hip dislocation approach in pediatric hip diseases. Clin Orthop Surg 2009;1(3):132–37. Description of a selection of adolescent patients with MO had a digastric surgical dislocation to remove osteochondromas at the femoral head-neck junction safely.

Siebenrock KA, Ganz R. Osteochondroma of the femoral neck. Clin Orthop. 2002;394:211–18. A description in adults of the Ganz surgical hip dislocation for excision of exostoses.

Weiner DS, Hoyt WA Jr. The development of the upper end of the femur in multiple hereditary exostosis. Clin Orthop. 1978;137:187–90. First substantial study of proximal femoral morphology in MO confirming high incidence of coxa valga and anteversion.

# **Key Evidence**

Bernard SA, Murphey MD, Flemming DJ, Kransdorf MJ. Improved differentiation of benign osteochondromas from secondary chondrosarcomas with standardized measurement of cartilage cap at CT and MR imaging. Radiology. 2010;255(3):857–65. A MRI study of osteochondromas indicating that a cartilage cap thickness of over 2 cm suggested a high probability of chondrosarcoma.

Duque Orozco M, Abousamra O, Rogers KJ, Thacker MM. Radiographic analysis of the pediatric hip patients with hereditary multiple exostoses (HME). J Pediatr Orthop. 2018;38(6):305–311. A radiographic analysis in over 100 hips with MO. In a longitudinal study, over 1/3 of hips exhibited features of subluxation at some point. The authors recommend regular follow-up in children with hip osteochondromas.

Porter DE, Lonie L, Fraser M, Dobson Stone C, Porter JR, Monaco AM, Simpson AHRW. Severity of disease and risk of malignant change in Hereditary Multiple Exostoses: A genotype-phenotype study. J Bone Joint Surg. [Br]. 2004;86-B:1041–46. A genotype-phenotype study indicating a worse phenotype in males and EXT 1 mutation individuals. EXT1 was also associated with a higher risk of chondrosarcoma development.

Yoong P, Mansour R, Teh JL. Multiple hereditary exostoses and ischiofemoral impingement: a case-control study. Skel Radiol. 2014;43(9)1225–30. A clinico-radiographic study linking ischiofemoral impingement symptoms with a reduced interval between these landmarks and oedema of quadratus femoris muscle.

Makhdom AM, Jiang F, Hamdy RC, Benaroch TE, Lavigne M, Saran N. Hip joint osteochondroma: systematic review of the literature and report of three further cases. Adv Orthop. 2014;2014:1802–54. A literature review of all surgical reports describing osteochondromas of the proximal femur and acetabulum, with and without dysplastic features. No series had more than six patients, so no firm conclusions could be drawn on optimum surgical approach or technique.

#### **Take Home Messages**

- All children with MO should have annual review with a pediatric orthopaedic surgeon until the end of skeletal growth. This should include clinical and radiographic assessments of the hip joints.
- Surveillance can be increased in frequency if warranted, by pain, more rapid changes in indices of deformity or for pre- and post-surgery review.
- Pain may come and go due to unnoticed trauma or inflammatory changes, and recent-onset pain alone is not an absolute indication for surgery.
- For younger children with features of mild hip dysplasia, consider careful observation since indices of dysplasia may improve in the second decade of life.
- Surgery for femoracetabular and ischiofemoral impingement is necessary in the presence of hip-joint subluxation. Removal of responsible osteochondromas is usually sufficient to improve pain and range of movement. Confluent, circumferential osteochondromas may be difficult to remove without risk of fracture or avascular necrosis.
- Many surgical approaches have been described, and none has shown more or less utility than any other. All reported series have very small numbers and therefore a pragmatic view should guide choice of surgery, determined by the location and extent of osteochondroma formation.

#### References

 Ahn J, Ludecke H-J, Lindow S, et al. Cloning of the putative tumour suppressor gene for hereditary multiple exostoses (EXT1). Nat Genet. 1995;11:137–43.

- Wuyts W, Van Hul W. Molecular basis of multiple exostoses: mutations in the EXT1 and EXT2 genes. Hum Mutat. 2000;15:220–7.
- ZakBM CBE, Esko JD. Hereditary multiple exostoses and heparan sulfate polymerization. Biochim Biophys Acta. 2002;1573:346–55.
- Stickens D, Clines C, Burbee D, et al. The EXT2 multiple exostoses gene defines a family of putative tumour suppressor genes. Nat Genet. 1996;14:25–33.
- Jennes I, Pedrini E, Zuntini M, et al. 2009 Multiple osteochondromas: mutation database Modb. Hum Mutat. 2009;30:1620–7.
- Francannet C, Cohen-Tanugi A, Le Merrer M, Munnich A, Bonaventure J, Legeai-Mallet L. Genotype-phenotype correlation in hereditary multiple exostoses. J Med Genet. 2001;38:430–4.
- Porter DE, Lonie L, Fraser M, Dobson Stone C, Porter JR, Monaco AM, Simpson AHRW. Severity of disease and risk of malignant change in Hereditary Multiple Exostoses: A genotype-phenotype study. J Bone Joint Surg Br. 2004;86-B:1041–6.
- Lind T, Tufaro F, McCormick C, Lindahl U, Lidholt K. The Putative Tumor Suppressors EXT1 and EXT2 Are Glycosyltransferases Required for the Biosynthesis of Heparan Sulfate. J Biol Chem. 1998;273:26265–8.
- McCormick C, Leduc Y, Martindale D, Mattison K, Esford LE, Dyer AP, Tufaro F. The putative tumour suppressor EXT1 alters the expression of cell-surface heparan sulfate. Nat Genet. 1998;19:158–61.
- Presto J, Thuveson M, Carlsson P, Busse M, Wilén M, Eriksson I, Kusche-Gullberg M, Kjellén L. Heparan sulfate biosynthesis enzymes EXT1 and EXT2 affect NDST1 expression and heparan sulfate sulfation. Proc Natl Acad Sci U S A. 2008;105:4751–6.
- Wei G, Bai XM, Gabb MMG, Bame KJ, Koshy TI, Spear PG, Esko JD. Location of the Glucuronosyltransferase Domain in the Heparan Sulfate Copolymerase EXT1 by Analysis of Chinese Hamster Ovary Cell Mutants. J Biol Chem. 2000;275:27733–40.
- 12. Jones KB, Piombo V, Searby C, Kurriger G, Yang BL, Grabellus F, Roughley PJ, Morcuende JA, Buckwalter JA, Capecchi MR, Vortkamp A, Sheffield VC. A mouse model of osteochondromagenesis from clonal inactivation of *Ext1* in chondrocytes. Proc Natl Acad Sci U S A. 2010;107:2054–9.
- Bishop JR, Schuksz M, Esko JD. Heparan sulphate proteoglycans fine-tune mammalian physiology. Nature. 2007;446:1030–7.
- Alvarez C, Tredwell S, De Vera MA, Hayden MR. The genotype-phenotype correlation of hereditary multiple exostoses. Clin Genet. 2006;70(2):122–30.
- Hecht JT, Hayes E, Haynes R, Cole WG, Long RJ, Farach-Carson MC, Carson DD. Differentiationinduced loss of heparan sulfate in human exostosis derived chondrocytes. Differentiation. 2005;73:212–21.
- Libshitz HI, Cohen MA. Radiation-induced osteochondromas. Radiology. 1982;142:643–7.

- Laing AJ, Dillon JP, Suiluman B, Mulcahy D. Osteochondroma development subsequent to traumatic hip dislocation. Orthopedics. 2008;31(1):84.
- Learmonth DJ, Raymakers R. Osteochondroma of the femoral neck secondary to a slipped upper femoral epiphysis. Arch Orthop Trauma Surg. 1993;112(2):106–7.
- Aguiar T, Dantas P. Arthroscopic Resection of Intra-Articular Osteochondromas of the Hip. Arthrosc Tech. 2014;3(3):e347–50.
- Ramos-Pascua LR, Sanchez-Herraez S, Alonso-Barrio JA, Alonso-Leon A. Solitary proximal end of femur osteochondroma. An indication and result of the en bloc resection without hip luxation. Rev Esp Cir Ortop Traumatol. 2012;56(1):24–31.
- Siebenrock KA, Ganz R. Osteochondroma of the femoral neck. Clin Orthop. 2002;394:211–8.
- Porter DE, Li F. Evidence-Based Treatment of Deformity in Multiple Osteochondromatosis. In: Alshryda S, Huntley JS, Banaszkiewicz P, editors. Pediatric Orthopaedics. New York: Springer; 2017. p. 499–518.
- Czajka CM, DiCaprio MR. What is the proportion of patients with multiple hereditary exostoses who undergo malignant degeneration? Clin Orthop Relat Res. 2015;473(7):2355–61.
- El-Fiky TAM, Chow AW, Li YH, To M. Hereditary multiple exostoses of the hip. J Orthop Surg. 2009;17(2):161–5.
- Ofiram E, Porat S. Progressive subluxation of the hip joint in a child with hereditary multiple exostosis. J Pediatr Orthop B. 2004;13(6):371–3.
- Duque Orozco M, Abousamra O, Rogers KJ, Thacker MM. Radiographic analysis of the pediatric hip patients with hereditary multiple exostoses (HME). J Pediatr Orthop. 2018;38(6):305–11.
- Porter DE, Benson MK, Hosney GA. The hip in hereditary multiple exostoses. J Bone Joint Surg Br. 2001;83-B:988–95.
- Garrison RC, Unni KK, McLeod RA, Pritchard DJ, Dahlin DC. Chondrosarcoma arising in osteochondroma. Cancer. 1982;49:1890–7.
- Porter G, Allard J. Wavy pelvis sign in CT of multiple hereditary osteochondromatosis. J Comput Assist Tomogr. 1992;16(1):126–8.
- Liu ZJ, Zhao Q, Zhang LJ. Extraskeletal osteochondroma near the hip: A pediatric case. J Pediatr Orthop B. 2010;19(6):524–8.
- Wang YZ, Park KW, Oh CS, Ahn YS, Kang QL, Jung ST, Song HR. Developmental pattern of the hip in patients with hereditary multiple exostoses. BMC Musculoskelet Disord. 2015;16(1):514.
- Weiner DS, Hoyt WA Jr. The development of the upper end of the femur in multiple hereditary exostosis. Clin Orthop. 1978;137:187–90.
- Ozaki T, Kawai A, Sugihara S, Takei Y, Inoue H. Multiple osteocartilaginous exostosis. A follow-up study. Arch Orthop Trauma Surg. 1996;15(5):255–61.
- 34. Moran MA, Krieg H, Boyle RA, Stalley PD. Bilateral total hip arthroplasty in Severe Hereditary

Multiple Exostosis: a report of two cases. Hip Int. 2009;19(3):279–82.

- Huvos AG. Bone Tumours—Diagnosis, Treatment and Prognosis. 2nd ed. Philadelphia: W B Saunders; 1991.
- Schmale GA, Conrad EU, Raskind WH. The natural history of hereditary multiple exostoses. J Bone Joint Surg Am. 1994;76-A:986–92.
- Porter DE, Simpson AHRW. The neoplastic pathogenesis of solitary and Multiple Osteochondromas. J Pathol. 1999;188:119–25.
- Bottner F, Rodl R, Kordish I, Winklemann W, Gosheger G, Lindner N. Surgical treatment of symptomatic osteochondroma. A three-to-eight year followup study. J Bone Joint Surg Br. 2003;85-B:1161–5.
- Ahmed AR, Tan T-S, Unni KK, Collins MS, Wenger DE, Sim FH. Secondary chondrosarcoma in osteochondroma: report of 107 patients. Clin Orthop Relat Res. 2003;411:193–206.
- Black B, Dooley J, Pyper A, Reed M. Multiple hereditary exostoses: An epidemiologic study of an isolated community in Manitoba. Clin Orthop Relat Res. 1993;287:212–7.
- Crandall BF, Field LL, Sparkes RS, Spence MA. Hereditary multiple exostoses. Report of a family. Clin Orthop Relat Res. 1984;190:217–9.
- 42. Exner GU, Von Hochstetter AR, Suter A. Malignant transformation in hereditary multiple cartilaginous exostoses. Acta Orthop Scand Suppl. 1991;62:62.
- Suzuki A, Ito S, Takechi H. Follow-up study of cartilaginous bone tumors. Acta Med Okayama. 1986;40:147–61.
- Voutsinas S, Wynne-Davies R. The infrequency of malignant disease in diaphyseal aclasis and neurofibromatosis. J Med Genet. 1983;20(5):345–9.
- Legeai-Mallet L, Munnich A, Maroteaux P, Le Merrer M. Incomplete penetrance and expressivity skewing in hereditary multiple exostoses. Clin Genet. 1997;52:12–6.
- Wuisman P, Jutte P, Ozaki T. Secondary chondrosarcoma in osteochondromas: medullary extension in 15 of the 45 cases. Acta Orthop Scand. 1997;68:396–400.
- Kivioja A, Ervasti H, Kinnunen J, Kaitila I, Wolf M, Bohling T. Chondrosarcoma in a family with multiple hereditary exostoses. J Bone Joint Surg Br. 2000;82-B:261–6.
- Pierz KA, Stieber JR, Kusumi K, Dormans JP. Hereditary multiple exostoses: one center's experience and review of etiology. Clin Orthop Relat Res. 2002;401:49–59.
- 49. Vanhoenacker FM, Van Hul W, Wuyts W, Willems PJ, De Schepper AM. Hereditary multiple exostoses: from genetics to clinical syndrome and complications. Eur J Radiol. 2001;40:208–17.
- Clement ND, Ng CE, Porter DE. Shoulder exostoses in hereditary multiple exostoses: Probability of surgery and malignant change. J Shoulder Elb Surg. 2011;20(2):290–4.
- 51. Altay M, Bayrakci K, Yildiz Y, Erekul S, Saglik Y. Secondary chondrosarcoma in cartilage bone

tumors: report of 32 patients. J Orthop Sci. 2007;12(5):415–23.

- Wicklund CL, Pauli RM, Johnston D, Hecht JT. Natural history study of hereditary multiple exostoses. Am J Med Genet. 1995;55:43–6.
- 53. Li F, Ngoh C, Porter DE. Chondrosarcoma transformation in heditary multiple exostoses: a systematic review and clinical and cost-effectiveness of a proposed screening model. J Bone Oncol. 2018;13:114–22.
- 54. Nystrom LM, DeYoung BR, Morcuende JA. Secondary Chondrosarcoma of the Pelvis Arising from a Solitary Exostosis in an 11-Year-Old Patient. A Case Report with 5-Year Follow-Up. Iowa Orthop J. 2013;33:213–6.
- 55. Ruggieri P, Angelini A, Montalti M, Pala E, Calabrò T, Ussia G, Abati CN, Mercuri M. Tumors and tumourlike lesions of the hip in the pediatric age: a review of the Rizzoli experience. Hip Int. 2009;19(6):S35–45.
- Malagon V. Development of Hip Dysplasia in Hereditary Multiple Exostosis. J Pediatr Orthop. 2001;21:205–11.
- Felix NA, Mazur JM, Loveless EA. Acetabular dysplasia associated with hereditary multiple exostoses: a case report. J Bone Joint Surg Br. 2000;82-B:555–7.
- Evans EL. Case of Multiple cartilaginous Exostoses associated with Congenital Dislocation of Hip. Proc R Soc Med. 1921;14:166.
- Higuchi C, Sugano N, Yoshida K, Yoshikawa H. Is hip dysplasia a common deformity in skeletally mature patients with hereditary multiple exostoses? J Orthop Sci. 2016;21(3):323–6.
- Makhdom AM, Jiang F, Hamdy RC, Benaroch TE, Lavigne M, Saran N. Hip Joint Osteochondroma: Systematic Review of the Literature and Report of Three Further Cases. Adv Orthop. 2014;2014:180254.
- Gore DR. Intra-articular osteochondromas of the hip joint in a child with multiple osteochondromas. Case report. Clin Orthop Relat Res. 1985;199:173–8.
- 62. Duque Orozco M, Abousamra O, Rogers KJ, Thacker MM. Magnetic Resonance Imaging in Symptomatic Children With Hereditary Multipl Exostoses of the Hip. J Pediatr Orthop. 2018;38(2):116–21.
- Yoong P, Mansour R, Teh JL. Multiple hereditary exostoses and ischiofemoral impingement: a case– control study. Skelet Radiol. 2014;43(9):1225–30.
- 64. Bernard SA, Murphey MD, Flemming DJ, Kransdorf MJ. Improved differentiation of benign osteochondromas from secondary chondrosarcomas with standardized measurement of cartilage cap at CT and MR imaging. Radiology. 2010;255(3):857–65.
- Gilday DL, Ash JM. Benign bone tumors. Semin Nucl Med. 1976;6(1):33–46.
- Jackson RL, Llaurado JG. Visualization by dynamic and static osseous scintigraphy of pelvic chondrosarcoma in multiple hereditary exostosis. Clin Nucl Med. 1987;12(2):113–5.
- 67. Bonnomet F, Clavert AFZ, Gicquel P, Clavert JM, Kempf JF. Hip arthroscopy in hereditary multiple

exostoses: A new perspective of treatment. Art Ther. 2001;17(9):E40.

- Guindani N, Eberhardt O, Wirth T, Surace MF, Fernandez FF. Surgical dislocation for pediatric and adolescent hip deformity: clinical and radiographical results at 3 years follow-up. Arch Orthop Trauma Surg. 2017;137(4):471–9.
- Jellicoe P, Son-Hing J, Hopyan S, Thompson GH. Surgical hip dislocation for removal of intraarticular exostoses: report of two cases. J Pediatr Orthop. 2009;29(4):327–30.
- Ettl V, Siebenlist S, Rolf O, Kirschner S, Raab P. Intraacetabular localisation of an osteochondroma causing subluxation of the hip joint. Z Orthop Ihre Grenzgeb. 2006;144(1):87–90.
- Woodward M, Daly K, Dodds R, Fixsen J. Subluxation of the Hip Joint in Multiple Hereditary Osteochondromatosis: Report of Two Cases. J Pediatr Orthop. 1999;19(1):119–21.
- Hussain W, Avedian R, Terry M, Peabody T. Solitary osteochondroma of the proximal femur and femoral acetabular impingement. Orthopedics. 2010;33(1):51.
- Shin SJ, Kwak HS, Cho TJ, Park MS, Yoo WJ, Chung CY, Choi IH. Application of Ganz surgical hip dislocation approach in pediatric hip diseases. Clin Orthop Surg. 2009;1(3):132–7.
- Viala P, Vanel D, Larbi A, Cyteval C, Laredo JD. Bilateral ischiofemoral impingement in a patient with hereditary multiple exostoses. Skelet Radiol. 2012;41:1637–40.
- 75. Li M, Luettringhaus T, Walker KR, Cole PA. Operative treatment of femoral neck osteochondroma through a digastric approach in a pediatric patient: a case report and review of the literature. J Pediatr Orthop B. 2012;21(3):230–4.
- Nisar A, Gulhane S, Mahendra A, Meek RM, Patil S. Surgical dislocation of the hip for excision of beningn tumours. J Orthop. 2014;11:28–36.
- Rebello G, Spencer S, Millis MB, Kim YJ. Surgical dislocation in the management of pediatric and adolescent hip deformity. Clin Orthop Relat Res. 2009;467(3):724–31.
- Sorel J, Schaeffer F, Homan AS, Scholtes VAB, Kempen DHR, Ham SJ. Surgical hip dislocation according to Ganz for excision of osteochondromas in patients with multiple hereditary exostoses. Bone Joint J Surg B. 2016;98-B(2):260–5.
- Chana-Rodríguez F, López-Capapé D, Rojo-Manaute J, Vaquero-Martín J, Alonso-Martín JM, González-Santander M, Villanueva-Martínez M. Ludloff approach for osteochondroma in the lesser trochanter in a young middle-distance runner. Eur Orthop Traumatol. 2011;2:83.
- Yu Y, Sun XB, Song XH, Tian Z, Zhou YJ. A novel surgical approach for the treatment of tumors in the lesser trochanter. Experiment Therapeut Med. 2015;10:201–6.
- Fitzgerald CW, Rowan FE, O'Neill SC, Mulhall KJ. A mountain among molehills: removing an

impinging large femoral neck osteochondroma in a man with hereditary multiple exostoses. BMJ Case Rep. 2014;09:2014.

- 82. Muzaffar N, Bashir N, Baba A, Ahmad A, Ahmad N. Isolated osteochondroma of the femoral neck presenting as hip and leg pain. A case study. Ortop Traumtol Rehabil. 2012;14(2):183–7.
- Inoue S, Noguchi Y, Mae T, Rikimaru S, Hotokezaka S. An external snapping hip caused by osteochondroma of the proximal femur. Mod Rheumatol. 2005;15(6):432–4.
- Bloem JL, Reidsma II. Bone and soft tissue tumors of hip and pelvis. Eur J Radiol. 2012;81(12):3793–801.
- Tachdjian MO. Pediatric orthopedics, vol. 2. Philadelphia: W. B. Saunders Company; 1990. p. 1172–90.
- 86. Ikeuch K, Hasegawa Y, Sakano S, Seki T, Matsuoka A. Eccentric rotational acetabular osteotomy for osteoarthritis of the hip due to hereditary multiple exostosis: report of two cases. J Orthop Sci. 2014;19(5):847–50.