

**THE OUTCOME OF RETAINING A STABLE
IMPLANT IN UNUNITED INFECTED FRACTURE
FOLLOWING OPEN REDUCTION AND
INTERNAL FIXATION OF CLOSED FRACTURES
IN LONG BONES**

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TABLES OF CONTENTS

List of tables	v
List of figures	vi
List of abbreviations	xi
Acknowledgement	xii
Abstrak	xiii
Abstract	xv
I. INTRODUCTION	1
I.1. Aim	4
I.2. Hypothesis	4
2. LITERATURE REVIEW	5
2.1. History.	5
2.2. Bone biology	7
2.3. Fracture healing	8
2.3.1. Pathophysiology	8
2.3.2. Diagnosis of union	10
2.3.3. Factors influence healing	12
2.3.3 (a). Systemic status of the patient	12
2.3.3 (b). Local pre injury limb status	12
2.3.3.(c) Nature of the injury	13
2.3.3.(d) Orthopedic fracture care	13

2.4. Infection around the implant	14
2.4.1. Incidence of infections around the implants	14
2.4.2. Pathophysiology	15
2.4.3. Diagnosis	18
2.4.3 (a). Clinical features	18
2.4.3.(b). Radiology	22
2.4.3.(c). Microbiology	26
3. DEFINITION	28
4. METHODOLOGY	31
5. RESULTS AND ANALYSIS	34
5.1. Age	36
5.2. Sex	37
5.3. Ethnic groups	38
5.4. Bone involvement	39
5.5. Site of infected fractures	41
5.6. Type of fractures	42
5.7. Type of implants	43
5.8. Onset of symptoms	44
5.9. Severity of infections	45
5.10. Organism cultured	46
5.11. Associated injuries	48
5.12. Associated medical problems	50
5.13. Outcome of treatment	52
5.14. Union time	53

6.	CASE ILLUSTRATION	55
	6.1. Case 2	55
	6.2. Case 3	60
	6.3. Case 5	64
	6.4. Case 11	68
	6.5. Case 16	73
	6.6. Case 26	76
	6.7. Case 27	79
	6.8. Case 29	82
7.	DISCUSSION	85
	7.1. Dermographic	85
	7.2. Site of bone	85
	7.3. Severity of fractures	87
	7.4. Type of implant	87
	7.5. Onset of symptoms	90
	7.6. Severity of infection	92
	7.7. Immunological status	94
	7.8. Microbiology	95
	7.9. Outcome of treatment	97
8.	SUMMARY AND CONCLUSIONS	98
9.	RECOMMENDATION	99
10.	LIMITATION	100
11.	REFERENCE	101
12.	APPENDIX	113

List of table

Table 5.1	Category of patient according to the stability of implant and fracture union in infected fracture.	34
Table 5.2	List of patients with infected implant	35
Table 5.3	The outcome base on sex	37
Table 5.4	Outcome of patient according to ethnic groups.	38
Table 5.5	Outcome of treatment base on bone involvement.	40
Table 5.6	Outcome of treatment base on site of infected fractures.	41
Table 5.7	Outcome base on type of fractures	42
Table 5.8	Outcome of treatment base on type of implant.	43
Table 5.9	Outcome base on onset of infections	44
Table 5.10	Outcome of treatment base on severity of infection	45
Table 5.11	Outcome base on type of organisms	47
Table 5.12	Outcome base on associated injuries.	49
Table 5.13	Outcome of patient base on associated medical problems.	51
Table 5.14	Number of patient with union and delayed union among the success group.	54

List of figures

Figure 2.1	Anatomical staging of osteomyelitis (Cierny and Mader ,1984)	20
Figure 2.2	Infection on the surface of implant	23
Figure 2.3	Florid callus reaction in a fracture fix with dynamic compression plate.	24
Figure 2.4.	Florid callus one month after fixation	24
Figure 2.5	Marked osteolysis with implant loosening	25
Figure 2.6.	Osteolysis with intact implant.	25
Figure 5.1	Age distribution of the study population	36
Figure 5.2	Distribution of sex	37
Figure 5.3	Ethnic groups distribution.	38
Figure 5.4	Type of bone involvement	39
Figure 5.5	Site of infected fracture	41
Figure 5.6	Type of fracture	42
Figure 5.7	Type of implant	43
Figure 5.8	Onset of symptoms	44
Figure 5.9	Severity of infection	45
Figure 5.10	Type of organisms	46
Figure 5.11	Associated injuries	48
Figure 5.12	Patient associated medical problems category	49
Figure 5.13	Outcome of treatment	52
Figure 5.14	Union time among the successful group	53

Figure 6.1 Radiograph of Case 2

Figure 6.1.1. Post operative radiograph 57

Figure 6.1.2. Two month after operation showing
early rarefaction 57

Figure 6.1.3. Four month after operation showing more
rarefaction and early loosening . 58

Figure 6.1.4. Five month after operation , the plate break
after patient start full weight bearing 58

Figure 6.1.5. After replating and insertion of bone graft 59

Figure 6.1.6. One year after injury showing union 59

Figure 6.2 Radiograph of Case 3

Figure 6.2.1. Post operative radiograph 61

Figure 6.2.2. One month post operation showing marked
osteolysis and early callus formation 61

Figure 6.2.3. Two month after operation showing
marked osteolysis 62

Figure 6.2.4. Six month after operation showing marked
medial callus and early loosening of proximal screws 62

Figure 6.2.5 Eighteen month after operation showing union 63

Figure 6.2.6. Two years after operation showing union with
loosening of almost all the screws 63

Figure 6.3 Radiograph of Case 5

Figure 6.3.1 Post operative radiograph 65

Figure 6.3.2. One month after operation showing minimal changes on radiograph 65

Figure 6.3.3 Two month after op showing exuberant callus with early loosening and angulation of distal fragment 66

Figure 6.3.4. Three month after operation showing angulation of fracture. Two loose screws have been removed 66

Figure 6.3.5 Ten month after operation showing solid union. 67

Figure 6.3.6. Two years after operation the plate were removed 67

Figure 6.4 Radiograph of Case 11

Figure 6.4.1. post operative radiograph 69

Figure 6.4.2 One month after operation showing early callus formation 69

Figure 6.4.3 after 4 month 70

Figure 6.4.4. after 7 month showing strong callus 70

Figure 6.4.5 after 9 months 71

Figure 6.4.6 after removal of nail 71

Figure 6.4.7 Two years after operation when he came with persistent discharge 72

Figure 6.4.8. After removal of sequestrum he was free of discharge 72

Figure 6.5 Radiograph of Case 16	
.Figure 6.5.1. postoperative radiograph	74
Figure 6.5.2. Two months after operation	74
Figure 6.5.3. Four month after operation showing rarefaction .	75
The lag screw have been removed due to loosening	
Figure 6.5.4. Eight month after operation showing	75
primary bone union	
Figure 6.6 Radiograph of Case 26	
Figure6.6.1 post operative radiograph	77
Figure 6.6.2. One month after operation	77
Figure 6.6.3 Two month after operation showing early osteolysis	78
Figure 6.6.4 Four month after operation showing minimal	78
medial callus	
Figure 6.7 Radiograph of Case 27	
Figure 6.7.1. post operative radiograph	80
Figure 6.7.2. Four month after operation showing locency	80
around the screws	
Figure 6.7.3. Seven month after operation showing marked	81
osteolysis and early loosening	
Figure 6.7.4.Eight month after operation showing more	81
bone resorbtion	

Figure 6.8 Radiograph of Case 29

Figure 6.8.1 .Post operative radiograph	83
Figure 6.8.2. One month after operation showing minimal changes from post operative radiograph	83
Figure 6.8.3. Two month post operation showing early loosening	84
Figure 6.8.4. Three month post operation showing loosening of all proximal screws	84

List of abbreviations

DCP	Dynamic compression plate
IM nail	Intra medullary nail
MRSA	Methicilline Resistance Staphylococcus Aureus
ICU	Intensive Care Unit
Gram -ve	Grams negative bacteria

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Abstrak

Kajian mengenai hasil rawatan mengekalkan implan yang stabil tetapi dijangkiti kuman pada kepatahan yang belum sembuh setelah pembedahan untuk kepatahan “close fracture” pada tulang panjang (long bones).

Ini merupakan kajian retrospektif keatas 30 orang pesakit yang mengidap jangkitan pada kepatahan yang belum sembuh tetapi mempunyai implan yang stabil. Kajian diadakan dari bulan Januari 1995 sehingga Disember 2000 di Hospital Universiti Sains Malaysia Kubang Kerian.

Kepatahan yang dijangkiti kuman dan belum sembuh merupakan dilema kepada kepakaran Ortopedik samada mahu menggantikan implan atau membiarkannya sehingga kepatahan sembuh. Tujuan kajian ini adalah untuk mengkaji hasil rawatan sekiranya implan dibiarkan sehingga kepatahan sembuh walaupun sedang dijangkiti kuman. Ia juga bertujuan untuk mengkaji faktor-faktor yang menyumbang kepada kegagalan rawatan cara ini dan jenis-jenis kuman penyebab jangkitan ini.

Keputusan menunjukkan kejayaan sebanyak 77% jika mengikuti rawatan cara ini. *Staphylococcus aureus* telah dijumpai pada 80% pesakit.

Walaupun bagaimanapun kajian ini menunjukkan jenis tempat dan tahap kepatahan, jenis implan, tahap jangkitan, jenis kuman, masalah perubatan dan kecederaan lain tidak menentukan kejayaan rawatan cara ini.

Berdasarkan keputusan ini menunjukkan pengejalan implan yang stabil tetapi dijangkiti kuman pada kepatahan yang belum sembuh boleh dijadikan cara rawatan awal bagi penyakit ini sehingga kepatahan sembuh.

Abstract

The outcome of retaining a stable implant in ununited infected fractures following open reduction and internal fixation of closed fractures in long bones.

This is a retrospective study of 30 patients with ununited infected fractures but with stable implant. The study was done between January 1995 to December 2000 in University Science Malaysia Kubang Kerian.

An infected ununited fracture is an orthopedic dilemma in deciding whether to remove the implant or retain it until union has been achieved. The aim of this study is to determine the outcome of patients where the stable implants were retained despite the presence of infection. This study is also aimed to identify the risk factors for failure of treatment and identify the microbiology pattern.

The result of this study showed a success rate of 77%. The commonest organism was *Staphylococcus aureus* which had been identified in 80% of patients. This study showed that site and severity of fracture, type of implant, onset and severity of infection, type of organism and associated medical problems and injuries does not significantly influence the outcome of patient with an infected ununited fractures of long bone with retained internal fixation.

I concluded that retaining an infected but stable implant in ununited fracture until the fracture has healed can be an initial treatment for all patients with infected fractures following open reduction and internal fixation of closed fractures in long bones.

1. INTRODUCTION

Osteomyelitis, unlike other infection is not consistently treated with success despite the extensive array of antibiotics now available. Although such drugs have improved the prognosis in acute hematogenous osteomyelitis, they have not been as successful in chronic osteomyelitis or in sepsis that develops around the implants. The frequent recurrence despite intensive treatment with both surgery and prolonged antibiotics suggest that many fundamental questions remain unanswered. The prevalent perioperative use of antibiotic has fostered the development of a low grade and delayed infection in contrast with fulminant sepsis. (Fitzgerald ,1983)

Often, patients with post traumatic osteomyelitis end up with chronic infection. It is characterized by foci in the bone which contains pus, infected granulation tissue, sequestra, draining sinus and resistant cellulitis. The inflammatory foci are surrounded by sclerotic bone with poor blood supply and covered by a thick, relatively avascular periosteum and scarred muscle and subcutaneous tissue. Antibiotics reach in such tissue mainly by diffusion.(Weiland et al ,1984)

Therefore, the goal of surgical treatment is to convert an infection with dead bone to a situation with well vascularized tissue that are readily penetrated by blood borne antibiotics.(Mader et al ,1993). However this surgical procedure will end up with instability of the fracture and a large tissue defect.

Stability of fracture is also important for fracture healing as well as controlling infection. (Warlock et al, 1994 ; Tetsworth and Cierny, 1999)

It had been shown that rigid stabilization of fracture is imperative in infected non union as infected fracture can unite either by callus or primary bone union. That's why most authors agreed that it is not necessary to remove the stable implant in an infected fracture. The idea is to get the fracture to heal first before tackling the sepsis. (Meyer et al,1975;Waldvogel and Vasey,1980 ; Jones W , 1982 ;Patzaki et al,1986).

The management of infected fracture with loose implant or when the fracture has already united is debridement and removal of implant. The dilemma confronting the surgeon concerns the removal or retention of metal in the presence of active infection in a fracture that is still not united .(Patzaki et al,1986 ; Perry CR ,1996). There are 2 strategies to overcome these problems .

The first technique is to keep the implant until the fracture has healed before tackling the infection. The second technique is immediate debridement of implant and necrotic tissue and stability is achieved by external fixator. The debrided defect is reconstructed by cancellous bone grafting ,local flap or free vascularized flap or distraction osteogenesis. (Ueng et al, 1999)

The first technique was claimed to have an unpredictable outcome that may end up with infective non union or persistent sinus discharge even after the fracture has healed. Although technically it is easier, it needs prolonged antibiotics and wound care. (Meyer et al ,1975; Patzaki et al ,1986 ; Kostuik and Harrington,1975; Kovacs et al,1973).

The second strategy claims to have a more predictable outcome of bony union and is free of infection. Wound care is easier and doesn't need prolonged antibiotics. However it is technically more demanding because it creates a large soft tissue and bony defect which needs secondary reconstructive procedure to cover it. (Ueng et al, 1999: Green and Dlabal,1983; Kelly,1984; Klemm,1993)

I.1. Aim

The purpose of this study is to determine :

1. the outcome of retaining an infected but stable internal fixation of long bones in fracture which has not united .
2. the risk factor for infected non-union or chronic osteomyelitis in patient treated with these methods.
3. the bacteriological pattern for infection following internal fixation of closed fractures in long bone.

I.2. Hypothesis

Infected long bones fracture with a stable implant will heal and the infection will resolve after removal of the implant.

2. LITERATURE REVIEW

2.1. History

Trueta (1940) emphasized the need for adequate debridement for treatment of orthopedic infection. Since then the principles of osteomyelitis surgery include atraumatic approach and removal of all necrotic and nonviable material.(Tetsworth and Cierny ,1999). Ritman and Paren (1974) experimental work support the concept that stabilization was beneficial in treating established post traumatic osteomyelitis and the stabilizing effect of implant outweighs the harm of their foreign body effect.

These 2 principles in treating infected fracture are difficult to meet without compromising each other. The earlier orthopedic surgeons tended to treat the fracture first by retaining the implant to provide stability and delaying the aggressive debridement after the fracture healed.

Kovac et al (1973) retained the nail in spite of infection and removed the implant only after the fracture had united . Kostuik and Harrington (1975) also suggested retaining the nail, but if the intramedullary nail was loose they suggested to change it with a bigger nail to achieve stability. They believed rigid intramedullary nailing is superior than plating in treating the infected and ununited fractures.

At the same time Mayer et al (1975) have used the compression plate with variable success in treating infected non union . Rosen (1979) did debridement of necrotic tissue and bone and filled it with bone graft before stabilizing it with compression plate in treating his patient. Muller and Thomas (1979) recognized the valuable use of external fixator in treatment of infected non union in tibia where there are poor skin condition and gross infection.

Green and Diab (1983) used external fixator and open bone graft technique after proper debridement. Kelly (1984) used external fixator, bone graft and muscle flap.

Klemm (1993) introduced the concept of pre formed gentamycin PMMA beads which produce high concentration of antibiotic in the infected area , eliminate dead space and eliminates daily dressing .

Ueng et al (1999) used bone graft or free vascularized graft ,external fixator and local antibiotic beads to treat infected fracture.

The milestone in treating the infected fracture occurred during 1980's with the introduction of rigid external fixator frame, new technique in tissue transfer and local antibiotic which made adequate debridement possible without compromising the stability or leaving a dead space.(Cierny ,1999)

2.2. Bone biology

The main function of bony skeleton is to provide a strong supportive and mechanically optimal structure for the soft tissues and muscles. It is composed of cellular and non cellular element.

The cellular element are derived from several stem cells line which include the osteoblast, osteocytes, osteoclast and mesenchymal osteoprogenitor cells. These varied cell lines form a dynamic reactive system central to bone growth, repair and remodelling.

The non cellular matrix is composed of an organic and nonorganic parts. The organics materials include collagen fibers, proteoglycans, glycoproteins, phospholipids and phosphoproteins. The collagens gives the bone tensile strength and flexibility. The inorganic part which make up 60% of the dry weight consist of calcium hydroxyapatite and osteocalcium phosphate. They gives the compressive strength to the bone. The tubular shape of the bone combines the strength and lightness.

The bone has a very rich blood supply reflecting the high metabolic activity of bone derived from nutrient, periarticular and periosteal systems. It continually remodel according to mechanical force acting upon it (Wolf's Law) (Webb and Tricker ,2000)

2.3. Fracture healing

2.3.I. Pathophysiology

Bone can regenerate itself when injured and unlike other tissue it can repair itself with bone (Webb and Tricker ,2000). Both biological and mechanical factors are important in fracture healing (Einhorn,1995).

The classical description divides 2 type of fracture healing which are primary and secondary healing. Primary healing occur when there is a combination of anatomical reduction, stabilization and compression of the fracture as occurs in a plate fixation. It is basically involves direct cortical remodelling, which is a formation of cutting cones.

The great majority of fractures undergo secondary healing, which requires some motion at the fracture site. This may be achieved in non operative treatment or a surgical procedure that retain some mobility. It follows the sequence describe by McKibbin. (McKibbin, 1978).

The original description of fracture healing was based on histological observations which suggest sequential phase of hematoma , inflammation ,callus formation and remodelling (McKibbin, 1978). These responses take place in the marrow, cortex, periosteum and external soft tissues. (Einhorn, 1998)

Fracture leads to disruption of blood supply and release of the cytokines that initiate healing process. These cytokines have a role in forming new blood vessels (angiogenesis), attracting (chemotaxis) and regulating the mesenchymal cells. (Webb and Tricker ,2000)

2.3.2. Diagnosis of union

Fracture union is a gradual process. It is difficult to decide the end point where risk of refracture is minimal. The definition ranges from clinical ,radiological to mechanical criteria.

Oni et al (1988) define union when all immobilisation aids had been discarded and unrestricted weight bearing was allowed.

Angliss et al (1996) defined union by bridging callus on serial radiograph while Chritensen et al (1980) define union as the disappearance of visible fractures lines and the development of slight amount of solid periosteal bridging callus.

Puno et al (1986) define union when pain, swelling, tenderness or motion at fracture site had disappeared and when there was partial or complete obliteration of the fracture line on plain radiograph.

Richardson et al (1994) define union when the sagittal plane stiffness is 15Nm/degree in his patients with tibial fracture treated with external fixator. He found out that risk of refracture were significantly less when union was judged biomechanically as compared to union judged clinically.

Oni et al (1988) on reviewing fracture tibia treated conservatively have shown that fracture union is not directly related to the size of callus. Furthermore fracture treated with rigid compression plate healed without callus and fractures treated with intramedullary nail healed with external callus. (Marsh, 1998)

Marsh (1998) also found out that there is no correlation between callus index and bending stiffness measurement.

I've decided to use definition by Puno et al since it is more practical in our setting.

2.3.3. Factors that influence healing (Hayda et al, 1998)

For the purpose of discussion the factors that influence fracture healing will be divided into systemic status of the patient, local limb status before the injury, the nature of the injury, and orthopedic fracture care.

2.3.3 (a). Systemic status of the patient

The increased rate of healing among children compared to an adults may be related to the vascularity of the periosteum. Malnutrition, anaemia ,diabetes mellitus and growth hormon deficiency have been shown to be associated with delayed union.

2.3.3 (b). Local pre injury limb status

Preexisting damage to soft tissue like previous trauma, surgery, irradiation, vascular disease and oedema all have potential effect on blood flow and oxygen delivery and thus influence bone healing. In addition low vascularity or low oxygen tension have been shown to shunt undifferentiated mesenchymal cells into a chondrogenic pathway. (Carter et al ,1998)

2.3.3.(c) Nature of the injury

The energy of impact, extent of soft tissue, nerve and vascular injury and compartment syndromes all have been shown to cause delayed union.

Infection causes intense inflammatory reactions increases the tissue damage and compromises the healing environment.

2.3.3.(d) Orthopedic fracture care

A gap of more than 2 mm will adversely effect healing. An inadequate immobilisation and disrupted neovascularization can impede bone healing. Torsional instability has been shown to cause non union whereas axial instability promotes healing.(Kenwright et al ,1991)

Mechanobiological studies have shown that bone formation is permitted in areas of low to moderate tensile strain, fibrous tissue is promoted in areas of moderate to high tensile strain and chondrogenesis is promoted in areas of hydrostatic compressive stress (pressure). (Carter et al ,1998)

Rand et al (1981) compared the effect of open intramedullary nailing and plating on blood flow and union. He found that blood flow reach higher level and remain elevated longer in nailing group. However fracture gain mechanical strength more slowly in nailing than in plate fixed fracture.

2.4. Infection around the implant

2.4.I. Incidence of infection

Almost all operative wounds are contaminated by bacteria and whether or not a clinical infection occurs depends on the extent of the contamination, local factors (presence of dead space, necrotic tissue or foreign bodies) and the body cellular and humoral defence mechanism (Pavel et al, 1974) . Dobbins et al (1988) found that 77% of the implants removed from fractures which were clinically not infected were colonized by bacteria .

In addition, Pavel et al(1974) noted that incidence of infection following a clean orthopedic surgery with prophylactic antibiotics was 2.8% as compared to placebo (5%). Fitzgerald (1994) noted the incidence of infection in a closed fracture was 0.7% as compared to open fracture (1.7%). Puno et al (1986) discovered 2.3% infection rate in treating closed tibial fractures with intramedullary nails. Court Brown et al (1992) had incidence of 1.8% infection rate following intramedullary nail for closed and open grade 1 tibial fractures.

2.4.2. Pathophysiology

The first step is entry of the pathogen which usually occurs following trauma or surgery. The bacteria must break the mechanical barrier like the skin and then colonize in the host tissue. Finally the clinical infection occurs when there is damage to the host.(Tsukuyama ,1999)

The traumatized tissue provides potential binding site for bacteria. *Staphylococcus aureus* has receptors for numerous host proteins e.g fibronectin, fibrinogen and laminin which helps them to adhere to the bone or the metal. Traumatized tissues also result in compromised blood supply and lead to tissue and bone necrosis and dead bone acts like a foreign body. In fracture it will also lead to instability which will cause further soft tissue damage , impaired healing and increased risk of infection .(Gustilo et al , 1990) .

Acute inflammation not only destroys and contains the spread of infection , proteolytic enzyme released by the phagocyte also damages the surrounding tissue. The influx of host defence cell and fluid infiltrate increases the pressure within the rigid confines of bone causing infarction of marrow. Generally these areas have poor vascular perfusion which is poorly penetrated by the antibiotics. (Tsukuyama, 1999)

Infection will also depend on the overall systemic trauma and additional effects of morbidity and local host damage . (Cierny and Mader, 1984; Hansis 1996; Mader, 1993) .

The fixation device acts as an additional foci for bacterial adherence. Studies have shown that as low as 50 contaminating organisms can result in infection in the presence of implant as compared to 10,000 organism in the absence of foreign body (Southwood et al, 1985) . It has been shown that the antibiotic resistance is related to these surface adhesion organisms as compared to the suspension organisms.(Naylor et al ,1990).

Once attached to host surfaces, many bacteria like Staphylococcus, Streptococcus and Pseudomonas have the ability to adhere firmly by production of biofilm. Biofilm forms strong bonds with the glycoprotein of tissue substrate. It protects the bacteria from the antibody, antibiotics and phagocytes and may be the key factors of difficulty in eradicating bacteria from the bone. (Gristina et al, 1983,1985,1991) .

The presence of implant will cause chronic inflammation which damages the tissues and directly protects the bacteria by reducing capillary flow and impairment of the polymorph functions to kill the organisms. (Petty et al, 1985; Printzen ,1996) .

The susceptibility of the bacteria to antibiotics is also reduced because of their reduced metabolic rate when attached to the implant. (Chuard et al ,1991)

Chronicity of infection is therefore due to biofilm, the presence of implant and ischaemic environment. Therefore for all these reason, operative treatment should be considered whenever possible . (Ciampolini and Harding ,2000)

However Widmer et al (1992) have shown that it is possible to cure implant related infection with Rifampicin without removing the implant .

2.4.3. Diagnosis

2.4.3. (a) Clinical features

Alteimeir et al described surgical wound as uninfected, possibly infected or definitely infected. Uninfected wounds heal without discharge. Possibly infected wounds are either inflamed without discharge or discharge without significant inflammation. A definitely infected wound is one with purulent discharge whether or not the organism is cultured (Trafton, 1984).

Centers for Disease Control (CDC) defines post operative infection as infection either deep or superficial occurring within 30 days after surgery or as late as 1 year if an implant is used (Peterson and Fitzgerald, 1994). Their criteria for deep infection are :

1. persistent drainage from drain placed deep into the fascia
2. spontaneous drainage of surgical wound or deliberate surgical opening associated with fever, pain and tenderness
3. abscess formation
4. presumed clinical diagnosis as determined by surgeon

The Orthopedic Trauma Association criteria for infection include (Puno et al, 1986):

1. presence of local sign of inflammation
2. presence of serous or purulent discharge or
3. direct or indirect bacterial confirmation

However Dobbins et al (1988) have cultured implants retrieved from asymptomatic patients and found bacteria in 77% of cases which suggests that adherent bacteria can exist for years in dormant state on implant without evoking the clinical sign of infection.

Gambhir et al (2000) also realised that definitive culture can be negative despite the overt appearance of deep infection.

The diagnosis of infected fracture is therefore mainly based on clinical judgment.

Gustilo thinks that any temperature elevation on the 3rd day after surgery or thereafter should arouse a strong suspicion of wound infection. It should be remembered that the most common potential complication in the management of open fracture or any open reduction and internal fixation of a fracture is infection.

JR Border (1987) thinks it is wrong to consider an infected fracture is equal to osteomyelitis since osteomyelitis literally means infection of bone and marrow whereas an infected fracture may just be an infection of the surrounding soft tissue, hematoma or around the implant.

McGraw and Lim (1988) classified deep infection as an intramedullary infection where there is purulent discharge or positive culture in the medullary cavity

which requires intramedullary reaming. They defined osteomyelitis when there is sequestrum which requires debridement and sequestrectomy.

Cierny and Mader (1984) consider infection on the surface of bone cortex or infection in the medullary cavity are as part of osteomyelitis.

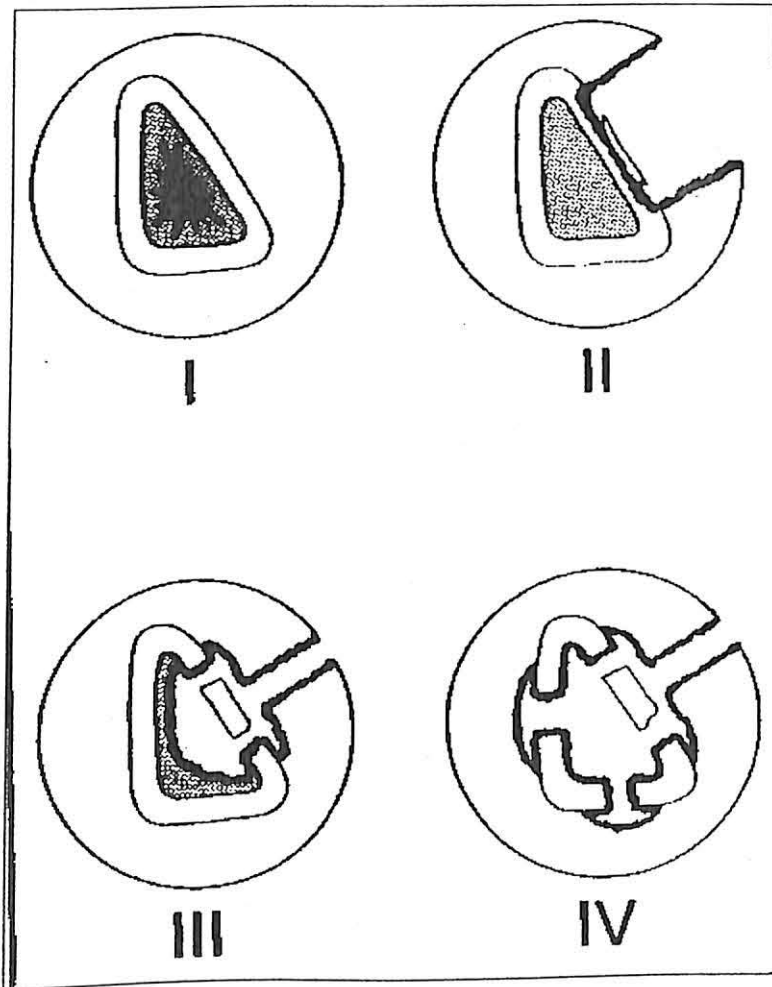


Figure 2.1. Anatomical staging of osteomyelitis (Cierny and Mader, 1984)

Stage I – intramedullary infections e.g. hematogenous osteomyelitis or infected intramedullary rods.

Stage II – limited to surface of bone e.g. infected plate

Stage III – well marginated by reactive or healthy bone and usually involves both medullary and periosteal surface e.g most infected fracture with stable implant

Stage IV – lesions are mechanically unstable either at presentation or after debridement e.g infected non union

2.4.3. (b). Radiology

Early radiological evidence of infection includes soft tissue swelling with distorted fascial planes and loss of fat interface. These findings can precede bone changes by several days. Periosteal reactions are also an early skeletal feature of osteomyelitis. Bone destruction present in the later phases ranging from permeative, geographic to moth-eaten appearance depending on the duration and rapidity of skeletal lysis. (David et al, 1987)

It is apparent that 3 types of bone reactions were observed.

1- No bony changes or reaction on the surface of implant

this occurs when infection involves only the surface of the implant without bone infection. (it is abscess around the implant rather than true osteomyelitis)

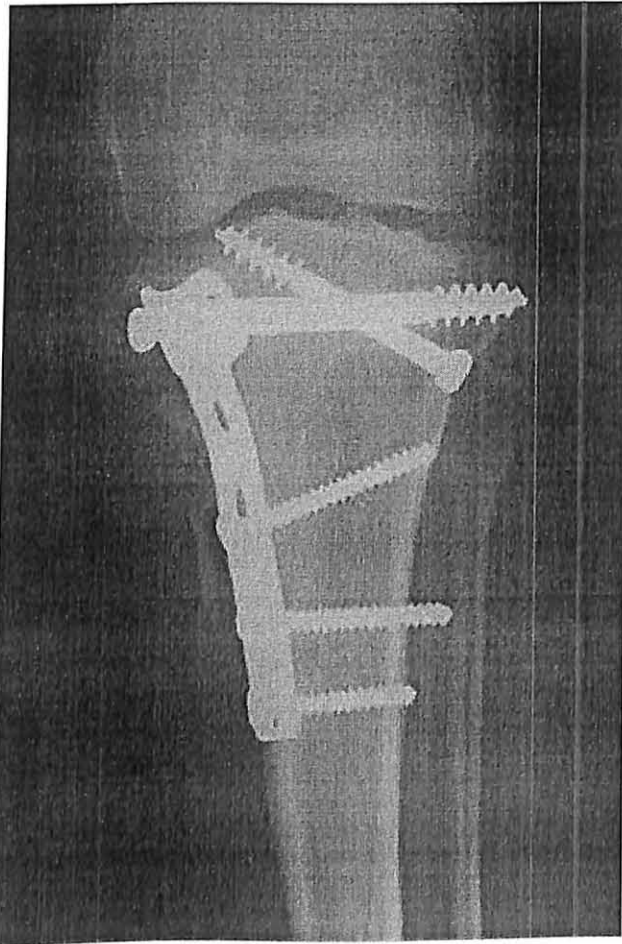


Figure 2.2 Infection on the surface of implant

2- Periosteal reactions

This mainly occurs when infection occurs following intramedullary nail but can also happen in plate fixation. The outcome is good because union is achieved even though the implant may become loose.

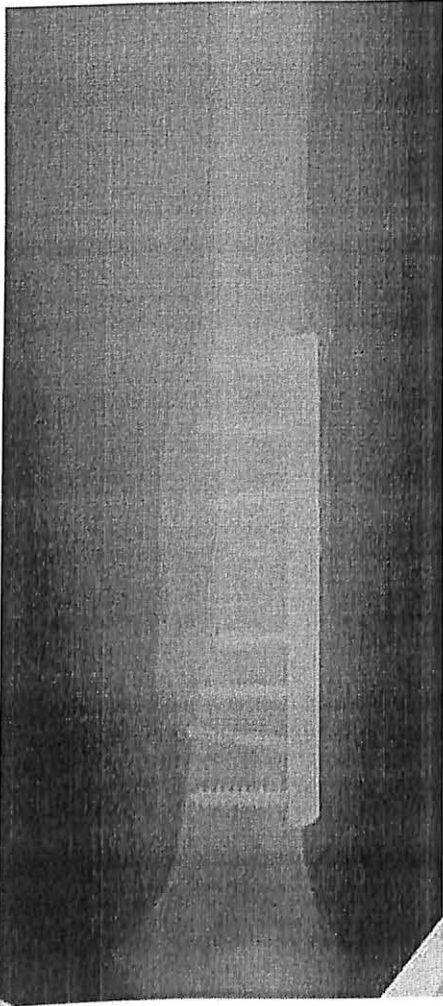


Figure 2.3 .Florid callus reaction in a fracture fix with dynamic compression plate.

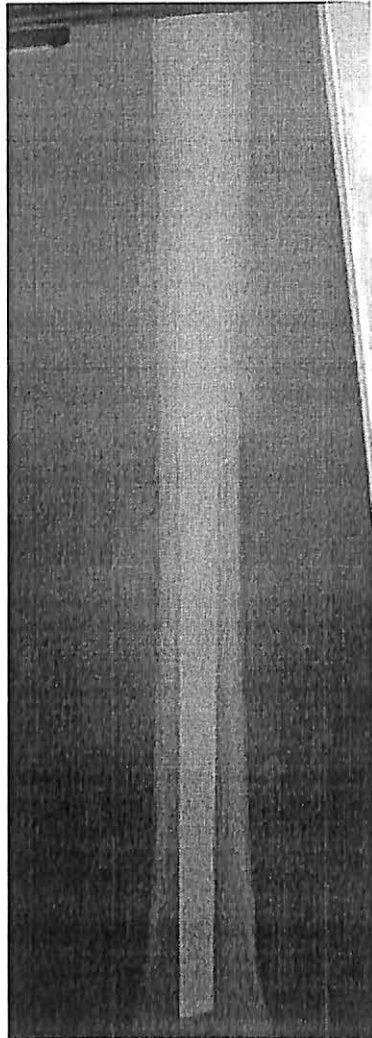


Figure 2.4. Florid callus one month after fixation.