



© J ORTHOP TRAUMA SURG REL RES

12(3) 2017

Review Article

The nexus of intra-articular fractures, osteoarthritis and clinical outcome.

Does early surgical intervention with anatomical reduction and stabilization of intra-articular fractures improve clinical outcome?

KULDIP SINGH DHILLON

KPJ Selangor Specialist Hospital, Selangor, Malaysia

Address for correspondence:

Kuldip Singh Dhillon

KPJ Selangor Specialist Hospital,

Selangor, Malaysia,

Tel: +60 3-5543 1111,

E-mail: dhillonks2001@yahoo.com

Statistics

| | |
|------------|----|
| Figures | 00 |
| Tables | 00 |
| References | 44 |

Received: 24.08.2017

Accepted: 03.10.2017

Published: 06.10.2017

Abstract

There has been a rapid proliferation, over the last two decades, of the number of products available for osteosynthesis in the treatment of intra-articular fractures. This rapid proliferation is due to a misplaced belief that anatomical reduction and stabilization of intra-articular fractures will reduce the incidence of post-traumatic osteoarthritis (OA) and that post-traumatic OA is invariably associated a poor clinical outcome.

The common belief that OA is a degenerative disease caused by wear of the joint has been debunked. Literature review shows that OA is a disease of all components of the joint and not the articular cartilage alone, where innate immunity plays a significant role.

Some joints that are injured have potential for repair while other joints do not have this potential and joints without potential for repair become prone to OA. Risk factors for OA are known but the relationship between these risk factors and the pathogenesis of OA remains unclear.

Some joints tolerate incongruity better than others; hence it is unnecessary to carry out extensive surgical procedures in joints which tolerate incongruity. Extensive soft tissue dissection which is often necessary for stabilization of fractures is known to cause denervation of joints and denervation of joint is now known to predispose the joint to OA.

Surgery can also be associated with serious complications and is not cost effective where joints tolerate incongruity well. In other joints where incongruity is poorly tolerated all attempts to achieve congruity should be made.

Good clinical outcome is possible despite the presences of OA in some joints. Management of intra-articular fractures should be tailored differently for each joint to achieve the best outcome with minimal complications and it should be cost effective.

Keywords: Stabilization of intra-articular fracture, Clinical outcome, Post-traumatic osteoarthritis

INTRODUCTION

There has been a rapid proliferation, over the last two decades, of products available in the market, for the treatment of intra-articular fractures. These products have been aggressively promoted by the devices industry based on a misplaced belief that that anatomical reduction and stabilization of intra-articular fractures will reduce the incidence of post-traumatic osteoarthritis (OA) and that post-traumatic OA is invariably associated a poor clinical outcome. As a result of this there has been a marked increase in the number of intra-articular fractures been treated by open reduction and internal fixation.

The devices industry aggressively promotes a belief that articular incongruity will lead to OA which is a progressive disease and that the disease will progress to a stage where a joint reconstruction will invariably be needed.

The belief that OA is due to degeneration of the articular cartilage caused by wear and tear has now been debunked. It is now known that incongruity of the articular surface does not invariably lead to OA of the joint. It is also now known that OA does not always progress to end stage disease which would require reconstructive surgery. The presence of OA is not synonymous with poor clinical outcome.

Often the poor results of surgical treatment never get published in the literature. One thing we can be certain of is that when there is less surgical intervention there would be less morbidity and mortality. This begs the question as to whether a less aggressive approach to the management intra-articular fractures would be compatible with good clinical outcome. To understand the link between intra-articular fractures, osteoarthritis and clinical outcome, the pathogenesis of OA has first to be elucidated.

PATHOPHYSIOLOGY OF OSTEOARTHRITIS

Although osteoarthritis is the commonest form of joint arthritis it is probably one of the least understood disease entity. It can produce pain and significant functional disability. The cost to society in terms of loss manhour and treatment is very high.

In the past osteoarthritis was classified as a degenerative disease of the joint which resulted from wear of the articular cartilage. However, in recent years there has been a renewed interest in this subject and this has lead to more research which resulted in reclassification of OA as a disease which affects all components of the joint rather than the cartilage alone. It has been aptly described as 'disease of the joint as an organ' by Loeser et al. [1]. Inflammatory mediators have been identified which lead to an abnormal remodelling of joint tissues after mechanical or other insult to the joint [1].

There are many factors besides joint trauma, joint incongruence and malalignment which predisposes a joint to OA. Some of these include obesity, sex, age, genetic phenotypes and metabolic disorders [1]. Studies of joints with OA show that besides changes in the articular cartilage, changes are also seen in the subchondral bone, capsule, synovium, blood vessels, nerves, muscles, bursae, and menisci (knee). The pathological process which leads to 'joint failure' is the same irrespective of the underlying cause of the disease [1]. The joint failure results from a failure of the cartilage haemostasis. A complex interaction between the synovium, subchondral bone and the articular cartilage maintains the cartilage haemostasis [2].

The adult chondrocytes in their normal resting unstressed state are quiescent in the hypoxic environment of the joint with little turnover of the collagen matrix (half-life of type II collagen is about 120 years) [2]. These chondrocytes and the synovial cells produce lubricants, lubricin and hyaluronan, which together with the smooth articular surface, provides low friction articulation for joint

movements [2]. Joint movement and joint loading are essential for joint cartilage haemostasis. Loss of joint movements as is seen with joint immobilization leads to increases in the protease levels with a corresponding loss of proteoglycan which in turn leads to joint damage.

OA changes start at the surface of the articular cartilage where shear stresses are the highest. Various stimuli including injury activate the usually quiescent chondrocytes. This activation of chondrocytes leads to proliferation of cells, cluster formation, and an increased production of both matrix proteins and matrix-degrading enzymes [2]. The activation of chondrocytes disrupts the haemostasis mechanism. Matrix remodelling begins with calcification of deeper layers of the cartilage, neurovascular invasion of the subchondral bone and the advancement of the 'tidemark'. As the matrix degrades certain receptors are stimulated and these receptors produce more proteinases which further degrades the matrix. The receptors also produce inflammatory cytokines and chemokines which kicks in an inflammatory response which is seen in joints with OA. There is some evidence that degradation products of articular cartilage may activate an innate immune response [2].

Chemokines which are associated with inflammatory arthritis and other autoimmune conditions are also produced by the joint and they play a role in cartilage homeostasis in joints with OA. Alarmins are also released by the damaged cartilage matrix leading to inflammation and more damage to the cartilage and other joint tissues. The joint cells also release Adipokines which contribute to immunity and inflammation and have a role to play in cartilage damage [2].

Most of the evidence gathered so far points to the fact that OA is an inflammatory immune disease in the spectrum between a normal state and an autoimmune disease like rheumatoid arthritis and that it is not a straightforward degenerative disease due to wear of the cartilage. Innate immunity appears to play an important role in activating inflammatory and catabolic events in the cartilage which leads to progression of the OA. Innate immunity is responsible for the synovitis seen in joints with OA. However, the factors which trigger this innate immunity which leads to synovitis and progression of the disease, however, remains elusive [3].

Considering the fact that innate immunity is the active player in OA, can restoration of joint congruity, stability and alignment prevent progressive degeneration of the joint?

Does restoration of joint congruity, stability and alignment prevent progressive degeneration of the joint?

Joint trauma is more frequently seen in the young and middle-aged individuals and when such adults develop OA it presents with a treatment challenge. In the elderly on the other hand OA is less of a treatment challenge because the elderly can accept modification of their lifestyle and reconstructive joint procedures can lead to good functional outcome.

Unfortunately to date there is no treatment available which can reduce the risk and progression of OA. For little known reasons the risk of post-traumatic OA varies with the joint involved and the individual involved [4]. Although the forces required to disrupt articular cartilage are higher than that required to fracture the subchondral bone, mechanical trauma when severe can cause cartilage damage [5].

Different joints and different individuals respond differently to articular cartilage injury. In some circumstances the cartilage can remodel and restore function whereas under other circumstances remodelling does not occur and the injury leads to progressive degeneration of the cartilage. At which point and when the injury becomes irreversible and leads to OA remains a mystery [4].

When the defects or step-offs in the articular cartilage are so large

that they produce malalignment and clinical instability, there is usually no potential for remodelling and repair. However, animal experiments show that small gaps and step-offs have the ability to repair. Factors such as the biological and mechanical properties of the cartilage, cartilage thickness, joint shape and congruity, and the severity of the initial trauma are believed to affect the capability of the articular cartilage to remodel but the relationship between these factors and the ability to remodel remains unclear. Neither is the relationship between rigid fixation of articular fractures and the ability of the cartilage to remodel, clear [4].

Since we now know that in some injuries there is potential for repair and in others there is no such potential, what then are the risk factors which create an environment where OA can develop?

RISK FACTORS FOR POST-TRAUMATIC OA

There are several factors which are well known to predispose a joint to post-traumatic OA after intra-articular fractures but the relationship between these factors and OA remains vague.

Severity of injury

It is a common belief that, the greater the initial injury, the higher is the risk of OA of the joint and this assumption is based on some in vitro and in vivo experimental studies [6]. There are no rigorous experimental studies to show the link between severity of injury and osteoarthritis. However, there are some observational studies which show that the risk of OA is higher in individuals who sustained severe injuries as reflected by gross comminution and displacement of fracture fragments. Whether the severity of injury or ineffective remodelling is responsible for OA of the joint following trauma remains unclear [7].

Incongruity of the articular surface

Incongruity of the articular surface is known to increase contact stresses at joint surfaces and it has been known for some time now that chronic increases in contact stress predisposes a joint to OA. Brown et al. experiments with human tibial articular surfaces showed that step-offs of the articular surface which are more than 1.5 mm do increase the contact stresses [8]. They found that 3 mm step-offs increased the contact stresses by 75%. They estimated that the tibial articular surface should be able to withstand twice the amount of force which it is subjected to, on a daily basis, without any harmful effects. They also found that the ability to withstand stress depends on the thickness of the articular cartilage. Areas of the tibial surface with greater thickness of articular cartilage are better able to withstand stress.

There are clinical studies available which show that tibial articular incongruity can lead to OA of the knee joint. What is lacking are clinical studies which show the critical level of step-off which will predispose the joint to OA [7].

Instability of the joint

Just like cartilage defects and step-offs, injuries to the joint capsule, ligaments and the menisci can also produce joint instability. Cruciate ligament laxity and surgical excision of menisci are well known predisposing factors for knee OA. Experiments show that a combination of joint instability and articular incongruity is more likely to cause progressive OA as compared either condition alone [7]. There is some evidence that instability with joint denervation, which can occur from capsular injuries and extensive surgery, is also more likely to produce OA. Although the exact mechanism by which joint instability causes OA is not known, it is most likely due to repeated shearing forces at the cartilage surface caused by instability which impairs cartilage healing and remodelling [7].

Age when the injury occurred

Age is a well-known risk factor for OA. Studies show that as the

age increases the the risk of post-traumatic OA increases. Cartilage remodelling and regeneration in response to injury declines with age due declining population of chondrocyte. This decline results from chondrocyte death and a poor response of the aging chondrocytes to anabolic stimuli [9]. Clinical studies show that there is a two to four-fold increase in the risk of OA in patients with articular fractures who are older than 50 years of age [7]. Studies also show that the incidence of OA in patients with anterior cruciate ligament laxity is higher after the age of 35 years as compared to those below that age [10]. Although age has been identified as a risk factor for OA, the actual reason why it is so remains undefined.

Joint susceptibility

Two factors, namely, the elastic modulus and the thickness of the articular cartilage affect the susceptibility of the joint to OA following trauma [7]. Studies show that, among the joints of the lower limbs, the ankle joint cartilage has the highest mean compressive modulus followed by the knee and hip joint. The hip joint probably has the lowest compressive modulus [11]. The higher stiffness of the articular cartilage predisposes the ankle joint to a higher incidence of OA. This has been reflected in clinical studies which show that the incidence of OA after plafond fractures of the tibia can be as high as between 70% to 75% [12]. The lower compressive modulus of the knee and hip cartilage is reflected by a lower incidence of OA after knee and hip fractures. The incidence of post-traumatic OA following tibial plateau fractures is between 23% to 44% [13] and following femoral condyle fractures is between 23% to 35% [14]. The incidence of post-traumatic OA is lowest with acetabular fractures where the incidence ranges between 11% to 38% [15]. This would mean that articular thickness and the elastic modulus of the cartilage is an important determinant of whether a joint will or will not develop OA after trauma and hence this could mean that the best treatment for each joint would be different.

The risk factors which predispose a joint to post-traumatic OA are well known, but how these risk factor contribute to the pathogenesis of OA has not been fully elucidated.

Articular incongruity, as we know now, predisposes the joint to OA, but does it also predispose to a poor clinical outcome?

Does failure to restore intra-articular congruity lead to poor clinical outcome?

Incongruity of the articular surface is a known risk factor for post-traumatic OA of the joints. A prevalent belief exists which associates OA with poor clinical outcome and this has prompted many surgeons to treat intra-articular fractures with early surgical intervention, anatomical reduction and stable fixation. Anecdotal evidence shows that intra-articular fractures are increasingly being treated by open reduction and internal fixation. Often the surgery involves extensive soft tissue dissection which can denervated the joint. Denervation of the joint creates an additional risk factor for OA.

This begs two questions: will restoration of articular congruity reduce the incidence of OA and will patients with articular incongruity have poor clinical outcome? Since the incidence of OA is joint dependent, starting the analysis with fractures of the distal radius which occur commonly would be logical.

Fractures of the distal radius

An observational retrospective study of the fractures of the distal radius by Forward et al. [16] throws some light on this topic. They studied 106 patients, who were less than 40 years of age at the time they sustained a fracture of the distal radius. They found that the functional outcome was good despite the existence of OA of the wrist. These patients were treated between 1960 and 1968 when fractures of the distal radius were routinely treated by close reduction and plaster immobilization. The mean follows up of these

patients was pretty long at 38 years with a range between 33 years and 42 years.

They found that only 5% of the patients had grade 3 OA, 30% had grade 2 OA and 68% had grade 0 or grade 1 OA (possible OA). None of the patients had a salvage operation of the wrist joint. Surprisingly the Disability of the Arm, Shoulder, and Hand (DASH) scores were the same as the population norm. The functional assessment using Patient Evaluation Measure (PEM) showed an impairment of less than 10%. The presences of a dorsal malunion did not affect the DASH and PEM scores but the grip strength was reduced to 89% as compared to uninjured side. In view of the good outcome of conservative treatment of distal radius fractures, the authors were of the opinion that it is not necessary to resort to invasive surgical intervention which can be associated with complications and is not cost effective. They recommended closed reduction with K-wiring, external fixation or cast immobilization.

A randomised controlled trial of 179 patients with distal radius fractures was carried out by Kreder et al. [17], to compare the clinical outcome between patients treated by close reduction and percutaneous fixation and those treated by open reduction and internal fixation. They followed up the patients for two years. They found that there was no significant differences between the two groups as far as anatomical reduction and range of wrist movements was concerned. Those patients who had close reduction of the fracture had a more rapid return of function and a better functional outcome as compared to those that had open reduction and internal fixation.

In 2003, Handoll and Madhok [18], carried out a Cochrane database review of surgical interventions for treating distal radial fractures in adults. They found no clear evidence that internal fixation of these fractures produces a better long-term outcome. In fact, they found some evidence to support the use of percutaneous pinning or external fixation in the treatment of distal radial fractures.

It is evident that the incidence of OA after distal radius is low despite the presences of articular incongruity and that despite the presences of OA the functional outcome is good.

This raises some interesting questions as to why there is a rapid proliferation of the number implants available for fixation of distal radius fractures and also why there is a worldwide increasing trend to treat these fractures surgically?

Fractures of the tibial plateau

Majority of the past publications on the outcome of treatment of tibial plateau fractures included small number of patients with a short follow up. In 2007, however, Rademakers et al. [19] published a retrospective study which analysed the long term radiological and functional outcome of surgical treatment of tibial plateau fractures. The study included 202 consecutive patients with tibial plateau fractures who had open reduction and internal fixation of the fractures. There were 112 males and 90 females with an average age of 46 years. At an average follow-up of 14 years (range 5 years to 27 years) 109 patients were available for analysis. In this group of patients, the authors found that, the mean range of knee motion was 135 degrees, the mean Neers score was 88.6 points and the mean HSS knee score was 84.4 points.

The incidence of OA was 31%, with no OA in 69% of the patients, 21% of the patients had mild OA, 7% had moderate OA and 3% had severe OA. Sixty four percent of the patients with moderate to severe OA had good to excellent Neers scores and 46% had good to excellent HSS scores.

Malalignment of more than 5 degrees was more often associated with moderate to severe OA as compared to normal alignment of the knee. Age of the patient did not seem to affect the outcome. Two

of the 202 patients (1%) developed progressive OA which required a knee replacement.

More recently in 2010, Manidakis et al. [20] published a retrospective review of 125 patients with tibial plateau fractures. One hundred and one patients were treated surgically and 24 patients had conservative treatment. The patients were followed up for an average 20 months (range 12 to 70 months). Radiological review showed residual varus in 9.6% and residual valgus in 8.8% of the patients. In 5% of the patients there was 2 cm or less of limb length inequality. They found that the American Knee Society score (AKSS) was good in 68.8%, fair in 24% and poor in 7.2% of the patients. In this study the incidence of OA was 26.4% and the incidence of knee replacement was 4% (5 patients).

Mehin et al. [21] in 2012 published a study which evaluated the incidence end stage OA in patients with tibial plateau fractures. Their data was obtained from the administrative database of a level I trauma centre where a higher than average number of patients with complex tibial plateau fractures are treated. They were also not aware if any of the patients had pre-existing OA when the fracture of the proximal tibia was sustained. They reviewed the data of 311 patients who were treated for tibial plateau fractures and were followed up in the hospital for 11 years. They did a 10-survival analysis for end stage OA in this group of patients. They found that the 10 years survival for end stage OA was 96%. In patients treated surgically the 10-year survival was 97% and for those treated conservatively it was 93%. In 4.5% of the patients the end stage OA required a reconstructive procedure such as a joint replacement, tibial osteotomy or an arthrodesis. A knee replacement (partial or total) was carried out in 2.8% of the patients.

This data is highly selective and slightly skewed, since it comes from a database of a hospital where a large number of patients with severe injuries are treated. The overall figures for endstage OA in the general population with tibial plateau fracture would be somewhat lower than this. However, the data does provide some very useful information.

Despite the scarcity of literature on the outcome of tibial plateau fractures, some useful information is available which shows that the incidence of post-traumatic OA following tibial plateau fractures varies from 26 to 31% with about 10% developing moderate to severe OA. There is some selective data (with some bias) which shows that the incidence of end stage OA is about 3% in patients who are treated surgically and 7% in patients treated conservatively. Contrary to popular belief the functional outcome of the treatment of intra-articular tibial plateau fractures is good in majority of the patients despite the presence of OA.

Ankle malleolar fractures

Functional outcome

According to reports in the literature the outcome of treatment of ankle fractures is good. In 2006, Egol et al. [22] published a report which defined the predictors of short-term functional outcome following ankle fracture surgery. Their study included 232 patients who had surgical treatment for ankle fractures. In 198 patients (85%) one year follow up data was available. The outcome was good with 88% of the patients having no pain or mild pain only and 90% of the patients had no limitation or a slight limitation only, in recreational activities. The prognostic factors for good functional outcome were young age, male sex, absences of diabetes mellitus, and a lower ASA class.

Lindsjö U [23] did a prospective study involving 321 patients who had fracture dislocations of the ankle and were treated surgically. He found excellent to good results in 82%, acceptable in 8% and poor results in 10% of the patients at 2 to 6 years follow-up. The decisive factors that influenced the clinical outcome were the type

of fracture, the accuracy of the reduction, and the sex of the patient. A good reduction with rigid fixation, early ankle mobilization and early weight bearing with a below knee walking support was associated with good outcome. Men were found to do better than females.

Other authors have published good outcome of ankle fractures without surgery. Bauer et al. [24] reported the natural history of ankle fractures in 143 patients who were treated conservatively without surgery. The average follow up of these patients was 29 years. Twenty patients had a Weber A, 103 patients Weber B and another 20 patients had Weber C fractures of the ankle. When the fractures were classified according to the Lange-Hansen (L-H), 100 patients had S-ER, 15 had S-AD, 14 had P-AB and another 14 had P-ER fractures of the ankle.

At 29 years follow up, 83% of the patients were symptom free and 16% had occasional ache in the ankle. There was no OA of the ankle in eighty two percent of the patients, six patients (4%) had moderate OA and 2 patients (1.3%) had severe OA. Two patient who had a severe form of ankle fracture (S-ER type IV) developed severe OA of the ankle. The authors were of the opinion that it is not necessary to have a perfect reduction of the ankle fractures to have a good functional outcome.

Donken et al. [25] followed up 276 patients with S-ER type II – IV fractures for 21 years and they found excellent or good results in 92% of their patients.

Open reduction and internal fixation of ankle fractures can be associated with serious complications although the incidence of complications is low. Nelson et al. [26] studied the California (USA) discharge database of patients who had internal fixation of ankle fractures between 1995 and 2005 and they found low short term complication rates which included a pulmonary embolism (0.34%), mortality (1.07%), wound infection (1.44%), amputation (0.16%) and reoperation for internal fixation (0.8%). The predictors of short term complications were open fractures, older age, diabetes mellitus and peripheral vascular disease. Although the complication rates are low, some of the complications, however, are serious.

At 5 years follow up, 0.96% of the patients required reconstructive surgery of the ankle in the form of arthrodesis or ankle replacement. The medium term (5 years) complications were more common in patients who had trimalleolar and open fractures of the ankle.

The overall incidence of post-traumatic OA of the ankle after ankle fractures is low (5.3% moderate to severe OA) and the functional outcome is excellent in most of the patients.

Fractures of the tibial plafond

The clinical outcome of treatment of tibial plafond fractures is unfortunately not as good as that of ankle malleolar fractures and the incidence post-traumatic OA after tibial plafond fractures is one of the highest among intra-articular fractures.

CLINICAL OUTCOME OF TREATMENT OF TIBIAL PLAFOND (PILON) FRACTURES

Babis et al. [27] carried out a study of 66 patients with 67 Pilon fractures, to shed some light on factors that influence the outcome of surgical treatment. They classified the fractures using the Rüedi and Allgöwer classification. There were 8 (11.95%) type I, 33 (49.25%) type II and 26 (38.8%) type III fractures. The patients were followed up for an average of 8.1 years (range 2 to 17 years).

Fifty of the 67 fractures were treated according to the AO principle, 9 had stabilization of the fibula with minimal fixation of the articular fragments and 8 had external fixation of the fractures. Seventeen of the type II and III fractures had bone grafting to fill bone gaps. Only the AO group had early mobilization of the ankle and the rest had

no mobilization of the ankle for 3 months. The outcome was based on subjective, objective and radiographic results based on Burwell and Charnley method [28].

Seven of the 8 (87.5%) type I fractures had good results regardless of the type of treatment or the quality of reduction. In type II fractures the subjective results were good in 72.7% and for type III in 50% of the fractures. The objective results were good in 69.7% of the type II and in 34.6% of the type III fractures. The radiographic results were good in 57.6% of the type II and in 15.4% of the type III fractures. The AO treatment group did significantly better than the other treatment group.

The severity of fracture, method of treatment and the quality of reduction of the fracture influenced the outcome of treatment of plafond fractures of the ankle. Greater severity of the fracture and poor reduction was associated with bad outcome. The AO method of treatment with early mobilization of the ankle was associated with better outcome. The probable reason why the fractures treated by the AO technique of internal fixation did better is because of lesser severity of injury in this group of patients. Patients with severe fractures with gross comminution and impaction and with open injury, poor skin and poor vascularity cannot be treated by internal fixation.

Etter and Ganz [29] studied the long term outcome of plafond fractures treated by internal fixation. Their retrospective analysis of 41 consecutive patients included 9.75% type I, 41.5% type II and 48.75% grade III fractures (Rüedi and Allgöwer classification). They reported good outcome in 66% and a fair outcome in 24% of the patients at an average of 10 years follow-up. Hence in 86% of the patients the outcome was satisfactory. Unlike other reports in literature, in this study 95% of the patients with type III fractures had a satisfactory outcome. This may partly have been due to the fact that 50% of the type III fractures in this study were from low velocity trauma. The severity of injury and poor fracture reduction were precursors of post-traumatic OA. The authors, however, found that the presence of severe osteoarthritis at follow-up did not correlate with poor subjective or objective results.

The outcome of treatment of high energy plafond fractures is generally poorer. Pollak et al. [30] did a retrospective cohort analysis of 80 (78%) of 103 eligible patients who were treated for high-energy plafond fractures with the objective of assessing their functional and general health outcome. The mean follow up was 3.2 years. The general health of the patients as measured with Short Form-36 (SF-36), was significantly poorer than age and gender-matched norms. Thirty-five percent of the patients reported ankle stiffness; 29% persistent swelling; and 33% ongoing pain. Forty three percent of the patients who were employed before the injury was unemployed at the last follow-up. Of these patients, 68% reported that fracture prevented them from working. The presence of two or more comorbidities, being married, annual personal income of less than \$25,000, lack of high-school diploma, and the use of external fixator for treatment correlated with poorer outcome.

The incidence of OA is higher in patients with plafond fractures. Harris et al. [31] in a retrospective review of 76 patients, with 79 plafond fractures, who were followed up for a mean of 26 months (range 24 to 38 months) found a 39% incidence of post-traumatic osteoarthritis of the ankle. The incidence of OA in patients with severe fractures which were treated with external fixation was higher.

In recent years a better outcome has been reported for high-energy plafond fractures with the use of minimally invasive operative technique and early physiotherapist led rehabilitation. Leonard et al. [32] in a prospective study of 32, C2 and C3 (AO) plafond fractures, treated by minimally invasive technique reported satisfactory reduction in all patients and excellent American Orthopedic Foot

and Ankle score (AOFAS) in 83% of the patients. The minimal follow-up in this study was 2 years. Patients with open fractures were excluded from the study.

The outcome of treatment of severe tibial plafond fractures is often difficult to predict. Williams et al. [33] followed up 29 patients with 32 plafond fractures for a minimum of 2 years (range, 24-129 months; average, 46.5 months) to assess the outcome based on radiographic arthrosis, subjective ankle scores, the Short Form-36 and ability to return to work. They found that the four outcomes did not correlate with each other. The outcome measures were influenced by socioeconomic factors. High scores were seen in patients with college degrees and lower scores in patients with work related injuries. The ability to return to work was affected by the patient's education levels rather than any of the outcome measurements. Improvement of symptoms was reported to occur over an average of 2.4 year.

PLAFOND FRACTURES, POST-TRAUMATIC OSTEOARTHRITIS AND ANKLE RECONSTRUCTION

The incidence of post-traumatic OA is high after plafond fractures. Harris et al. [31] in a retrospective review of 76 patients, with 79 high-plafond fractures, at a mean follow up 26 months (range 24 to 38 months), found a 39% incidence of post-traumatic osteoarthritis of the ankle. The incidence of post-traumatic OA following tibial plafond fractures is higher on longer term follow up. Marsh et al. [34], in a study of 56 plafond fractures in 52 patients, were able to follow-up 31 patients with 35 fractures between 5 and 12 years after the injury. They found the incidence of joint space narrowing (grade II OA) in 57% and severe OA (grade 3 OA) in 17% of the ankles. Although 74% of the ankles had significant OA, the presence of OA did not correlate with the clinical outcome. Two ankles (5%) were arthrodesis at a follow-up of between 5 to 12 years.

Rüedi and Allgöwer [35] reported a rate of ankle arthrodesis of 5% (four of seventy-five) at an average of nine years after the injury. Bourne et al. [36] reported a 17% rate of arthrodesis at an average of fifty-three months after plate fixation, with majority of the fusions performed in patients with severely comminuted fractures. Ovadia and Beals [37] reported a 12% rate of arthrodesis or replacement of the ankle at an average of fifty-seven months. These studies were done in the seventies and eighties and it appears that the incidence of reconstructive procedures for end stage OA is falling with improved treatment as reported by Marsh et al. [34] in 2003.

In patients with tibial plafond fractures the incidence of OA is high (about 70%) on long term follow up, especially in the more comminuted fractures but the presences of OA does not correlate with the clinical outcome. The clinical outcome is improving with better less invasive surgical techniques. It appears that with plafond fractures of the distal tibia the quality of reduction does seem to affect the clinical outcome.

Acetabular fractures

Articular incongruity of the weight bearing dome of the acetabulum is poorly tolerated and it appears to influence the outcome of treatment of fractures of the acetabulum. A good reduction of the fracture is essential for good to excellent long-term outcome and a good reduction can often be obtained by conservative means (traction). Sen and Veerappa [38] carried out a long-term outcome study of conservative treatment of displaced (more than 3 mm displacement) fractures of the dome of the acetabulum in 32 patients. They were able to obtain good reduction of the fracture in 56.3% of the patients. The average follows up was 4.1 years with a range between 2 to 12 years. The patients were evaluated using the Merle de'Aubigne and Postel clinical scoring and Matta's radiologic scoring system. They found that in patients where they were able to obtain good reduction the clinical scores were good to excellent in 83.3% of the patients.

Heeg et al. [39] did a retrospective review of 57 patients with acetabular fractures who were treated conservatively and followed for an average of 7.9 years. They found that the overall functional result was satisfactory in 75% of the patients. In patients with displaced fractures of the dome which could be reduced to less than 2 mm of displacement, the results were good to excellent.

The clinical outcome of acetabular fractures is adversely affected by the complexity of the fracture, older age, associated involvement of the femoral head and operative complications. The ability to obtain an anatomical reduction is influenced by complexity of the fracture, age of the patient and the interval between the injury and the reduction of the fracture. Matta [40] reviewed 259 patients with 262 displaced fractures of the acetabulum who had open reduction and internal fixation within 3 weeks of the injury. The patients were followed up for an average of 6 years (2 years to 14 years). In 71% of the hips an anatomical reduction was achieved. The clinical outcome was excellent in 40%, good in 36%, fair in 8% and poor in 16% of the patients. Osteonecrosis of the femoral head was seen 3% and progressive wear of the femoral head in 5% of the hips. Six percent of the patients had hip replacement and 2% had a hip arthrodesis. The author concluded that in many of the patients with complex acetabular fractures the hip joint can be preserved and post-traumatic osteoarthritis can be avoided if an anatomical reduction is achieved.

There is scarcity of good literature on the long-term outcome of treatment of acetabular fractures. However, in 2005, Giannoudis et al. [41] did a meta-analysis to evaluate the clinical outcome of treatment of displaced fractures of the acetabulum. They analyzed a total of 3670 displaced acetabular fractures which were treated operatively. They found that the most common long-term complication was OA of the hip which occurred in 20% of the patients. Avascular necrosis of the femoral head and heterotopic ossification occurred in less than 10% of the patients. The incidence of reoperation, which was mainly for a total hip replacement, was 8%. At an average of 5 years follow up, 75% to 85% of the patients had good to excellent results. Iatrogenic nerve injuries were seen in 8% of the patients. Wound infections were seen in 4.4% and thromboembolic complication were seen in 4.3% of the patients. The authors were of the opinion that patients with displaced articular fractures of the acetabulum should be treated at tertiary medical institutions where surgeons with experience can achieve good reduction of the fractures through minimal surgical approach.

Overall the clinical outcome of treatment of acetabular fractures is good to excellent in 75% to 85% of the patients. The incidence of OA is about 20% and the incidence of end stage OA which requires reconstructive procedure is less than 8%.

DISCUSSION

Articular incongruity and OA

A review of the literature shows that the extent of articular fracture fragment displacement (incongruity) is not the only factor that affects the outcome of treatment of intra-articular fractures. The outcome varies depending on the joint involved and also different parts of the same joint has different tolerance to step-off of the articular surface. In patients with fractures of the distal radius the presence of OA does not negatively affect the clinical outcome. Tibial plateau incongruity is well tolerated with good clinical outcome. The outcome of treatment of ankle malleolar fractures is good even in the absence of a good reduction. For acetabular fractures the preservation of congruity of the weight bearing dome is important for good outcome and the involvement of the posterior wall appears to be a bad prognostic factor [42]. For the treatment of acetabular (especially the dome) and tibial plafond fractures, restoration of congruity appears to be important. The presences of radiological osteoarthritis changes in the joint does not correlate

with clinical outcome and most patients can have good clinical outcome despite the presences of OA [43,44].

CONCLUSION

In the past osteoarthritis has been labelled as a degenerative disease of the joint which is caused by 'wear and tear'. However, research in recent years shows that it is not degenerative disease of the articular cartilage but it is a disease of the joint as an organ and it has an element of innate immune disorder where factors which have not been fully elucidated affect the ability of the body to repair and heal articular cartilage. Many in the medical fraternity believe that joint incongruity from intra-articular fractures will produce OA which will progress to endstage disease which would require a reconstructive procedure. This belief has resulted in an aggressive invasive approach to the management of intra-articular fractures. Incongruity of articular surface is only one of the known risk factors

for post-traumatic OA. Certain joints tolerate incongruity well and this includes the wrist, ankle and the knee. The superior dome of the acetabulum and the tibial plafond does not tolerate incongruity as well. Hence the management of intra-articular fractures should be tailored differently for each joint. The incidence of post-traumatic OA is low after intra-articular fractures, with the exception of the tibial plafond fractures, contrary to what is commonly believed. Furthermore, the presence of OA does not correlate with clinical outcome. Most patients have a good clinical outcome despite the presence of OA. A less aggressive and less invasive approach should be taken in the treatment of intra-articular fractures.

Research is currently focused on elucidating the basic mechanism by which OA develops so that OA can be detected early before radiological changes become manifest and ultimately to develop disease-modifying drugs as has been done for diseases such as Rheumatoid arthritis.

References:

- Loeser R.F., Goldring S.R., Scanzello C.R., et al.: Osteoarthritis: a disease of the joint as an organ. *Arthritis Rheum.* 2012;64:1697-1707.
- Houard X., Goldring M.B., Berenbaum F.: Homeostasis mechanisms in articular cartilage and role of inflammation in osteoarthritis. *Curr Rheumatol Rep.* 2013;15(11):375.
- Ru Liu-Bryan.: Synovium and the innate inflammatory network in osteoarthritis progression. *Curr Rheumatol Rep.* 2013;15(5):323.
- Marsh J.L., Buckwalter J., Gelberman R., et al.: AOA Symposium: Articular Fractures: Does an anatomic reduction really change the result? *J Bone Joint Surg.* 2002;84A:1259-1271.
- Repo R.U., Finlay J.B.: Survival of articular cartilage after controlled impact. *J Bone Joint Surg.* 1977; 59A:1068-1075.
- Buckwalter J.A.: Articular cartilage injuries. *Clin Orthop.* 2002;402:21-37.
- Buckwalter J.A.: Mechanical injuries of articular cartilage. In Finerman G (ed). *Biology and Biomechanics of the Traumatized Synovial Joint.* Park Ridge, IL, American Academy of Orthopaedic Surgeons. 1992;83-96.
- Buckwalter J.A., Brown T.D.: Joint Injury, Repair, and Remodeling Roles in Post-Traumatic Osteoarthritis. *Clin Orthop Relat Res.* 2004;423:7-16.
- Brown T.D., Anderson D.D., Nepola J.V., et al.: Contact stress aberrations following imprecise reduction of simple tibial plateau fractures. *J Orthop Res.* 1988;6:851-862.
- Martin J.A., Buckwalter J.A.: Roles of articular cartilage aging and chondrocyte senescence in the pathogenesis of osteoarthritis. *Iowa Orthop J.* 2001;21:1-7.
- Sommerlath K., Lysholm J., Gillquist J.: The long-term course after treatment of acute anterior cruciate ligament ruptures: A 9 to 16 year follow up. *Am J Sports Med.* 1991;19:156-162.
- Shepherd D.E.T., Seedhom B.B.: The 'instantaneous' compressive modulus of human articular cartilage in joints of the lower limb. *Rheumatology.* 1999;38:124-132.
- Marsh J.L., Weigel D.P., Dirschl D.R.: Tibial plafond fractures: How do these ankles function over time? *J Bone Joint Surg Am.* 2003;85-A(2):287-295.
- Weigel D.P., Marsh J.L.: High-energy fractures of the tibial plateau: Knee function after longer follow-up. *J Bone Joint Surg Am.* 2002;84(9):1541-1551.
- Rademakers M.V., Kerkhoffs G.M., Sierevelt I.N., et al.: Operative treatment of 109 tibial plateau fractures: Five- to 27-year follow-up results. *J Orthop Trauma.* 2007;21(1):5-10.
- Rademakers M.V., Kerkhoffs G.M., Sierevelt I.N., et al.: Intra-articular fractures of the distal femur: A long-term follow-up study of surgically treated patients. *J Orthop Trauma.* 2004;18(4):213-219.
- Giannoudis P.V., Grotz M.R., Papakostidis C., et al.: Operative treatment of displaced fractures of the acetabulum: A meta-analysis. *J Bone Joint Surg Br.* 2005;87(1):2-9.
- Forward D.P., Davis T.R., Sithole J.S.: Do young patients with malunited fractures of the distal radius inevitably develop symptomatic post-traumatic osteoarthritis? *J Bone Joint Surg Br.* 2008;90(5):629-637.
- Kreder H.J., Hanel D.P., Agel J., et al.: Indirect reduction and percutaneous fixation versus open reduction and internal fixation for displaced intra-articular fractures of the distal radius: a randomised, controlled trial. *J Bone Joint Surg [Br]* 2005;87-B:829-836.
- Handoll H.H., Madhok R.: Surgical interventions for treating distal radial fractures in adults. *Cochrane Database Syst Rev.* 2003;(3):CD 003209.
- Rademakers M.V., Kerkhoffs G.M., Sierevelt I.N., et al.: Operative treatment of 109 tibial plateau fractures: five- to 27-year follow-up results. *J Orthop Trauma* 2007;21:5-10.
- Manidakis N., Dosani A., Dimitriou R., et al. Tibial plateau fractures: functional outcome and incidence of osteoarthritis in 125 patients. *International Orthopaedics.* 2010;34:565-570.
- Mehin R, O'Brien P, Broekhuysen H, et al. Endstage arthritis following tibial plateau fractures: average 10 years follow-up. *Can J Surg.* 2012;55:87-94.
- Egol K.A., Tejwani N.C., Walsh M.G., et al.: Predictors of short-term functional outcome following ankle fracture surgery. *J Bone Joint Surg [Am].* 2006;88-A:974-979.
- Lindsjö U. Operative treatment of ankle fracture-dislocations. A follow-up study of 306/321 consecutive cases. *Clin Orthop Relat Res.* 1985;(199):28-38.
- Bauer M, Jonsson K, Nilsson B. Thirty-year follow-up of ankle fractures. *Acta Orthop Scand.* 1985;56:103-106.
- Donken C.C., Verhofstad M.H., Edwards M.J., et al.: Twenty-one-year follow-up of supination-external rotation type II-IV (OTA type B) ankle fractures: a retrospective cohort study. *J Orthop Trauma.* 2012;26(8):e108-114.
- Nelson F., SooHoo., Krenke L., et al.: Complication Rates Following Open Reduction and Internal Fixation of Ankle Fractures. *J Bone Joint Surg Am.* 2009;91(5):1042-1049.
- Babis G.C., Vayanos E.D.: Papaioannou and Pantazopoulos T. Results of treatment of tibial plafond fractures. *Clin Orthop Relat Res.* 1997;341:99-105.
- Burwell HN, Charnley AD.: The treatment of displaced fractures of the ankle by rigid internal fixation and early mobilization. *JBJS* 1965;47B:634-660.
- Etter C., Ganz R.: Long-term results of tibial plafond fractures treated with open reduction and internal fixation. *Arch Orthop Trauma Surg.* 1991;110(6):277-283.
- Pollak A.N., McCarthy M.L., Bess R.S., et al.: Outcomes after treatment of high-energy tibial plafond fractures. *J Bone Joint Surg Am.* 2003;85(10):1893-1900.

33. Harris A.M., Patterson B.M., Sontich J.K., et al.: Results and outcomes after operative treatment of high-energy tibial plafond fractures. *Foot & Ankle International*. 2006;27:256-265.
34. Leonard M., Magill P., Khayyat G.: Minimally-invasive treatment of high velocity intra-articular fractures of the distal tibia. *Int Orthop*. 2009;33(4):1149-1153.
35. Williams T.M., Nepola J.V., DeCoster T.A., et al.: Factors affecting outcome in tibial plafond fractures. *Clin Orthop Relat Res*. 2004;423:93-98.
36. Marsh J.L., Weigel D.P., Dirschl D.R.: Tibial Plafond Fractures: How do these ankles function over time? *J Bone Joint Surg*. 2003;85A(2):287-295.
37. Rüedi T.P., Allgöwer M.: The operative treatment of intra-articular fractures of the lower end of the tibia. *Clin Orthop*. 1979;138:105-110.
38. Bourne R.B., Rorabeck C.H., Macnab J.: Intra-articular fractures of the distal tibia: the pilon fracture. *J Trauma*. 1983;23:591-596.
39. Ovidia D.N., Beals R.K.: Fractures of the tibial plafond. *J Bone Joint Surg Am*. 1986;68:543-551.
40. Sen R.K., Veerappa L.A.: Long-term outcome of conservatively managed displaced acetabular fractures. *J Trauma*. 2009;67(1):155-159.
41. Heeg M., Oostvogel H.J., Klasen H.J.: Conservative treatment of acetabular fractures: the role of the weight-bearing dome and anatomic reduction in the ultimate results. *J Trauma*. 1987;27(5):555-559.
42. Matta J.M.: Fractures of the acetabulum: accuracy of reduction and clinical results in patients managed operatively within three weeks after the injury. *J Bone Joint Surg Am*. 1996;78(11):1632-1645.
43. Giannoudis P.V., Grotz M.R., Papakostidis C., et al.: Operative treatment of displaced fractures of the acetabulum. A meta-analysis. *J Bone Joint Surg Br*. 2005;87(1):2-9.
44. Giannoudis P.V., Tzioupis C., Papathanassopoulos A., et al.: Articular step-off and risk of post-traumatic osteoarthritis. Evidence today. *Injury*. 2010;41(10):986-995.