Chapter 17

Complications Following Total Hip Arthroplasty

Asim Rajpura and Tim Board

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/53030

1. Introduction

Total hip arthroplasty (THA) is an increasingly common and successful operation, with 76,759 procedures logged in the National Joint Registry for England and Wales in 2010 [1]. Overall satisfaction rates rank amongst the highest of any joint replacement procedure, with over 90% reporting a good to excellent overall outcome [2, 3].

Complications related to THA can be classified as either procedure specific or systemic. Advances in technology, anaesthesiology and surgical technique have resulted in an overall temporal decrease in complication rates despite the increasing incidence of co-morbidities in the patient population [4]. Table 1 highlights rates of complications most commonly encountered after THA.

2. Systemic / non-surgical

2.1. Thromboembolic complications

Deep Vein Thrombosis

Distal deep vein thrombosis (DVT) can range from being asymptomatic, to resulting in long term valvular damage resulting in chronic venous insufficiency. Proximal propagation can result in more serious pulmonary embolism. The overall incidence of DVT, including both radiologically diagnosed asymptomatic DVT and symptomatic DVT, post THA in early studies was reported to be as high as 70% without any form of prophylaxis [15]. Recent systematic review of several randomised control trials concerned with DVT prophylaxis has estimated this figure to be around 44% [16]. The recent FOTO study has shown a symptomatic DVT rate of 1.3% in THA patients with extended duration [36 day) chemical prophylaxis [8].

The overall combined incidence of asymptomatic and symptomatic DVT with prophylaxis has not declined with time, converse to the findings with knee arthroplasty in which the incidence has declined significantly [17]. This may be due to the increasing frequency of co morbidities within patients undergoing THA which act as risk factors for DVT.

Systemic		Procedure Specific	
Complication	**Rate**	**Complication**	**Rate**
Subclinical Fat Embolism	90%[5]	Dislocation (Posterior approach with repair)	0.49%[6]
Symptomatic Fat Embolism	Unknown	Leg length discrepancy (patient perceived)	30%[7]
Symptomatic Deep Vein Thrombosis with prophylaxis	1.3%[8]	Infection	1.08%[9]
Symptomatic Pulmonary Embolism with prophylaxis	0.5 – 0.6%[10]	Aseptic Loosening	2% failure rate at 15 years (Corail uncemented stem) [11] 3.2% failure rate at 30 years (Exeter Cemented Stem)[12]
Mortality	0.29 – 0.6%[4]	Periprosthetic Fracture (Postoperative femoral)	1.1%[13]
Myocardial Infarction	0.5%[10]	Heterotopic Ossification (Grade III/IV)	3 – 7%[14]

Table 1. Complication rates for Total Hip Arthroplasty

THA is thought to mainly affect 2 limbs of Virchow's triad, namely hypercoagulability and venous stasis. Activation of the coagulation cascade begins during surgery, primarily during preparation and insertion of the femoral prosthesis, with cemented prostheses providing a greater stimulus than uncemented implants [18]. Whether this increases the incidence of DVT with cemented fixation is unclear as the evidence is inconclusive [15, 19]. Venous haemodynamics are also altered not only during surgery, but also for up to 6 weeks post operatively [20]. Significant reductions in venous capacitance and outflow are seen in both legs, with greater changes seen in the operated leg, and this has been shown to correlate directly with the incidence of postoperative DVT [20]. Complete femoral vein occlusion has also been noted during THA, particularly during the posterior approach when the limb is internally rotated and flexed for operation on the femur [18].

Numerous risk factors for postoperative DVT have been identified. Major risk factors in approximate order of importance include: hip fracture, malignancy, antiphospholipid syndrome, immobility, previous history of DVT, use of selective oestrogen receptor modulators, oral contraceptives, morbid obesity, stroke, atherosclerosis and a ASA greater than 3 [21]. However 50% of patients who develop DVT have no identifiable clinical risk factor [21]. Genetic predispositions include antithrombin III and protein C deficiency and prothrombin

gene mutation [21]. In order to aid recognition of 'at risk' patients, the National Institute for Health and Clinical Excellence (NICE) has published a table of relevant patient related risk factors as shown in Table 2 [16].

Active cancer or cancer treatment	Active heart or respiratory failure
Acute medical illness	Age over 60 years
Antiphospholipid syndrome	Behcet's disease
Central venous catheter in situ	Continuous travel of more than 3hours approximately 4weeks before or after surgery
Immobility (for example, paralysis or limb in plaster)	Inflammatory bowel disease
Myeloproliferative diseases	Nephrotic syndrome
Obesity (body mass index > 30kg/m2]	Paraproteinaemia
Paroxysmal nocturnal haemoglobinuria	Personal or family history of VTE
Pregnancy or puerperium	Recent myocardial infarction or stroke
Severe infection	Use of oral contraceptives or hormonal replacement therapy
Varicose veins with associated phlebitis	
Inherited Thrombophilias for example:	
High levels of coagulation factors (for example, Factor VIII)	Hyperhomocysteinaemia
Low activated protein C resistance (for example, Factor V Leiden)	Protein C, S and antithrombin deficiencies
Prothrombin 2021A gene mutation	

Table 2. Patient related risk factors for Venous Thromboembolism [16]

Prophylaxis against DVT begins with the type of anaesthesia used. Regional compared to general anaesthesia has been shown to reduce the risk of DVT post THA by over 50% [22, 23]. This is thought to be due to the relative hyperkinetic blood flow seen in the lower limbs during regional anaesthesia compared to general anaesthesia, and the stabilising effect of local anaesthetics on the cell membranes of vascular endothelium and platelets [24]. Mechanical and chemical prophylaxis remains a somewhat contentious issue, with various differing opinions existing regarding prophylaxis regimes. Numerous randomised controlled trials exist supporting the use of mechanical methods such as pneumatic compression devices (figure 1) and chemical methods such as low molecular weight heparins and fondaparinux, a factor Xa inhibitor [25-34].

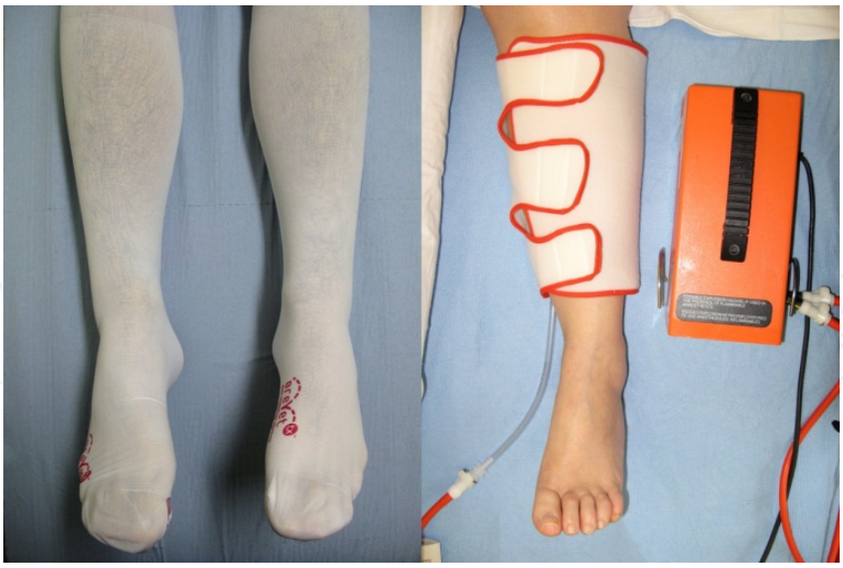

Figure 1. Left: Thrombo-Embolic Deterrent Stockings, Right: Flotron pumps (Huntleigh Healthcare Ltd, Luton, UK)

There has been recent increasing interest in oral factor Xa inhibitors such as Rivaroxaban and Apixaban. The RECORD trial has demonstrated greater effectiveness for oral Rivaroxaban compared to subcutaneous Enoxaparin with equal side effect profiles [35, 36]. Pooled analysis of the ADVANCE-2 and ADVANCE-3 trials has also demonstrated greater efficacy for oral Apixaban compared to subcutaneous Enoxaparin [37]. There has been some recent concern however regarding the increased rate of wound complications, specifically with the use of Rivaroxaban [38]. A retrospective analysis by Jensen et al. demonstrated a greater return to theatre rate for wound complications such as prolonged drainage and haematoma associated with the use of Rivaroxaban compared to Tinzaparin [38]. The authors suggest that trial data to date has not fully evaluated the complications profile of Rivaroxaban, as only major bleeding was used as a primary outcome measure, and further randomised trials are necessary to examine rates of surgical complications.

Current recommendations by National Institute for Health and Clinical Excellence (NICE) in England state that, THA patients should be offered mechanical prophylaxis in the form of intermittent pneumatic compression devices or compression stockings, and chemical prophylaxis with either low molecular weight heparin, Fondaparinux, Rivaroxaban or Dabigatran. This should be continued for 28-35 days post operatively [39].

2.2. Pulmonary embolism

DVTs that propagate proximally have the potential embolise to the lungs resulting in pulmonary emboli (PE). Mild emboli can be asymptomatic, whereas massive embolism can be fatal, and PE is one of the leading causes of mortality post THA. Rates for symptomatic pulmonary embolism in recent large case series of primary THA in which chemical prophylaxis was used, has been between 0.51-0.6% [10, 40]. In the absence of prophylaxis this is estimated to be around 3%, with approximately 6% of symptomatic PEs post THA result in fatality [16].

http://dx.doi.org/10.5772/53030

As with DVT, both mechanical and chemical methods such as pneumatic compression pumps and low molecular weight heparins have been shown to provide effective prophylaxis against symptomatic PE [25, 41-44]. However due to the low rate of fatal PE, trials and even meta-analyses have failed to demonstrate statistically significant effects on the rate of fatal PE by using thromboembolic prophylaxis [45]. Power analysis indicates a trial involving 67,000 patients would be needed to demonstrate a statistically significant difference [46].

Vena Caval filters as shown in figure 2 can also be used to prevent migration of venous emboli into the pulmonary circulation. However no RCTs exist supporting their use in surgical patients and significant complications such as pneumothorax, air embolism and arteriovenous fistulae can develop either during their placement or post procedure [47]. UK NICE guidelines therefore recommend their use in patients with recent or existing thromboembolic disease in whom anticoagulation is contraindicated [39].

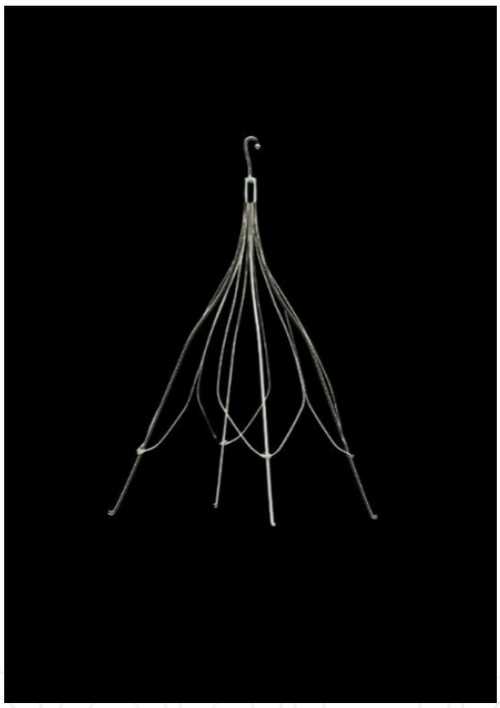

Figure 2. Retrievable Inferior Vena Caval Filter, courtesy of Cook Medical

2.3. Fat embolism

During insertion of the femoral component, rises in intramedullary pressure can force medullary fat and marrow contents into the venous circulation via the metaphyseal vessels [48-50]. Fat and marrow embolus can then pass into and through the pulmonary circulation depending on the size of the emboli [51, 52]. Large emboli can lodge within the pulmonary circulation leading to pulmonary hypertension and haemodynamic instability. Trans-pulmonary passage of micro-emboli can result in cerebral embolism potentially causing neurological complications [51, 53].

Subclinical fat embolisation can been detected in up to 90% of patients undergoing THA [5]. However the exact incidence of fat embolism syndrome characterised by the classic triad of respiratory insufficiency, neurolgic symptoms and upper body petechiae is unknown [54].

Measures to reduce the risk of fat embolism include medullary lavage to reduce the fat load during cement pressurisation [55]. Vacuum cementation techniques using drainage cannulae have also been shown to be effective in reducing the intramedullary pressure rises during cementation therefore reducing the risk of emboli [56]. Treatment of established fat embolism syndrome is essentially supportive, frequently requiring intensive care unit admission for respiratory support.

3. Mortality and cardiorespiratory complications

Published rates for mortality following primary THA are low, ranging from between 0.29% to 0.6% [4, 10]. Mortality rates have declined slightly with time despite the increasing incidence of relevant co-morbidities [4]. Cardiovascular complications account for the most common cause of death [57].

Age has been identified as one of the strongest predictors of post operative mortality after joint arthroplasty [10]. Octegenarians have been shown to have a mortality rate 3.4 times higher than patients between 65-79 years of age and were 2.4 times more likely to suffer a post operative myocardial infarction [58]. Other significant risk factors for post operative mortality and morbidity include male sex, smoking and higher American Society of Anesthesiologists' (ASA) grade which is representative of relevant significant co-morbidities such as artherosclerosis, diabetes, renal impairment and valvular disease [10, 59, 60]. Greenfield et al. found that the incidence of morbidity after THA varied from 3% to 41% when comparing those with the lowest and highest incidence of co-morbidities [61]. The role of anaesthesia is somewhat controversial. Some studies suggest regional compared to general anaesthesia may reduce the risk of thromboembolic and cardiorespiratory complications and short term mortality [62, 63]. Others have shown no difference between the 2 groups in terms of morbidity and mortality [64]. Therefore no conclusive evidence exists supporting one form of anaesthesia, but the overall consensus would appear to favour regional techniques [54].

4. Procedure specific / surgical complications

4.1. Dislocation

Dislocation is the 3rd most common cause for revision after THA [65]. Published rates for dislocation after primary THA vary widely between 0.2% to 7% [66]. Up to 70% of dislocations occur early within 6 weeks [67]. Early dislocation carries a better prognosis compared to late dislocation which is defined as occurring after 3 months, as late dislocation usually

has a multifactiorial aetiology including component wear and soft tissue laxity [68, 69]. Approximately a third of dislocating THAs managed conservatively after the first episode will go on to become recurrent dislocators [67]. Risk factors for dislocation can be classified as either patient, surgery or implant related.

4.2. Patient related risk factors

Patients with neuromuscular and cognitive disorders such as cerebral palsy, muscular dystrophy and dementia, have been shown to have higher rates of dislocation [70]. Fracture as the primary indication for surgery is the indication most strongly linked with dislocation [71]. This is thought to be due to the lack of capsular hypertrophy normally seen with osteoarthritis which provides additional stability. Previous hip surgery of any sort has also been shown to double the risk of dislocation [68]. Factors such as height, weight, age and sex of the patient have not been conclusively shown to affect the rates of dislocation [67].

4.3. Surgical risk factors

Surgical factors include surgical approach, soft tissue tension, component design and orientation, and surgeon experience. The majority of dislocations occur in a posterior direction and therefore the posterior approach has been deemed to be the approach with the highest risk of dislocation. Early data supported this theory with Woo et al reporting a rate of 5.8% for posterior approach compared to 2.3% for an antero-lateral approach [68]. However recent research investigating the role of posterior capsular and external rotator repair has shown comparable rates to other approaches [72, 73]. A recent meta-analysis has shown a reduction of the dislocation rate from 4.46% to 0.49% by carrying out a posterior soft tissue repair [6]. Therefore with meticulous soft tissue repair, surgical approach should have little effect on dislocation rates. Besides the posterior structures, the glutei and joint capsule also provide soft tissue tension reducing dislocation risk. Therefore following a transtrochanteric approach, trochanteric non union greater than 1cm can result in abductor insufficiency increasing the rates of dislocation by over 6 fold [68]. Inadequate offset is another factor affecting soft tissue tension and has been shown to increase dislocation risk [74].

4.4. Implant related factors

Component positioning and design both play key roles in reducing dislocation risk. “Safe zones” for acetabular cup position are defined as an abduction angle of 40° ± 10° and anteversion of 20° ± 10° [75, 76]. With a posterior approach reduced cup anteversion has been shown to be a major risk factor for dislocation [77]. Archbold et al. have suggested the use of the transverse acetabular ligament as a landmark to judge cup anteversion [78]. Using this technique they reported a 0.6% dislocation rate using a posterior approach with soft tissue repair. How this relates to the traditionally defined safe zones is currently being examined. Femoral component positioning has been less well studied. Recent studies have suggested the use of a ‘combined anteversion’ technique in which the acetabular and stem combined anteversion should be 35° ± 10° [79, 80].

Femoral head size also affects stability. Larger heads provide more favourable head-neck ratios, reducing possible impingement, and seat deeper within the acetabulum requiring a greater 'jump distance' to cause dislocation as illustrated in figure 3. Such advantages have been validated using cadaveric and computer modelling [81-83]. Clinical data from both the Norwegian and Australian joint registries has also shown a reduction in rates of revision for dislocation with increasing head size [84, 85].

Surgeon experience is another factor that has been identified in influencing dislocation rates. Hedlundh et al. found that surgeons who had performed less than 30 THAs had a double rate of dislocation compared to more experienced surgeons [86]. A recent systematic review has also demonstrated reduced dislocation rates with increased surgical volume [87].

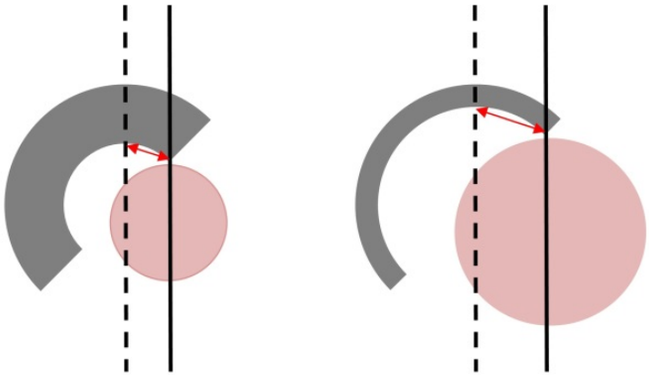

Figure 3. Jumping Distance highlighted by red arrow demonstrates distance the head needs to travel before dislocation occurs. Increasing head size increases this distance

5. Management

Management of dislocation initially involves closed reduction which is usually successful in the majority of cases. This should be performed ideally under anaesthesia with muscle relaxation to reduce the chance of damage to the femoral head [88, 89]. Some surgeons advocate the use of an abduction brace after reduction but little evidence exists supporting their use.

Indications for operative intervention include recurrent or irreducible dislocation, component malposition, soft tissue laxity and dislocation due to impingement. Strategies during revision include component realignment, removal of osteophytes causing impingement, modular component exchange to increase head size and improve head-neck ratio, liner exchange if worn and addressing soft tissue laxity using capsulorrhaphy, trochanteric advancement or tendon allografts.

Selected patients unsuitable for major revision surgery can be treated with posterior lip augmentation devices (PLAD) as shown in figure 4. These consist of a C shaped piece of UHMWPE and a steel backing plate, and are applied to the posterior lip of the acetabulum and held in place with up to 5 screws. This constrains the head within the augmented sock-

et. Contraindications to its use include gross component malalignment and loosening. McConway et al. reviewed 307 recurrently dislocating THAs treated with PLADs [90]. Persistent instability occurred in only 5 patients [1.6%) and there was no evidence of accelerated loosening affecting the acetabular component.

Salvage procedures for failed revision or uncorrectable aetiology include the use of constrained cups or conversion to bipolar hemiarthroplasty. However both of these procedures are associated with poor functional outcome and constrained cups can result in premature loosening [70]. Therefore their use is usually reserved for low demand patients. The final salvage option is Girdlestone resection for the unreconstructable hip.

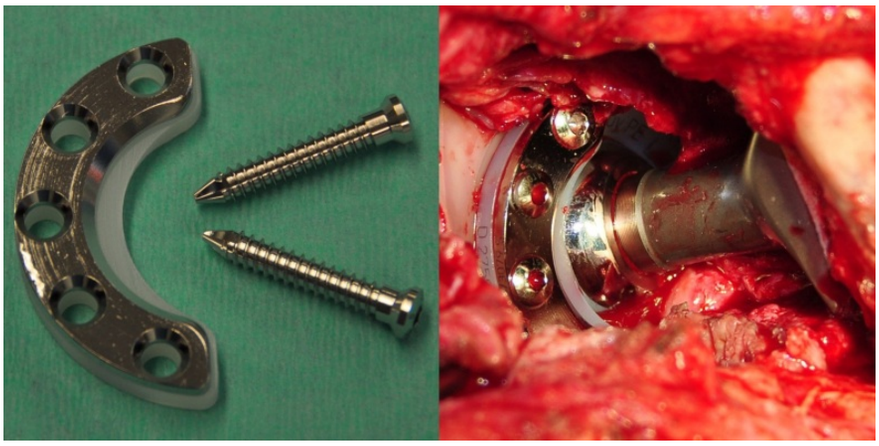

Figure 4. Posterior Lip Augementation Device (PLAD, Depuy, UK)

5.1. Leg length discrepancy

Leg length discrepancy (LLD) is the most common cause of patient dissatisfaction and subsequent litigation after THA [91]. LLD can result in nerve palsies, abnormal gait, lower back pain and reduced functional outcome [92]. Wylde et al. showed up to 30% of patients after primary THA can have a perceived LLD, but only 36% of these had an anatomic LLD greater than 5mm [7].

Nerve palsies are potentially the most serious complications of LLD. Sciatic and peroneal nerve palsies have both been associated with limb lengthening. Edwards et al suggested sciatic and peroneal nerve palsies are associated with lengthening greater than 4 and 3.8cm respectively [93]. Farrell et al however found an average lengthening of only 1.7cm was a significant risk factor for nerve palsies [94]. Therefore safe limits for limb lengthening before traction nerve palsies develop are yet to be defined, and it may be that any minor degree of lengthening may make the nerve more susceptible to other trauma [94].

Minor LLD less than 1cm is usually well tolerated by patients. However LLD greater than 2cm has been shown to significantly affect the gait cycle, increasing physiological demand [95]. LLD greater than 3cm in the elderly was shown to cause significant increases in heart

rate and quadriceps activity in the lengthened limb, which may be especially relevant in patients with cardio-respiratory co-morbidities [95].

Avoiding potential problems with LLD begins with patient history and examination. It is crucial to determine patient perceived leg length in order to counsel the patient effectively regarding likely outcomes. True leg length can then be determined, measuring from the ipsilateral anterior superior iliac spine to the medial malleolus, followed by apparent leg length by measuring from the umbilicus to the medial malleolus. Apparent leg length can be affected by pelvic obliquity secondary to either lumbar spine pathology or contractures about the hip. Significant LLD due to fixed pelvic obliquity secondary to chronic lumbar spine pathology cannot usually be corrected as it may involve significant shortening or lengthening. With pelvic obliquity secondary to contractures, the true length only needs to be corrected as after the THA the pelvis will balance with time [96].

Radiographs can also be used to determine leg length by referencing the position of the lesser trochanter in relation to a line drawn across the inferior aspect of the pelvis as shown in figure 5. Templating can then be carried out to determine the correct level of the neck cut for the femoral prosthesis and the position of the acetabular component in order to determine the new hip centre. Both of these directly affect leg length.

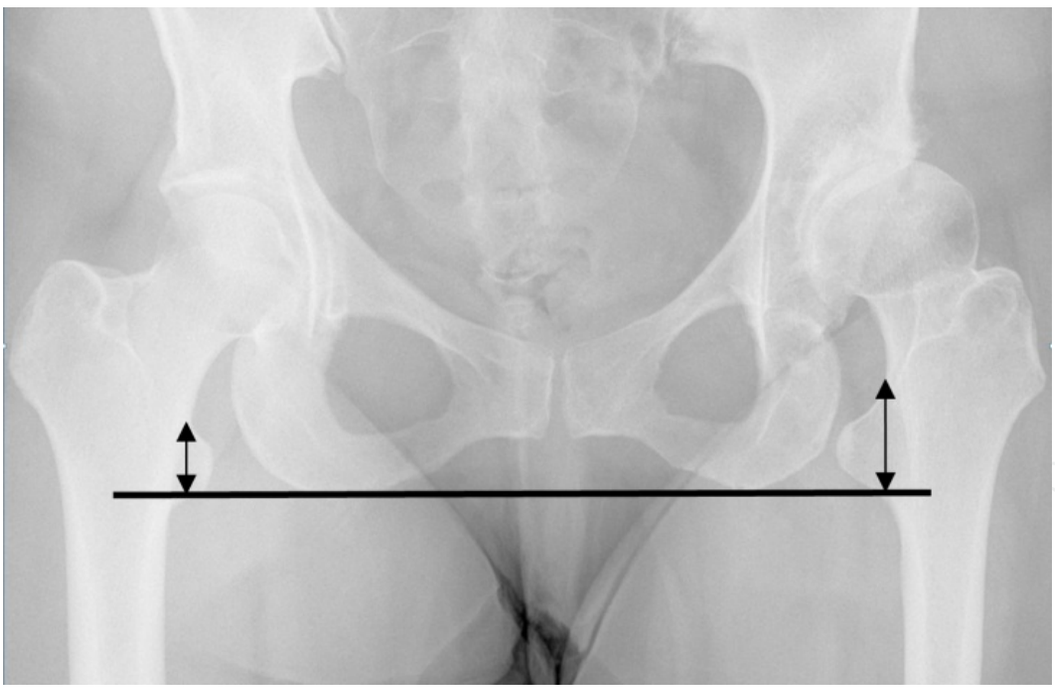

Figure 5. Radiographic estimation of LLD can be made by measuring vertically from the top of the lesser trochanter to a line drawn across the inferior margin of the pelvis

Intraoperative methods include the use of measurements taken from reference pins placed in the pelvis to a mark on the greater trochanter [97-99]. Mihalko et al described using a large fragment screw placed above the superior rim of the acetabulum and marking a point on the greater trochanter a fixed distance from this prior to dislocation. After insertion of the

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prostheses this distance was rechecked giving an indication of leg length changes [98]. Shiramizu et al used a similar method but with a steimann pin in the ilium and a custom calliper to measure the distances [99]. They found a mean LLD of only 2.1mm with this method.

Minor LLD postoperatively can be treated using a shoe raise. Prescription of such devices should be delayed for 3-6 months to allow any residual pelvic tilt secondary to contractures to resolve as the soft tissues can progressively relax. Failure of conservative measures and symptoms such as severe pain, nerve palsies and instability can necessitate surgical intervention. Shortening can be treated using soft tissue release and exchange of modular heads to give modest changes in leg length or more extensive surgery such as exchange of the femoral component to give greater neck length or offset. Lengthening can also be treated with component exchange but secondary procedures such as trochanteric advancement or the use of larger heads or stems with increased offset may be needed to maintain stability [100].

5.2. Infection

Infection post THA is potentially one of the most catastrophic and challenging to treat complications. During the early development of THA, Charnley reported a deep infection rate of 9.4% in unventilated operating theatres [101]. This initial unacceptably high deep infection rate stimulated the development of several prophylactic measures including ultraclean laminar air flow ventilation and peri-operative antibiotics. With the aid of such measures, infection rates in the UK between 1993 and 1996 fell to 1.08% [9].

5.3. Prophylaxis

Bacterial contamination of theatre air was initially recognized as a risk factor for post operative sepsis by Lister in 1867 [102]. Charnley later introduced the concept of ultraclean air flow ventilation that produces less than 10 colony forming units per cubic meter [103]. His reported infection rates in THA fell to 1% with the use of such enclosures. A MRC trial published in 1982 demonstrated a deep sepsis rate of 0.6% with ultraclean ventilation compared to 1.5% with conventional ventilation [104]. The use of ultraclean air ventilation during joint arthroplasty has subsequently become universally adopted practice within the UK [105].

The use of peri-operative antibiotics during THA is also a common prophylactic measure. Early trials using cloxacillin in THA found a 12% infection rate without prophylaxis compared to 0% with [106]. Currently cephalosporins are commonly used prophylaxis for THA. There is however a gradual move away from these due to the emergence of MRSA and problems with Clostridium difficile infection. Alternative regimens include flucloxacillin and gentamicin, or vancomycin and gentamicin. There is no conclusive evidence with regards to the optimal antibiotic regimen or duration of administration. However no benefit of extended prophylaxis beyond 24 hours has been demonstrated [107]. Therefore antibiotic regimes should be ideally guided by local microbiological knowledge so locally prevalent organisms can be targeted. Other measures shown to reduce rates of infection or bacterial load include the use of occlusive clothing, exhaust suits, pulsed wound lavage, preoperative showering and reducing theatre traffic [105].

5.4. Pathogenesis

Infection can arise by direct bacterial contamination at the time of surgery or later haematogenous spread. Staphylococcus aureus was the most common causative organism in an early series published by Charnley [108]. Coagulase negative staphylococci have become increasingly prevalent over the years with a recent series showing such organisms responsible for 58% of infections [109]. This is thought to be due to the effect of antibiotic use on bacterial flora [105]. Risk factors for periprosthetic infection include obesity, revision surgery, inflammatory arthritis, open skin lesions on the affected limb, blood transfusion, urinary infections and high ASA score [110].

Pathogenesis begins with bacterial adhesion. Primary adhesion occurs due to physical interactions (hydrophobic/electrostatic) between the bacteria and prosthetic surface. This is followed by bacterial aggregation through membrane adhesion molecules and generation of exopolysaccharides which form a glyocalyx or biofilm surrounding the bacteria [111]. This biofilm is thought to protect the bacteria from antibiotics and host defences [112].

5.5. Classification

Periprosthetic infections can be classified into 4 main catogeries [113]. Early postoperative infection is one that becomes apparent within one month of the procedure. Late chronic infection presents later than 1 month after operation and has an insidious course of gradual onset of pain and swelling with minimal systemic symptoms. Acute haematogenous spread results in an acute onset of symptoms associated with a documented or suspected bacteraemia. The final type is positive intra-operative culture, this is an occult infection diagnosed by positive cultures taken at time of revision surgery.

5.6. Diagnosis

Diagnosis of peri-prosthetic infection can be extremely challenging. Hip pain is the most consistent symptom. Presence of systemic symptoms such as fevers or rigors can be very variable. Examination may reveal local wound tenderness, signs of inflammation, discharge, sinuses and a painful range of movement.

Plain radiographs may show evidence of osteopenia or osteolysis, periostiitis and endosteal scalloping. However none of these can reliably differentiate between infection and aseptic loosening. Radionucleotide scanning using technetium or gallium can also been used. Technetium uptake reflects active bone turnover and gallium binds to transferrin, accumulating in inflammatory foci. Technetium scanning has a greater sensitivity than gallium for infection but their inability to differentiate infection and aseptic loosening limits their application [114, 115]. However its relatively high negative predictive value can make technetium bone scanning a useful initial screening test [116]. 18F-Fluoro-deoxyglucose [18-FDG) PET scanning is a newer technique that has increased sensitivity and specificity for infection. Pooled data from recent studies demonstrate a sensitivity of 85.5% and a specificity of 92.6% for periprosthetic infection [117]. Availability of PET scanners however still remains poor. Using radio-labelled white cells or immunoglobulins is another technique which has shown

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improved sensitivity and specificity relative to traditional three phase bone scans. Their widespread availability combined with the lack of established diagnostic criteria for 18-FDG PET scans, makes labelled white cell scans the current nuclear medicine investigation of choice for periprosthetic infection [118].

Blood investigations include ESR, CRP and Interleukin-6. ESR and CRP are non specific inflammatory markers and therefore can be elevated by concurrent illnesses. In the absence of such conditions an ESR greater than 30 mm/hr has a sensitivity and specificity of 82 and 85% respectively for peri-prosthetic infection, and the values for a CRP greater than 10mg/l are 96% and 92% [119]. Elevated levels of interleukin-6 have also been associated with periprosthetic infection with a sensitivity and specificity of 100% and 95% in one study [120]. However other chronic inflammatory conditions such as rheumatoid arthritis and other illnesses such as AIDS and Multiple Sclerosis can also cause elevated levels.

Cytological and microbiological analysis of hip aspirate taken under sterile conditions can give useful information regarding not only the presence of infection but also the potential offending organism. Ali et al. have shown a sensitivity and specificity of 0.82 and 0.91 for radiologically guided guided hip aspiration. However recent antibiotics can affect cultures and therefore antibiotics must be stopped for at least 2 weeks prior to aspiration.

5.7. Treatment

The aims of treatment of an infected prosthesis are eradication of infection and restoration of function. The classification by Tsukyama et al. can be used to help guide treatment [113]. Acute infections either presenting as early infection or acute haematogenous spread can be treated by component retention and thorough debridement, irrigation and intravenous antibiotics. However such treatment must be undertaken within 2 weeks of onset of symptoms [121]. Success rates between 50-74% have been reported with such a strategy [113].

Late chronic infection is best treated with full revision. This can be performed as a single stage exchange arthroplasty or a 2 stage exchange procedure. Originally described by Bucholz, a single stage procedure involves prosthesis removal, soft tissue debridement and lavage, followed by re-implantation of a new prosthesis if a clean uninfected bed is achieved, followed by appropriate antibiotic therapy [122]. Review of 1299 cases treated with single stage revision showed an 83% success rate at an average follow up of 4.8 years [123]. Factors associated with successful outcome were good general health of the patient, absence of wound complications after the primary procedure, methicillin sensitive organisms and infection with organisms sensitive to antibiotics within the cement [123]. Advantages of a single stage procedure include lower patient morbidity and lower incidence of complications such as fracture and dislocation. However 2 stage procedures have consistently demonstrated higher success rates compared to single stage procedures [124-128]. Thus 2 stage exchange still remains the most common strategy.

2 stage procedures involve initial prosthesis removal, soft tissue debridement and insertion of an antibiotic loaded cement spacer. This can be in the form of an articulating spacer allowing some range of movement and reducing soft tissue contracture. This is followed by

appropriate antibiotic therapy and a usual interval of 6 weeks prior to reimplantation of the definite new prosthesis. Success rates of between 87 to 94% have been reported with cemented 2 stage revision [124, 125, 128].

6. Nerve and vessel injury

The overall incidence of nerve injury after THA is estimated to be around 1% [129]. Sciatic nerve palsies account for 79% of all cases, followed by femoral nerve palsies (13%), combined femoral and sciatic nerve palsy (5.8%) and obturator nerve palsy (1.6%) [129]. In the majority of cases (47%) the aetiology is unknown. Other causes include traction (20%), contusion (19%), haematoma (11%) and dislocation (2%), with laceration only accounting for 1% of all nerve palsies [130]. Risk factors for nerve injury include female sex, revision surgery and developmental dysplasia of the acetabulum [129].

When the sciatic nerve is affected, it most commonly involves the common peroneal division. This is thought to be due to the lower amount of connective tissue present between the funiculi and its relatively tethered position at the sciatic notch compared to the tibial branch [129, 131]. These factors are thought to make the peroneal branch more susceptible to trauma and traction. The use of the posterior approach has traditionally been associated with increased risk of sciatic nerve damage. However a Cochrane review in 2006 found no difference in the incidence of nerve palsy between the posterior and direct lateral approaches [132]. Femoral nerve palsy is less common and is usually secondary to direct compression, usually due to a malpositioned retractor [130].

Indications for surgical intervention in a patient with nerve palsy include haematoma causing compression, palsy associated with excessive lengthening and palsy that can be definitely attributed to implanted metalwork. Electrodiagnostic studies can be helpful in determining the level of the lesion. Outcomes of nerve palsies are variable, with 40% of patients showing a good recovery, 45% of patients having mild residual motor or sensory symptoms and 15% left with a dense motor or sensory deficit [129]. Partial nerve lesions and maintenance of some motor function are good prognostic indicators, with recovery possible for up to 3 years after the initial insult [133].

Vascular injury during THA is extremely rare. Published incidence varies between 0.04 to 0.08% [134, 135]. As opposed to knee arthroplasty, vascular injury in THA is usually the result of direct trauma either during component insertion or removal [136]. Risk factors include revision surgery, previous vascular injury or surgery and pre-existing atherosclerosis [137]. The majority of vascular injuries are arterial but venous injury has been described [138]. Venous injuries however may be under diagnosed as they may run a relatively benign course remaining undetected.

The majority of vascular injuries are either the result direct trauma from acetabular retractors or acetabular screw insertion [130]. Wasielewski et al. have described an acetabular quadrant system to help guide safe screw insertion [139]. The postero-superior and poster-

inferior quadrants are the safest zones for screw insertion as they have they areas of greatest bone stock [139].

6.1. Wear and aseptic loosening

Aseptic loosening is the most common cause for revision surgery, accounting for 75% of revision cases [140]. Aseptic failure occurs as a result of a chronic inflammatory reaction secondary to particulate wear debris eventually resulting in osteoclast activation, osteolysis and loosening [141].

The pathogenesis begins with the generation of wear particles from the bearing surface, and also non bearing surfaces such as the interface between acetabular shell and the liner insert, known as backside wear. The morphology of the wear debris is dependent on the type of implant used. Particles from polyethylene bearing surfaces can vary from submicron in size to several millimetres. The average size of polyethylene debris has been shown to be around 0.5 μm and it is this submicron sized particle that has been shown to have the most bio-reactivity [142, 143]. The rate of generation of the wear particles has also been shown to correlate with the degree of osteolysis [142]. Inadequate initial fixation can also contribute to loosening by generating micromotion and increasing the rate of generation of particulate debris [144]. This highlights the importance of good cementation techniques in reducing the risk of aseptic failure. Pressure within the joint fluid has also been suggested to contribute to osteolysis. Increased joint fluid pressure in animal models has been shown to induce bone loss at the prosthesis bone interface possibly by interfering with bone perfusion causing osteocyte death [145, 146]. Increased joint fluid pressures have been noted in THAs undergoing revision and pressure waves generated by load bearing have been demonstrated in retroacetabular lytic lesions [147, 148]. Thus increased fluid pressures may directly contribute to osteolysis and also perpetuate the dissemination of the wear debris throughout the prosthesis bone interface, enhancing the biological response.

The primary response to wear debris is predominantly macrophage mediated. The exact mechanism of macrophage activation is still unclear. Macrophages can be activated as a result of either phagocytosis of particulate matter and also possibly through cell membrane interactions with particulate matter [149]. Macrophage activation causes the release of pro-inflammatory cytokines and growth factors including TNF-α, Interleukin-1, TGF-β and RANKL [150]. This results in the production of a pseudomembrane at the bone cement prosthesis interface consisting of macrophages, fibroblasts and lymphocytes within a connective tissue matrix [151]. TNF-α and Interleukin-1 both promote osteoclastic differentiation and activation, but it is the up regulation of the RANK/RANKL pathway that is the key to activating osteoclastogenesis and subsequent osteolysis [143]. Recent studies have suggested individual genetic susceptibility to osteolysis may exist via single nucleotide polymorphisms in the implicated cytokine genes, possibly by altering the magnitude of the biological response [152-155].

Alternative bearing surfaces can be used to reduce wear rates, debris generation and subsequent osteolysis. Highly crosslinked polyethylene, ceramic on ceramic and metal on metal bearings have all been shown to have reduced wear rates compared to standard ultra-high

molecular weight polyethylene (UHWPE) [156-164]. However there are concerns regarding the increased bioreactivity of crosslinked polyethylene debris compared to standard UHWPE which may offset the benefits of reduced volumetric wear [165, 166]. Volumetric wear is also lower with metal on metal bearings. However as the particle size is much smaller, usually between 20-90nm, the overall surface area is much larger compared to UHWPE raising concerns of possible increased bioreactivity.

Pain is usually the primary presenting symptom of aseptic failure. Gross acetabular loosening can cause groin pain whereas thigh pain can indicate femoral loosening [167]. Early loosening however may also be asymptomatic merely detected on routine follow up radiographs. Clinical signs may include inability to straight leg raise, shortening of the leg due to subsidence and increasing external rotation of the leg if the femoral stem twists into retroversion. Investigations for aseptic loosening are similar to those for infection discussed earlier. Blood inflammatory markers such as CRP are usually normal with aseptic loosening [168]. Radiological tests include plain radiography, subtraction and nuclear arthrography and bone scintigraphy. Meta-analysis has shown similar diagnostic performance for all of these tests and therefore suggests plain radiographs and bone scintigraphy as the tests of choice due to their lower risk of patient morbidity [169]. CT 3D imaging is also useful for the evaluation of lytic lesions as plain 2 dimensional radiographs can underestimate the size of the lesion as demonstrated in figure 1 [170, 171].

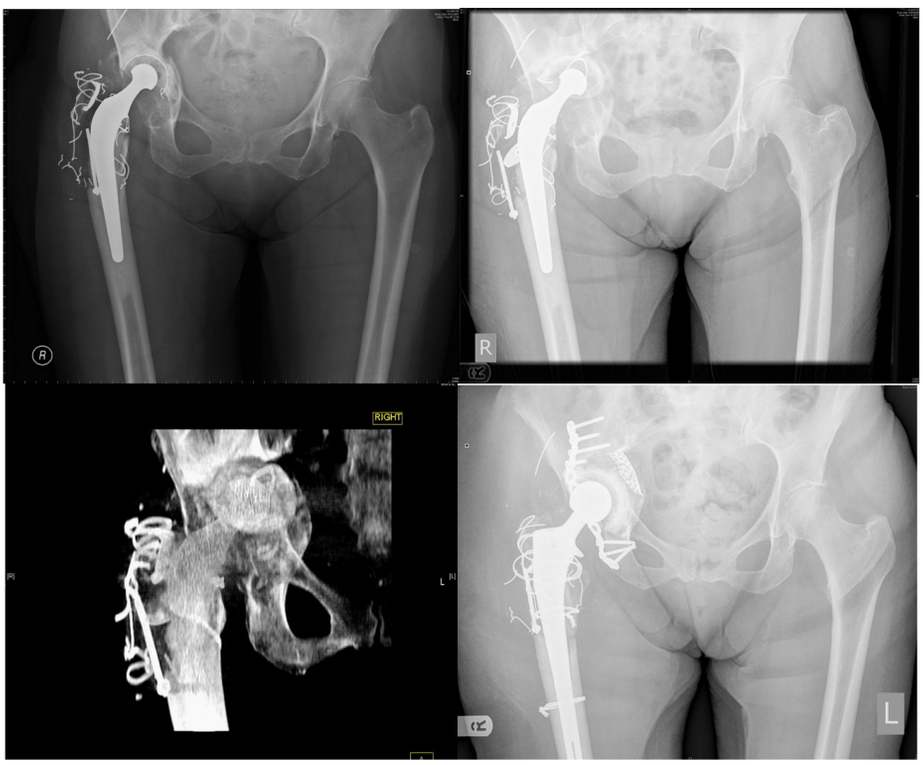

Figure 6. Top left & right, Progressive acetabular osteolysis over 1.5 years, **Bottom left,** 3D CT reconstruction demonstrating lesion and pelvic discontinuity, **Bottom right,** defect reconstructed with mesh and bone graft and plate to posterior column to address discontinuity

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Treatment of aseptic loosening is guided by the severity of the patient's symptoms and the rate and volume of osteolysis. Indications for surgical treatment in asymptomatic patients are progressive osteolysis and risk of catastrophic mechanical failure such as periprosthetic fracture. Nonsurgical treatment using bisphosphonates and anti cytokine therapy such as anti-TNF-α to prevent progression of osteolysis has been suggested. However their efficacy is yet to be determined [172]. Goals of surgical treatment include removal of wear debris and also the wear generator, reconstruction of the osseous lesion and restoration of mechanical stability [173]. This can involve exchange of bearing surfaces, bone grafting of lytic lesions and revision of loose components.

7. Bearing specific complications

7.1. Ceramic on ceramic bearings

Ceramic articulations have become increasingly popular due to their low wear profile and good biocompatibility. However potential complications of ceramic bearings include chipping and incomplete seating of ceramic liners during insertion, fracture and bearing generated noise.

Currently all ceramic acetabular bearings consist of a modular ceramic liner which is inserted into a metal shell implanted into the acetabulum. Incomplete seating of the liner due to soft tissue interposition or deformation of the metal shell has been reported [174, 175]. Thus extra care and good visualisation of the acetabulum is imperative when inserting a modular ceramic liner. Chipping during impaction has also been reported and this can also be secondary to deformation of the metal shell [176]. Using titanium sleeved or recessed ceramic liners has been shown to reduce such risks [177].

Risk of fracture for modern 3rd generation ceramic bearings is extremely low. Willman et al. found a fracture rate of 0.004% for femoral heads manufactured after 1994 [178]. Fracture of both the liner and femoral head have however been reported [179, 180]. Head fracture has been associated with improper handling during implantation. Contamination of the stem-ball interface with blood or soft tissue has been shown to significantly reduce the load required for inducing fracture [181]. Impingement of the femoral neck on the edge of ceramics liner is thought to be a major risk factor for liner fracture [179, 182]. Therefore correct positioning of the acetabular component is especially important for ceramic bearings.

Noise generated from ceramic bearings is a recently described phenomenon. Published rates of "squeaky" ceramic bearings range from 2.7% to 20.9% [183, 184]. Component malposition has been implicated [185]. However recent studies have found no association between cup inclination and version and the incidence of squeaking [183, 184]. Short neck length is the only factor that has been associated with squeaking, possibly due to impingement or micro-seperation to due increased joint laxity [184]. Revision of squeaking hips has revealed evidence of stripe wear but there is currently no evidence to suggest squeaking is a precursor for ceramic fracture [186, 187].

7.2. Metal on metal bearings

Metal on metal bearings also have superior wear rates compared to standard UHMWPE [161, 163, 164]. However there is increasing concern regarding metal ion toxicity and hypersensitivity type reactions. Volumetric wear is considerably lower for metal bearings compared to UHMWPE, but the absolute number of particles generated is estimated to be 13500 times higher [188]. Therefore the total surface area is considerably higher. Thus the bioreactivity of metal wear particles may be higher than polyethylene or ceramic debris, and the nanometre scale of the particles and dissolution of metal ions allows distant transport, raising concerns of systemic toxicity.

The possibility of systemic toxicity has raised interest in serum metal ion levels in patients with modern metal on metal bearings. Recent studies using standardised measurement techniques have reported mean serum chromium levels of between 0.86 – 17.7 μg/L [189-192]. Safe levels of serum metal ion levels have however yet to be determined [193]. Concerns regarding carcinogenesis and immune suppression secondary to raised blood metal ion levels have been raised [194, 195]. Teratogenicity is also another potential concern as transplacental crossage of metal ions has been demonstrated [196]. However, currently no conclusive evidence exists supporting these theories [197, 198]. A positive correlation between cup inclination and blood metal ion levels has been demonstrated with metal on metal bearings [191, 199]. This is probably due to increased edge loading with increasing cup inclination and serum metal ion levels have been suggested as a tool to monitor the performance of metal on metal bearings [190]. Therefore metal ion exposure can be minimised with proper cup orientation.

Local tissue reactions to metal on metal articulations have also been reported [200-202]. Metal ions are thought to induce an immune reaction leading to tissue necrosis and osteolysis. This is in contrast to UHMWPE which induces a macrophage reaction to particulate wear debris. Willert et al. has called this unique reaction, aseptic lymphocytic vasculitic associated lesions (ALVAL) [200]. Histologically this reaction is characterised by perivascular lymphocytic infiltration and plasma cells. Clinical presentation can vary between chronic groin pain to extensive tissue necrosis forming pseudotumours [201]. Exact incidence of such tissue reactions is unknown but is estimated to be around 1% [201]. Risk factors associated with the development of these adverse reactions include small component size and component malposition [203]. Stemmed metal on metal hip replacements also appear to have a higher rate of revision and their use has now been discouraged [204].

8. Periprosthetic fracture

Periprosthetic fractures can occur either intraoperatively or in the postoperative period. Overall, periprosthetic fractures more commonly affect the femoral component of the THA. Data from the largest published series by Berry et al. reports the incidence of intra- and postoperative femoral fracture as 1% and 1.1% respectively [13]. Rates of intraoperative fracture after cementless fixation are higher, 5.4% for primary THAs and 21% for revision surgery [13].

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The treatment of unstable postoperative periprosthetic femoral fractures is now almost always operative. Loosening, non-union, varus malunion and morbidity associated with prolonged immobility have made conservative management unpopular [205]. Treatment can be guided by using the Vancouver classification which is the most widely accepted system for classifying such fractures [206]. This system takes into account three main factors, site of the fracture, stability of the implant and quality of the surrounding bone. Type A fractures occur in the trochanteric region and are subdivided into type A_G and A_L fractures. A_G fractures involve the greater trochanter and usually stable and can therefore be treated conservatively with protected weightbearing. A_L fractures involve the lesser trochanter, and are also usually insignificant unless a large portion of the calcar is involved potentially affecting implant stability, in which case revision THA may be necessary. Type B fractures occur around or just distal to the stem and are subdivided into type B_1, B_2 and B_3 fractures. B_1 fractures have a well fixed stem and can be treated with open reduction and internal fixation. Combined plate and cerclage wire systems are commonly used for such fractures. Type B_2 fractures have a loose stem but good bone stock. These are usually revised with long stem implants bypassing the fracture, and can be augmented by plates, cables and strut allografts to improve stability. Type B_3 fractures have a loose stem and poor stock stock. These are the most difficult to treat and require either revision THA with structural allografts to reconstitute the proximal femur, distally fixed long stemmed implants of custom proximal femoral replacement. Type C fractures occur distal to the stem. The stem can therefore essentially be ignored and the fracture treated with standard open reduction and internal fixation.

Acetabular fractures are somewhat less common with reported intraoperative rates ranging between 0.02-0.4% [207, 208]. Data regarding postoperative fractures is currently not available [13]. The majority of intraoperative acetabular fractures occur during acetabular insertion especially during impaction of pressfit cementless components [209]. Underreaming by greater than 2mm has been suggested to significantly increase fracture risk [210].

The aims of treatment of intraoperative acetabular fractures include stabilizing the fracture and preventing further propagation and maintaining component stability [209]. Techniques include plating the anterior and posterior columns and using bone graft and jumbo revision cups if there is marked bone loss. Treatment of postoperative fractures follows similar principles. Early postoperative fractures with stable cups and minimally displaced fractures, especially around uncemented implants with supplemental screw fixation, can be treated conservatively. Unstable cups require revision with fixation of the fracture. Late presenting fractures are frequently associated with osteolysis and therefore usually require revision with bone grafting [211].

9. Heterotopic ossification

Heterotopic ossification (HO) is the abnormal formation of mature lamellar bone within extraskeletal soft tissues. HO is most commonly asymptomatic, merely detected on follow up radiology. When symptomatic, stiffness is the most common presentation. Pain and soft tis-

sue signs such as localised warmth, mild oedema and erythema are uncommon but can cause confusion raising concerns over infection [212].

Early changes of HO within the soft tissues can be detected after 3 weeks on bone scan and plain radiographic changes can take 6 weeks to become apparent [212]. Extensive bone deposition can occur within 3 months, but full maturation takes up to one year [213]. The abductor compartment is most commonly affected. HO is most commonly classified using the Brooker classification [214]. This is based upon plain anteroposterior radiographs of the pelvis and is outlined in figure 7.

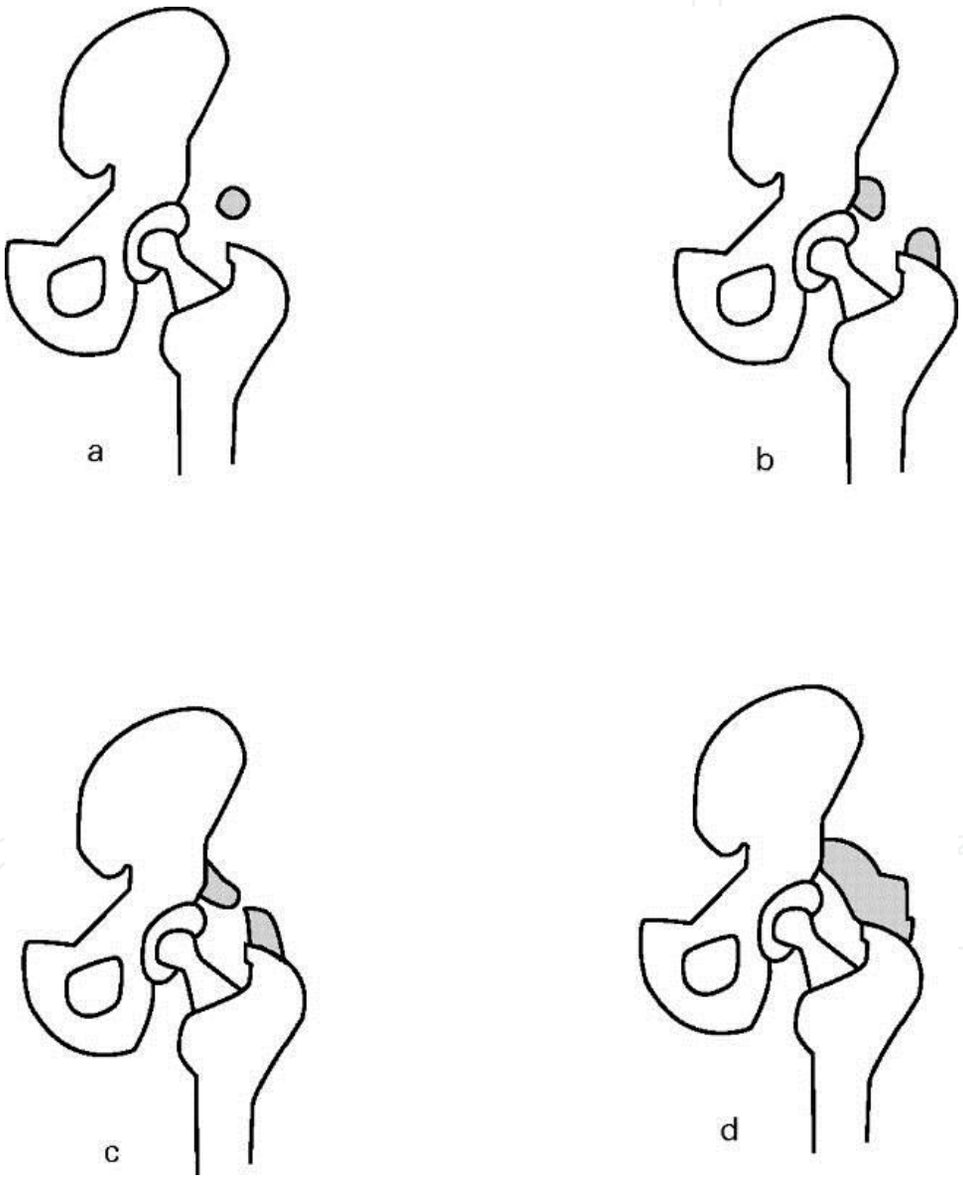

Figure 7. Brooker classification showing a) grade 1: islands of bone within the soft tissues about the hip, b) grade 2: bony spurs from either the femur or the pelvis, with a gap of more than 1 cm between opposing bony ends, c) grade 3: the gaps between the spurs are less than 1 cm and d) grade 4: apparent ankylosis of the hip due to the heterotopic ossification.

http://dx.doi.org/10.5772/53030

The pathophysiology is believed to involve inappropriate differentiation of pluripotent mesenchymal stem cells into osteoblasts, causing the excess bone formation [215]. Overexpression of bone morphogenetic protein-4 has been implicated [216, 217].

Incidence of clinically significant HO is reported to be between 3 – 7% [218, 219]. Risk factors include male gender, previous history of HO, pre-existing hip fusion, hypertrophic osteoarthritis, ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis, Paget's disease, post traumatic osteoarthritis, osteonecrosis and rheumatoid arthritis [14]. Surgical factors include extensive soft tissue dissection, haematoma and persistence of bone debris. Evidence implicating the role of surgical approach is debatable [14].

Treatment of symptomatic patients can initially involve intensive physiotherapy during the maturation phase. The efficacy of this treatment is however yet to be determined. Surgical management involves excision of the HO after maturation of the bone is allowed, followed by appropriate prophylaxis. Improvements in range of motion in all planes has been reported with surgical excision [220].

Patients at high risk of HO should be given prophylaxis either in the form of non steroidal anti-inflammatory medication (NSAIDs) or radiotherapy. Preoperative radiotherapy, 4 hours before, or post operative radiotherapy within 72 hours has been shown to be the most effective method of prophylaxis [221-223]. This involves a single dose of between 7 – 8 Gy. Combination therapy with NSAIDs and radiotherapy can be considered in patients at highest risk of HO such as patients undergoing excision of symptomatic HO [14].

10. Conclusion

- Complications following total hip arthroplasty can be classified into procedure specific or systemic. On the whole complication rates have fallen with time due to improved surgical and anaesthetic technique.
- The most common symptomatic systemic complication is DVT and data suggests that DVT rates post THA have not fallen with time.
- The most common cause for revision is aseptic loosening. Registry data suggests up to 75% of revision surgery may be due to aseptic loosening.
- Infection is one of the most feared complications. Rates with prophylactic measures such as antibiotics and clean air enclosures have however dropped significantly to below 1%.
- Leg length discrepancy is one of the most common causes of patient dissatisfaction and is the most common cause of litigation in the USA.

Despite the potential wide range of complication that can occur after THA, it remains one of the most successful orthopaedic interventions

List of abbreviations used

THA: Total Hip Arthroplasty

DVT: Deep Vein Thrombosis

PE: Pulmonary Embolism

THA: Total Hip Arthroplasty

PLAD: Posterior Lip Augmentation Device

UHMWPE: Ultra High Molecular Weight Polyehtylene

LLD: Limb Length Discrepancy

TGF-β: Transforming Growth Factor – Beta

RANKL: Receptor activator of nuclear factor kappa-B ligand

UHMWPE: Ultra High Molecular Weight Polyethylene

TNF-α: Tumour Necrosis Factor – Alpha

ALVAL: aseptic lymphocytic vasculitic associated lesions

HO: Heterotopic Ossification

Author details

Asim Rajpura and Tim Board*

*Address all correspondence to: tim@timboard.co.uk

Wrightington Hospital, Hall Lane, Appley Bridge, Wigan, Lancashire, UK

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