Traumatic Avascular Necrosis of the Femoral Head

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14.1 Introduction

Avascular necrosis (AVN) of the femoral head is a rare but devastating complication following fractures of the proximal femur or hip dislocation. Traumatic AVN derives from the interruption of the blood flow to the femoral head, which is mainly supplied by the medial circumflex femoral artery (MCFA) in the adult hip [1, 2]. The incidence of AVN depends on the fracture pattern and the integrity of the deep branch of the MCFA. While fractures with close proximity to the nutrient vessels (femoral head and neck fractures) have a considerable risk for AVN, AVN rarely occurs in fractures which do not interfere with the MCFA (intertrochanteric or femoral shaft fractures). In addition to traumatic interruption of the femoral head blood supply, the MCFA can be injured iatrogenically. Symptoms and radiographic changes in hips with AVN of the femoral head usually occur late and often months after the trauma. There is no curative treatment and therefore prevention is most important.

This article (1) describes the pathophysiology of AVN with a special focus on the vascular anatomy of the femoral head, (2) reports on the different modalities to assess the integrity of the nutrient vessels, (3) compares the different injury patterns and their associated risk of AVN, and (4) provides information to prevent iatrogenic damage to the blood supply of the femoral head.

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14.2 Blood Supply to the Femoral Head

14.2.1 Vascular Anatomy of the Femoral Head

Precise knowledge of the vascular anatomy of the femoral head helps to understand the etiology of traumatic AVN. The deep branch of the MCFA provides the main blood supply to the femoral head [1, 2]. Most commonly, it arises from the deep femoral artery or, alternatively, from the common femoral artery (Fig. 14.1). There are five constant branches of the MCFA. Of these, the most relevant for the femoral head perfusion is the deep branch of the MCFA [1, 2]. It runs posteriorly towards the intertrochanteric crest between the pectineus muscle medially and the iliopsoas tendon laterally (Fig. 14.1). The deep branch of the MCFA then runs along the inferior border of the obturator externus muscle, which is the most important structure to protect the course of the MCFA (Fig. 14.1). As long as the obturator externus muscle is in continuity, the deep branch of the MCFA is not under relevant tension even with the femoral head dislocated [1]. After crossing the obturator externus tendon posteriorly, a constant trochanteric branch is given off which runs between the quadratus femoris muscle and the triceps coxae (gemelli and obturator internus muscles; Fig. 14.1). The deep branch of the MCFA continues cranially and ventrally to the triceps coxae muscles and penetrates the joint capsule at the level of the superior border of the gemellus superior muscle (Fig. 14.1). At the posterosuperior aspect of the femoral neck, it splits up into four to five retinacular vessels (Fig. 14.1). The retinacular vessels lie extraosseously but intracapsularly and enter the head 2–4 mm lateral to the bone-cartilage junction.

Multiple anastomoses with the MCFA exist [1]. The most important anastomosis regarding femoral head perfusion is a branch of the inferior gluteal artery, which runs along the

K.-H. Koo et al. (eds.), *Osteonecrosis*,
DOI 10.1007/978-3-642-35767-1_14,

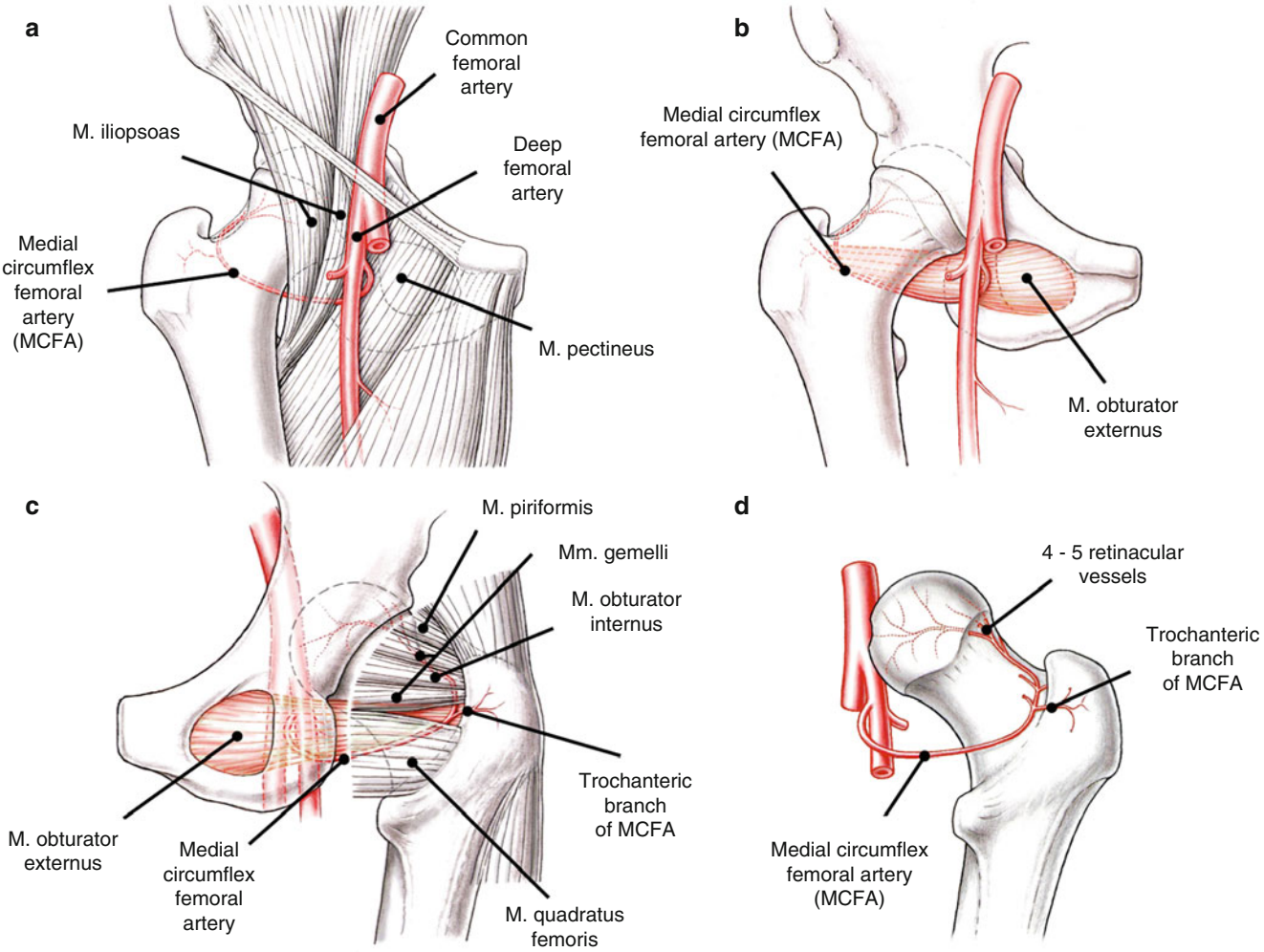

Fig. 14.1 Vascular anatomy of the femoral head. (**a**) The deep branch of the medial circumflex femoral artery (*MCFA*) runs towards the intertrochanteric crest between the pectineus muscle medially and the iliopsoas tendon laterally. (**b**) The MCFA then runs along the inferior border of the obturator externus muscle. (**c**) After crossing the obturator externus tendon posteriorly, a trochanteric branch is given off which runs between the quadratus femoris muscle and the triceps coxae (gemelli and obturator internus muscles). The deep branch of the MCFA continues cranially and ventrally to the triceps coxae muscle and enters the joint capsule at the level of the superior border of the gemellus superior muscle. (**d**) At the posterosuperior aspect of the femoral neck, it splits up into four to five retinacular vessels which enter the head (Reprinted with permission Tannast et al. [3])

piriformis muscle [1, 4]. It could be shown that this vessel has the capacity to compensate for an interruption of the blood supply of the afferent portion of the MCFA [5]. The lateral circumflex femoral artery or the ligamentum teres arteries contribute very little to head perfusion [2]. Usually, the intraosseous blood flow cannot compensate for an intracapsular injury to the deep branch of the MCFA. Therefore, in clinical practice, viability of the femoral head directly depends on the integrity of the MCFA.

14.2.2 Assessment of the Integrity of the Nutrient Vessels of the Femoral Head

Damage to the MCFA can theoretically occur by direct traumatic injury from the accident (i.e., rupture of the vessel), kinking due to fracture and/or joint dislocation, thrombosis, vasospasm, or iatrogenic injury (e.g., forced closed reduction, surgical approach, hardware insertion).

14.2.2.1 Preoperative Modalities for Detection of an Interrupted Blood Supply to the Femoral Head

A reliable, clinically routinely usable, preoperative technique to diagnose an injury of the MCFA with interruption of the blood supply to the femoral head does not exist. Direct visualization of the nutrient vessels to the femoral head is possible by angiography [5, 6]. It can show the interruption of the MCFA but is unable to show the ultimate perfusion of femoral head. Two historic studies [5, 6] tried to correlate the angiographic results in proximal femoral fractures with the occurrence of

AVN at most recent follow-up. In hips with preserved blood supply to the femoral head in the angiography, AVN occurred very rarely. In contrast, if the preoperative blood supply to the femoral head was interrupted in the angiography, this did not necessarily end up in an AVN. In these cases, the blood supply can sometimes even be restored by simple closed internal rotation of the hip in femoral neck fractures or after open reduction and internal fixation [6, 7]. This supports the theory of a transient kinking and/or vasospasm of the nutrient vessels. Despite its utility, angiography has not become part of the routine clinical follow-up. Nowadays, CT angiography may be a promising noninvasive alternative to conventional angiography [8]. The MCFA and its anastomoses can be visualized in detail with this technique [8]. However, similar to classic angiography, the terminal intraosseous blood flow to the femoral head is not visible. The usability of this technique in clinical routine has not yet been proven.

There are two imaging modalities that can show the preoperative femoral head vascularity with proximal femoral fractures. The first modality is the dynamic magnetic resonance imaging (MRI), which monitors quantitatively the flow of an intravenously applied contrast agent in the femoral head [9]. The signal intensity of the fractured side before and after application of gadolinium is then compared to the unaffected side. Similarly, bone scintigraphy (as the second modality) uses an intravenously applied radiographic tracer [10, 11]. Unlike dynamic MRI, this method reveals more qualitative results. Despite relative promising results of these two techniques, neither has found its way into clinical routine use.

14.2.2.2 Intraoperative Assessment

One of the most reliable and technically simple methods to assess femoral head perfusion is intraoperative drilling of the femoral head [7]. After reduction of the fracture, two to four 2.0 mm drill holes are made at the base of the femoral head to assess femoral head bleeding. This requires an open approach to the hip and usually takes up to 2 min until reliable bleeding from the femoral head may be observed. If a closed reduction and percutaneous fixation is attempted, the retrograde blood flow through the cannulated screws can provide a relatively reliable assessment of the femoral head perfusion [12]. More sophisticated methods such as laser Doppler flowmetry [13] and intramedullary oxygen tension measurements [14] have been described but basically offer a lower sensitivity and specificity for prediction of AVN in comparison to direct drilling of the femoral head.

14.3 Injury Patterns of the Proximal Femur

The incidence of traumatic AVN of the femoral head depends on the fracture pattern and its proximity to the deep branch of the MCFA (Table 14.1). While the femoral head and neck fractures have the highest incidence of AVN (up to 40 % [18]), the incidence decreases for intertrochanteric fractures (1–5 % [17, 18, 23]), and AVN rarely occurs in femoral shaft fractures [24] (Table 14.1). Femoral head necrosis has also

Table 14.1 Selected literature reporting on incidence of avascular necrosis of the femoral head following fractures of the proximal femur or traumatic hip dislocation

Study	Fracture pattern	Treatment	Number of hips (patients)	Mean age with range (year)	Incidence of AVN (%)	Results
Giannoudis et al. [15]	Head	23 % nonsurgical 77 % surgical	453 (450)	39 (6–81)	12	Systematic review 3.7 and 2.2 times increased risk for AVN for posterior or trochanteric flip approach compared to anterior approach
Guo et al. [16]	Head	18 % nonsurgical 82 % surgical	176 (176)	n.a.	13	Systematic review Incidence of AVN of 16 % for posterior, 13 % for trochanteric flip, and 8 % for anterior approach
Moon and Mehlmann [17]	Head, neck, intertrochanteric	Surgical and nonsurgical	360 (360)	10 (1–16)	21[a] I: 38 II: 28 III: 18 IV: 5	Systematic review Fracture type[a], displacement, age, and treatment (open vs. closed reduction) are risk factors for AVN
Yeranosian et al. [18]	Head, neck, intertrochanteric	Surgical and nonsurgical	n.a. (935)	n.a. (1–19)	23[a] I: 40 II: 27 III: 20 IV: 5	Systematic review Fracture type[a], treatment (open vs. closed reduction), delay in treatment (>24 h), and use of fixation are risk factors for AVN; no association with decompression

(continued)

Table 14.1 (continued)

Study	Fracture pattern	Treatment	Number of hips (patients)	Mean age with range (year)	Incidence of AVN (%)	Results
Garden [19]	Neck	100 % surgical	406 (n.a.)	n.a.	23	Case series Incidence of AVN depends on quality of reduction; definition of alignment index; no AVN inside normal range of index; 100 % incidence of AVN with varus (<150°) or valgus (>185°) malreduction
Holmberg et al. [20]	Neck	3 % nonsurgical 97 % surgical	2,251 (n.a.)	74 (39–99)	12	Case series Increased incidence of AVN in hips with operative treatment (12 %) compared to nonoperative treatment (2 %)
Johnson and Crothers [21]	Neck	26 % pinning 74 % intramedullary nailing	153	72 (n.a.)	14	Case series Increased incidence of AVN in hips with intramedullary nailing (16 %) compared to pinning (8 %)
Strömqvist et al. [22]	Neck	100 % surgical	n.a. (300)	78 (18–98)	7	Case series Increased incidence of AVN in hips with displaced neck fractures (9 %) compared to non-displaced fractures (3 %)
Aguado-Maestro et al. [23]	Intertrochanteric	100 % PFN	n.a. (200)	n.a.	1	Case series Patient series with intertrochanteric fractures treated with PFN and 1 out of 200 with AVN
Orler et al. [24]	Shaft	100 % intramedullary nailing	17 (17)	13 (8–15)	n.a.	Systematic review of case reports with AVN following intramedullary nailing of femoral shaft fractures
Brav [25]	Dislocation	93 % closed reduction 7 % ORIF	523 (517)	25 (3–75)	26	Case series Incidence of AVN depends on direction of dislocation, associated fractures, delay in treatment (>12 h)
Epstein [26]	Dislocation	Closed reduction and ORIF	242 (242)	n.a.	18	Case series Incidence of AVN depends on associated fractures
Hougaard and Thomsen [27]	Dislocation	Closed reduction and ORIF	100 (98)	39 (16–89)	13	Case series Incidence of AVN depends on delay of treatment (>6 h)

n.a. not available, *AVN* avascular necrosis, *PFN* proximal femoral nailing, *ORIF* open reduction internal fixation
[a]Incidence for the type of Delbet classification [28]: I = head, II = medial neck, III = lateral neck, and IV = intertrochanteric fracture

been reported following traumatic dislocations with an incidence ranging up to 26 % [25–27].

Fractures of the proximal femur are divided into head, neck, intertrochanteric, and femoral shaft fractures. For each fracture pattern, the etiology of AVN, the anatomical relation to the MCFA, and the incidence of AVN reported in literature are summarized.

14.3.1 Femoral Head Fractures

Among the different fracture types of the proximal femur, femoral head fractures have the highest incidence for AVN ranging up to 40 % (Table 14.1) [18]. Femoral head fractures typically are related to direct mechanical damage to the intraosseous blood flow from the retinacular vessels (Fig. 14.2). The vascularity of the femoral head fragment can only be reestablished by diffusion from the viable femoral head portion. Based on two systematic reviews [15, 16], an increased risk for AVN has not been found for a specific subtype of femoral head fracture or concomitant traumatic dislocation. However, in both reviews [15, 16], the risk of AVN was associated with the type of approach used for surgical treatment (Table 14.2). The highest incidence of 16 % was found for the posterior approach, followed by the trochanteric flip approach with 13 % and the anterior approach with 8 % [15, 16]. These results suggest that an iatrogenic lesion to the nutrient vessels of the femoral head may play an important role. The posterior approach to the hip can potentially be dangerous for the blood supply to the femoral head if the topographical course of the MCFA is not fully understood and respected (Fig. 14.1).

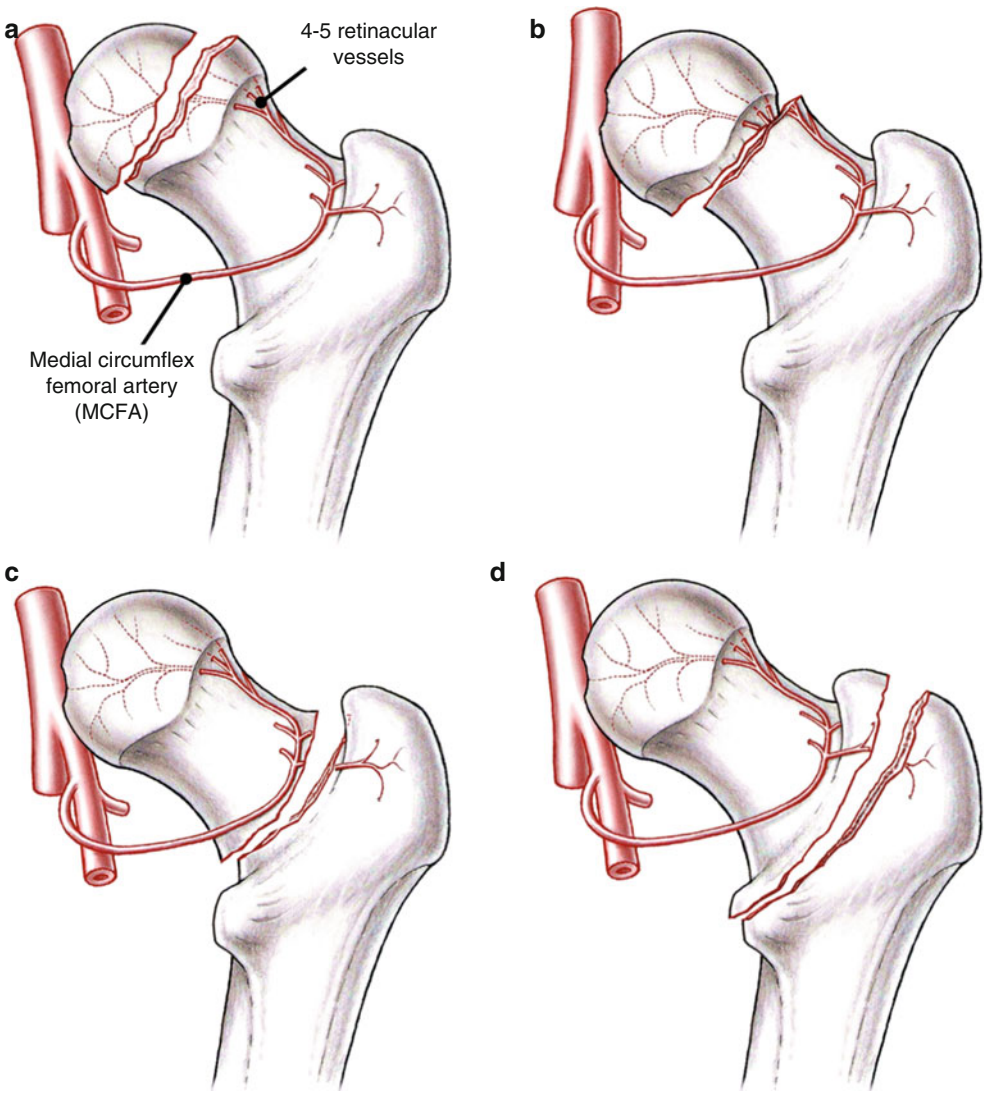

Fig. 14.2 (**a**) Femoral head fractures have the highest incidence of avascular necrosis (AVN) of the femoral head ranging up to 40 % [18] and typically are related to direct mechanical damage to the retinacular vessels. (**b**) In medial neck fractures, direct mechanical trauma to the nutrient vessel results in increased risk for AVN compared to (**c**) lateral neck fractures which less likely interfere with the MCFA. (**d**) AVN rarely occurs in hips with intertrochanteric fractures because these fractures usually do not interfere with the nutrient vessel

If a trochanteric osteotomy is conducted, the osteotomy should exit just anterior to the most posterior insertion of the gluteus medius muscle in order to protect the deep branch of the MCFA [34].

14.3.2 Femoral Neck Fractures

AVN in femoral neck fractures is typically the result of an injured retinaculum (Fig. 14.2). Several risk factors have been reported that can be attributed to the integrity of the retinacular vessels. This includes the location of neck fracture [17, 18], fracture dislocation [13, 17, 22], quality of reduction [19], delay of treatment [18, 29], and type of treatment [17, 18, 20, 21] (Table 14.2).

14.3.2.1 Location of Femoral Neck Fracture

Medial femoral neck fractures have an increased incidence of AVN (28 %) in comparison to lateral neck fractures (18 %, Table 14.1) [17, 18]. The increased incidence for medial neck fractures can be explained by the very close topographical relationship of the retinacular vessels and the fracture (Figs. 14.2 and 14.3). Lateral neck fractures show an increased distance between the fracture and the terminal branches of the MCFA; therefore, the fracture has less potential to damage the vessel (Fig. 14.2).

14.3.2.2 Fracture Dislocation

Fracture dislocations of the femoral neck can be classified as varus/valgus, dorsal/ventral, and rotational (Fig. 14.4).

A varus dislocation without dorsal angulation inevitably causes tension on the retinacular vessels (Fig. 14.4). This can lead to an impairment of the femoral blood supply [6, 13]. Eventually, the vulnerable retinaculum ruptures with large dislocations. A valgus impaction without dorsal angulation theoretically relaxes the retinaculum (Fig. 14.4). Isolated valgus impactions up to 30° do not impair the blood supply to the femoral head [6]. However, large valgus impactions are associated with AVN [19] and attributed to kinking of the retinacular vessels.

Table 14.2 Fracture pattern with corresponding risk factors and incidence of avascular necrosis of the femoral head

Fracture pattern	Risk factor for AVN of femoral head	Description/incidence of AVN
Head	Surgical approach [15, 16]	Posterior approach with incidence of 16 %, trochanteric flip with 13 %, and anterior approach with 8 %
Neck	Location of fracture [17, 18]	Medial neck fractures with incidence of 28 % compared to lateral fractures with 18 %
	Fracture dislocation [13, 17, 22]	Adults: dislocated fractures with incidence of 9 % compared to non-dislocated fractures with 3 %
		Children: dislocated fractures with incidence of 25 % compared to non-dislocated with 9 %
	Quality of reduction [19]	Anatomical reduction with incidence of 7 % compared to severe valgus/varus with 100 %
	Delay of treatment [18, 29]	Adults: increased incidence of 16 vs. 0 % for a delay of >12 h
		Children: four times increased risk for a delay of >24 h
	Type of treatment [17, 18, 20, 21]	Adults: incidence of 12 % for surgical treatment vs. 2 % for nonsurgical treatment, 16 % for open reduction vs. 8 % for closed reduction
		Children: 2.5 times increased risk for surgical vs. nonsurgical treatment, 2.7 times increased risk for open vs. closed reduction
Intertrochanteric	n.a.	
Shaft	Young age [24, 30, 31]	Patients aged between 8 and 15 years; only two case reports with patients older than 20 years
	Antegrade femoral nailing [32]	Nail insertion in the piriformis fossa results in complete disruption of the MCFA in 57 %
		In all case reports nail insertion was performed with reaming of the femoral medullary canal
		Implants designed for adults but used in children
Dislocation	Direction of dislocation [25]	Posterior dislocations with incidence of 29 % compared to 9 % for anterior dislocations
	Concomitant fracture [25, 26]	Dislocation without fracture with incidence of 22 % compared to 60 % for concomitant femoral head or acetabulum fracture
	Delay of reduction [25, 27]	Increased incidence of 59 vs. 5 % for a delay of >6 h or 57 vs. 18 % for a delay of >12 h
	Surgical approach [33]	Three times increased risk for ORIF using posterior vs. anterior approach

AVN avascular necrosis, *MCFA* medial circumflex femoral artery, *ORIF* open reduction internal fixation, *n.a.* not available

A dorsal tilt of the femoral head can lead to a kinking of the posterosuperiorly located retinaculum (Fig. 14.4). A reperfusion of the femoral head can be achieved by simple (closed) internal rotation of the leg [6] or by anatomical open reduction and internal fixation [6, 13]. An additional varus dislocation can aggravate the kinking of the retinacular vessels [6].

Rotational errors at the level of the femoral neck can typically occur when performing closed reduction [35] or by excessive torque from an implant (e.g., a dynamic hip screw [36]). Rotational errors are hard to detect with fluoroscopy or conventional radiographs but can have serious consequences on the retinaculum. A flexion of the head fragment can stretch the retinaculum (Fig. 14.4). However, a large extension of the head fragment may compromise the head vascularity by kinking of the vessel (Fig. 14.4).

14.3.2.3 Quality of Reduction

Quality of reduction was found to be a significant predictor for AVN (Table 14.2) [19]. Severe residual varus and valgus deformities are associated with a higher incidence of AVN [19]. The same principles as mentioned above (see Sect. 14.3.2.2) apply for the integrity of the vascularity.

14.3.2.4 Delay of Treatment and Tamponade Effect

Delay of treatment of more than 12–24 h is associated with a higher risk of AVN (Table 14.2). This could be attributed to a tamponade effect of the retinacular vessels by hemarthrosis. Because the retinacular vessels are extraosseous and intracapsular, they may be compressed by increased hydrostatic pressure resulting from the fracture hematoma. This tamponade effect can occur in both displaced and nondisplaced fractures [37–39]. This would explain the presence of AVN in undisplaced fractures [17]. The time limit of 12 h is somewhat arbitrarily chosen but could be reproduced in a canine animal model [40].

14.3.2.5 Type of Treatment

An increased incidence of AVN was found for neck fractures treated surgically compared to hips with nonsurgical treatment (Table 14.2). Additionally, the incidence of AVN was increased in hips with open reduction and internal fixation compared to hips with closed reduction only (Table 14.2). With an open treatment the MCFA can potentially be iatrogenically damaged. However, type of treatment might also be confounding with previously mentioned risk factors such as location of fracture or fracture dislocation.

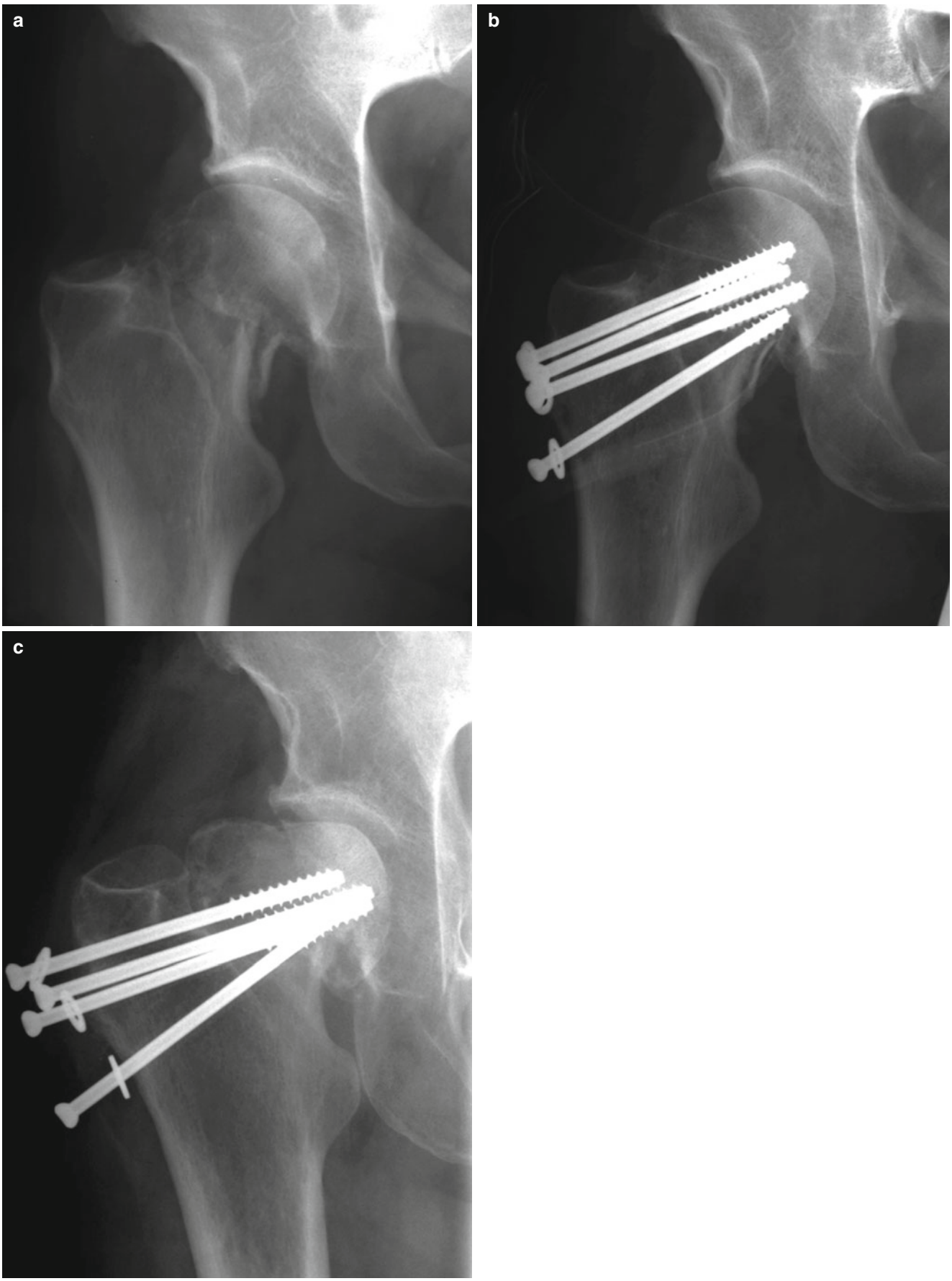

Fig. 14.3 (**a**) A 49-year-old male patient with a medial femoral neck fracture. (**b**) He underwent closed reduction and screw fixation resulting in insufficient reduction and stabilization. (**c**) At a 1.2 year follow-up, avascular necrosis of the femoral head occurred necessitating total hip arthroplasty

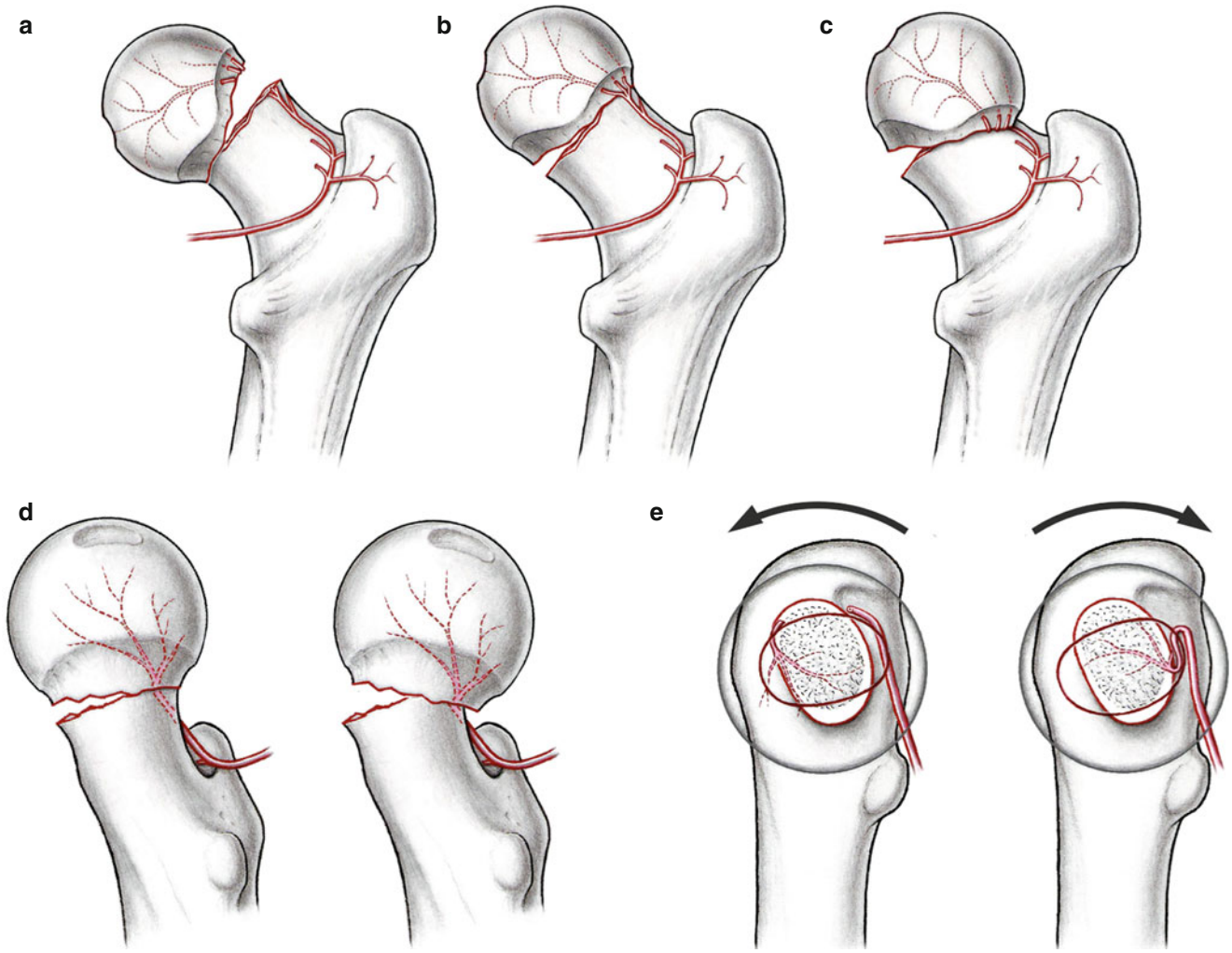

Fig. 14.4 Neck fractures with (**a**) varus dislocation cause tension and possibly rupture of the retinacular vessels. (**b**) A mild valgus dislocation (<30°) relaxes the retinacular vessels and does not impair the blood supply [6]. (**c**) However, a severe valgus dislocation results in kinking of the retinacular vessels and impaired blood supply [19]. (**d**) A mild dorsal tilt usually does not impair perfusion of the femoral head. However, a severe dorsal tilt and, particularly, when in combination with a varus dislocation, leads to impairment of the head perfusion. (**e**) Rotational errors can compromise the head vascularity. Flexion of the head can stretch the retinacular vessels and result in rupture. Extension of the femoral head can result in impaired blood supply by kinking of the vessel

14.3.3 Intertrochanteric and Greater Trochanteric Fractures

Intertrochanteric fractures are rarely associated with AVN, and the incidence ranges from 1 to 5 % (Table 14.1). Generally, these fractures do not interfere directly with the anatomical course of the deep branch of the MCFA in the adult hip (Fig. 14.2). Associated risk factors have therefore not clearly been described based on large patient cohorts. Assumed risk factors that can compromise the vascular supply are high-energy trauma, associated fractures of the base of the femoral neck, and iatrogenic vascular damage [41].

Three case reports exist for isolated fractures of the greater trochanter and femoral head necrosis [42–44]. Two patients were treated conservatively [43, 44], while one patient was treated with open reduction and screw fixation [42]. Interestingly, all cases occurred in children aged between 12 and 13 years. In adults, greater trochanteric fractures typically involve avulsion fractures of the tip. In the pediatric population, the fracture often involves the trochanteric growth plate. This results in a larger fragment of the greater trochanter which extends far medial in the femoral neck [45] and may compromise the blood supply of the MFCA (Fig. 14.5).

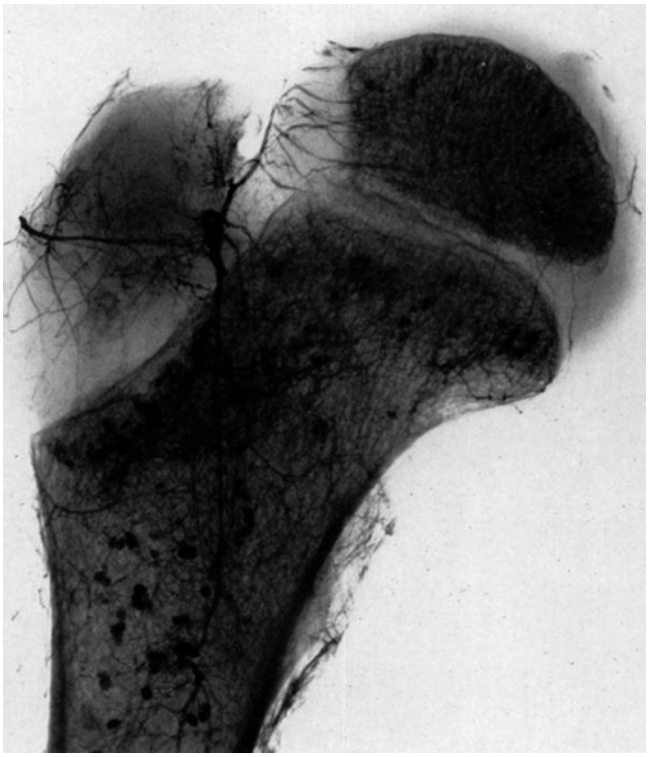

Fig. 14.5 Radiograph of the proximal femur and angiography of the medial circumflex femoral artery (MCFA) of a 6-year-old male. Both the epiphyseal and trochanteric growth plates are clearly visible. All cases of a greater trochanteric fracture resulting in AVN of the femoral head occurred in children. These fractures often involve the trochanteric growth plate. This results in a larger fragment of the greater trochanter which extends far medial in the femoral neck and may compromise the blood supply of the MFCA [45] (Reprinted with permission Trueta [45])

14.3.4 Femoral Shaft Fractures

Although femoral shaft fractures generally do not interfere with the course of the MCFA, a few cases of AVN following antegrade intramedullary nailing of the femur have been reported [24]. In these cases an iatrogenic damage to the MCFA at the location of nail insertion at the proximal femur has been described [32]. For geometrical and biomechanical reasons, the piriformis fossa has been recommended as nail insertion point of straight femoral nails. However, the piriformis fossa has a high risk for iatrogenic damage to the MCFA due to its close proximity (Fig. 14.6). In a cadaver study, nail insertion in the piriformis fossa resulted in damage to the nutrient vessel in all cases with complete disruption of the MCFA in 57 % [32]. In addition, intramedullary femoral reaming may put the vascular supply of the femoral head at risk (Table 14.2). The tip of the greater trochanter was suggested as a more appropriate insertion point with the use of an anatomically shaped femoral nail [32]. The problem

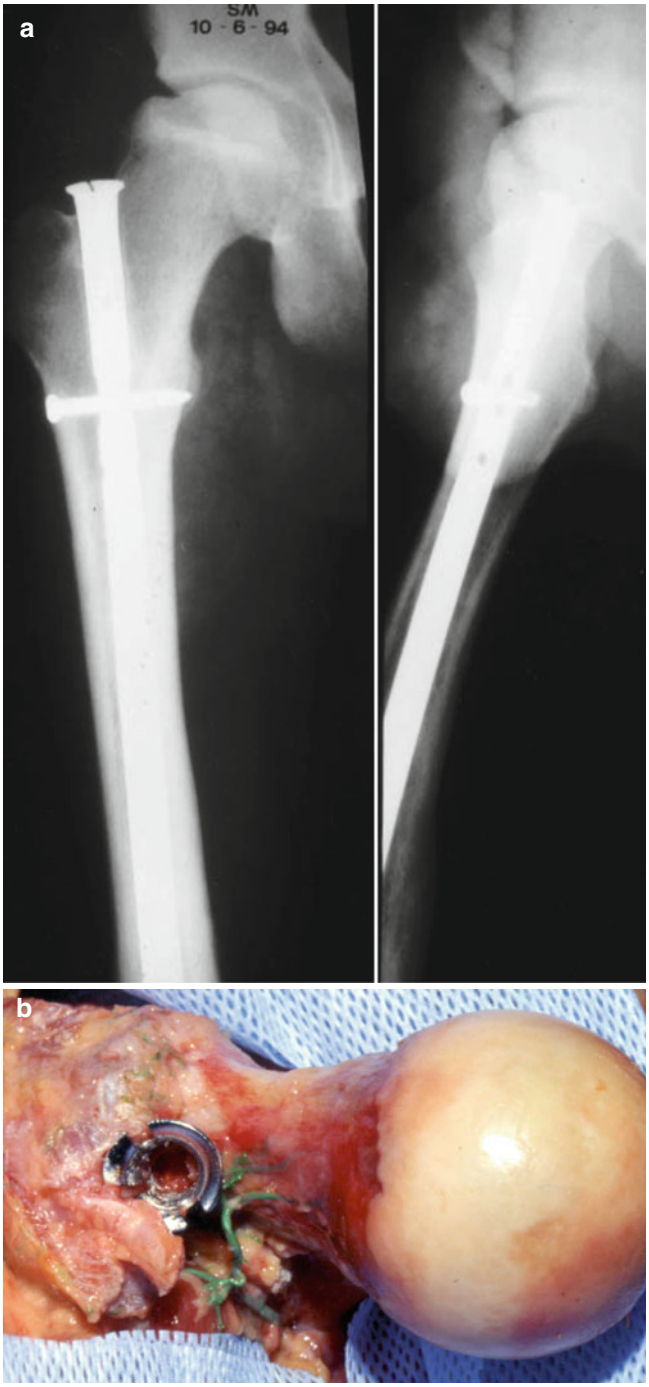

Fig. 14.6 (**a**) Fourteen-year-old male patient who sustained a femoral shaft fracture which was treated with antegrade intramedullary nailing. A very medial nail insertion point was chosen in close proximity to the medial circumflex femoral artery (MCFA). Eight months postoperative, avascular necrosis of the femoral head occurred. (Reprinted with permission Orler et al. [24]) (**b**) The risk of avascular necrosis of the femoral head following antegrade intramedullary nailing of the femur depends on the location of nail insertion [32]. In a cadaver study the use of the piriformis fossa as location of insertion with its close proximity to the MCFA showed damage to the nutrient vessel in all cases with complete disruption of the MCFA in 57% [32] (Reprinted with permission Dora et al. [32])

can be aggravated when oversized implants for adults are used in the pediatric population, which represent the majority of these cases with femoral shaft fractures and AVN of the femoral head [24] (Table 14.2).

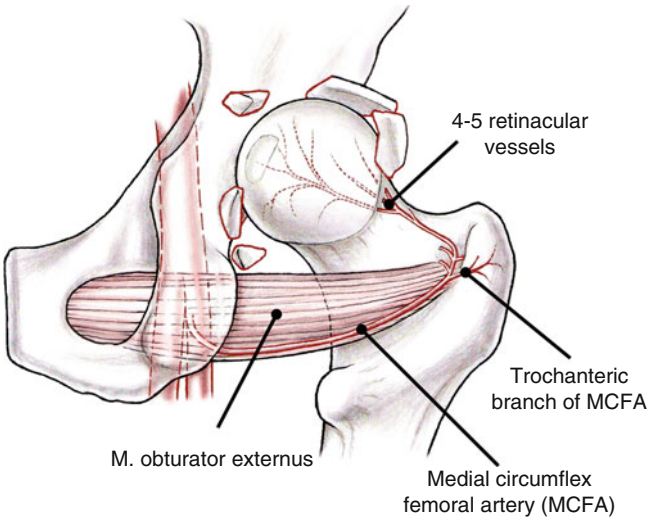

Fig. 14.7 The obturator externus muscle protects the deep branch of the MCFA in hips with traumatic dislocation [1]. Its integrity ascertains blood supply to the femoral head in hips with traumatic dislocation [46]

14.3.5 Traumatic Hip Dislocation

Traumatic hip dislocation is not necessarily associated with AVN. It could be shown that the obturator muscle is the most important structure to protect the MCFA in the dislocated position (Fig. 14.7) [1]. This fact is based on cadaver experiments for surgical hip dislocations [1]. As long as the obturator externus muscle is in continuity, the deep branch of the MCFA is not in danger or under relevant tension even with the femoral head dislocated [34, 47, 48]. Since the obturator externus muscle typically remains intact in traumatic hip dislocations [46], additional factors have to contribute to the reported incidence of AVN up to 26 % (Table 14.1) following traumatic hip dislocations. These additional risk factors for AVN include direction of dislocation [25], concomitant fracture [25, 26], delay of reduction [25, 27], and surgical approach [33] (Table 14.2).

Posterior traumatic hip dislocations have an increased incidence for AVN compared to anterior dislocations (29 vs. 9 %; Table 14.1) [25]. A concomitant femoral fracture may mechanically harm the nutrient vessels to the femoral head. The incidence of AVN for dislocations without fractures is zero to 22 % (Fig. 14.8) [46]. In contrast, the incidence for dislocations with a concomitant femoral head or acetabulum fracture was 66 % [25]. Early reduction may reduce the incidence of AVN [25, 27].

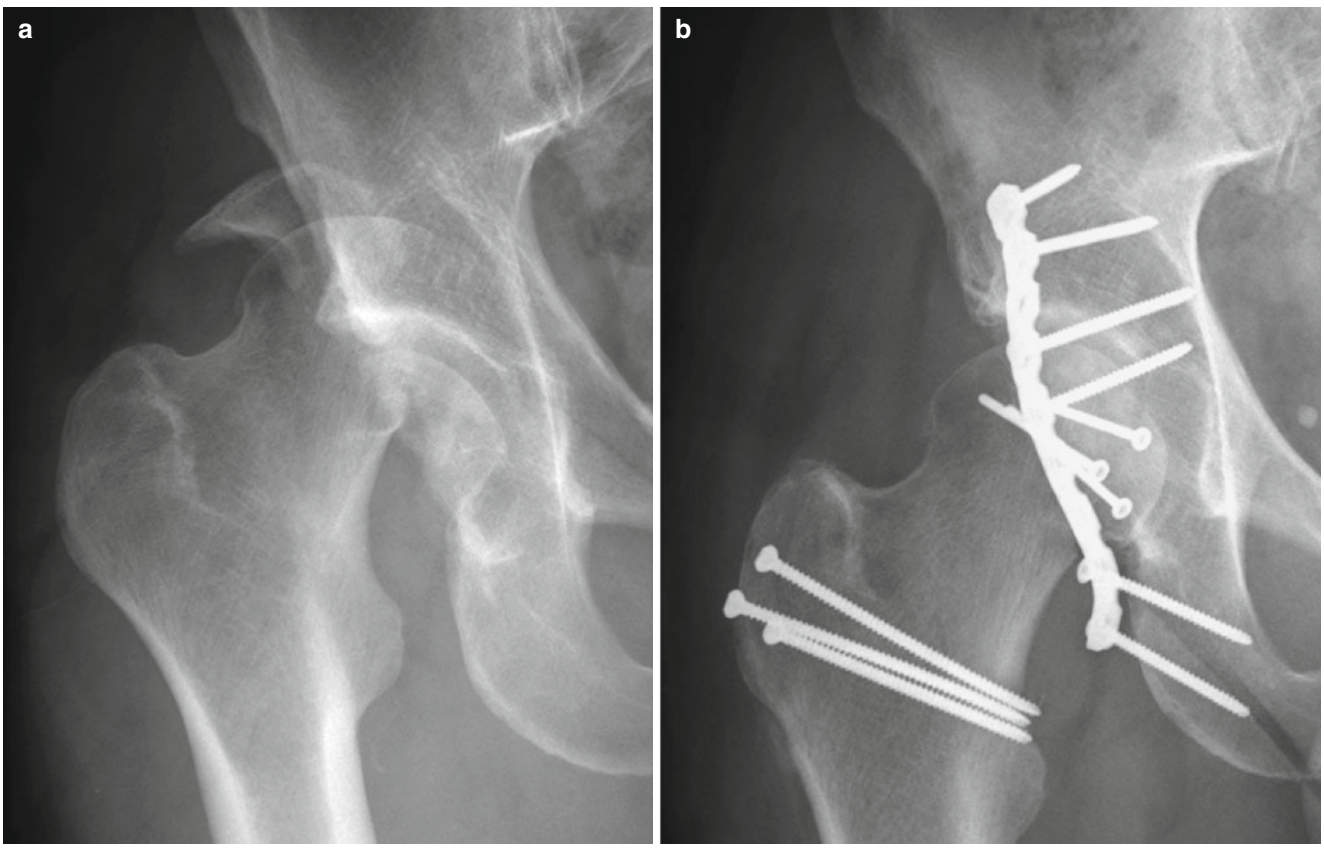

Fig. 14.8 (**a**) A 59-year-old male patient with traumatic posterior hip dislocation with a fracture of the femoral head and posterior acetabular wall. (**b**) Open reduction with internal fixation of the femoral head and posterior wall was performed using a trochanteric flip approach. (**c**) Half a year postoperative, avascular necrosis of the femoral head occurred necessitating total hip arthroplasty

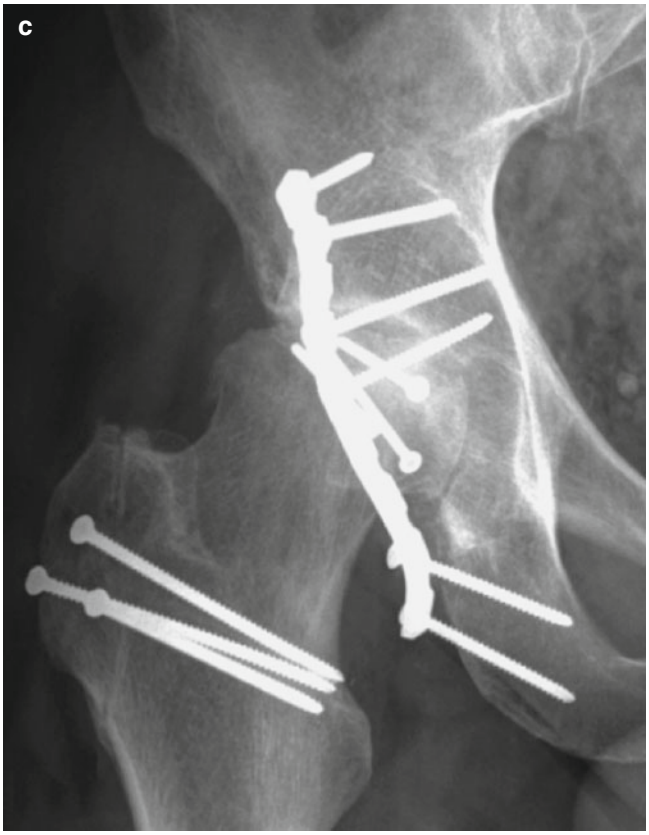

Fig. 14.8 (continued)

Based on laser Doppler studies, the femoral head blood supply can be reduced in the dislocated position, but will normalize after reduction in all hips [47]. With a delay of 6 or 12 h for reduction, the incidence increases from 5 to 59 % [27] or from 18 to 57 % [25], respectively. When performing a standard Kocher-Langenbeck approach (with or without detachment of the short external rotators), the MCFA can potentially be iatrogenically harmed. This could explain the three times higher incidence of AVN in traumatic hip dislocations with a posterior approach for internal fixation in comparison to the anterior approach [33].

14.4 Summary

Traumatic AVN of the femoral head derives from the interruption of the blood supply to the femoral head. The MCFA plays the key role for maintenance of the femoral head viability. The incidence of AVN varies greatly among the different fracture types of the proximal femur. It is directly correlated to the proximity of the fracture site with the topographical course of the MCFA. Preoperative noninvasive assessment of intact vascularity of the femoral head is theoretically possible with various imaging modalities. However, none of these methods has found its way into clinical routine use. The most sensitive and specific method to determine an intact vascularity is bleeding of the femoral head after intraoperative drilling. Lack of bleeding may be due to an interruption of the afferent blood supply to the femoral head, from either a definitive (e.g., rupture) or transient (e.g., vasospasm, kinking) stop of the MCFA.

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