

**FACTORS INFLUENCING THE OUTCOME OF
ACUTE HAEMATOGENOUS OSTEOMYELITIS
(AHO) AT THE UNIVERSITY TEACHING
HOSPITAL, LUSAKA**

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of the Requirement for the Degree of Master of Medicine in
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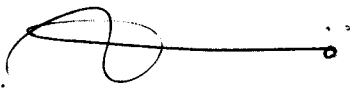
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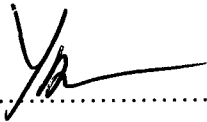
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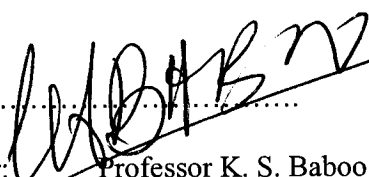
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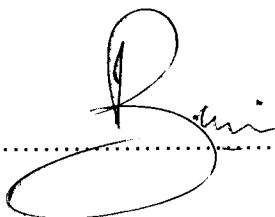
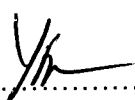
LIST OF ABBREVIATIONS

Hb	-	Haemoglobin
ESR	-	Erythrocyte Sedimentation Rate
AHO	-	Acute Haemogenous Osteomyelitis
COM	-	Chronic Osteomyelitis
SO	-	Sub Acute Osteomyelitis
WCC	-	White Cell Count
CRP	-	C-Reactive Protein
MRI	-	Magnetic Resonance Imaging
GRZ	-	Government of the Republic of Zambia
ARVT	-	Anti-Retroviral Therapy
Prn	-	When need arises
IV	-	Intra-Venous
UTH	-	University Teaching Hospital
SCD	-	Sickle Cell Disease
HIV	-	Human Immunodeficiency Virus
AIDS	-	Acquired Immunodeficiency Syndrome
ELISA	-	Enzyme Linked Immuno Sorbet Assay

APPROVAL

The University of Zambia approves this dissertation of Dr. Maundo Makunika Chowa in partial fulfillment of requirements for the award of the Master of Medicine in Orthopaedic Surgery.

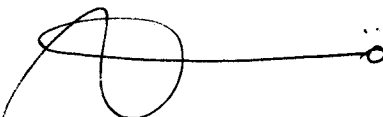
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ABSTRACT

This is a cross sectional review study of 112 patients with Acute Haematogenous Osteomyelitis (AHO) of long bones of the upper and lower extremities who were seen in the orthopaedic clinic at the University Teaching Hospital in Lusaka between July, 2002 and August, 2003. The results show that AHO is more common in children than in adults; ratio 4:1 and in males than females, ratio 2:1.

Delayed Presentation (one week after onset of symptoms) was seen in 64% (72) patients and Early Presentation (within 5 days after onset of symptom) in 36% (40) patients. The results show a poor outcome of AHO in 48% patients in the Delayed Presentation group compared to 6% in the Early Presentation group. Delayed Presentation was caused by high poverty levels and failures of the referral health system. Lower socioeconomic status was seen in 69% of AHO patients. All the patients in the Delayed Presentation group had inappropriate initial treatment because Primary Medical Attendants, Clinical Officers (63%) and Traditional Healers (37%) failed to recognise AHO and refer for adequate initial treatment. Poor outcome was seen in 61% of the inappropriate treatment group. In the Early Presentation group, 68% patients had inadequate treatment (no admission, no surgery and oral antibiotics) and 22% had adequate treatment (admission, surgical drainage and intravenous antibiotics). The results showed good outcome in 44% in the adequate initial treatment group compared to 13% in the inadequate initial treatment group.

Associated HIV infection was seen in 72% of the adults with AHO. The result showed 78% poor outcome of AHO associated with HIV. Sickle cell disease was seen in 14% children. The results showed that 83% of patients with AHO associated with sickle cell disease had a poor outcome. Poor nutrition status was seen in 26% of all children in the study. The results record 43% poor outcome in the poor nutritional status groups.

Bacteriological studies yielded 73% aerobic gram positive staphylococcus. The results showed 100% sensitivity to Ceftaxime and Vancomycin. These drugs are expensive and not available in state sponsored hospitals. The prohibitive cost and non-availability of appropriate antibiotic treatment contributed to poor outcome in all treatment groups.

INTRODUCTION

Poor outcome of AHO is marked by the development of chronicity, physical disability and uncosmetic appearance. Factors responsible for the poor outcome include; **Delayed Presentation** of acute cases of AHO and **low head immunity** due to poverty and malnutrition in the population ¹¹. Other factors include **mis- diagnosis** of children presenting with a limb pain and a fever by most primary medical attendants. The genesis of AHO is enhanced by the presence of a recent throat, skin or urinary tract infection and recent trauma. These causes confusion in the diagnosis by most primary medical attendants resulting in inadequate and inappropriate treatment. Failure to recognise AHO, can lead to patients receiving inappropriate treatment.

In Africa, high poverty levels force patients to seek traditional health providers as a first choice. **Inadequate treatment** associated **immunosuppressive** disease and collapsing health systems are listed as causes of poor outcome in most series ¹². In Zambia the picture is not far from this. Data is not available on the relationship of these factors on the outcome of osteomyelitis. This study has been undertaken to determine the influence of late presentation, inadequate treatment, inappropriate treatment, sickle cell disease, diabetes and HIV/AIDS on the morbidity and mortality outcome of osteomyelitis. The study will also investigate the causes of delay in presentation, social economic status and nutritional status of children presenting with AHO.

Background: The University teaching Hospital (UTH) is tertiary hospital serving a population approximately 4 million in and around Lusaka. UTH has 1200 beds capacity and is the largest hospital in Zambia. Orthopaedic work is split between two units with 64-bed capacity. All cases of AHO are treated by general surgeons before

being referred to orthopaedic wards and clinic. Patients with AHO are referred to UTH mostly by clinical officers in Lusaka urban clinics.

AIMS AND OBJECTIVES

General Objective

To study the factors that influence the outcome of osteomyelitis of long bones in patients presenting to the Orthopaedic Unit, Department of Surgery, University Teaching Hospital, Lusaka.

Specific Objectives

1. To study the outcome of osteomyelitis in Delayed Presentation.
2. To investigate the causes of Delayed Presentation at UTH.
3. To study the outcome of osteomyelitis treated inadequately and inappropriately.
4. To study the outcome of osteomyelitis associated with poor nutritional status, HIV diseases and sickle cell disease.
5. To investigate the common bacteriological finding from pus swabs from sinuses and determine sensitivity patterns.

RATIONALE

The study is intended to provide information on factors that influence the outcome of osteomyelitis. The study will determine whether delayed presentation, inadequate treatment or inappropriate treatment are associated with a high frequency of poor outcome. The study will review, investigate whether associated debilitating diseases like HIV/AIDS, Diabetes and Sickle Cell disease are responsible for poor outcome. The study will also determine the prevalence of aetiological factors like poor social economical status, poor nutritional status, trauma and recent bacterial infections. In addition the factors responsible for delayed presentation will be investigated. These include failure to recognize and refer cases of acute osteomyelitis and initial consultations with traditional healers. The study will also determine the frequency of staphylococcus Aureus and sensitivity pattern from discharging sinuses. This will give guidance on antibiotics, which must be used empirically before the organism responsible for the acute infection is known.

LITERATURE REVIEW

The prevalence and outcome of osteomyelitis varies greatly from one region to another¹. In the developed world haematogenous osteomyelitis is seen with less frequency than in the past and is more responsive to adequate treatment. Adequate treatment include use of sophisticated imaging techniques, which confirm and localize the earliest lesion of osteomyelitis combined with surgical drainage and use of new antimicrobial agents with wide spectrum and high bioavailability in bone tissue^{1, 3}. Effective rational use of intravenous potent antibiotics and timely surgical intervention, in combination are responsible for improved morbidity and mortality outcome^{3,4}. Other factors responsible for improved outcome of osteomyelitis in the developed world are; .1) high host immunity, .2) high standards of living and .3) informed society that seeks modern medical intervention early⁴. However, preventable morbidity still remains a problem in the face of advanced health care systems^{3,7}. With the sporadic occurrence of acute haematogenous osteomyelitis in developed countries orthopaedic surgeons are now mainly concerned with prevention of infection when implants are used in fracture management, prosthetic joint replacement surgery²⁷.

In contrast, acute haematogenous osteomyelitis is very common in developing countries. Osteomyelitis patients may occupy up to one third of beds in orthopaedic wards. Here the disease is more severe and has a long protracted course affecting mainly children from poor families²¹.

In general, the disease has a poor devastating outcome; accounting for a large number physical disability, frequent re-admissions, re-operations and absenteeism and school stoppage in children³⁴.

HISTORICAL ASPECTS

Before the advent of antibiotics and antimicrobials, acute haematogenous osteomyelitis had mortality rates of between 45% and 50% ¹⁵. The natural course of the disease was radically altered with the introduction of sulphonamides in the 1930's. The mortality rates began to approach 20% and morbidity of 50%. The search for a magic bullet against infections culminated in the discovery of penicillin in 1944. At that time, Penicillin Sensitive Staphylococcus and Streptococcus organism caused the majority of the acute haematogenous osteomyelitis. With wide spread use of Penicillin in the 1950's excellent outcomes were reported with mortality rate falling to between 0 and 1%. In the 1960's morbidity and mortality began to rise due to two factors: .

- 1) The emergence of bacterial resistance to Penicillin and .
- 2) Most surgeons had abandoned surgical treatment in view of reported excellent outcome with use of Penicillin alone ²⁵.

In the 1970's controversy in the treatment of osteomyelitis had centred on:

- (1) What constitutes correct chemotherapy and how far chemotherapy alone can cure the patient and;
- (2) The role of early surgery whether it improved the outcome or caused actual harm.

In 1970, Blockley and Watson suggested that exploration of the metaphysis would result in damage of the epiphyseal growth plate and that the sequelae was too high a price to pay for a disease of certain diagnosis and a good chance of cure by antibiotics only¹⁵. Craven, Pugsley and Blomer's (1970) added further that 95 percent of patients seen within three days do not need surgical exploration.

In 1974, Mollan and Piggot came out strongly in favour of early drainage. Their review of patients with primary bone infection who presented to two Belfast hospitals from 1960 to 1974 revealed that in 59 out of 66 (83 %) surgical explorations yielded pus in the acute stages of the disease and no damage to the epiphysis was reported. They concluded that surgery is an essential therapeutic and diagnostic procedure to be carried out early as a routine in all but the mildest of cases. Routine exploration, drainage and culture of pus should be part of the **adequate management** of acute osteomyelitis ^{36,37}.

To date it is generally agreed that osteomyelitis is an emergency and adequate management requires drainage of pus from either the Metaphysis, Subperiosteal space or Soft tissue combined with rational use high potent intravenous antibiotics.

INCIDENCE

Osteomyelitis in the western world is uncommon. In the United Kingdom, an average 20 new cases of osteomyelitis are reported annually from large to sizeable centres ^{4,12}. These figures are much higher in developing countries.

In South Africa, Hoffman reported an incidence of 92 new cases of osteomyelitis every year at a Red Cross children's hospital in Cape Town. This is about five times the incidence quoted in Western series.

DEFINITIONS AND CLASSIFICATIONS

It's generally agreed, that the term Acute Osteomyelitis refers to a pyogenic bacterial bone infection affecting the Metaphysis, Intramedulla, Epiphysis, Cortex, Periosteum or the surrounding soft tissue ^{12,15,20}.

Nonetheless, osteomyelitis is still classified according to behaviour of causative microbial organism into; 1) **pyogenic osteomyelitis** caused by pus forming organism and **non-pyogenic osteomyelitis** caused by organisms that produce a less aggressive, granulomatous type of infection²⁴. Other Classification of osteomyelitis are based on the duration and mechanism of origin. Based on the mechanism of origin, osteomyelitis is classified as exogenous or haematogenous. **Exogenous Osteomyelitis** is caused by direct inoculation of organism through open fractures (**post-traumatic osteomyelitis**) orthopaedic surgical wound (**postoperative osteomyelitis**) or by contiguous spread from infected local soft tissues. Haematogenous form results from bacteraemia or septicaemia.

Haematogenous Osteomyelitis is further sub-classified into Acute, Post acute, and Sub acute or chronic. **Acute Osteomyelitis** is the earliest stage of the disease occurring 0- 72 hours from onset of symptoms characterized by an acute inflammation and build up of intraosseous pressure especially in the Metaphysis. The term acute osteomyelitis when used in clinical practice usually implies acute haematogenous osteomyelitis^{6,9}. Post acute osteomyelitis also known as late acute osteomyelitis occurs after 3-5 days from onset of symptoms and is marked by presence of pus either in the Metaphysis, Intra medulla or subperiosteal space.

Sub acute Osteomyelitis occurs in two forms; .1) antibiotic altered acute osteomyelitis which occurs from 5 – 10 days from the onset of symptoms attack and 2) Primary Sub acute form of Pyogenic Osteomyelitis in which there has been no acute attack and the patient has not received antibiotic before admission to hospital^{10,17}. The term Chronic Osteomyelitis refers to bone infection that has gone untreated or failed to

respond to treatment^{27,30,34}. Chronic Osteomyelitis occurs 10 days from onset and is marked by remissions and acute exacerbations in years to come.

The eponym **Brodies Abscess** refers to a special form chronic osteomyelitis characterized by radiolucent area surrounded by a sclerotic margin (FIGURE 1). It is a form antibiotic modified acute attack in which the virulence of bacteria and host resistance are evenly balanced³¹. Another form of chronic osteomyelitis is known by the eponym hypertrophic sclerotic osteomyelitis seen as high density hard bone “ivory bone” which may be enlarged “elephant bone” usually with obliteration of intramedullary cavity (Fig 2). A sequestrum is a segment of devitalised bone surrounded by pus and granulation tissue nearly always harbours bacteria. An involucrum usually forms around the infected dead bone and consist of newly formed reactive bone.

(Fig 3). A sequestrum and involucrum are radiological hallmarks of chronic osteomyelitis.

AETIOLOGY AND THE PATHOGENESIS

Haematogenous osteomyelitis is a disease of childhood occurring most frequently between the ages of 5 and 15 years³⁶. The ends of long bones of the lower extremities are involved more often than the upper extremities¹. Males are affected about 3-4 times as frequent as females³⁵.

(a) The Organism

Aerobic gram-positive Staphylococcus is the most infecting organism accounting for about 70% - 80% in most series⁵. The predilection of

staphylococcus aureus for bone tissue is thought to be due to specific bone sailoprotein receptors, which interacts with the staphylococcus cell ¹⁰.

The outcome of the bone lesion depends on a combination factors like the virulence of staphylococcus, local resistance and tissue reaction. The most commonly identified (Gram-negative) bacteria are; Esherichia coli, Pseudomonas and Klebsiella species. Uncommon forms of osteomyelitis due to salmonella (most common in sickle cell disease), Brucellosis, sarcoidosis, coccidiomycosis, Blastomycosis, actinomycosis Bacteroides (Anaerobic), Mycobacterium Tuberculosis and syphilis have been described in literature ^{7,8,28,35}.

Acute haematogenous osteomyelitis is caused by bacteraemia, which is a common occurrence in childhood. A recent throat, respiratory, aural or urinary infection is present in 25% to 50 % cases ²⁷. In most debilitating disease like HIV and diabetes, bone infections are polymicrobial (a mixture of aerobic and anaerobic organism). Staphylococcus aureus is still the most common, followed by Bacteriodes and streptococci species (Group D Streptococci, and Enterococci). Bacteriodes are the most common anaerobic organism followed by Peptococci ³⁴.

(b) Predisposing Factors

Factors that predispose an individual to osteomyelitis are well known. The particular problems of the neonate namely; relative lower resistance to infection and umbilical sepsis predispose to neonatal osteomyelitis^{9,12}. The association of the male child with trauma, the old and diabetic patients with poor peripheral blood supply are known factors²⁵. In old age and diabetic

patients poor blood supply to the extremities is due to arteriosclerosis and diabetic arteriopathy respectively is a predisposing factor¹².

In Sickle cell disease, vaso occlusive events lead to osteonecrosis followed by secondary infection with Bacteria ¹⁶. Chronic debilitating illness, acquired immunodeficiency's and immuno-suppressant therapies like corticosteroids, cancer chemotherapy and irradiation predispose to bacterial infection including osteomyelitis². Skeletal infections also occur frequently in intravenous drug abuser ¹⁴. The association of AHO with trauma explains the high incidence of AHO in the trauma prone male child²⁵.

(c) The Trauma Factor

Morrisy et al, demonstrated that bacteraemia alone without localized tissue trauma had failed to produce osteomyelitis in animal models ⁴. Trauma, howbeit-minor causes damages to the blood vessels in surrounding tissues bone resulting vascular occlusion from micro-thrombi. This leads to vascular stasis and plasma transudation. This establishes a favourable media for growth of bacteria in the site of trauma ⁴.

(d) The Anatomical Factor in Outcome and Aetiology

In children, acute haematogenous osteomyelitis begins in the metaphysis of long bones ⁶. This is probably due to: 1) this is where the blood flow is greatest during the period of skeletal growth. 2) The rapidly growing cells near the growth plate have poorly developed reticular endothelial system (phagocytic system), 3) The delicate vessels are easily injured even with trivial trauma, resulting in **thrombosis, stasis and transudation**, which favour growth of bacteria ^{4,25}. In 1921, Hobo described vascular loops (hairpin

arrangement) of arterioles that take sharp bends at the physis and empty into venous lakes (sinusoids).

The transition from high flow arterioles to low flow venous sinusoid creates turbulences, which favours lodging of bacteria and thrombi in the area ²⁵. Electron microscopic studies have shown end-arteries which when occluded resulted in rapid osteonecrosis of the area supplied. The difference in outcome of osteomyelitis between infancy, childhood and adult is due to the variation in the pattern of vascular supply of the metaphyseal bone.

During infancy before the age 18 months the **Metaphyseal** arterioles perforate the immature growth plate hence infantile osteomyelitis is frequently complicated by septic arthritis and epiphyseolysis. After 18 months the mature growth plate becomes a vascular barrier separating metaphyseal circulation from epiphyseal circulation, (Fig. 1). The growth plate is an effective barrier to the spread of infection but if the metaphysis lies partly or wholly within the joint pyogenic arthritis is not an uncommon sequelae. Metaphysis that lie wholly or partly within the joint capsule are the upper metaphysis of the humerus, all the metaphysis at the elbow, and the upper and lower metaphysis of femur. In childhood wide extensive stripping of the periosteum occurs resulting in large sequestrum. In adulthood, the metaphyseal and epiphyseal circulation communicate across the scar of previous growth plate. This permits organism to reach the subchondral bone and may precipitate a septic arthritis ^{6,9}. Extensive stripping of the periosteum is not a common feature in adult onset haematogenous osteomyelitis since the periosteum is fibrous and

adherent to bone by the strong ligaments of Sharpey. Hence a large sequestrum is not a common outcome in adult onset AHO.

(e) The Immunologic Factors

There are two important immunological aetiological factors about bone; 1) There is a poorly developed reticulo endothelial system with lack of tissue macrophages around the physis of long bones, 2) With trauma, and development of infection, there is thrombosis and occlusion of metaphyseal vessels which excludes migratory fighting cells from reaching the area. These two factors explain the pathophysiological mechanism that is responsible for the 'Locus Minoris Resistantiae' (reduced local resistance) described by Morris⁴.

Defective cellular and humoral systemic immunities are associated with poor outcome osteomyelitis. Rare unusual organisms are commonly involved. Osteomyelitis may be the first presentation of an acquired immune deficiency syndrome such as leukemia and HIV infection². Susceptibility to developing osteomyelitis is increased in systemic chronic debilitating disease that alters the systemic immunological status of the patient.

PATHOLOGY OUTCOME

The pathological outcome of bone infection depends on; 1) the interplay between the virulence of bacteria involved, 2) the host's cellular and humoral response, 3) the anatomic site involved and 4) the tissue reparative responses²⁶. Changes in infected bone always include loss of bone tissue due to the effect of inflammation or reparative reaction and disuse osteoporosis. Bone death occurs as a result of two factors 1)

cytotoxic effects of bacterial toxins and 2) ischaemia from thrombosis and vascular stasis ^{6,9}. The pathological outcome of AHO is complicated by suppuration, bone necrosis and sclerosis of bone.

(a) Suppuration

An acute inflammatory response is initiated by products of tissue damage and bacterial degradation, (Fig. 2).

Vascular changes caused by products released in tissue damage result in exudation of fluid and cells. The first cellular action comes from induced polymorph nuclear leukocyte (PMNs) activity. Circulating PMNs infiltrate the site and begin to ingest and digest organism and debris with the help of lysosomes. Collagenase present in lysosomes is responsible for extensive cartilage damage seen pyogenic arthritis accompanying osteomyelitis.

Release of lysosomal enzymes is thought to be brought about by lipid peroxidation. Exogenous Vitamin C has been known to inhibit lipid peroxidation and may be employed to limit the extent of damage ²⁷.

The next phase of cellular reaction is proliferation and infiltration of lymphocytes. Interleukin I stimulate proliferation of lymphocyte. B and T lymphocytes produce antibodies in addition to functioning as T-helper and T suppressor cells in their interaction with macrophages. The inflammatory reaction result in the formation of pus (suppuration) in a confined space with resultant rise in intraosseous pressure. The pus escapes through the Volkmann canals to the cortical surface forming a subperiosteal abscess, which may extend to the soft tissue ²⁷.

(b) Bone necrosis

Bone dies when its blood supply is cut off by:

- (1) thrombosis of the end-arteries;
- (2) by stripping of the periosteum which supplies two-thirds of the outer cortex, and;
- 3) The rise in intramedullary pressure cause damage the endosteal supply which supplies one-third of the inner cortex ⁹.

The other factor that causes necrosis is the cytotoxic effect of the products of bacteria and tissue degradation.

The extent of bone death depend on; 1) the extent of periosteal stripping, 2) the site of initial infection, 3) the host response and 4) virulence of the infecting organism ⁶.

(c) New Bone Formation

Two types of bone formation occurs: 1) Appositional bone formation and; 2) Metaplasia within fibrous scar tissue.

The reparative process begins with infiltration of new blood vessels, which bring in immature fibroblasts. Products of initial inflammatory response like serotonin, bradykinins promote proliferation and maturation of fibroblasts ²⁷. Serotonin stimulates collagen production whilst Histamine is a depressor of fibroblast activity. Local prostaglandins influence Metaplasia of fibroblast into osteoblast within the fibrous scar resulting in immature ephemeral bone formation.

Therapeutic doses of indomethacin and corticosteroids inhibit bone healing the former is an inhibitor of prostaglandins ²⁷.

New bone formation under the periosteum produces the involucrum visible on x-rays as thin shell around the 10th day. The encasing involucrum grows and expands to become protective against pathological fractures. In areas where the periosteum is killed by bacterial toxins the involucrum is deficient resulting in formation of holes (cloacae) ⁹.

CLINICAL SPECTRUM AND DIAGNOSIS

There are two thoughts on the natural course of AH osteomyelitis;

- 1) the first thought which is widely believed is that acute osteomyelitis progress' to sub acute, then chronic osteomyelitis if not diagnosed early and treated adequately.
- 2) The second thought rather contravertial widely discarded suggest that chronic osteomyelitis is a different disease entity from the acute form.

(a) **Acute Haematogenous Osteomyelitis.**

Paediatric AHO usually occurs in the first decade. With peak incidences occurring around 7-8 years and is most common in boys¹⁸. The diagnosis is essentially clinical. The commonest complaint is a painful limb with reluctance to use it. One third to one-half of children will give a recent history of injury¹⁶. One quarter to one-half will give a history of a recent Infection¹⁷. Clinical suspicion is confirmed if the child points with one finger to the commonest site of osteomyelitis usually around the knee. A useful clue is that the pain and tenderness is out of proportion to the extent of reported trauma or swelling. Fever and malaise in acute osteomyelitis are usually profound. It must be borne in mind that AHO can present without these cardinal signs.

(b) Late acute Haematogenous Osteomyelitis

Lushke and Frey reported that late acute haematogenous osteomyelitis (defined as AHO presenting 3-5 days after onset of symptoms) was the most common stage of presentation in their study done in Namibia³⁰. Swelling and warmth around the affected site are the commonest features. Loss of bone density due to increased cellular activity may be seen on plain radiographs as areas of rarefaction.

(c) Sub acute Haematogenous Osteomyelitis

Sub acute haematogenous osteomyelitis can arise from an acute attack or as a primary sub acute haematogenous osteomyelitis^{17,29}. The former is the most common. Primary sub acute osteomyelitis is due to a host –pathogen relationship in which there is increased host resistance and decreased virulence of the causative organism. These children appear less ill and display less toxicity than children with acute osteomyelitis²¹. Diagnosis is largely dependent on clinical suspicion. Plain radiographs will show variable types of appearances like sequestra, lytic and sclerosis.

(d) Chronic Osteomyelitis

Chronic osteomyelitis is characterized by: 1) a dead infected bone; 2) surrounded by sclerotic relatively avascular bone enveloped by scared soft tissues. 3) Continuous drainage of pus from sinuses. The diagnosis of chronic osteomyelitis is based on clinical, laboratory and imaging studies.

(e) Brodies Abscess

A brodies abscess is a localized form of sub acute osteomyelitis that occurs mostly in long bones of lower extremities of young adults. Most brodies

abscess occurs in the metaphysis, (Fig 3). Brodies abscess present with intermittent pain of long duration with local tenderness over the affected area. The diagnosis is based on careful examination of plain radiographs to exclude neoplastic lesions ⁹.

(f) Neonatal osteomyelitis

Osteomyelitis is commonly accompanied by septic arthritis frequently occurs in the upper end of the femur. Predisposing factors include prematurity, skin and umbilical sepsis and delivery by caesarean section. The neonate presents with lethargy and lack of interest in feeding. Swelling and pseudoparalysis of an affected limb are the most significant local signs. Osteomyelitis may occur with septicaemia, meningitis or jaundice ^{30,36,37}.

(g) Sickle Cell Osteomyelitis

Children suffering from sickle cell disease have reduced resistance to infection. During a crisis, aseptic bone infarction affecting several bones at once occurs. This may become secondary infected with bacteria commonly salmonella. The tibia and femur are affected more commonly ^{16,28,33}. (Fig 4).

(h) Osteomyelitis in HIV Disease

Adult onset acute haematogenous osteomyelitis of the long bones of HIV patients is now common. The disease is often bilateral affecting tibia and femur more commonly. The presentation is either sub acute or chronic with little periosteal reaction ^{3,19,22}.

(I) Sclerosing Osteomyelitis of Garre

Sclerosing hypertrophic osteomyelitis is a chronic form of disease in which the bone is infected and thickened. The thickened cortex obliterates the

medulla in most cases. (Fig 5). Patients present with intermittent pain of moderate intensity of long duration. Swelling and tenderness may be found ⁹.

DIAGNOSTIC APPROACH

(a) Acute Haematogenous Osteomyelitis

(i) Bacteriology

The diagnosis is largely clinical though bacteriological studies will help in identifying the causative organism. Blood culture is positive in about 50% of the cases and should be taken before administration of antibiotics ^{11,30}. Pus cultures obtained from the metaphyseal aspirate or subperiosteal abscess will identify the organism in most cases. Since culture results will be ready after 48 hours an immediate gram stain will help differentiate between gram-positive or gram-negative organism ³⁷.

(ii) Haematological Studies

Haemoglobin estimation does identify anaemia and the real need for transfusion. A white blood count (WCC) is variable to be of diagnostic significance ²⁵. The ESR is useful as monitoring tool rather than aiding diagnosis. Sick cell status can be diagnosed on Blood smears. This will raise the possibility of salmonella infection. C-reactive protein is better than ESR in reflecting effectiveness of therapy and predicting recovery.

(iii) Radiographic Evaluation

Plain x-rays may show a deep soft tissue swelling and loss of tissue planes by the 3rd-day but no bone changes, (Fig. 7).

The usual finding is negative plain x-rays even up to the 14th day. Technetium-99 bone scanning shows increased uptake early in acute haematogenous osteomyelitis. There is 4 to 20% false negatives rate with bone scanning ^{1,27}. In addition increased uptake also indicate trauma, tumour and other infection like cellulites. Magnetic resonance imaging (MRI) has a better tissue resolution is more useful in differentiating cellulites and osteomyelitis.

(iv) Differential Diagnosis

Differential diagnosis of AHO includes septic arthritis, rheumatic fever, sickle cell crisis and leukemic bone pains. With septic arthritis swelling and tenderness are centred on the affected joint. In patients older than 18 months of age AHO rarely crosses the physis (Fig 7).

Any attempt to move the joint is met with resistance. Rheumatic fever has a more insidious onset and produce more systemic symptoms. In sickle cell crises previous history of bone pain or jaundice is present in most cases ^{16,33}. The AHO is often misdiagnosed as malaria when profound fever and malaise are present.

(b) Sub acute Osteomyelitis

(i) Bacteriology

Sub acute osteomyelitis is due to organism of reduced virulence. Only a few positive cultures will be obtained when antibiotics have been administered before. Blood cultures are usually negative ¹⁷.

Bone biopsies are positive in 60% of the cases. *Staphylococcus aureus* and *streptococcus epidermidis* are predominant organisms identified in sub acute osteomyelitis ²⁹.

(ii) Haematological Studies

While blood cell count is usually normal in most of the cases. The ESR .is elevated in about 50% of the case ²⁵.

(iii) Radiological Evaluation

Radiological bone lesions, which may be difficult to differentiate from lesions due to other diseases, are commonly seen ⁹.

(iv) Differential Diagnosis

Differentiative lesions of sub acute osteomyelitis from primary bone tumours sometimes can be difficult. Neoplasms such as eosinophilic granuloma, Erwing's sarcoma and osteogenic sarcoma should be considered in the differential diagnosis. In many series, 50% of cases of sub acute osteomyelitis are confused with a tumour. The diagnosis is often established by an open biopsy and culture ^{9,29}.

(b) Chronic Osteomyelitis

(i) Bacteriology

Chronic bone infection usually yields multiple organisms. Secondary infections are usually common such that sinus track cultures do not correlate with cultures obtained at bone biopsy. Organisms isolated include *staphylococcus Aureus* *streptococci* and anaerobes (*bacteriodes* and *peptococci*) ³⁹. The cause of hypertrophic sclerosing osteomyelitis is not known but it is thought to be a low-grade anaerobic bacteria ¹⁸.

(ii) Haematological Studies

Laboratory studies generally are non-specific and give no indication of severity of infection. In most studies, ESR and CRP are elevated and WCC is elevated in 35%^{25,27}.

(iii) Radiological Evaluation

Radiographical studies are used to confirm and help to plan surgical treatment. On Plain radiographs signs of cortical destruction and periosteal reaction strongly suggest the diagnosis of osteomyelitis. Sino grams can be performed to determine the origin of pus if a sinus tract is present. Gallium isotope bone scan shows increases uptake where bacteria and leukocytes are present and is useful in follow up examination after surgery²⁵. Indium III – labeled leukocyte scans are also useful in evaluating chronic osteomyelitis²⁷.

CT scan provides excellent definition of cortical bone and sequestra. MRI shows areas of bone edema surrounding a focus of active infection.

(iv) Differential Diagnosis

The differential diagnosis of chronic osteomyelitis includes neoplasms, aseptic necrosis of bone, Pyomyositis and cellulitis. The Gold standard for diagnosis of chronic osteomyelitis is a biopsy with culture and sensitivity^{9,49}. Neoplasms are usually associated with great destruction of bone and characteristic radiological features⁹.

TREATMENT AND OUTCOME

The treatment of chronic bone infection centers on a combination of antimicrobial chemotherapy and surgical removal of infected tissues. General principles of treatment include: 1) Correction of anaemia and dehydration if present. 2)

Appropriate bactericidal antimicrobials. 3) Drainage of pus or removal of necrotic tissue and infected dead bone. 4) Protective splintage to reduce pain and protect the limb from deformities and pathological fractures ⁴⁹.

(a) Acute Haematogenous Osteomyelitis

The principles of treatment are: 1) Identification of the organism. 2) Selection of appropriate antibiotic. 3) Delivery of antibiotic in sufficient concentrations and for a sufficient duration. 4) Arresting the process of tissue destruction by surgical decompression ^{20,25}.

(i) Antibiotic Therapy

Penicillin-resistant staphylococcus organism is responsible for the majority of cases of osteomyelitis. In most series primary organism were found to be sensitive to ciprofloxacin, cefotaxime and chloramphenicol. The duration and route of administration of the antibiotic has been the subject of considerable debate. At one time children with AHO were treated routinely for 6 weeks with intravenous antibiotic in a hospital setting.

Todate, the attainment of appropriate serum concentration by a combination of intravenous antibiotic followed by oral antibiotics has become standard treatment ^{20,25,30}.

(ii) Surgical Interventions

The need to drill the cortex in acute osteomyelitis is disputed by some authors³⁷. Acute haemogenous osteomyelitis is a surgical emergency

requiring urgent drainage of pus from either the metaphyseal focus or subperiosteal space. Drainage of the metaphyseal or medulla focus decompresses the metaphysis and stops the build up of intraosseous tension, which is a factor in further bone damage.

Operative drainage of the subperiosteal abscess will limit stripping of periosteal blood supply. In addition, routine drilling or aspirations of bone increased the chance of obtaining the offending organism in AHO³⁶.

(iii) Outcome

If AHO is not treated adequately, or if treatment fails it progresses to the chronic form of the infection with devastating disastrous outcome^{25,36,37}. Osteomyelitis in infants occurs near a major joint with sequelae of septic arthritis³². Osteomyelitis of the proximal femur spread to the hip frequently and result in destruction and ankylosis of the hip (Fig. 12). The common sequelae include focal destruction of the femoral head and dislocation. Destruction of the growth plate result in genu varus or valgum and limb-length inequality. Chronic osteomyelitis cost the patient and health care providers in terms of re-admission and recurrent operations.

(b) Subacute Haematogenous Osteomyelitis

The lesion must be treated by careful thorough curettage. Antibiotic are only useful as an adjuvant to surgery.

(i) Antibiotic Therapy

Subacute haematogenous osteomyelitis must be treated with intravenous antibiotics for 48 hours before surgery followed by a 6-week course of oral antibiotics. Rug and Cole had 87% success rate with this treatment regime ⁴. Handy et al reviewed 44 patients with sub acute osteomyelitis and found no difference in outcome between conservative and surgical treatment ¹⁷.

They suggest open biopsy and curettage for aggressive appearing lesions or for those that do not respond to antibiotic treatment. If a sinus tract is present the organism can be determined and antibiotics given for 2 weeks before the operation.

(ii) Surgical Intervention

Later a sequestra may be sub-acute osteomyelitis present. This must be removed through a wide incision and opening made into the bone. If the necrotic and granulation tissue are adequately removed the wound may be closed primarily with a one-gram streptomycin or cloxacillin into the bone cavity. In 90% of cases this procedure leads to primary healing within three to four weeks ^{9,17}. Sometimes pus forms inside the bone in a cavity, the aim is to remove the pus by opening the bone ‘saucerisation and curettage’.

(iii) Outcome

Mortality is rare. But morbidity is common especially if treatment is inadequate, or the organism is resistant to the antibiotics used. Metastatic infection to the brain or to the lungs has been known to occur ^{9,24}.

(c) Chronic Osteomyelitis

The hallmark of chronic osteomyelitis is infected dead bone within a relatively avascular soft tissue envelop. This requires adequate surgical debridement and removal of dead bone otherwise eradication is impossible ⁴⁴. Chronic osteomyelitis levels deformities and uncosmetic scurrying are indicators of poor outcome of AHO.

(i) Antibiotic Therapy

Systemic antibiotics have a role pre-operatively. Most authors recommend antibiotic two days before surgery. Duration of antibiotics postoperatively varies with clinical response. In cute i ntermittent exacerbations pus s wabs from discharging sinuses can be obtained, organism isolated and appropriate antibiotics employed ²¹. Chronic osteomyelitis run for a long course and use of systematic antibiotics for a long time results in systemic side effects.

Klenn, et al reported good outcome in use of antibiotics impregnated polymethylmethocrylate (PMMA) beads in treatment of chronic osteomyelitis ^{38,48}. This type of treatment when combined with surgery delivery high potent local levels of antibiotic for a long time with low serum levels and low systemic toxicity. Hyper baric oxygen has been used an adjuvant to surgery and in eradicating anaerobic infection with good result ¹⁸.

(ii) Surgical Intervention

Surgical intervention includes sequestrectomy, saucerization and curettage and reconstruction surgery of bone and soft tissue defects.

(1) Sequestrectomy and Curettage

The sequestrum and infected granulation must be removed in total through an appropriate size of cortical opening without compromising bone stability. Adequate surgery involves, removal of sequestra, granulation tissue (pyogenic membrane), and sinus tract. Sclerotic bone usually obliterates the medullary cavity at the site of infection. The medullary cavity must be re-canalized by reaming to bring new vascularised tissue which help fight infection. Skin closure must be done loosely without tension. If closure is not possible wound must be left open closed later or left for skin grafting. The margin of the cortical window must be flattened to avoid pressure on the skin flaps ^{6,9,24}.

(2) Use of Reconstruction Surgery in Chronic Osteomyelitis:

The Papineau Technique

Large bone defects require complex reconstructive surgery.

Pand, et al reported 89% success rate with the papineau technique in treatment of 41 patients with chronic osteomyelitis ⁹. This is done in stages. The first stage is to achieve a granulating bone cavity by removal of necrotic tissue and avascular sclerotic bone and with or without stabilization with an external fixator.

The next stage is placement of autogenous bone usually from the ileac crest in the granulating bed. Once bone grafting is achieved skin closure or skin grafting is done.

(3) Reconstruction Surgery use of in Chronic Osteomyelitis:

Osteocutaneous Flaps

When chronic osteomyelitis has been surgically treated by sequestrectomy and radical excision of sclerotic bone, large cavities or bone gaps remain. In areas like the lower limb between the knee and calcaneum a free or pedicle peroneal muscle and bone flap can be used to cover the defect.

In other instances a gastrocnemius muscle flap is used for defects of the proximal third of the tibia and the soleus muscle used to cover defects of the middle third. The reported success ranges from 66% to 100% ⁹. When chronic osteomyelitis is complicated by segmented bone loss of the tibia a free fibular graft - tibialisation of fibula can be used to bridge the deficit ^{4,9}.

(4) Reconstruction Surgery:

The Illazorov Technique

Stabilization by Illazorov system of external fixator allows radical resection of infected bone followed by lengthening of immature union. Other uses include correction of angulatory deformities, (Fig 8).

(iii) Outcome

Chronic osteomyelitis is difficult to cure. It follows one of several courses. In some patients will present with intermittent acute exacerbations, while in other infection remain dormant and flares up years later. After years of persistent drainage epidermoid carcinoma in sinus tract has been known to develop ⁹. Perpetuation of chronic osteomyelitis is due to residue sequestrum or infected granulation in enclosed cavities surrounded by sclerotic bone.

When septic arthritis occurs it leads to chondrolysis, epiphysiolysis and avascular necrosis when the hip is involved.

Ankylosis of the joint and limb shortening following epiphysiolysis and chondrolysis is the usual end outcome ³². Poor outcome of osteomyelitis is marked by; chronic discharging sinuses, sequestrum surrounded by involucrum, pathological fractures, joint destruction and contractures. Amputations and death are the worst outcome ³⁰. (Fig. 12)

MATERIALS AND METHODS

This cross sectional review or survey study recruited 112 patients who presented with AHO of long bones to the Orthopaedic Unit, Department of Surgery at the University Teaching Hospital Lusaka. The study was conducted over a period of 1 year from July 2002 to August 2003.

The Subjects

112 patients were included, 76 males and 36 females, (Fig. 15). The criteria for inclusion to study were:

- (a) Age groups: children from 0 - 21 years and adults from 21 – 90 years. There were 87 children and 25 adults.
- (b) Only patients with AHO of the long bones of the upper and lower extremities were included
- (c) Signed consent by guardian or patients over 21 years and above.

The criteria for exclusion was:

- (a) Osteomyelitis secondary to open fractures or following surgery.
- (b) Osteomyelitis of the small long bones of the feet and the hands;
- (c) Non-consenting patients.

Eight 8 patients with osteomyelitis complicating open fractures, 3 occurring in implants, 2 with osteomyelitis of the fingers and 2 osteomyelitis of the feet were excluded.

A diagnosis of osteomyelitis was made on the basis of the patient's history, physical examination and radiological evaluation.

Ethical Considerations

A written consent was signed by the guardian of the patient or the patient if 21 years and above after careful explanation of the study. In the consent form (Appendix I), the nature of the study, the benefits and safety of the procedures to be carried out were clearly explained. The Ethics Committee of the University of Zambia, School of Medicine, approved this study.

Data Collection

A clinic day was created on Mondays for the purpose of recruiting, follow up and collection of samples. Blood and Pus swabs were collected with minimal discomfort to the patients.

For each subject, data were collected about sex, age, and social economic status (in terms of income, education, water and energy source). The nutritional status of the patient was assessed in terms of height, weight and mid arm circumference for age as well as physical signs of malnutrition namely hair changes, body swelling and lethargy.

Patients or guardians were questioned on the duration of symptoms at the time of presenting to the first medical personnel contacted. Patient's notes from urban clinical officers' referral letters; casualty officers and general surgeons were reviewed for information on duration of symptoms, initial diagnosis, associated factors and initial treatment. The information was entered on a proforma. (Appendix ii).

Presentation and Classification:

The study adopted the classification of outcome used by Luschke and Frey in Namibia and Rashkoff and William in South Africa for easy comparison of results.

Based on the duration of symptoms at presentation to a medical or clinical officer, AHO was classified into Early Acute Osteomyelitis, Late Acute Osteomyelitis and Neglected Acute Osteomyelitis^{30,46}.

The patients were then evaluated for the outcome using an outcome scoring system which takes into account the chronicity (disease outcome), physical disability (functional outcome) and cosmetic disfigurement. Early acute osteomyelitis was defined as, acute osteomyelitis that presented to a medical or clinical officer within the first three days from the onset of symptoms. Late acute osteomyelitis was defined as acute osteomyelitis that presented to a medical or clinical officer after 3 to 5 days after onset of symptoms.

Neglected acute osteomyelitis was defined as osteomyelitis that presented to a medical or clinical officer 1 week after the onset of symptoms and those who had traditional treatment. In this study 18 (16%) patients presented with early acute osteomyelitis, 22 (20%) with late acute osteomyelitis and 72 (64%) with neglected acute osteomyelitis.

For the purpose of comparing results categories in Early AHO and Late Acute AHO were classified as **Early presentation** and category Neglected Acute AHO as **Delayed presentation**.

Sinus tract Swabs:

The swabs were collected at time of recruitment in patients who had discharging sinus, or at the time of incision and drainage or exploration of the metaphysis. The swabs were inoculated immediately into culture media and incubated at 37°C for 48 hours. 59 Pus swabs collected from sinus tracts were subjected to gram staining, culture and sensitivity testing.

Blood specimen:

Blood collected from 25 consenting adult patients was subjected to HIV testing using ELISA and confirmation with western blot. 11 of the adult's patients were judged to be stage IV in terms of WHO clinical criteria for HIV.

In patients who were over 21 years an HIV test was done after professional pre test counselling by the home based care group UTH and the researcher. Post test counselling done where necessary. 18 patients who tested positive were referred for inclusion on the GRZ-ARV scheme. Haematological studies included 65 full blood counts and 70 erythrocyte sedimentation rates. These numbers are lower because at times samples were not processed due to non-functioning of automated counters. Another reason is that some laboratory reports were excluded because they were incomplete.

Surgical intervention:

During the study, surgery was necessary in 26 patients who had sequestra and 7 patients with brodie's abscess. Due to constraints, only 12 operations were done, 8 due to sequestrum removal, 4 on brodie's abscess and 2 patients underwent bone drilling for Late AHO.

Bone biopsies:

Collection of bone tissue from chronic osteomyelitis was abandoned after 5 consecutive samples were reported no growth. This was due to poor handling of samples during transportation and laboratory.

Radiographs:

Plain radiographs were obtained in Anterior - posterior and lateral views for classification and planning of surgical intervention.

Treatment:

Patients case notes and where this was not available, the patient or guardian, were questioned to determine the initial treatment at the time the patient presented first to a clinical officer or medical officer. The initial treatment was grouped into;

- (a) Adequate, 09 patients
- (b) Inadequate, 31 patients
- (c) Or inappropriate management, 72 patients

Adequate initial management was defined as, admission, resuscitation, iv fluids, iv highly potent, high dose antibiotics and surgical intervention to explore and drain the pus. **Inadequate initial management** was defined as treatment with no admission, oral antibiotics and without appropriate surgical intervention.

In appropriate initial management was defined as osteomyelitis treated as another disease (misdiagnosis) by primary medical attendants and those treated by traditional methods. This is the same group as delayed presentation.

Data Analysis

An objective system of assessing outcome was employed since most of the patients were children for a subjective system of assessment to be used. The system was based on modification of the system used by Luschke and Frey in their study of 55 children with haematogenous osteomyelitis ³⁰. The following where considered;

- (i) Chronic pain**

 - No pain - good outcome
 - Pain under control - satisfactory outcome
 - Pain resulting in atalgic gait - poor outcome
- (ii) Discharging Sinus**

 - No discharging sinuses - good outcome
 - Healed sinuses - satisfactory outcome
 - Discharging sinuses - poor outcome
- (iii) Limb Shortening**

 - No limb shortening - good outcome
 - Limb shortening (less than 2.5cm) - satisfactory outcome
 - Limb shortening greater than 2.5cm) - poor outcome
- (iv) Angular Deformity**

 - No angular deformity - good outcome
 - Angular deformity (not requiring surgery) - satisfactory outcome
 - Angular deformity (requiring surgery) - poor outcome
- (v) Use of Mobility Aids**

 - No mobility aids - good outcome
 - Mobility aids needed occasionally - satisfactory outcome
 - Mobility needed all the time - poor outcome
- (vi) Joint Contractures**

 - No contracture - good outcome
 - Contractures in functional position - satisfactory outcome
 - Contracture interfering with limb function - poor outcome

The results where judged as **good, satisfactory** and **poor** depending on presence of the most severe outcome of any of the above 6 items.

RESULTS I

Patients

One hundred, 100 (89%) Patients were referred from Lusaka urban and rural clinics. Four, 4 were from Central Province and 6 from Southern and 1 from Luapula and Eastern Province each. The primary medical attendants were 76 (68 %) clinical officers, 32 (29%) traditional medical attendants, 04 (4%) medical officers and 02 (2%) by nurses. (Fig. 2 and Figure 17).

Presentation

All 112 (100%) patients had complained of pain, 98 (88 %) limping or loss of function, 79 (71%) local swelling and 77 (69%) temperature above 37.5°C or body hotness was reported in (Table 4 and Fig 18). Forty 40 (36%) patients presented to a primary medical attendant within the first 5 days of onset of symptoms, 18 Early AHO and 22 Late AHO were placed under **Early Presentation**. All 72 (64%) neglected acute osteomyelitis (who presented to a primary medical attendant one week after the onset of symptoms) were placed under **Delayed Presentation**. The primary medical attendants in the delayed presentation group were 36 (74%) clinical officers, 25 (22%) traditional healers and 02 (2%) by medical officers and 02 (2%) by nurses (Table 2). The result showed 28% of patients in the Early Presentation group had good outcome, 48% satisfactory outcome and 24% poor outcome. In the Delayed Presentation group, only 13% had good outcome, 26% satisfactory outcome and poor outcome was high (61%).

Treatment

There were 09 (11%) who treated adequately, 31 (39%) patients were treated inadequately and 62 (50%) patients were treated inappropriately. The results show 44% good outcome and 66% satisfactory outcome in the adequate treatment group. There was no poor outcome in the adequate treatment group. Poor outcome was high in the inadequate treatment group (32%). Only 13% had good outcome and 55% had satisfactory outcome in the inadequate treatment group.

Socio-Economic Status

After considering the level of education, income, water and energy sources, patients were grouped into lower, middle and upper classes. Most patients in the lower socio-economic group had only attained primary level of education, had an income less than K250, 000.00 and lived in unhygienic areas with primitive source of energy and water. In this study, there were 77 (69%) patients in the lower social economic group 29 (26%) in middle class and 06 (5%) in the upper class (Table 6 and Fig. 19).

Recent Infection

Patients were asked if they had a sore throat, skin infection, gastroenteritis or urinary tract infection weeks prior to onset of limb pain (the commonest symptom of AHO in this study). This study showed 53 (48%) had a recent infection. The breakdown of recent infection was as follows: 28 (52%) had a fever treated as malaria, 11 (21%) respiratory tract infection, 08 (15%) gastrointestinal disturbance and 06 (11%) skin rashes. (Table 7).

Associated Debilitating Condition

Patients were questioned and records reviewed to establish the presence and frequency of factors predisposing to poor outcome osteomyelitis. In this study, 57 (66%) children had associated predisposing factors and all 25 (100%) adults had associated predisposing factors. The breakdown of associated predisposing factors in 87 children is as follows: 23 (26%) had a poor nutritional status, 22 (25%) reported history of trauma and 12 (13%) had Sickle Cell Disease and 30 (34%) associated predisposing factors were unknown.

In the 25 adult patients the breakdown of associated predisposing factors were as follows: 18 (72%) were HIV Seropositive, 04 (16%) had diabetes and 03 (12%) were above 70 years old (Table 21,9). The results showed high poor outcome in poor nutritional status (43%), sickle cell disease (83%) and HIV infection (78%). Good outcome was low, poor nutrition status (17%) and no good outcome in sickle cell disease and HIV infection.

Radiological Evaluation

On plain radiographs, the site of the bone lesion, extent of bone damage and bone reaction was assessed. A sclerotic bone lesion with or without obliteration of medullary cavity was the commonest, 51 (46%) films (Fig 10). There were 26 (23%) patients with sequestration and involucrum was seen in 26 (23%) films (Fig. 10)

Twenty eight 28 films (25%) had multiple lytic areas and poor reaction of the periosteum and 07 (6%) had a Brodie's abscess. In patients who had more than one radiological feature, the most prominent appearance has been recorded. (Table 9 and Fig 29).

Radiological evaluation revealed that the proximal tibia was the commonest site of osteomyelitis with 25 (21%) films. 22 (19%) was in the proximal femur, 16 (14%) in the distal femur, 13 (11%) distal tibia, 11 (9%) shaft of femur, 09 (8%) shaft of tibia, 07 (6%) proximal humerus, 03 (3%) the shaft and distal humerus and 02 (2%) for proximal and shaft of the femur. Four 4 patients had osteomyelitis in 2 sites and 1 in 3 sites.

(Table 20).

Bacteriological Findings

A total of 59 pus swabs were collected. Staphylococcus was isolated in 43 (73%). Other aerobic isolates were 06 (10%) Enterobacter spp, 04 (8%) with Escherichia Coli. 03 (5%) proteus, 02 (3%), Actinobacteria and 1 (2%). TABLE 17. Penicillin-resistant staphylococcus was the commonest (Table 18)

RESULT II

Data analysis (spread sheets and graphs) was done using Microsoft excel versions6.0, 2000)

Table 1: Results of the age and sex distribution

AGE GROUP	AGE	FEMALE	MALE	TOTAL	PERCENTAGE
1	0 - 5	8	06	14	13%
2	6 -10	18	11	29	26%
3	11 - 15	14	7	21	19%
4	16 - 20	6	8	14	13%
5	21 - 25	6	2	8	7%
6	26 - 30	03	0	03	3%
7	31 - 35	02	0	02	2%
8	36 - 40	06	01	07	6%
9	41 - 45	04	03	07	6%
10	46 - 50	01	02	03	3%
11	51 - 55	00	00	00	0
12	56 - 60	00	00	00	0
13	61 - 65	00	00	00	0
14	66 - 70	00	00	00	0
15	71 -75	00	01	01	1%
16	76 - 80	01	01	01	1%
17	81 - 85	00	00	00	0
18	86 - 90	01	01	01	1%

FIGURE 14: RESULTS OF THE AGE DISTRIBUTION

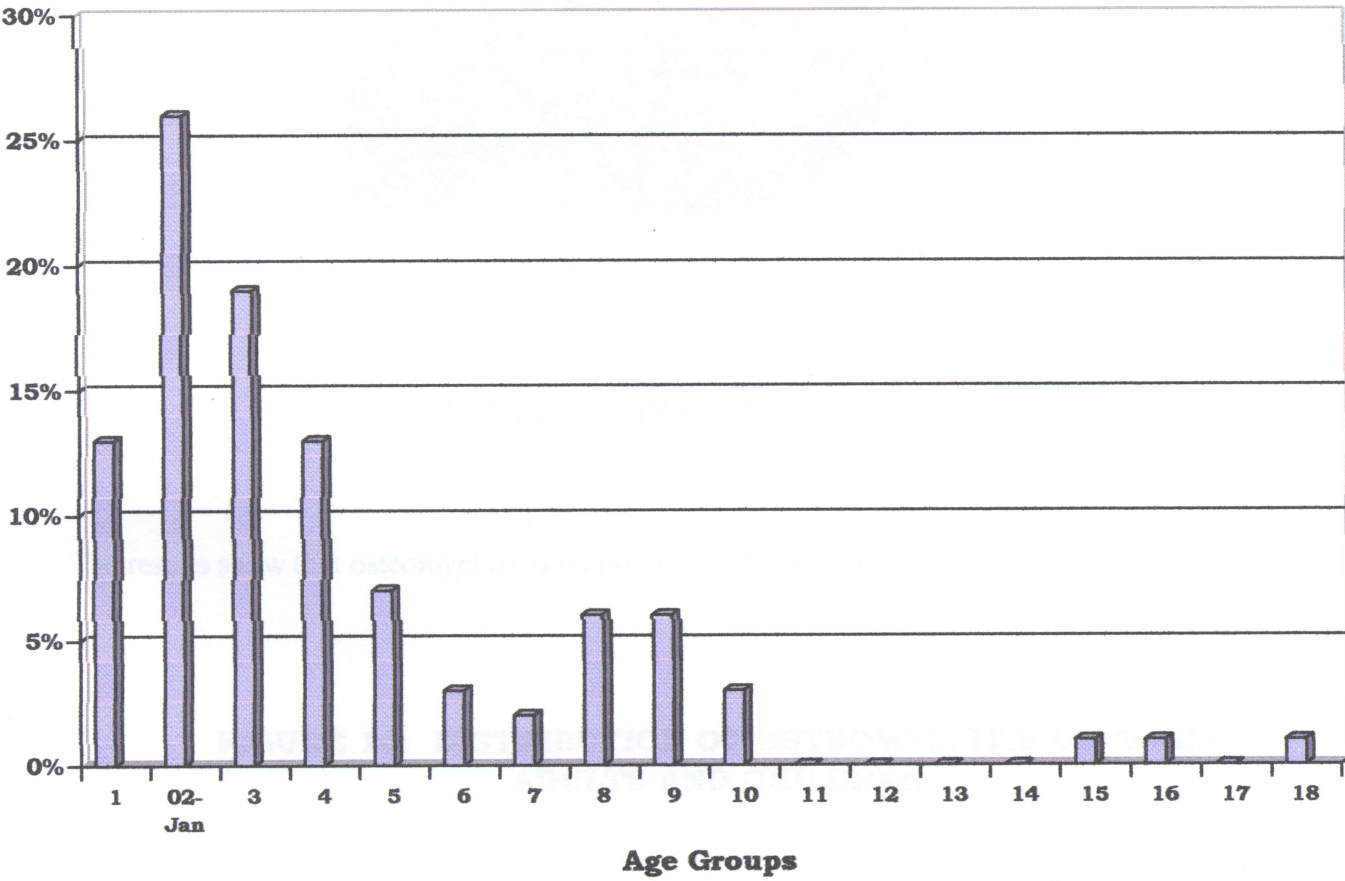
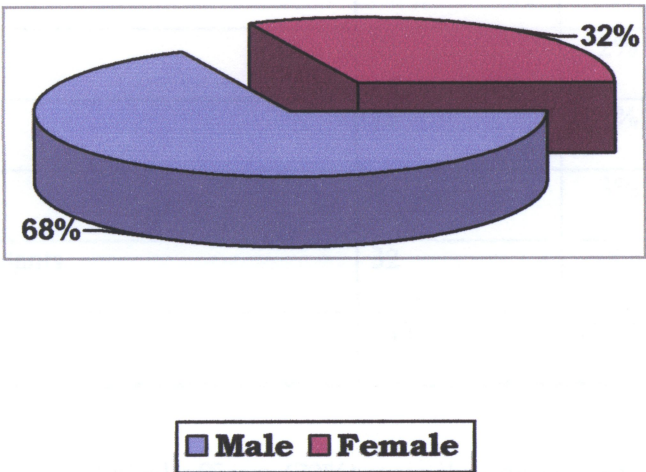
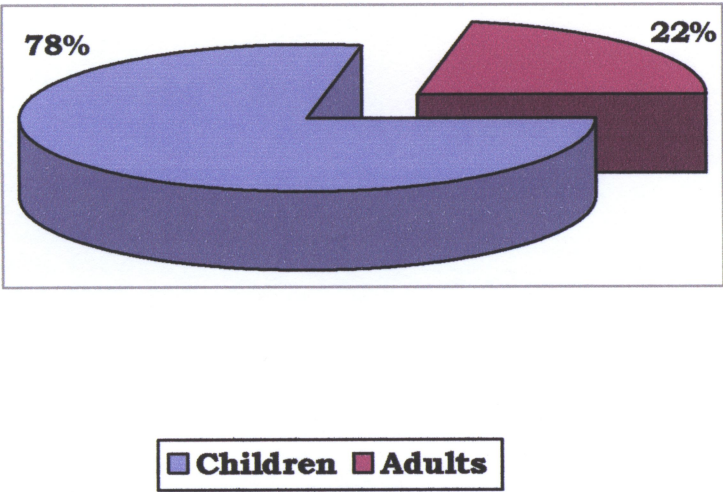


FIGURE 15: RESULTS: SEX DISTRIBUTION



The results show that osteomyelitis is commoner in males (ratio 2:1)

FIGURE 16: DISTRIBUTION OF OSTEOMYELITIS BETWEEN ADULTS AND CHILDREN



n = 25 adults above 21 years at onset of symptom; n = 87 children below 21 years at onset of symptoms. The results show that osteomyelitis is commoner in children (ratio 3 ½:1)

TABLE 2: RESULTS: PRIMARY MEDICAL ATTENDANTS OUTSIDE UTH

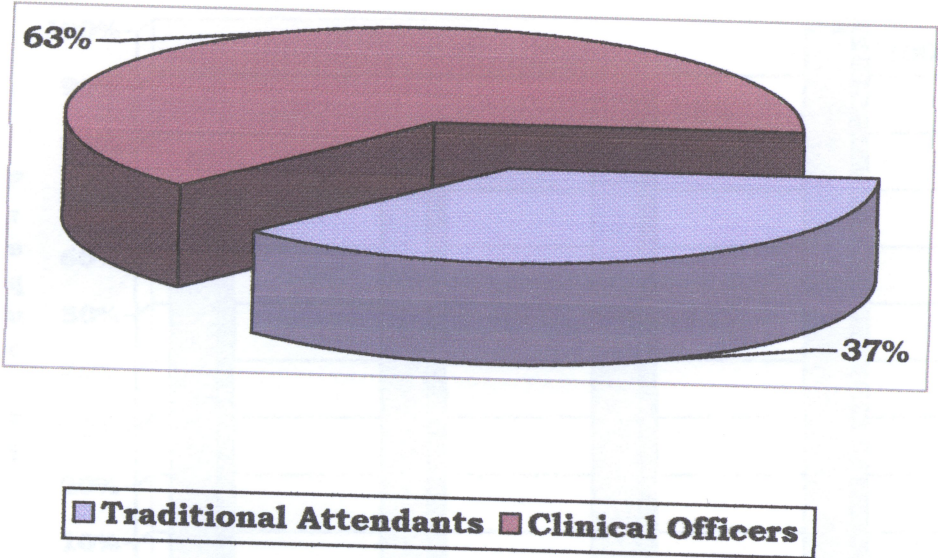
CATEGORY OF HEALTH WORKER	NUMBER	FREQUENCY
Nurses	02	2%
Clinical Officers	76	68%
Medical Officers	04	3%
Traditional Attendants	32	28%
Total	112	100%

The results shows that clinical officers (72%) were the commonest primary medical attendants followed by traditional healers (22%).

TABLE 3: RESULTS: PRIMARY MEDICAL ATTENDANTS IN PATIENTS WITH DELAY PRESENTATION (n=68) EXCLUDING 2 BY MEDICAL OFFICERS AND 2 BY NURSES

	number	percentage
Traditional Medical Attendants	25	37%
Clinical Officers	43	63%
Total	68	100

FIGURE 17: FREQUENCY OF PRIMARY MEDICAL ATTENDANTS

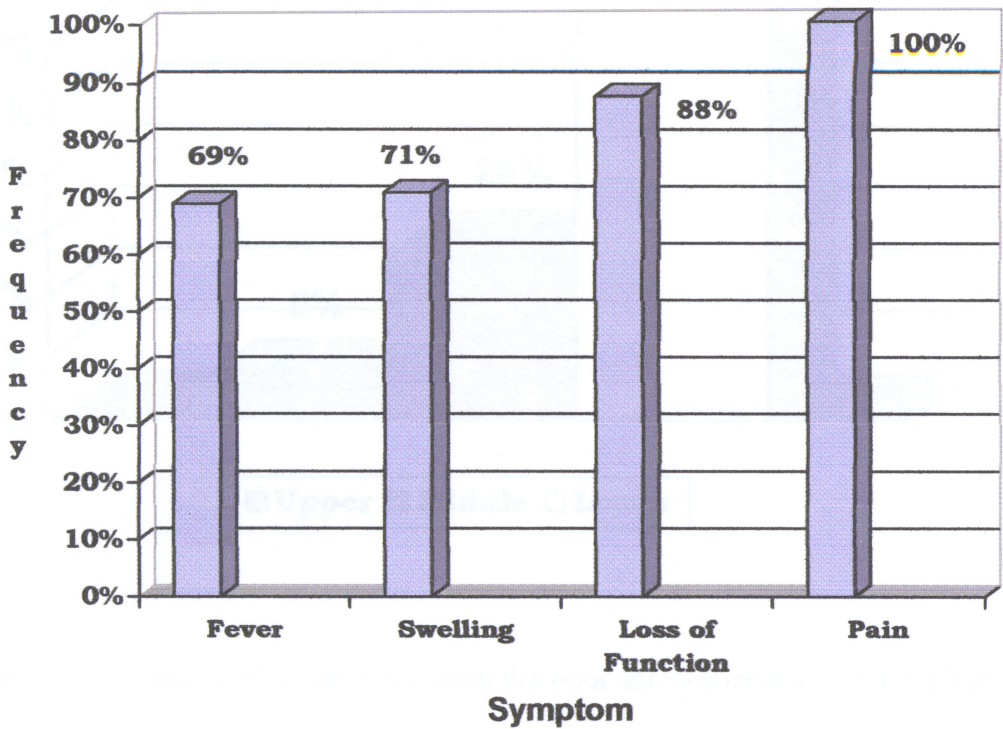


The results show that the clinical officer caused 63% delay in presentation compared to (37%) by traditional healers.

TABLE 4: RESULTS: FREQUENCY OF PRESENTING SYMPTOMS (n=112)

	NUMBER OF CASES	TOTAL NUMBER OF PATIENTS	percentage
Pain	112	112	100
Swelling	79	112	71
Loss of function or limping	98	112	82
Body hotness or temperature >37.5°C	77	112	69

FIGURE 18: GRAPH COMPARING FREQUENCY OF SYMPTOMS

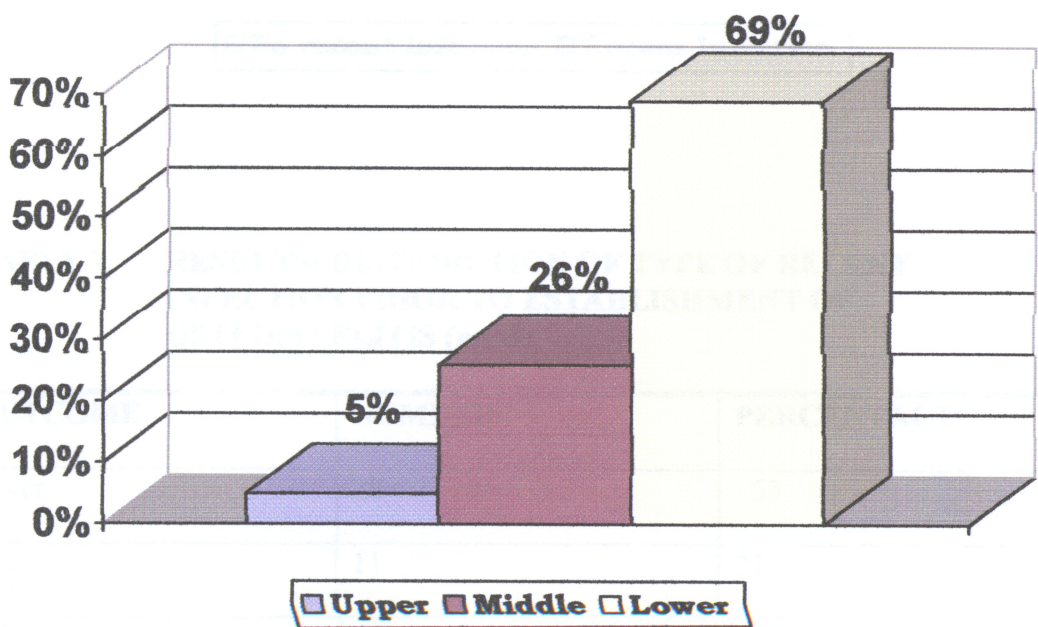


The results showed that pain was present at the onset of disease in all the patients 100% and functional disturbances in 88%. Fever, 69% and swelling 71% are least common at the onset of the disease.

TABLE 6: RESULTS: DISTRIBUTION OF OSTEOMYELITIS AMONG THE SOCIO-ECONOMIC CLASSES

CLASS	number
Upper	06 (5%)
Middle	29 (26%)
Lower	77 (69%)

FIGURE 19: FREQUENCY OF OSTEOMYELITIS AMONG SOCIAL ECONOMIC GROUPS



The results show that most children are from the poor socioeconomic group with poor education, poor sanitation and poor nutrition with high levels of disease burden.

FIGURE 20: COMPARING THE FREQUENCY OF PATIENTS WITH RECENT INFECTION AND NO RECENT INFECTION

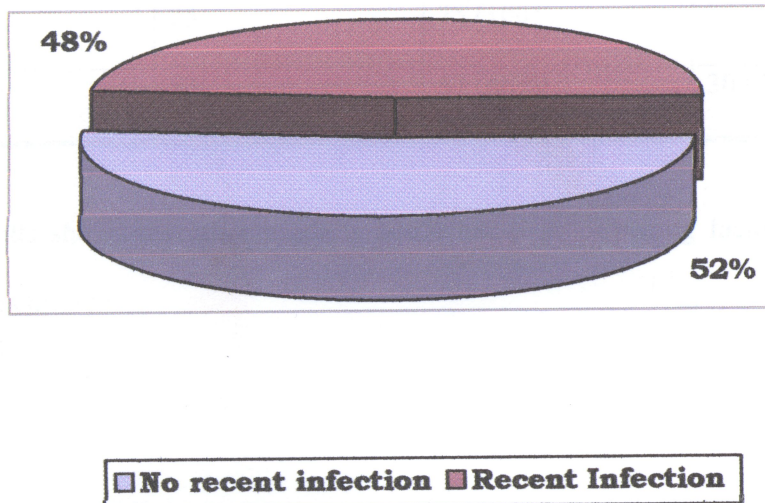


TABLE 7: RESULTS: DISTRIBUTION OF TYPE OF RECENT INFECTION PRIOR TO ESTABLISHMENT OF OSTEOMYELITIS (n=53)

OUTCOME	NUMBER	PERCENTAGE
Fever	28	53
RE	11	21
GE	08	15
Skin sepsis	06	11
Total	53	100

Results show that fever was the commonest premorbid condition at 53%.

**TABLE 8: RESULTS: COMPARING PREVALENCE OF ASSOCIATED
PREDISPOSING FACTORS BETWEEN ADULTS AND
CHILDREN**

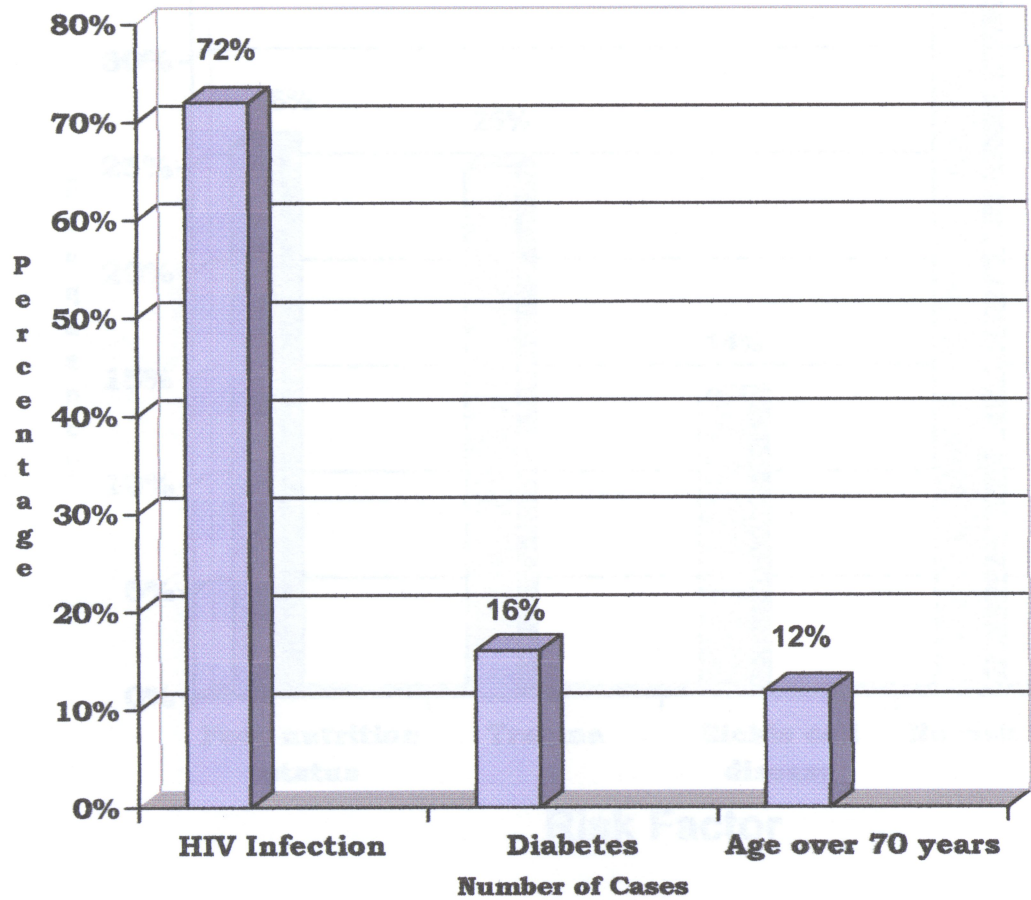
	NUMBER	RISK FACTOR	NO RISK FACTOR
Adults	25	25 (100%)	
Children	87	57 (66%)	30 (44%)

The results shows that all the adults 100% had a predisposing factor compared to (66%) in children.

TABLE 9: DISTRIBUTION OF ASSOCIATED RISK FACTORS IN ADULTS OVER 21 YEARS (n=25)

	NUMBER OF CASES	TOTAL number OF ADULTS	FREQUENCY
HIV seropositive	18	25	72%
Diabetes	04	25	16%
Elderly	03	25	12%

FIGURE 21: FREQUENCY OF ASSOCIATED PREDISPOSING FACTORS IN ADULTS



Results show that HIV was the commonest predisposing condition at 72% in adults.

TABLE 10: DISTRIBUTION OF ASSOCIATED RISK FACTORS IN CHILDREN BELOW 21 YEARS (n=57)

	NUMBER OF CASES	TOTAL number OF children	FREQUENCY
Poor nutrition status	23	87	26%
Trauma	22	87	25%
Sickle cell disease	12	87	14%
No risk factor	30	87	35%

FIGURE 22: FREQUENCY OF PREDISPOSING FACTORS IN CHILDREN (n=87)

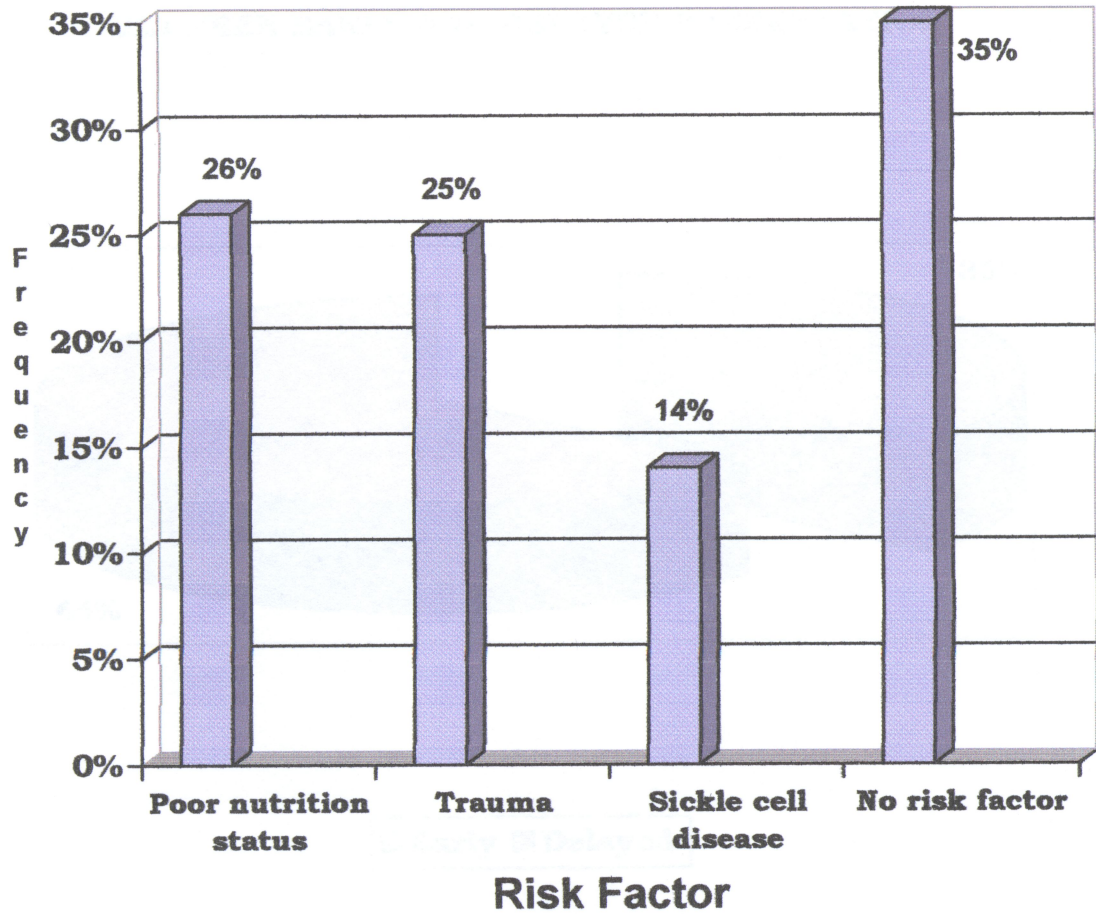
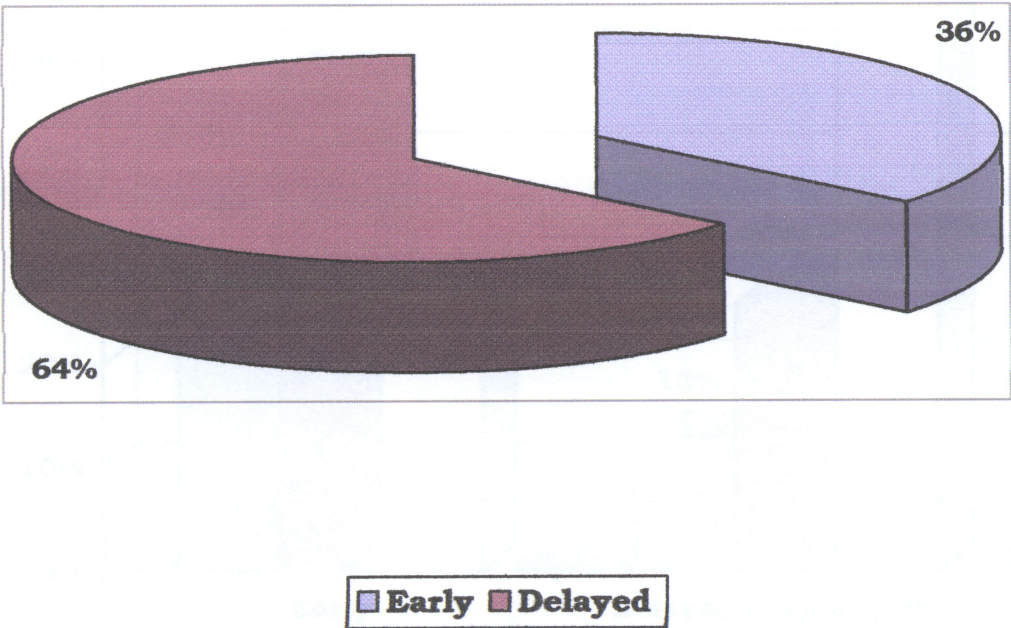


TABLE 11: RESULTS: DISTRIBUTION OF PATIENTS BETWEEN EARLY AND DELAYED PRESENTATION (n=112)

PRESENTED	NUMBER OF CASES	FREQUENCY
Early	40	36%
Delayed	72	64%

FIGURE 23: COMPARING THE FREQUENCIES BETWEEN EARLY AND DELAYED PRESENTATION

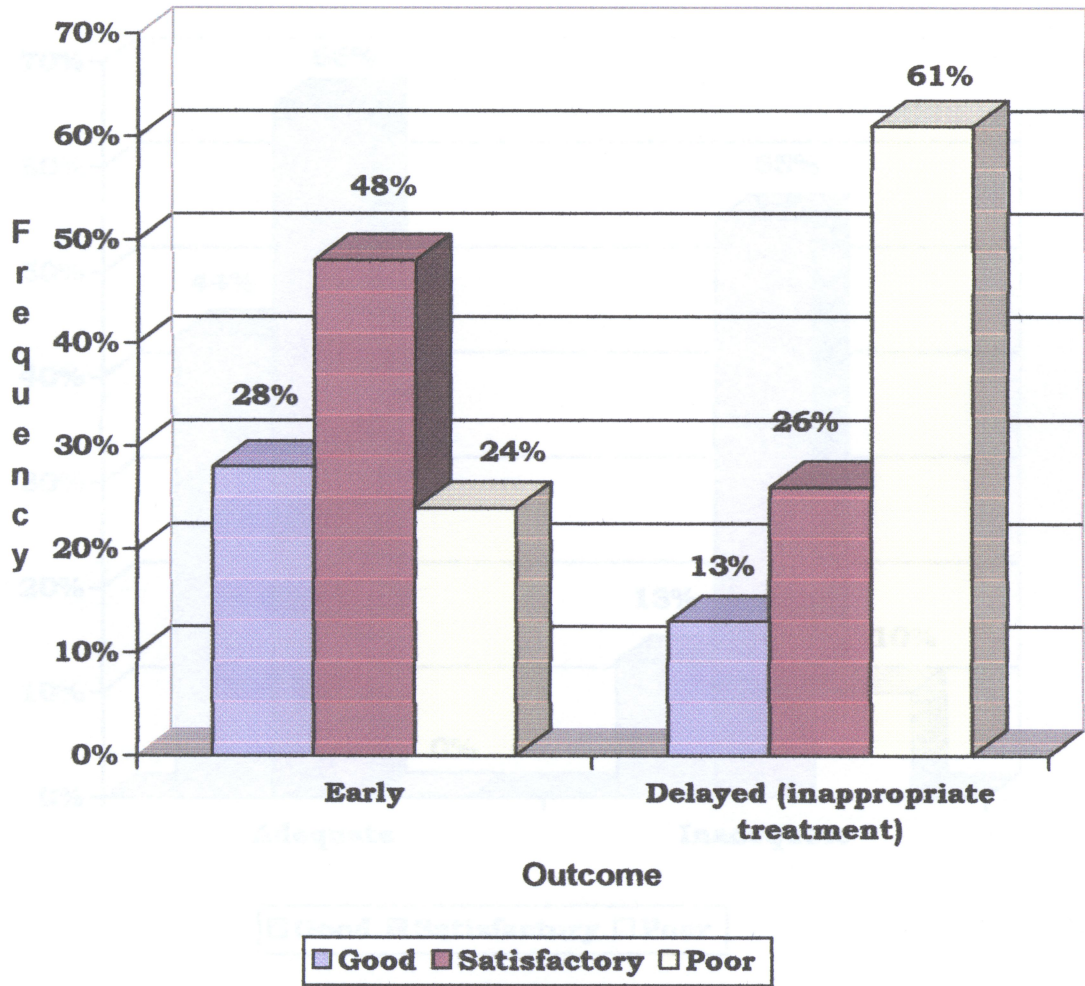


The results shows very high number of delayed presentation (Neglected AHO), (64%)

TABLE 12: RESULTS: THE OUTCOME OF OSTEOMYELITIS IN EARLY AND DELAYED PRESENTATION

PRESENTATION/ OUTCOME	NUMBER OF CASES	GOOD	SATISFACTORY	POOR
Early	40	11 (28%)	19 (48%)	10 (24%)
Delayed	72	09 (13%)	19 (26%)	44 (61%)

FIGURE 24: GRAPH COMPARING THE FREQUENCY OF OUTCOME IN EACH GROUP (EARLY n=40, DELAYED N=72)

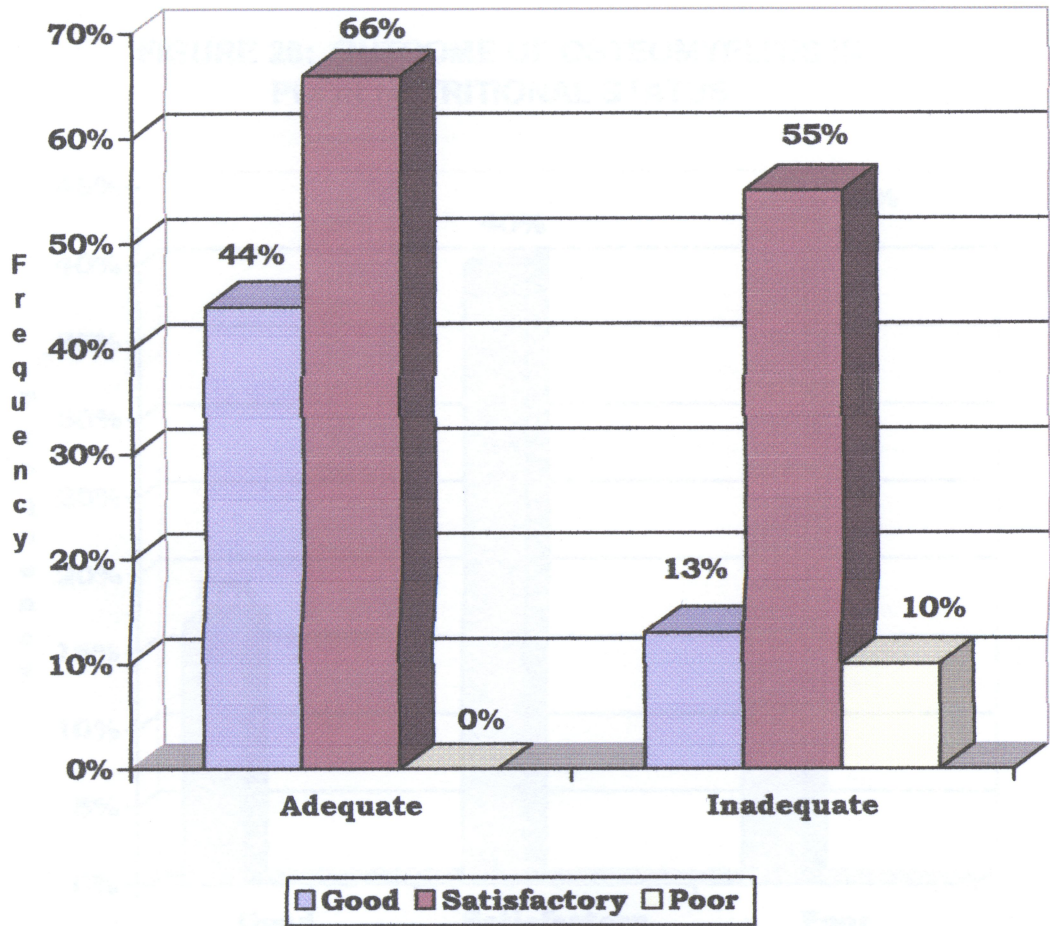


The results show a high poor outcome (61%) in delayed presentation (Neglected AHO).

TABLE 13: RESULTS: THE TREATMENT OF OSTEOMYELITIS IN PATIENTS WHO PRESENTED EARLY (n=40)

PRESENTATION/ OUTCOME	NUMBER OF CASES	GOOD	SATISFACTORY	POOR
Adequate	09	04 (44%)	05 (66%)	
Inadequate	31	04 (13%)	17 (55%)	10 (32%)

FIGURE 25: COMPARING THE FRQUENCY OF OUTCOME IN EACH GROUP (ADEQUATE n=09, INADEQUATE n=31)

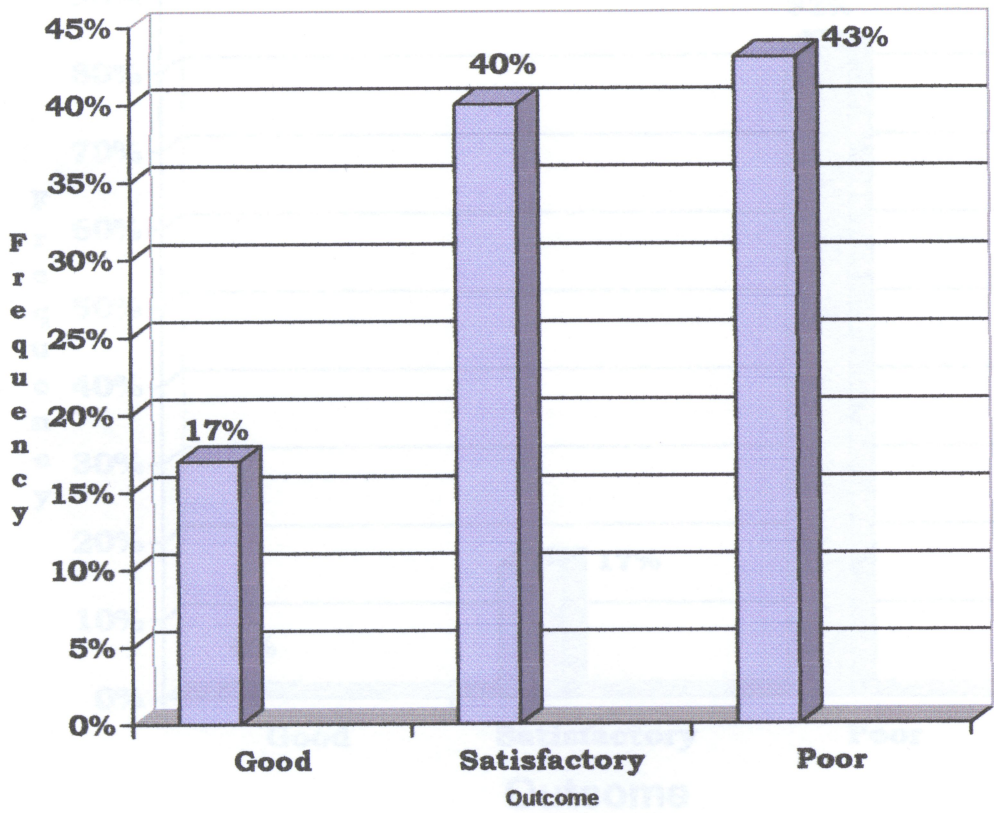


The results show that adequate treatment is associated with (44%) good outcome compared to 13% in inadequate treatment.

TABLE 14: RESULTS: THE OUTCOME OF OSTEOMYELITIS IN CHILDREN WHO HAD POOR NUTRITION STATUS (n=23)

OUTCOME	NUMBER	FREQUENCY
Good	04	(17%)
Satisfactory	09	35 (40%)
Poor	10	52 (43%)
Total	23	100%

FIGURE 26: OUTCOME OF OSTEOMYELITIS IN POOR NUTRITIONAL STATUS

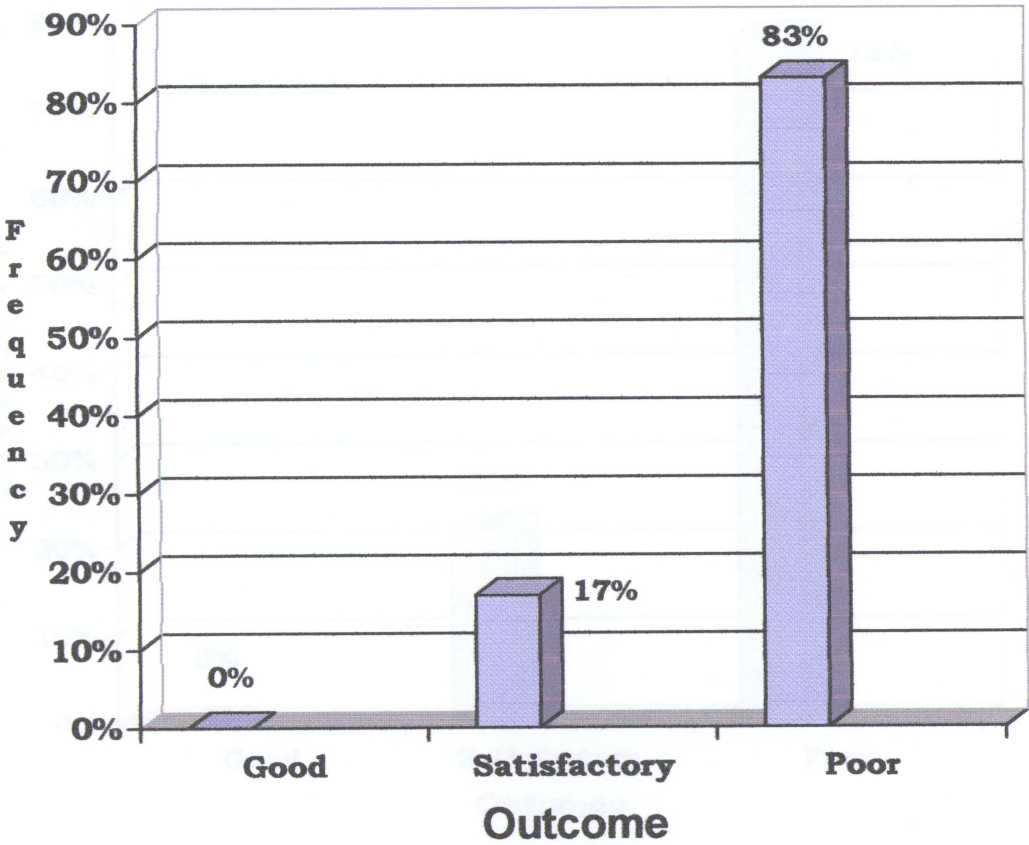


The results show a high poor outcome (43%) in children with poor nutritional status.

TABLE 15: RESULTS OF THE OUTCOME OF OSTEOMYELITIS IN CHILDREN WHO HAD SICKLE CELL DISEASE (n = 12)

OUTCOME		percentage
Good	0	0
Satisfactory	02	17
Poor	10	83
Total	12	100

FIGURE 27: OUTCOME OF OSTEOMYELITIS IN SICKLE CELL DISEASE PATIENTS (n=12)

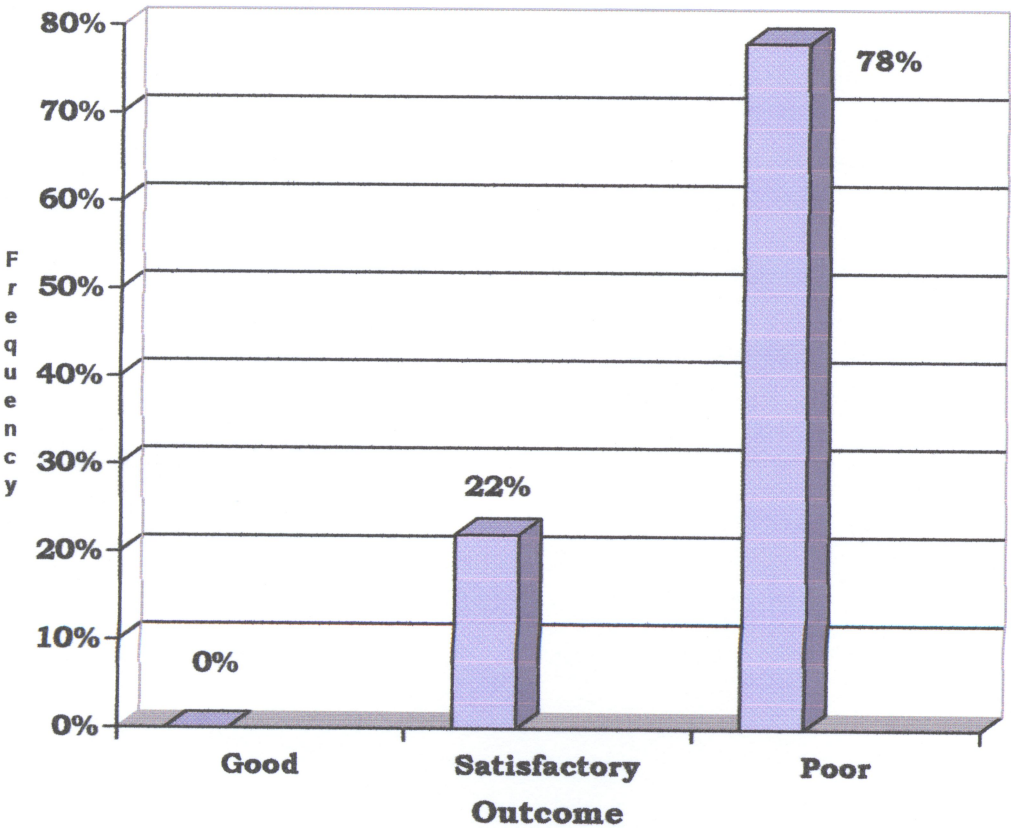


The results show a high poor outcome (83%) in children with sickle cell disease.

TABLE 16: RESULTS: THE OUTCOME OF OSTEOMYELITIS IN ADULTS WHO HAD HIV INFECTION (n=18)

OUTCOME	NUMBER	PERCENTAGE
Fair	0	0
Poor	4	22
Very poor	12	78
Total	18	100

FIGURE 28: OUTCOME OF OSTEOMYELITIS IN HIV DISEASE

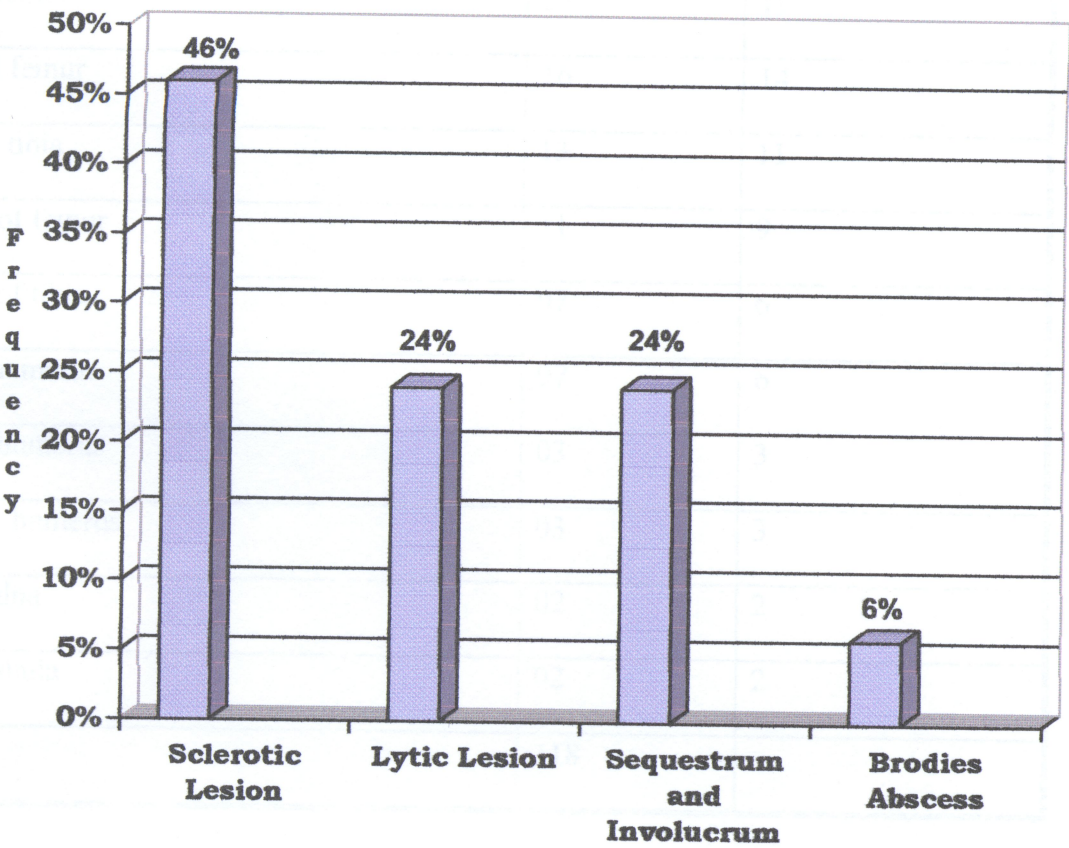


The results show poor outcome 78% in patients who had HIV infection

TABLE 19: RADIOLOGICAL EVALUATION OF OSTEOMYELITIS

	NUMBER	FREQUENCY
Sclerotic Hypatrophic lesion	51	46
Lytic lesion	27	24
Sequestrum and involucrum	27	24
Brodies abscess	07	6
Total	112	100

FIGURE 29: FREQUENCY OF TYPE OF RADIOLOGICAL PRESENTATION



The results show that sclerotic hypertrophic lesions is the commonest lesion (46%) in this study.

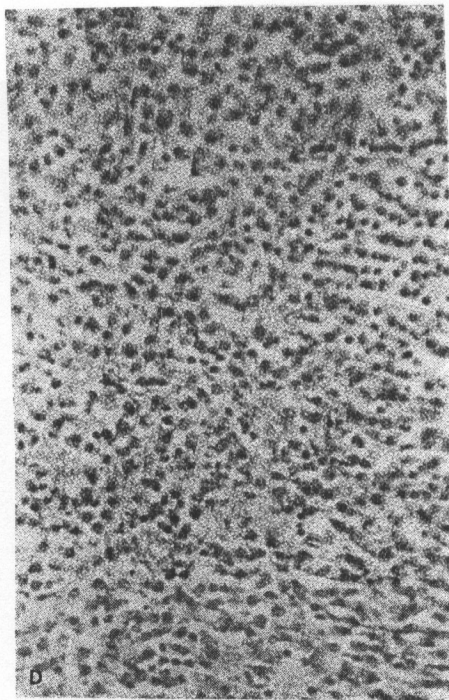
TABLE 20: RESULTS: SITES OF OSTEOMYELITIS

SITE	NUMBER	FREQUENCY
Prox tibia	25	21
Prox femur	22	19
Distal femur	16	14
Distal tibia	13	11
Shaft of femur	11	9
Shaft of tibia	07	6
Prox humerus	07	6
Shaft humerus	03	3
Distal humerus	03	3
Prox ulna	02	2
Prox fibula	02	2
Total	118	

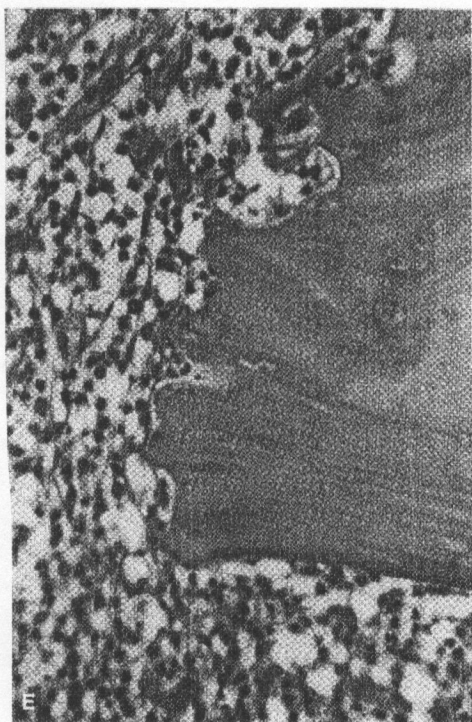
The results show that the proximal tibia (21%) is the commonest site of the initial infection followed by proximal femur (19%).



Figure 1: The mature growth plate acting as Barrier to spread infection to the epiphysis in 3 year old child.



B



C

Figure 2: A - Lower power view of marrow infiltration with sounding dead bone.
 B - High power view show acute inflammatory cellular reaction
 C - High power view showing inflammatory cells destroying necrotic lamellar Bone.



Figure 3: A brodies abscess; a radiolucent area surrounded by sclerotic bone containing a sequestrum



Figure 4: Extensive destruction of the shaft and metaphysis of the tibia in sickle cell disease child



Figure 5: Sclerosing Osteomyelitis of Garre. Note hypertrophy and obliteration of Medullary Cavity



Figure 6: Osteomyelitis affecting the Distal Femur medial Condyle in a Diabetic Patient.



A



B

Figure 7A: A - Normal x-rays done on the 1st day

B - Extensive bone destruction with sequestrum seen 6 weeks later



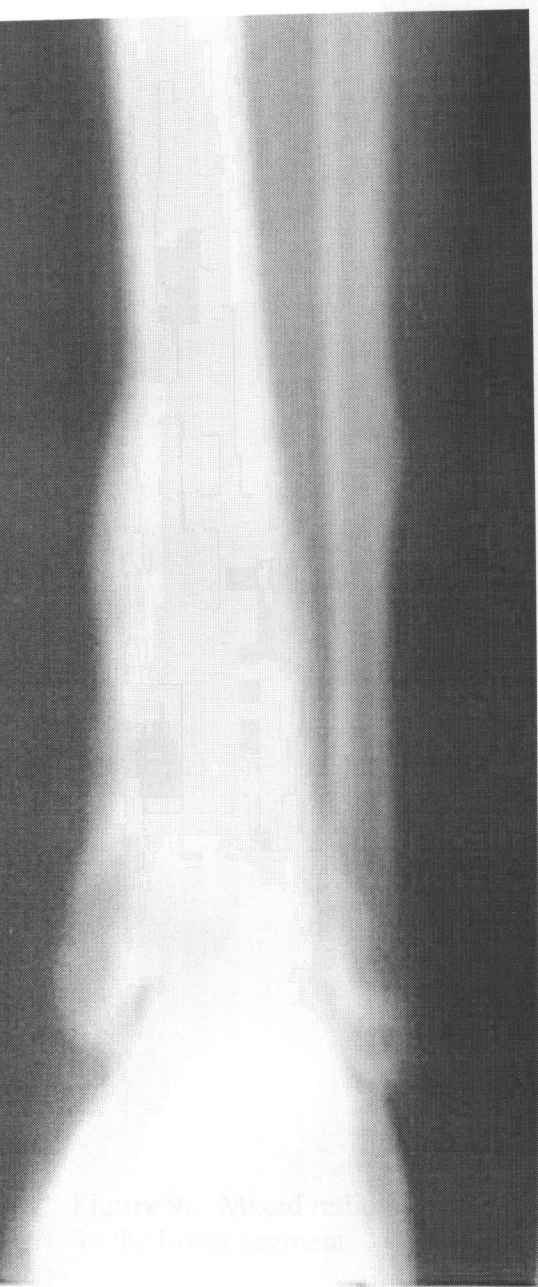
A



B

Figure 7B: A - Normal x-rays taken 1 day after onset of osteomyelitis

B - Extensive destruction of the Fibula seen after 2 weeks



A

B

Figure 8: A - TB osteomyelitis of the Distal Tibial and ankle.

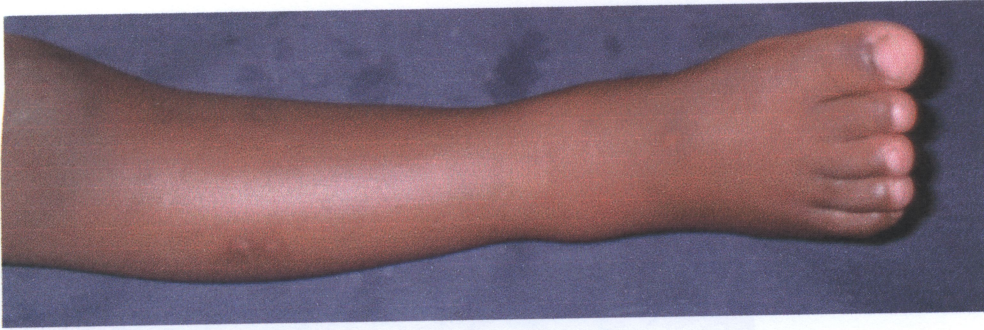
B - Signs of recovery after TB Chemotherapy Confirming suspicion of TB



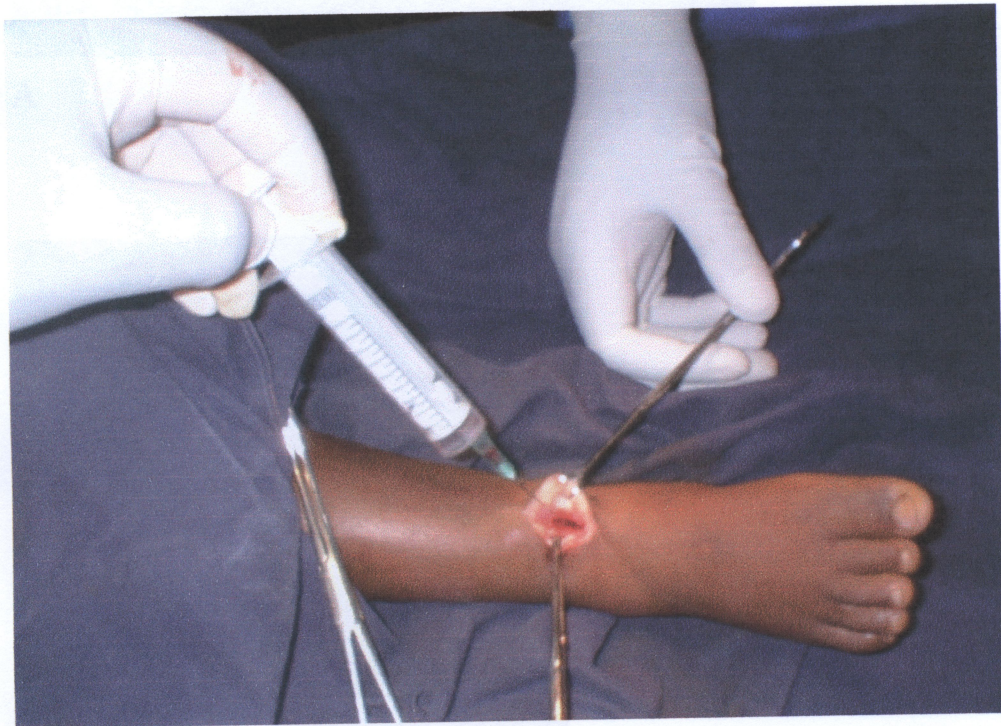
Figure 9: Mixed radiologic picture, Multiple lytic areas or cavities and a sequestrum in the lower segment



Figure 10: Pathological fracture in sclerotic Hypertrophic osteomyelitis of the femur



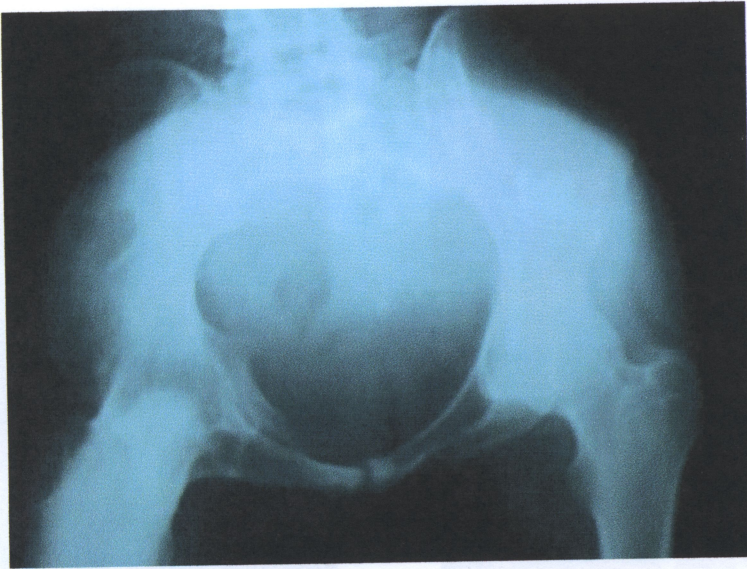
A



B

Figure 11: A - Normal looking limb in a child 3 years old with acute osteomyelitis 36 hours After onset.

B - Exploration of the metaphysis yielded seropurulent fluid from which *Staphylococcus* was isolated.



A



B

Figure 12: A - Ankylosis complicating osteomyelitis of the right proximal femur

B - The result is a severe shortening of 7cm in the limb



A



B

Figure 13: A - Ilizarov System used to correct varus bowing of a leg with a Tibia Defect Post Osteomyelitis. Tibialisation of the fibula is planned after Correction of varus deformity
 B - 5cm shortening being reduced simultaneously

DISCUSSION

The study has identified a number of factors, which affect the morbidity and mortality outcome in patients who present with acute haematogenous osteomyelitis at UTH. These are; inadequate treatment, Delayed Presentation, Associated Debilitating disease and failure to recognise and refer early for surgical treatment by primary medical attendants. At UTH, osteomyelitis patients are referred from peri urban clinics by clinical officers. The initial management of AHO is done by general surgeons who later refer to orthopaedic surgeons for continued management.

Age and Sex Distribution

The sex distribution results in this study was 76 males and 36 females (ratio 2-1). This agrees with findings of other researchers. Hoffman and Goldsmith showed that males outnumber females (3:1) in South Africa ¹⁵. Mollan and Piggot had 52 males to 41 girls ratio almost 1:1 in the Belfast study ³⁶. Other series report Ratios of 3-4 boys to 1 female^{27, 42}.

The age range in this study was 0-90. The youngest was three months and the oldest was a 90 years old male. The results show that the age range 6-10 had the highest frequency. The mode was 7 years.

When the age from 0-50 is considered, the median value was 8 years and the mean was 6 years. This result is similar to results recorded in literature. Morrissey et al explained that the frequency is high in this age group because of association to trauma ⁴⁹. In this study history of trauma was reported in 22 (25%) children.

Presentation and Site of Osteomyelitis

In this study limb pain was reported in all 100% of the patients 71% had reported swelling at the site of osteomyelitis 88% and reported limping or loss of function in and body hotness or temperature above 37.5⁰0 was seen in 69% patients. Associated joint swelling was seen in 20%. The results are similar to observation by Mollan and Piggot. In their series of 93 patients, limb pain was seen in 96% patients and that not all cardinal signs of osteomyelitis were seen in all the patients ³⁷.

The frequency of Poor outcome was high in patients with delayed presentation. Patients with early presentation had a high frequency of good and satisfactory outcome despite being treated inadequately. This is because some had received oral antibiotics before presenting. Out of 40 patients only 9 had adequate treatment. Out of 40 patients who presented early, only 09 (23%) patients were treated adequately. The results showed that 04 (44%) of patients in the group had good outcome and 05 (64%) had satisfactory outcome. Luschke and Frey in their study of 23 patient who presented early and were treated adequately 19 (83%) had satisfactory results. These results demonstrated the importance of early and aggressive treatment, which combines surgery and antibiotics.

31 (78%) of the patients who presented early were treated inadequately (with antibiotics alone). 55% had satisfactory results.

The proximal tibia (21%) and proximal femur was (19%) and distal femur (14%) were the most common affected sites. Hoffman and Goldschmidt found that the distal femur (23%) proximal tibia (18%) and (17%) distal tibia was the most common site. The Tibia

is prone to trauma and has poor blood in its subcutaneous position. This study showed a surprising high level of proximal femur osteomyelitis. This is due to contribution from complications of septic arthritis of the hip.

Associated Predisposing Factors

Predisposing factors for haematogenous osteomyelitis were seen in 65% of the 87 children less than 21 years old. The result is not far from other results in literature. Luschke and Frey in Namibia reported 66% prevalence of predisposing factors in children ³⁰. In this study 25% onset was associated with trauma, 26% had a poor nutritional status and 14% had sickle cell disease. It was difficult to assess the history of recent fever, UTI, throat and GE.

In 25 adults, 21 years and above, 18 (72%) were HIV positive, 04 (14%) had diabetes mellitus, 03 (12%) were elderly patients, 70 years and above. In 1992, Professor Jellis reported adult onset haematogenous osteomyelitis 16 out of 147 HIV positive orthopaedic patients ¹⁹. In this study, 18 (70%) out of 25 patients with adult onset haematogenous osteomyelitis were HIV positive. Defective humeral and cellular immunity is associated with increased incidence of bacterial infections. This partly explains the frequent occurrence of osteomyelitis in HIV, diabetes malnourished children, infants and elderly patients.

Premorbid Condition Recent Infection

Fifty-three 29 (33%), patients had a recent infection in the week before onset of the limb pain. From the literature review there is a history of recent or concurrent infection in

25% to 50% of the patient of haematogenous osteomyelitis ^{15,27}. A recent or concurrent infection is the source of bacterium, which predispose to osteomyelitis.

Challenges in Diagnosis

The clinical picture remains the most important factor in making the diagnosis. In acute osteomyelitis x-rays are negative ²⁰. Eighteen, 18 patients had presented with early acute osteomyelitis. Only 1 patient was referred to orthopaedics and received adequate treatment as defined in this study. 22 patients presented with late acute osteomyelitis. Only 08 were referred within 3-5 days and received adequate treatment. The rest were either misdiagnosed (inappropriate treatment) or treated inadequately and referred late to orthopaedic surgeons. Out of 72-delay presentation, 02 were seen by medical officers, 43 by clinical officers, 02 nursing officers and 25 were treated by traditional healers It is reported in literature that in Africa most patients present late they seek traditional treatment initially ⁴¹. 40 (36%) were diagnosed as something else before clinical diagnosis of osteomyelitis was made. 19 (17%) were diagnosed as malaria, 14 (13%) as soft tissue trauma, 06 (5%) as sickle cell crisis, 07 (6%) as pyomyositis, 06 (5%) as pneumonia, and 03 (3%) as abscess. Failure to recognize osteomyelitis was contributed to delay in presentation of osteomyelitis.

Erythrocyte sedimentation is not helpful as diagnostic aid but as monitoring tool. Out of 40 patients with acute and post acute ESR was raised >50 mm/hr in (14) 35% by the at the time presentation.

In this study delay was due to many factors, lack of money to transport the patient, failure to recognize osteomyelitis and self-treatment at home. Six 06 (67%) of patients who

presented with late acute the osteomyelitis had a good result 03 (33%) had 43 (60%) of the patients who presented with neglected osteomyelitis had poor outcome.

Sensitivity Patterns Of Staphylococcus

Thirty-two 32 patients (74%) of the isolates were due to staphylococcus. The results showed 100% sensitivity to Ceftaxime andVancomycin, 97% to Ciproflorxacin, 94% to Chloramphenical and Oxacillin. Resistance outcome was, 87% to Penicillin, 60% to Tetracycline, 31% to Ampicillin and 21% to Gentamycin .At UTH Cloxacillin is frequently used in osteomyelitis.

CONCLUSION

The study has shown the factors that are associated with high morbidity at UTH. These are comparable to other studies elsewhere. HIV infection has worst outcome in terms of function and chronicity. Neglected acute osteomyelitis (Delayed Presentation) defined as a case presenting over one week after onset had highest percentage of Poor outcome. Osteomyelitis in sickle cell disease had poor outcome in terms of physical disability. Children who are malnourished had a lower than expected poor outcome. This is because there were no cases of severe malnutrition like Marasmus and Kwashiorkor.

Many children were from the poor families around Lusaka. High numbers of poor were recorded in this group. Most of them saw a traditional health attendant or stayed at home due to lack of money for transportation to a medical centre. The study showed clinical officers failed to recognize osteomyelitis and refer osteomyelitis. Early for adequate treatment.

Inappropriate management was associated with high frequency of poor outcome. This was comparable with other studies in the region. However, this study could not show that bone-drilling leads to improved outcome of metaphyseal osteomyelitis because patients are mainly referred after the acute stages of the infection. Chronic osteomyelitis is associated with devastating outcome of the disease. Three 3 patients had large bone defects of the tibia requiring Tibialisation of the Fibula later and 4 patients had severe shortening and 3 had ankylosed hips. The question of bacterial virulence factor osteomyelitis remains unanswered in this and many other studies. It is obvious that a less virulent organism will present with less profound symptoms and would naturally present

late with sequelae of chronic osteomyelitis. This is seen in tuberculosis osteomyelitis, sacoidosis and fungal osteomyelitis. The host tissue resistant determines what pattern of bone lesions as seen on x-ray. A sclerotic lesion was the commonest in this study and signifies an attempt to contain the infection by a stronger immunity.

Nonetheless, this type of lesion is associated with much acute exacerbation later. Large bone death and sequestration is indication of the virulence of initial infecting organisms usually staphylococcus. The sequestrum is later surrounded by involucrum, granulation and necrotic tissue and cannot be reached by antibiotics. Therefore adequate surgical management requires removal of all dead bones, necrotic tissue and infected granulation. The most of sclerotic bone must be removed by reaming. In Sclerotic hypertrophic osteomyelitis reaming and re canalisation of the medulla brings in new blood supply that quickens healing.

The study did prove however that most of the patients had profound symptoms at onset. This suggests that they had an acute osteomyelitis. It can be concluded therefore that chronic osteomyelitis is part of continuous diseases that starts as an acute osteomyelitis. The other evidence is that staphylococcus is the commonest isolates in acute osteomyelitis and chronic osteomyelitis.

In addition a sequestrum in a dead space in bone favour secondary invasion by anaerobic organisms, which can be eradicated by bringing new blood supply. Such methods of surgical treatment are employed in 12 patients with chronic osteomyelitis who were operated on. In general chronic osteomyelitis is a difficult disease to eradicate. It is better to prevent its occurrence by early recognition and treatment of acute stages of infection.

RECOMMENDATIONS

Looking at the results in this study, the following recommendations are being made:

1. Diagnosis and treatment charts or algorithms for osteomyelitis should be posted in local urban clinics to help primary medical attendants in recognising AHO. This has been done for diseases like pneumonia and diarrhoea. This will help the clinical officer and other officers managing these centres to recognize and refer osteomyelitis for early adequate management. This will reduce the levels of poor outcome (physical disability, chronicity and uncosmetic appearance) seen in patients at UTH.
2. Medical students and clinical officers and health care providers should be taught that not all cardinal signs of AHO are present initially. Thus any child who present with pain around a major joint with a questionable history of trauma with or without a fever should be explored surgically for the presence of pus in the joint, metaphysis or subperiosteal space.
3. Clinical officers, paediatricians should be aware of a febrile child whose symptoms are not responding to 3 days of antibiotics or antimalarials. Such a child should be considered for bone infection. Investigation to locate the site of infection, should include bone scans with isotope labelled leukocytes.
4. A child below the age of 15 years who presents with trauma to the lower limb without a normal bone findings with or without soft tissue swelling should be considered for prophylactic antibiotics cover and probably with anti platelet treatment like Aspirin.

5. Adequate treatment for AHO should be based on the sensitivity patterns. The empirical antibiotic treatment being proposed is IV chloramphenicol. Cefotaxime or Ciprofloxacin should be considered as a first choice when available.

48 hours later the patient should be treated according to the culture and sensitivity results. The patient should be treated with intravenous antibiotics until symptoms and signs have shown improvement. Follow up oral antibiotics should be given for 3-6 weeks or until patient is judged to have clinical improvement or by drop in ESR.

6. The timing of surgery can prevent disabilities in chronic osteomyelitis. Bone stability must be achieved with adequate involucrum before a sequestrum can be removed. Other methods of treating disability in children include papineau technique, Ilizarov technique and osteocutaneous flaps must be employed to manage and prevent gross disability in children.

OSTEOMYELITIS OF LONG BONES RESEARCH
PROFORMA

STUDY No: _____

A. PERSONAL DATA: AGE/SEX _____ months/years M/F
Duration of osteomyelitis _____
Age at first presentation _____

B. HISTORY

FROM; PATIENT/GUARDIAN/MOTHER/FATHER/MEDICAL RECORDS.

1. History (Clinical presentation); SYMPTOMS; FEVER, PAIN, LOCAL SWELLING, JOINT SWELLING, A LIMP, OTHER _____
2. Duration of symptoms (at first presentation) _____
3. Primary Medical Attendant NURSE/CLINICAL OFFICER / MEDICAL OFFICER/ TRADITIONAL _____
4. Preceding Morbid Condition: Pneumonia (chest infection)/Urinary tract infection/Gastroenteritis/Throat Infection/Skin Sepsis/Malaria. TRAUMA a Fall/a kick (reported by child/Adult) Other _____
5. Associated Risk factors: SCD, PEM, DM, Clinical AIDS, Previous bone infection, ORIF, OTHER _____
6. Physical Findings; Nutrition Status: Body weight _____ Height _____
7. Systemic Features: General conditions, ill, very ill, Toxic. _____
Body Temperature _____
8. Local Features: LOCALISED SWELLING (ABSCESS) DIFFUSE SWELLING (CELLULITIS), JOINT SWELLING, DISCHARGING SINUS, OTHER _____
9. Treatment Given: Working Diagnosis _____ Admission YES/NO Intravenous antibiotics YES/NO Injectable Antibiotics YES/NO Oral Antibiotics YES/NO Antimalarials YES/NO IV Fluids YES/NO Blood Transfusion YES/NO. Surgical intervention: NIL/ BONE DRILLING/ INSCISION AND DRAINAGE/ ARTHROTOMY/SEQUESTRECTOMY/ SAUCERISATION/ CURRETAGE.

C. PRESENT STATUS

1. Diagnosis: Acute/Intermediate/Chronic.
2. Radiological Findings in favor of Active Infection: Nil/Difficult to tell/ Periosteal elevation (reaction)/Lytic areas (Cavitations)/ Sclerotic areas/ Brodies Abscess/ Sequestrum/ Involucrum. Bone and part involved: PROX TIBIA/ PROX FEMUR/ DISTAL FEMUR/ TIBIA SHAFT OTHER _____

3. LABORATORY FINDINGS:

RVD test +VE 1+2 /-VE
Haemoglobin level _____
WCC _____ DC L _____ PMN _____ M _____ B _____ E _____
ESR _____

4. Physical Disability: Joint stiffness/Severe contracture; SHOULDER / ELBOW /HIP/ KNEE/ ANKLE other _____
Pathological fractures, Muscle wasting, Pressure sores, Limb shortening, Mobility aids (CRUTCHES WHEELCHAIR) OTHER _____

5. Socioial economic impact : Number of admissions _____ Number of operations _____ Education: Grade attained _____ other _____

PROFORMA

STUDY No:

Pre operative assessment: Bone involved

Number of operations _____ haemoglobin level _____ RV test _____
 +VE/-VE Radiological finding _____

Pre operative antibiotic	iv/im/po
--------------------------	----------

Operator: JRMO/ SHO/ REGISTRAR/ SENIOR REGISTRAR/ CONSULTANT

Operation: SEQUESTRECTOMY/ SAUCERISATION/ CURRATAGE/ AMPUTATION

OTHER

Intra operative finding: PUS/ SEQUESTRUM/ SCLEROTIC BONE (obliterating the Medullary cavity) OTHER

Done: Removal of sequestrum partially/in total, Curettage of cavity, Removal of sclerotic bone from Medullary cavity, Lavage with saline/antibiotics, Pus Swab, biopsy OTHER

	mg/	iv/	im
Intra operative antibiotics:			

Tourniquet time: **HAEMOSTASIS:TIE/DIATHERMY.**

Post operation POP YES/NO

IN PATIENT FOLLOW UP: Post op Complication; SWELLING (requiring release of dressing or casts) / BLEEDING MILD,MODERATE (Requiring change of dressing with in 24 hours) SEVERE (Requiring blood transfusion)

OTHER

AT 2 WEEKS (date _____) Wound healed YES/NO, Persistent Discharge YES/NO Soft tissue pus collection YES/ NO. Joint movement FULL/REDUCED/FIXED, **Muscle wasting/contracture:**MID LIMB DIAMETER

Treatment: SPLINTS/PARTIAL WEIGHT BEARING/ FULL WEIGHT BEARING/
PHYSIOTHERAPY OTHER

AT 4 WEEKS (date)

Findings

Treatment

AT 3 MONTHS (date)

Findings

Treatment

AT 6 MONTHS (date)

Finding

Treatment

AT 1 YEAR (date)

Findings

Treatment

OSTEOMYELITIS OF LONG BONES
BACTERIOLOGY

STUDY No: _____

Sample: Code:
URINE A
BLOOD CULTURE B
SUPERFICIAL PUS C
DEEP PUS D
BONE BIOPSY CULTURE E

Has patient taken antibiotics in the last 2 weeks? YES/NO

If yes, which antibiotics ? _____

Date last taken ____/____/____

Culture results:

Sample/Organism code	1	2	3
Code			
A	_____	_____	_____
B	_____	_____	_____
C	_____	_____	_____
D	_____	_____	_____
E	_____	_____	_____

Sensitivity test result

S = sensitive R = resistant

Antibiotics	/Sensitivity			OUTCOME AFTER TREATMENT
	1	2	3	
Cefotaxime	___/___/___			_____
Penicillin	___/___/___			_____
Cloxacillin	___/___/___			_____
Co-trimoxazole	___/___/___			_____
Chloramphenicol	___/___/___			_____
Ciprofloxacin	___/___/___			_____
Amoxacillin	___/___/___			_____
Ampicillin	___/___/___			_____
Gentamicin	___/___/___			_____
Erythromycin	___/___/___			_____
Nalidixic acid	___/___/___			_____
Nitrofuratoin	___/___/___			_____
Ceftriaxone	___/___/___			_____
Oxacillin	___/___/___			_____
Vancomycin	___/___/___			_____
Methicillin	___/___/___			_____
_____	___/___/___			_____
_____	___/___/___			_____
_____	___/___/___			_____

INFORMED CONSENT FORM

Study Title: Factors influencing the outcome of osteomyelitis (bone infection) at UTH in Lusaka

Introduction: This is to inform you that I am carrying out a research which is looking at the factors that influence the outcome of osteomyelitis. In order for you to participate in this study you will be required to give blood samples which will be collected from your forearm Without discomfort. In case of any pain or problem doctors and nurses present at the time will attend to you immediately. Full blood counts, ESR and HIV testing will be done on the blood collected. You will be provided with pre and post test counselling for possible implications. A pus swab will be collected from the wound overlying the osteomyelitis. After that a surgical operation will be done on the site of your osteomyelitis to take a piece of bone for isolating organisms responsible for your disease. During this time you will enjoy all the medical services and medication entitled to you through the hospital

Possible Benefits: Care will be taken to isolate the probable cause of disease through the laboratory facilities for which you will not have to incur extra personal expenses. You will also be kept under supervision during your medication which will be changed according to your response and sensitivity patterns of the isolated organisms. There is no direct monetary gain by virtue of you participating in this study.

Possible disadvantages: You will be required to stay in the hospital at least for three (03) days after the operation. You will also experience some pain after your operation which will be taken care of by the doctors on the ward.

Confidentiality: All the records and results of laboratory findings will be kept in strict confidence. Confidentiality will be maintained throughout the study. You shall be informed if there is any other treatment required out side the domain of this study.

Voluntary participation: *Your participation in this study is purely voluntary. In case you wish to withdraw from the study you will be allowed to do so without prejudice. Your giving consent does not violate your right to withdraw.*

If you have any questions or need any clarification please call or write to me on the phone number and address given below:

Dr. Maundo Makunika Chowa,
University Teaching Hospital,
Department of Surgery,
RW 1X, LUSAKA.
Mobile Phone: 097-779701

I have fully understood the procedures and implications of the study:

Name of the participant/Guardian.....

If guardian relationship with participant.....

Signature or thumb print.....

Date:.....

Witnessed by.....

Date

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