“ROLE OF MRI IN OSTEOMYELITIS”

By

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ABSTRACT

BACKGROUND & OBJECTIVES

Osteomyelitis is an infection and inflammation of the bone.

It is classified by the severity of the inflammation and length of time the infection has been present. Osteomyelitis is an important cause of morbidity.[1]

Imaging plays a crucial role in establishing a timely diagnosis and guiding early management, with the aim of reducing long-term complications.

Only a few studies of osteomyelitis have been conducted in India to find out early diagnostic criteria. Thus, this study is undertaken to determine the early diagnostic features of osteomyelitis on MRI and provide true anatomical extent of the infection for guidance towards proper treatment and management.

AIMS & OBJECTIVES OF THE STUDY:

The aim of this study is to evaluate the early detection of osteomyelitis through Magnetic Resonance Imaging (MRI) scan and know the extent of soft tissue and bone involvement.

SOURCE OF DATA:

The patients attending/referred to the Radiology department or admitted to B.L.D.E. (DEEMED TO BE UNIVERSITY) Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura; with clinical suspicion of osteomyelitis.

METHOD OF COLLECTION OF DATA:

The study is based number of patients, who are visiting Department of Radio Diagnosis from the period of between NOVEMBER 2016 to APRIL 2018. Consent taken for each case.

RESULT: In our study series of 45 cases, we got 32 cases of infective etiology (osteomyelitis), 37 cases showed soft tissue changes, 41 cases had bone marrow changes, 39 cases in which fluid was present, 25 cases had intraosseous findings, 21 cases showed cortical bone destruction and 36 cases were showing contrast enhancement after administration.

INTERPRETATION: MRI is a revolutionary imaging modality that helps in evaluating the normal anatomical structures, normal variants, distribution features, localization and assessing the extent of various pathologies affecting the bone.

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INTRODUCTION

Osteomyelitis is inflammation of the bone marrow secondary to infection, which can progress to osteonecrosis, bone destruction and septic arthritis. It is an important cause of permanent disability in both children and adults worldwide.

The typical clinical presentation of osteomyelitis with pain, erythema and oedema of the affected part is nonspecific and can be caused by a multitude of other diseases. Poor feeding and irritability may be the only symptoms present in infants. Serum inflammatory markers may be normal, especially in neonates and patients with chronic osteomyelitis. For these reasons, imaging plays an integral role in establishing the diagnosis of osteomyelitis and characterizing the extent of disease spread.

The importance of imaging goes beyond making the initial diagnosis as radiologists are able to perform image-guided abscess aspirations and bone biopsies to direct further management, and follow-up scans are often required during the course of treatment to ensure resolution of infection.

This study provides an overview of the imaging of osteomyelitis, focusing on the radiological features.

The diagnostic imaging of osteomyelitis can require the combination of diverse imaging techniques for an accurate diagnosis.

IMAGING IN DIFFERENT MODALITIES:

Conventional radiography has low sensitivity and specificity for detecting acute osteomyelitis. As many as 80% of patients in the first two weeks of onset of infection will have a normal radiograph.[(2)]

Sonography is useful to diagnose subperiosteal fluid collections, periosteal involvement, and surrounding soft tissue abnormalities. Its main limitation is that it

cannot assess bone marrow.[3]

The evaluation of osteomyelitis with CT is limited by its poorer soft tissue resolution compared to MRI. CT is poor in demonstration of bone marrow oedema, which means that a normal CT does not exclude early osteomyelitis. Other limitations of CT are ionizing radiation exposure and image degradation by streak artefact when metallic implants are present. [1]

Magnetic resonance imaging is the most sensitive and most specific imaging modality for early detection of osteomyelitis. It provides superb anatomic detail and accurate information of the extent of the infectious process and soft tissue involvement.[2]

Nuclear medicine imaging can detect osteomyelitis very early and allow imaging of the whole skeleton to look for multiple sites of infection but nuclear medicine studies are not cost effective. [4]

There will be a particular emphasis on MRI because it is the imaging modality of choice for the investigation of suspected osteomyelitis owing to its high sensitivity for detecting early osteomyelitis, excellent anatomical detail and superior soft tissue resolution in current evidence based guidelines.

AIMS AND OBJECTIVES:

- To evaluate the early detection of osteomyelitis by Magnetic Resonance Imaging (MRI) scan.
- To study the extent of soft tissue and bone involvement.
- To compare plain X-Ray, Ultrasonography, Computed Tomography scan findings with Magnetic Resonance Imaging.

METHODOLOGY

This study evaluating the efficacy of MRI in the diagnosis of osteomyelitis was done on 45 cases. This study was conducted during the period between NOVEMBER 2016 to APRIL 2018 in Radiology department B.L.D.E. (DEEMED TO BE UNIVERSITY)Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

Source of Data:

The patients attending/referred to the Radiology department or admitted to B.L.D.E. (DEEMED TO BE UNIVERSITY) Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura; with clinical suspicion of osteomyelitis.

SAMPLE SIZE:

With sensitivity of detecting osteomyelitis on MRI 97 %, at 95 % confidence level and at +/-5 margin of error, the sample size is 45.

$$n = \frac{z\sim^2 p(100-p)}{D^2}$$

Where zα – z value at α level ***p- sensitivity for bone lesions***

d- margin of error

Hence minimum 45 cases of osteomyelitis will be included in the study.

SELECTION OF PATIENTS:

INCLUSION CRITERIA:

All patients presenting with clinical suspicion of osteomyelitis irrespective of age sex.

EXCLUSION CRITERIA:

1. Patients who are claustrophobic.
2. Patients who have bone implants.
3. Patients with chronic kidney disease.
4. Patients who do not consent to the examination.

Preparation of Patients:

Prior to performing the scan particularly in infants and children less than six years, sedation was usually required. The purpose of sedation was to avoid motion artifact and to ensure a MRI scan of diagnostic quality.

From six years onwards the need for sedation generally decreased. Sedatives used in our institution were Pedichloryl syrup administered orally or Injection Midaz administered intravenously in the dose of 0.1 – 0.3 mg/kg dose.

Patients were kept nil orally 4 hours prior to the procedure to avoid complications of contrast In infants the last feed before the procedure was omitted.

MRI Machine:

All the MRI scans were performed at our institute on **PHILIPS ACHIEVA 1.5 TESLA**, available at Radiology department B.L.D.E. (DEEMED TO BE UNIVERSITY) Shri.B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

Patients were scanned in the axial, coronal and sagittal planes. Scanning involved the whole infected part of the bone using dedicated MRI surface coils. The higher signal-to- noise ratio produced by these coils allows for improved spatial resolution; both of these factors improve the diagnostic ability of MR study.

Flexible coils are those that wrap around and conform to the anatomic area of interest. They offer improved patient comfort. It is important that the coil be centered over area of primary interest. Restraint bands should be used to restrict movement of the coil with respiratory or gross patient motion.

Imaging Planes and Technique

Depending on body dimensions, images were obtained in four or five subsequent table positions. Parallel imaging technology was used, with generalized autocalibrating partially parallel acquisition through integrated parallel acquisition techniques. All MR examinations included coronal two-dimensional fast short inversion time inversion-recovery (STIR) images and non enhanced and contrast material–enhanced fat-suppressed two-dimensional T1-weighted fast spinecho (SE) images.

On the basis of the coronal images, dedicated axial images were obtained of specific sites suspected of having pathologic processes. Contrast-enhanced MR images were obtained after the intravenous administration of 0.1 mmol per kilogram of body weight of gadoliniumbased contrast material (gadopentetatedimeglumine, Magnevist; Schering, Berlin, Germany).

Imaging parameters of the two-dimensional fast STIR sequence were as follows: 7320/79/150 (repetition time msec/echo time msec/inversion time msec); echo train length, 19; section thickness, 5 mm; gap, 1 mm; flip angle, 140°; bandwidth, 305 Hz/pixel; field of view, 479 mm; and matrix, 384. Imaging

parameters of the two-dimensional T1-weighted fast SE sequence were as follows: 591/11 (repetition time msec/echo time msec); echo train length, three; section thickness, 5 mm; gap, 1 mm; flip angle, 180°; bandwidth, 200 Hz/pixel; field of view, 480 mm; and matrix, 384. Imaging parameters of the two-dimensional fat-suppressed T1- weighted fast SE sequence were as follows: 591/11; echo train length, three; section thickness, 5 mm; gap, 1 mm; flip angle, 180°; bandwidth, 200 Hz/pixel; field of view, 479 mm; and matrix, 256. **Contrast material**–enhanced imaging is useful for the evaluation of soft-tissue complications such as sinus tracts, abscesses, and necrosis, and it provides invaluable information for preoperative planning of limited limb resection. It should be performed with a turbo gradient-echo sequence because of the speed and uniformity of fat suppression that sequence provides. After turbo gradient-echo imaging, we usually obtain at least four additional views: one image in each plane and an additional delayed image in the key plane. Obtaining this delayed image is particularly important because the slow blood flow in diabetic patients may lead to false-negative findings due to a lack of enhancement. Caution should be exercised in patients with renal failure because of the potential for gadolinium-induced nephrogenic systemic fibrosis. The American College of Radiology recommends that gadolinium-based contrast material not be administered to patients with a severely reduced glomerular filtration rate (<30 mL/min).

Indications for intravenous gadolinium contrast

Gadolinium is a contrast agent that causes enhancement of tissues according to their degree of vascularity. This enhancement is best assessed on FS-T1 sequences. When investigating suspected osteomyelitis, there are various clinical contexts in which gadolinium is useful. If a possible abscess or sinus tract is seen,

post-contrast FST1 sequences will allow further characterisation. Contrast is also indicated in suspected epiphyseal infection because the unenhanced images may appear normal. Contrast administration is essential for differentiating an abscess from a phlegmon, which is a solid inflammatory mass. Overall, there is a low threshold for gadolinium administration and we routinely obtain post-contrast sequences for patients with suspected osteomyelitis at our institution.

Statistical Analysis: All the data were expressed in percentages.

REVIEW OF LITERATURE

BRIEF HISTORICAL BACKGROUND

HOW THE TERM "OSTEOMYELITIS" WAS COINED (TERMINOLOGY):

Various terms have been used by different authors to denote inflammation of bones. They have all been based on the pathogenic theories held at the time they started to be used.

"Cold" of the bones was the term used by Albucasis (Abulquasim Ez-zahrawy) in the eleventh century.[5] Cold was considered the aetiological factor of the disease by many authors prior to the discovery of bacteria.[6]

"Corruption" of the bones, was the term used by Avicenna (his real name is Ibn-Sina) and most Arab authors.[7]

"Necrosis" was the term used by Gross in 1830.[8] In his opinion the disease was mainly the result of necrosis. This was believed to be either due to local causes, such as blows, wounds, fractures and burns, or to general causes.

"Typhus" of the bone was the term used by Chassaignec in 1853, recognizing only the fulminating cases in which the patient quickly falls into "typhoid state".[9]

" Carbuncle" of the bone, was the term coined by Pasteur in l860.[10] He noticed that -the organisms were the same, both in the ordinary abscesses and bone infection.

"Osteitis" was the term used by Nowicki in 1931[11] and preferred by Dennison in 1948.[12] They believed that the inflammation involved mainly the bony tissues and

that the bone marrow played only a small role in the suppuration. Periostitis, osteitis and myelitis were terms applied by some authors to denote inflammation of the separate bone structures: periosteum, cortex or medulla respectively. The term "Osteomyelitis" was reserved to the combination of infection of the cortex and medulla.[13] "Osteoperiostitis" was the term commonly used by other authors denoting that the main lesion was found to affect the cortex and periosteum.[12] "Periosteomyelitis" was the term used by Starr in 1922[14] denoting that all the bone structures are affected in the process.

"Osteomyelitis" is the term more universally employed nowadays in relation to any bone inflammation. It was the term coined by Nelaton in 1846.[15] Wilensky in 1934 stated that this term was not in use before Nelaton.[16] According to leveuf in 1947, Lannelongue was the first to use this term in 1879 because he thought that the initial infection took place in the bone marrow.[17]

Hunter in 1786 was the first to describe the mechanism of sequestrum formation. He pronounced the importance of the periosteal blood supply in calling for early incision of subperiosteal abscesses, "to prevent as much as possible the separation of the periosteum".[18]

Dorsey in 1818 wrote at length on necrosis of bone.[19]

The cause of osteomyelitis remained obscure until Pasteur in 1860 illustrated osteomyelitis could be caused by the presence of living microorganisms[20]. Before him, osteomyelitis cause was thought to be "severe cold". Soon the common offending organism became known and named Staphyle which means bunch of

grapes and Kokkos meaning grain both Greek words coined the term "Staphylococcus"[21].

The first detailed description of localized abscess formation in long bones came by Benjamin Brodie in 1819 [22]. He considered it a residuum of a previous low grade osteomyelitis.

By the year 1890, Chyne described a case of osteitis in the radius in which at operation a sequestrum was removed without a trace of pus being encountered.[23] Garre's description of similar cases came in 1893 after which the condition became known by his name.[24]He named it "Sclerosing non suppurative osteomyelitis".[23]
The nature of the disease was completed by the discovery of Xrays, by Roentgen in 1896 which facilitated the understanding of the bone changes.[25] Tomographic studies were used to demonstrate deep-seated small cavities. Planographic studies helped to demonstrate small sequestra. [26] Diagnosis of the bone changes in the early stages of acute osteomyelitis did not benefit from Roentgen discovery except recently when the soft tissue changes occurring early in acute osteomyelitis became understood. [27],[28] Advances in imaging have added much to the understanding and diagnosis of different stages of osteomyelitis by ultrasonography, [3],[29] computed tomography [30],[31] and MRI.[32]

FIRST REPORTED CASE

Being one of the ancient ailments, osteomyelitis required several years of research and treatment trials to control by antibiotic therapy but the chronic form is still pretty hectic to regulate.

The most primitive and first case of the disease was the fracture of spinal regions in a dimetrodon Permian species of reptile, which was present nearly 291 - 250 million years before and was described by Moodie.[33] In that case a proper and evident form of infection was present at the place of coarsened bone with an inflated part on the top of the injury, depicting that the wound caught infection. The tissue specific proof is also there to support the Statement.

During the era of Hippocrates around 460-370 BCE [34], the contamination of bone after some injury has been identified, however medical imaging of acute form of bloodstream mediated osteomyelitis was identified afterwards.

Bromfield [6] during 1773 first elaborated that bones 'can be putrefied or rancid in the interior locations at first and then from some exterior wound, also from the contaminated animal fluids'. He coined the term "Abcessus in medulla", and suggested that it wouldn't be very fast to permit it out only when we know that the stuff is beneath the periosteal (envelope of bones except the joint).

Sequelae of Osteomyelitis in the skeletons and mummies of ancient Egyptians were met with. Elliot-Smith and Dawson in 1924 [35] stated that mastoiditis and alveolar abscesses were frequently met with in the ancient Egyptian mummies.

BONES[36]

Skeletal system consists of bones which provide mechanical support for tendons, joints and ligaments and act as calcium, phosphate reservoir, and protect vital organs from damage and maintenance of normal mineral homoeostasis. Bone being a dynamic tissue undergoes repair and renewal throughout the life by the process called bone remodelling. Abnormalities in the remodelling process that

compromise the mechanical strength, structure or architecture of bone, which leads to clinical symptoms, such as deformity, fracture and pain, and phosphate and calcium homoeostasis abnormalities are the major causes in most diseases of bone.

EMBRYOLOGY OF BONES

During embryonic life bones develop by two processes intramembranous ossification (flat bones such as the calvariae of the skull, mandible and maxilla) in which the bone forms by differentiation of mesenchymal stem cells directly into bone cells and endochondral ossification (long bones of the limbs, ribs, pelvis and vertebrae) in which the bone first forms as a cartilage template, which is subsequently invaded by vascular tissue containing osteoprogenitor cells. The cartilage is then removed and replaced by bone, which extends from centres of ossification situated in the middle and at the ends of the developing bone. A thin remnant of cartilage remains present at each end of the bone during childhood and this is referred to as the growth plate or epiphyseal plate (Figure 1 and 2). Growth of the skeleton depends on division of cartilage cells (chondrocytes) within the growth plate. This takes place in the so-called ‘proliferative zone’ near the end of the bone; the newly formed chondrocytes migrate downwards towards the centre of the bone, where they become enlarged in the hypertrophic zone. The hypertrophic chondrocytes then die and the surrounding matrix calcifies before being removed by osteoclasts and replaced by mature bone. During puberty, the rise in circulating concentrations of sex hormones causes cell division in the growth plate to cease. This causes the cartilage remnant to disappear as the epiphyses fuse and longitudinal bone growth stops.

ANATOMY AND MICROANATOMY OF BONES [37]

"The skeleton consists of two main structural types of bone (Figure 1). Cortical bone is dense with a low surface area and forms an envelope around the marrow cavity. It is formed from Haversian systems, which consist of concentric lamellae of bone tissue surrounding a central canal that contains blood vessels. Trabecular or cancellous bone (also known as spongy bone) has a lower density and a larger surface area than cortical bone. Trabecular bone fills the centre of the long bones, flat bones and vertebrae and consists of an interconnecting meshwork of bony trabeculae, separated by spaces that are filled with bone marrow. The majority of bone in the skeleton (80%) is cortical. These differences are relevant clinically since trabecular bone has a high surface area and is remodelled more rapidly than cortical bone. This means that bone is lost more rapidly from sites rich in trabecular bone under conditions of increased bone turnover.

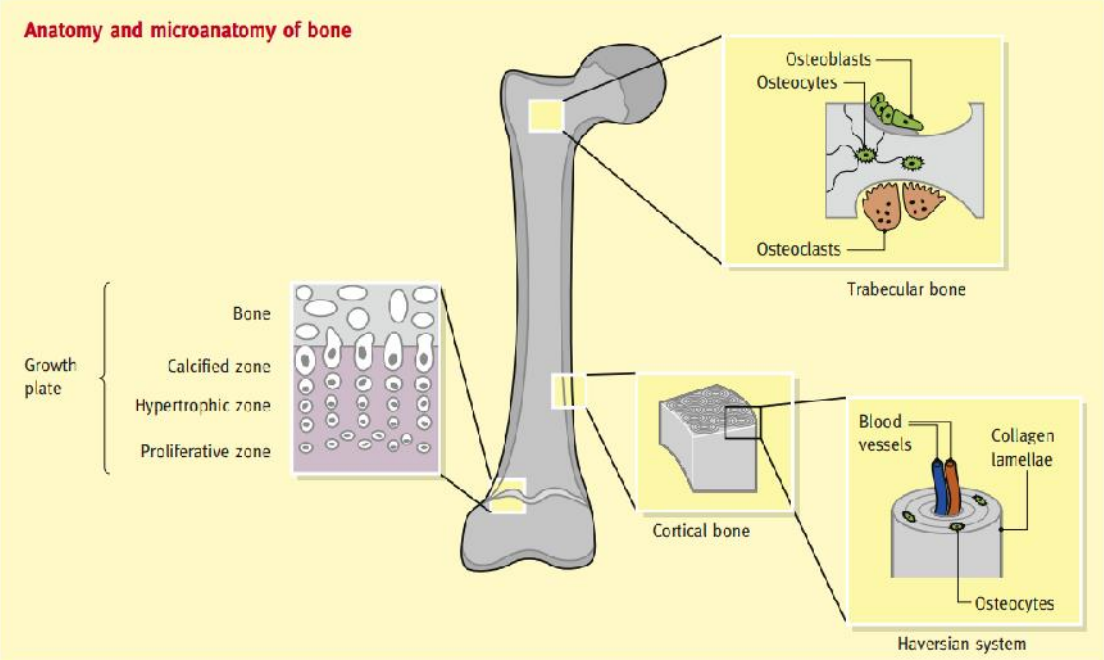


Figure 1

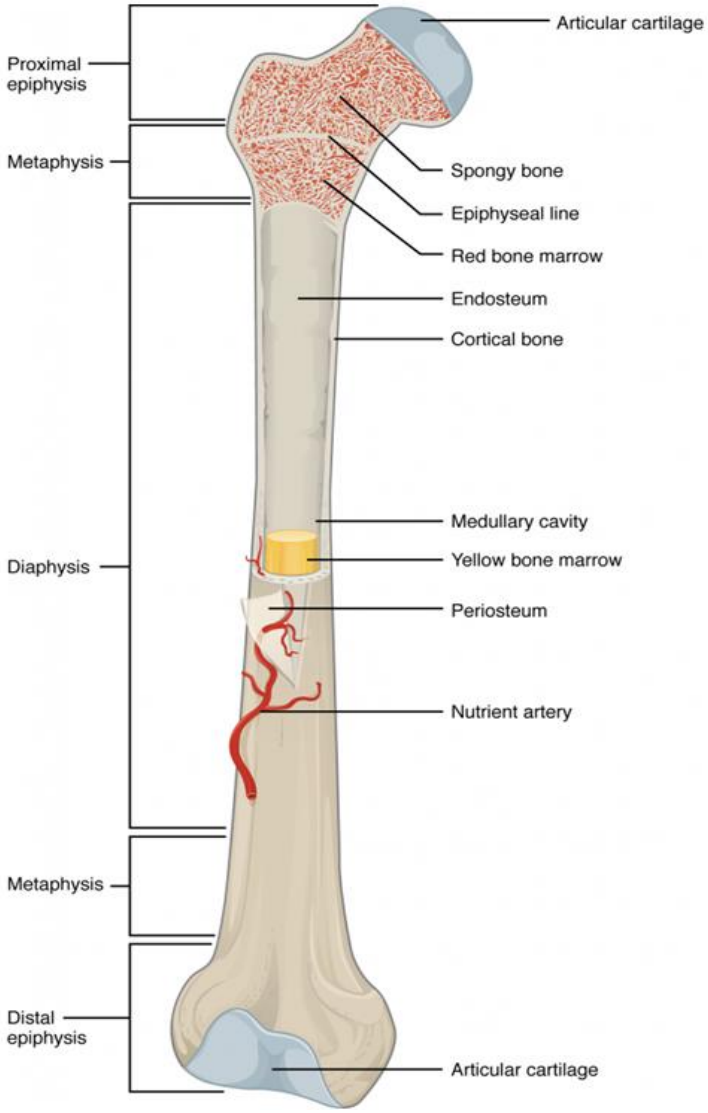


Figure 2

COMPOSITION OF BONE

The main organic component of bone matrix is type I collagen, a fibrillar protein formed from two collagen alpha (I) 1 peptide chains and one alpha 2 (I) chain, wound together in a triple helix. Collagen is synthesized as a propeptide, but following its secretion from the osteoblast, the amino- and carboxyl-terminal

fragments are cleaved off by proteolytic enzymes in the extracellular space. The triple helical domains that remain selfassemble in a staggered configuration to form collagen fibrils. Subsequently, individual collagen molecules within these fibrils become linked to one another at each end by specialized covalent bonds, called pyridinium cross-links, which help to give bone its tensile strength. When bone is broken down, these cross-links are released into the extracellular fluid and their concentrations can be measured in blood or urine, providing biochemical markers of bone resorption. Bone matrix also contains small amounts of other collagens and several non-collagenous proteins and growth factors. Non-collagenous proteins such as fibronectin are involved in mediating attachment of bone cells to the matrix. Growth factors also play a role in regulating bone cell activity. Particularly important is transforming growth factor beta (TGFb), which is buried in bone matrix, where it acts as a coupling factor to promote bone formation, and from which it is released and activated during bone resorption.

The organic component of bone forms a framework upon which mineralization occurs. Mineralization confers on bone the property of mechanical rigidity, which complements the tensile strength and elasticity derived from bone collagen. Bone mineral is composed mainly of calcium and phosphate laid down in the form of hydroxyapatite [Ca10 (PO4)6 (OH2)] crystals.

BONE REMODELLING

Bone is constantly being repaired and renewed through life as the result of bone remodelling, which is taking place in about 10% of the adult skeleton at any one time (Figure 3). Bone remodelling commences with attraction of osteoclast precursors in peripheral blood to the remodelling site. The mechanisms that trigger this event are

unclear, but it is thought that osteoclasts are attracted to areas of skeletal microdamage by chemotactic factors released from damaged bone. The osteoclast precursors start to differentiate and fuse together to form multinucleated osteoclasts. The most important regulators of osteoclast differentiation are summarized in Figure 4 and table 1.

Once formed, mature osteoclasts attach to the bone surface by forming a tight sealing zone over the bone surface, demarcated by a so-called actin ring, which forms around the periphery of the cell. Osteoclasts then resorb bone by secreting hydrochloric acid and proteolytic enzymes into the space underneath the sealing zone, through a specialized membrane called the ruffled border.

The osteoclast contains specialized proton and chloride pumps, which are responsible for secretion of hydrochloric acid into the extracellular space. Under normal circumstances, the acid dissolves hydroxyapatite and allows proteolytic enzymes access to bone matrix, whose components it degrades. When bone resorption is complete, osteoclasts move away from the bone surface and undergo programmed cell death (apoptosis) in the so-called ‘reversal phase’, which heralds the start of bone formation.

Bone formation begins with the attraction of osteoblast precursors to the site that has undergone resorption. The key players are shown in Figure 2. These cells are derived from mesenchymal precursors in bone marrow. Osteoblast precursors differentiate into mature osteoblasts in response to the transcription factors Cbfa1 and osterix (Osx), which binds to the promoter of several osteoblast specific genes such as osteocalcin, type I collagen and alkaline phosphatase, causing the cells to assume an osteoblast-like phenotype. Other growth factors, including bone morphogenic

proteins, are also thought to promote bone formation by encouraging proliferation and differentiation of osteoblast precursors to form mature osteoblasts.

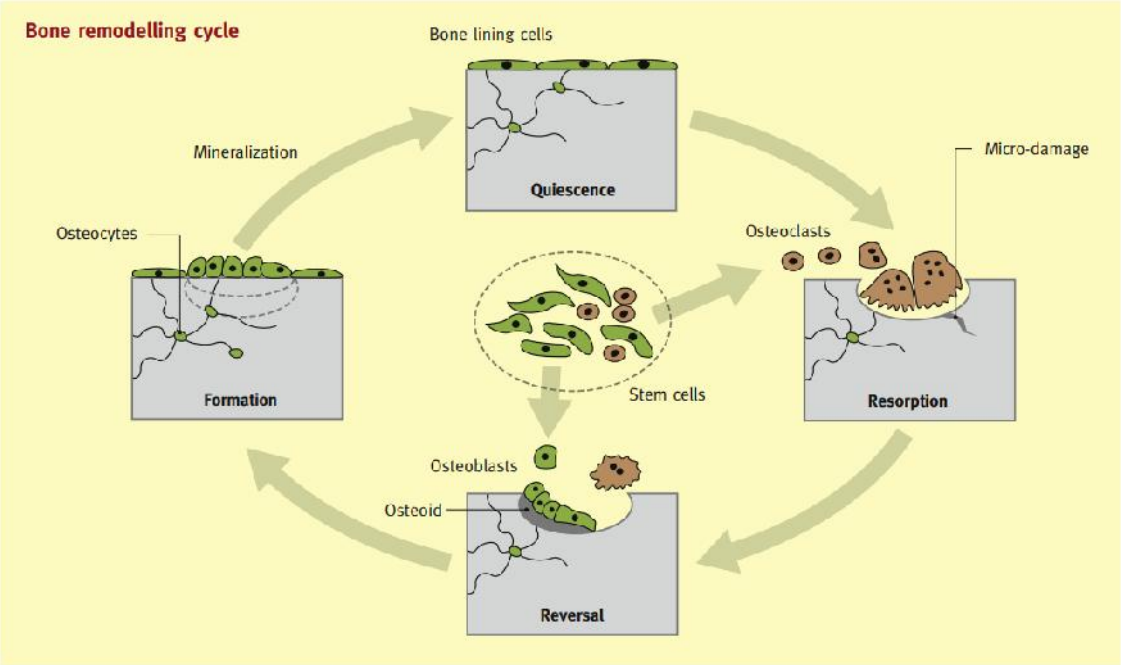


Figure 3

Mature osteoblasts lay down uncalcified bone matrix (osteoid) onto the bone surface and this subsequently calcifies after a period of about 10 days to form mature mineralized bone. During bone formation, some osteoblasts become trapped within bone matrix and differentiate into osteocytes, which interconnect with one another and with cells on the bone surface by long cytoplasmic processes that run through canaliculi in the bone matrix.

Cell type	Function	Location
Osteogenic cells	Develop into osteoblasts	Endosteum, cellular layer of the periosteum
Osteoblasts	Bone formation	Endosteum, cellular layer of the periosteum, growing portions of bone
Osteocytes	Maintain mineral concentration of matrix	Entrapped in matrix
Osteoclasts	Bone resorption	Endosteum, cellular layer of the periosteum, at sites of old, injured, or unneeded bone

Table 1

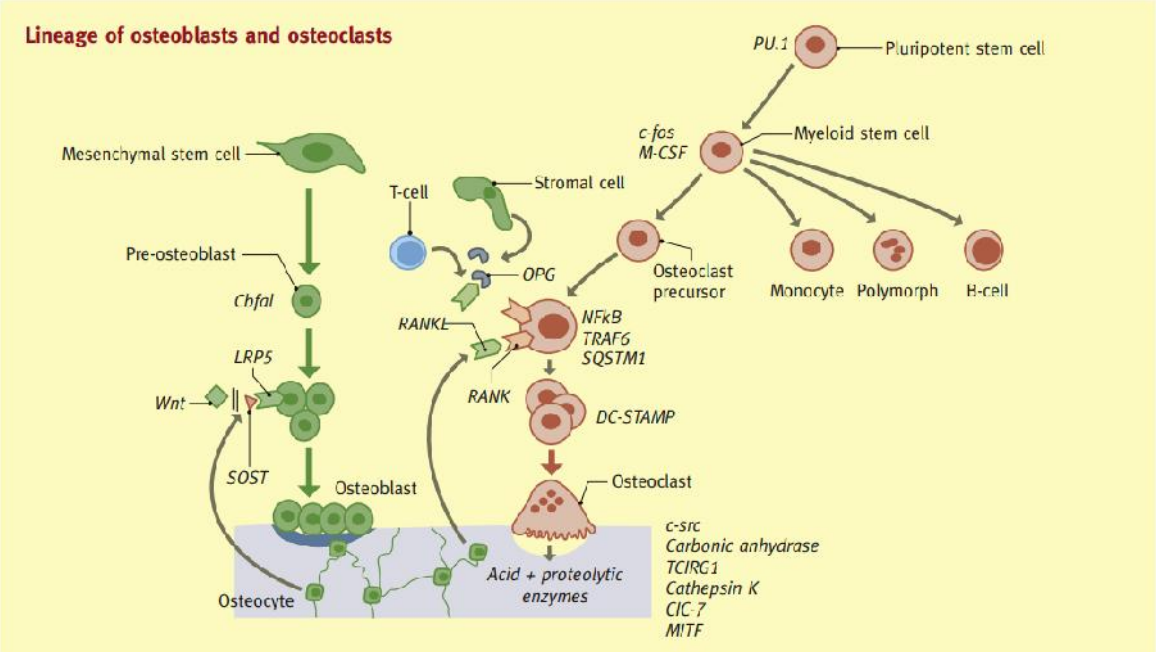


Figure 4

REGULATION OF BONE REMODELLING

The process of bone remodelling is regulated by various circulating hormones and other local regulatory factors. Mechanical loading increases bone formation and suppresses bone resorption, whereas bone resorption is increased by inflammatory cytokines such as interleukin-1 (IL-1), tumour necrosis factor (TNF) and RANKL, which are released during inflammation and play a role in local and systemic osteoporosis associated with inflammatory diseases, such as rheumatoid arthritis. Calciotropic hormones, such as parathyroid hormone (PTH) and 1,25 dihydroxyvitamin D, act together to increase bone remodelling, allowing skeletal calcium to be mobilized for the maintenance of plasma calcium homoeostasis. Bone remodelling is increased by other hormones such as thyroid hormone and growth hormone, but suppressed by oestrogen and androgens. “

INTRODUCTION TO OSTEOMYELITIS

Osteomyelitis is inflammation of the bone marrow secondary to infection, which can progress to osteonecrosis, bone destruction and septic arthritis in infants.[38]

GENERAL CLINICAL PRESENTATION

Generally one may distinguish the three clinical stages of osteomyelitis, although in clinical practice there may be some overlap. The routes of contaminations may vary in those clinical stages. In children hematogenous spread is the most common route of infection, whereas in adults spread from a direct contamination, contiguous source or post-operative infection is more frequent. Furthermore, clinical manifestations may also differ according to the age of the patients. The clinical findings in infants are often more pronounced including local pain, reduced

movement, swelling or difficulty in moving the affected limb, especially in the acute phase. On the contrary, in adults the clinical onset is often more insidious. In children bones such as tibia and femur are the most common sites of infection, whereas the axial skeleton is most frequently affected in adults.

Laboratory findings typically show an increase of c-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) especially in acute osteomyelitis in children whilst the white blood count may be normal. The evolution of the CRP levels correlate with the response to the therapy. Cultures are essential for accurate treatment but in acute setting only half of the blood cultures are positive impeding the diagnosis.

PATHOGENESIS:

"Osteomyelitis is due to infection by a variety of microorganisms via different mechanisms. Staphylococcus aureus is the causative organism in up to 80% of cases of osteomyelitis. Other common pathogens include Staphylococcus epidermidis and Enterobacter species. Certain organisms predominate in specific clinical settings, such as Salmonella species in sickle-cell patients and Pseudomonas or Klebsiella in intravenous drug users. Fungal osteomyelitis most commonly occurs in immunocompromised patients."[(39)]

Predisposing to bacterial proliferation metaphyseal vessels contain slow-flowing blood. Hence, the metaphysis is a common site for haematogenous osteomyelitis. In children over 18 months of age the growth plate forms a barrier between the metaphyseal and epiphyseal vessels. However, in infants under 18 months and in adults, transphyseal vessels are present which provide a route for infection to communicate between the epiphysis and metaphysis. In acute

osteomyelitis, a collection of pus gets surrounded by granulation tissue and reactive bone, forming an intraosseous abscess. Raised intramedullary pressure which is secondary to pus leads to rupture of the cortex, creating a defect (cloaca), which drains pus from the bone to the surrounding tissues. This can cause a subperiosteal abscess with elevation of the periosteum, as well as soft tissue abscesses. In chronic osteomyelitis, disruption of the intraosseous and periosteal blood supply leads to formation of a necrotic bone fragment, known as a sequestrum, which is surrounded by pus and granulation tissue. A reactive shell of new bone forms around the sequestrum and is known as an involucrum. A sinus tract, which drains pus from bone to the skin surface, may be present in both acute and chronic osteomyelitis.

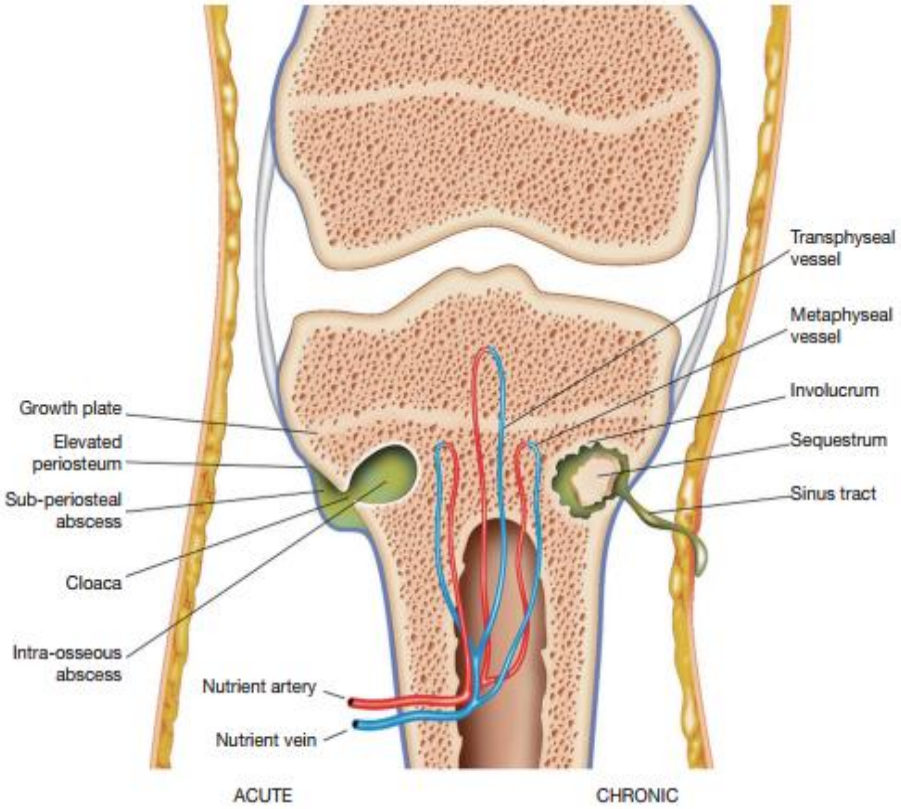


Figure 5

ROUTES OF DISEASE SPREAD:

Haematogenous spread[40]:

"Blood-borne organisms, usually bacteria, are deposited in the medullary cavity of metaphysic and form a nidus of infection."

Contiguous spread[41]:

"Infections originating from soft tissues and joints can spread contiguously to bone. This often occurs in the context of vascular insufficiency, such as in patients with diabetes mellitus or peripheral vascular disease."

Direct inoculation[41]:

"Direct seeding of bacteria into bone can occur as a result of open fractures, insertion of metallic implants or joint prostheses, human or animal bites and puncture wounds."

TYPES OF OSTEOMYELITIS[42]:

- **ACUTE**: Infection has been present for less than 6 weeks.
- **SUBACUTE**: "Brodie's abscess" is a type of subacute osteomyelitis. It appears as a well-defined lucent area in cancellous bone with smooth, rounded geographic margins and a thick sclerotic ring that may merge imperceptibly with the surrounding bone[43]. The lesion appear lobulated with lucent, serpentine tracts extending along the bone. Typically 1-4 cm in size.
- **CHRONIC**: "Infection has been present for more than 6 weeks. If the acute infection is inadequately treated, there will be progression of disease to chronic osteomyelitis. The pathological features of chronic osteomyelitis are a result of osteonecrosis, caused by disruption of the intraosseous and periosteal blood

supply during the acute stage of disease. The diagnostic features of chronic osteomyelitis are involucrum, sequestrum and cloacae"[44].

MAGNETIC RESONANCE IMAGING: PHYSICS AND SEQUENCES

HISTORICAL PERSPECTIVE

Magnetic Resonance Imaging is a relatively newer technology in imaging. It was independently discovered by Felix Bloch and Edward Purcell during the year 1946. Later in the year 1952 they were awarded the Nobel Prize for the same. The use of MR for imaging required a method for spatial localization. In 1973, Paul Lauterbur, a chemist and an NMR pioneer at the State University of New York, Stony Brook, showed how this could be done by applying a linearly varying magnetic field to the body.[45]

The first human MRI examination occurred in 1977. Mansfield and Maudsley [46] in 1977 and Hinshaw et al in 1978[47] were the first to publish human in vivo images. Hawkes et al. in 1980 first demonstrated the multiplanar facility of MRI. The most significant advancement in MRIs occurred in 2003, when the Nobel Prize was won by Paul C. Lauterbur and Peter Mansfield for their discoveries of using MRIs as a diagnostic tool.[48]

THE BASIC PHYSICS OF MRI

Magnetic Resonance Imaging using the principle of nuclear magnetic resonance creates images of the human body and aides in medical diagnosis. It can generate thin-section images of any part of the human body - from any angle and direction. Using MRI it is possible to make such a picture of the human body when the body is exposed to an electromagnetic field.

BIOPHYSICAL BASIS

OF MRI

Nuclear magnetic resonance began within physics, at a confluence among particle physics, condensed matter physics, spectroscopy, and electromagnetic.[48] The human body is primarily made of fat and water. Numerous hydrogen atoms are present in the fat and water molecules, thus making the human body approximately 63% hydrogen atoms. Each hydrogen atom has a nucleus comprised of a single proton. The proton possesses a property called spin which can be thought of as a small magnetic field and will cause the nucleus to produce an NMR signal. MRI is based on magnetic spin properties of nuclei, particularly the hydrogen nuclei and how these nuclei recover after excitation with radiofrequency electromagnetic waves. [49]

When an unpaired electron of hydrogen atom placed in a strong external magnetic field is interrogated with radiofrequency pulse, it releases energy. This released energy is detected by a receiver coil which is then converted into an image by 3D Fourier transformation. [50]

T1 relaxation (spin-lattice relaxation) is due to release of energy into the surrounding tissues. It depends on the time the nuclei take to recover 63.2% of longitudinal magnetization. Chemical nature & physical state of a substance, liquid surrounding the protons, mobility of the protons, magnetic field strength and temperature influence the T1 value.

T2 relaxation (spin-spin relaxation) is due to exchange of energy between spins. It is determined by the time taken by the signal to lose 63% of its initial intensity in the transverse plane due to dephasing.

First, a steady state of magnetism is created within the human body by

placing the body in a steady magnetic field. Second, the body is interrogated with radio waves to change the steady-state orientation of protons. Third, the MRI machine stops the radio waves and registers the body's electromagnetic transmission. Fourth, the transmitted signals are used to construct internal images of the body by computer programs. An MRI image is not a photograph. It is actually a computerized map or image of signals emitted by the human body. [51]

IMAGING

HARDWARE

An MR system consists of the following components:

1) A powerful magnet to generate the static magnetic field.

2) Homogenizing coils (called shim coils) to make the magnetic field as equally distributed as possible.

3) An RF coil for radio signal transmission into the body part which is being scanned.

4) A receiver coil which detects the returning radio signals (echo).

5) Gradient coils for detecting and providing spatial localization of the signals.

6) A computer system for reconstruction of the final image from radio signals received.

The basic hardware component on current imagers is the magnet which produces the B_o field for the imaging procedure. Within the magnet are the gradient coils for producing a gradient in B_o in the X, Y, and Z directions. Within the gradient coils is the RF coil. The RF coil produces the B_1 magnetic field necessary to rotate the spins by 90^o, 180^o, or any other value selected by

the pulse sequence. The RF coil also detects the signal from the spins within the body.[52]

The patient is positioned within the magnet by a computer controlled patient table. The table has a positioning accuracy of 1 mm. The scan room is surrounded by an RF shield. The shield prevents the high power RF pulses from radiating out through the hospital. It also prevents the various RF signals from television and radio stations from being detected by the imager. Some scan rooms are also surrounded by a magnetic shield which contains the magnetic field from extending too far into the hospital. The magnet shield is an integral part of the magnet in newer magnets.[53]

The scanning operation is controlled from a central computer. This specifies the shape of gradient and radiofrequency waveforms, and timings to be used, and passes this information to the waveform generator, which outputs the signals and passes them to be amplified and sent to the coils. The NMR signal, once it has been phase sensitively detected, is turned to a digital signal by an analogue to digital converter. The digital signal is then sent to an image processor for Fourier transformation and the image is displayed on a monitor.

The raw data, that is the signal before Fourier transformation, is stored to enable the application of corrections to the data in post processing. To allow the use of fast Fourier transformation, matrix sizes of 2^n are usually used.

The array processor, located on some imagers, is a device which is capable of performing a two-dimensional Fourier transform in fractions of a second. The computer off loads the Fourier transform to this faster device. The operator of the imager gives input to the computer through a control console. An imaging sequence is selected and customized from

the console. The operator can see the images on a video display located on the console or can make hard copies of the images on a film printer.

The signal intensity on the MR image might be determined by four basic parameters:

1) Proton or spin density,

2) T*1* relaxation time,

3) T*2* relaxation time, and

4) Flow.

Proton density is the concentration of protons (hydrogen atom nuclei) in the tissue in the form of water and macromolecules (proteins, fat, etc). The T*1* and T*2* relaxation times define signal behavior after excitation as well as the way the protons revert back to their resting states (equilibrium) after the initial RF pulse excitation.[54]

IMAGE ARTIFACTS

An image artifact is any feature which appears in an image which is not present in the original imaged object. An image artifact is sometime the result of improper operation of the imager, and other times a consequence of natural processes or properties of the human body. It is important to be familiar with the appearance of artifacts because artifacts can obscure, and be mistaken for, pathology. Therefore, image artifacts can result in false negatives and false positives.

Artifacts are typically classified as to their source, and there are dozens of image artifacts. The following table summarizes a few of these.[55]

Artifact	Causes
RF Offset and Quadrature Ghost	Failure of the RF detection circuitry
RF Noise	Failure of the RF shielding
B_o Inhomogeneity	Metal object distorting the B_o field
Gradient	Failure in a magnetic field gradient
Susceptibility	Objects in the FOV with a higher or lower magnetic susceptibility
RF Inhomogeneity	Failure or normal operation of RF coil, and metal in the anatomy
Motion	Movement of the imaged object during the sequence
Flow	Movement of body fluids during the sequence
Chemical Shift	Large B_o and chemical shift difference between tissues
Partial Volume	Large voxel size
Wrap Around	Improperly chosen field of view
Gibbs Ringing	Small image matrix and sharp signal discontinuities in an image
Magic Angle	Angle between B_o and dipole axis in solids.

Table 2: MRI Artifacts

IMAGING OF OSTEOMYELITIS

CONVENTIONAL RADIOGRAPHY

"The evaluation usually begins with plain radiographs in all patients suspected of having osteomyelitis. Plain radiographs initially show soft tissue changes, muscle swelling, and blurring of the soft tissue planes.

Typical early bony changes include: periosteal thickening, lytic lesions, endosteal scalloping, osteopenia, loss of trabecular architecture, and new bone apposition. Late changes include Periostitis, involucrum formation, and sinus tracts due to subperiosteal abscess with lifting of the periosteum, new bone formation, and soft tissue fistulas."[(1)]

Dr. Marie A. Capitanio and DR. John A. Kirpatrick [(56)] in the year 1970 studied the early Roentgen observations in acute osteomyelitis on patients presenting to Department of Radiology and Pediatrics with a clinical suspicion of osteomyelitis. They discovered that within first 3 days alterations in Roentgen appearance of soft tissues take place with a small, local, deep, soft tissue swelling in the region of metaphysis which is followed by obliteration of lucent planes between muscles and later by involvement of more superficial muscles and soft tissue edema. If it goes undetected Roentgen findings will involve bone destruction and perisosteal new bone formation after 10-12 days.

Modic T. Modic et al in 1985 concluded in a study of 37 patients plain radiographs had a specificity of 57% and sensitivity of 82% while MRI it was 92% and 96% respectively [(57)].

In a study conducted in 1986 by Vung D. Nguyen et al on 14 patients ring sequestrum was a positive characteristic of chronic osteomyelitis and was seen in 13 patients on plain radiography with a sensitivity of 93%. However plain radiography failed to locate the pathology in its earlier stages.

In 1995 Diego Jaramillo et al observed in a study of 84 subjects to assess the roentgen findings of osteomyelitis concluded that plain radiographs detect deep soft tissue swelling and loss of soft tissue planes as early as 48 hours but bone destruction cannot be ascertained before 8-10 days after the onset of symptoms and conventional radiographs are insensitive to destruction of less than 30% of bony matrix. [58]

M. Beth McCarville et al in 2015 [59]in "a study of 60 patients suspected of having osteomyelitis or Ewing sarcoma (EWS) described te plain radiographic features. Typical osteomyelitis included a cavity in bone and cortical sequestrum. A wide transition zone was seen in 23 subjects with EWS and 25 with osteomyelitis . Lamellar periosteal reaction was seen in 13 subjects with EWS and five with osteomyelitis, spiculated periosteal reaction was seen in five subjects with EWS and one with osteomyelitis, and a serpiginous medullary or cortical was noted in none of the subjects with EWS and in four subjects with osteomyelitis. They concluded that plain radiograph lacks the ability to clearly distinguish between EWS and Osteomyelitis."

Cohen M. et al in the year 2018 evaluated the specificity of plain radiographs and MRI in 32 pathology proven cases of forefoot osteomyelitis. 9 cases were positive on both modalities and MRI identified 1.2 additional bone segments of disease on an average. There was surgical agreement with X-ray in 3 out of 31 cases (9.7%) and with MRI in 17 out of 31 cases (55%).[60]

ULTRASONOGRAPHY

"Ultrasound allows evaluation of soft tissue swelling due to oedema or fluid collection, and hyperaemia can be detected using Doppler techniques. Periosteal thickening and sub-periostal collections can also be seen. Ultrasound has high sensitivity for detection of intra-articular fluid. It can be useful for detecting non-radioopaque foreign bodies foreign bodies."[61]

In one of the first case studies Ashok Kumar Nath et al in 1991 "performed ultrasound in 25 patients clinically suspected of osteomyelitis and concluded that sonographic diagnosis of osteomyelitis was made if fluid was present in direct contact with the bone, without intervening soft tissues. This was thought to represent an inflammatory exudate dissecting in a subperiosteal and/or extraperiosteal location. Ultrasonographically 15 patients were found to have osteomyelitis, which was proved either by surgical drainage or needle aspiration. Seven patients had soft-tissue abscesses, one had cellulitis and two patients had no abnormality."[62]

C. Howard, M. Einhorn et al in 1993 [3] "reviewed the ultrasound findings in 59 children suspected of having bone infection. Twenty-nine were eventually proved to have acute haematogenous osteomyelitis and 26 of these showed characteristic ultrasound findings. Ultrasound examination was able to detect the presence of subperiosteal pus and thus indicated the need for surgical treatment."

In the year 1994 Edward T Mah et al performed "a study involving 77 cases and stated that deep tissue swelling which is an accurate sign of acute osteomyelitis can be differentiated from superficial soft tissue swelling of cellulitis on ultrasound. Periosteal thickening and elevation, subperiosteal fluid collection and abscesses can

be visualized on ultrasound. Hence ultrasound is a useful diagnostic tool for early detection and management of osteomyelitis."[63]

In 1995 N. Wright et al [61]described the importance of Ultrasound (US) in identifying subperiosteal abscess formation and cortical thickening in osteomyelitis in study consisting 9 children.

In the year 2005 Quamar Azam et al performed "a prospective study on 55 children with osteomyelitis of limbs to evaluate how ultrasound might be useful in early diagnosis of osteomyelitis. In all cases showing sub-periosteal accumulation of fluid Ultrasound guided aspiration was performed, and the aspirate was sent for culture and sensitivity report. Surgical drainage was undertaken in patients in which a sub-periosteal abscess was demonstrated. Anechoic fluid accumulation contiguous with bone was highly suggestive of osteomyelitis, whereas presence of soft tissue between the bone and the fluid suggested a non-osseous origin of the fluid. Sub-periosteal accumulation of fluid was seen in 42 cases (76.3%). A subperiosteal abscess with periosteal reaction was demonstrated in 35 children (63.63%). Colour Doppler study revealed increased vascular flow within or around the affected periosteum in all cases. Concurrent involvement of a joint was noted in 13 cases. The study concluded that Ultrasound being a rapid, cheap, easily available, non-ionising and reasonably accurate diagnostic modality also helps in localising the lesion for diagnostic aspiration."[64]

James W. Tsung et al [65]in the year 2008 described through 5 case reports that the sensitivity of plain radiography for joint effusion is as low as 20%, whereas ultrasound is the method of choice do detect even small amounts.

Rebecca L. Vieira, MD and Jason A. Levy, MD in the year 2009 did a "study on twenty-eight patients and concluded that bedside ultrasonography had a sensitivity of 80% (95% confidence interval [CI] 51% to 95%), a specificity of 98% (95% CI 85% to 99%) to detect joint effusion and sub-periosteal collection."[66]

In 2011 *Anton Delport, MD and Suzanne S. Long, MD* [67] observed "the diagnostic accuracy of ultrasound in various musculoskeletal infections. Ultrasound being an affordable, rapid, portable, and sensitive imaging modality can be used as the primary imaging modality, or as an adjunct to other imaging modalities, and can improve the outcome of patients with musculoskeletal infections by allowing more rapid diagnosis and enables accurate, real-time guidance of fine needle aspiration which can reduce the risk of contamination of other compartments. Ultrasound was useful in evaluating for septic arthritis, periarticular or soft tissue abscess, septic bursitis, and tenosynovitis and can be helpful in diagnosing osteomyelitis but cannot be used a sole imaging modality."

COMPUTED TOMOGRAPHY

"CT provides excellent multiplanar reconstructions of the axial images hence allowing delineation of even the most subtle osseous changes. CT is superior to MRI for the detection of sequestra, cloacas, involucra, or intraosseous gas and can help in the guidance of needle biopsies and joint aspiration; furthermore, it is also valuable in cases of vertebral osteomyelitis.

Although CT may show changes earlier than plain radiographs do, CT is less desirable than MRI because of decreased soft tissue contrast as well as exposure to ionizing radiation."[68]

In 1995 E.C. Orpe, L. Lee and M.J. Pharoah studied 11 cases and when the CT scans were analysed, detection of sequestra improved from 45 to 91% which is feature of chronic osteomyelitis.[69]

A Vasil'ev et al in 2002 [70] "performed radiation studies in 121 patients of different age (4 to 75 years) for limb osteomyelitis. All the patients underwent routine X-ray study and computed tomography (CT), 26 patients had X-ray fistulography; 8, linear tomography; 10, CT fistulography; 6, scintigraphy, and 15, ultrasound study. Spiral CT has proven to be the most effective technique for diagnosing limb osteomyelitis as compared with routine X-ray study: the accuracy of computed tomography study was 96.7%, its sensitivity, 99.1%, and specificity, 80.0%."

In the year 2005 Termaat MF et al "in a systematic review and meta-analysis assessing the accuracy of different imaging techniques for the evaluation of chronic osteomyelitis, concluded that CT yielded a sensitivity of 0.67 with a 95% confidence interval (0.24 to 0.94), and specificity of 0.50 (0.03 to 0.97)."[71]

In 2016 La Fontaine J et al in "multi modality study of 110 patients who met the study's criteria: 52 CT patients and 58 MRI patients. The sensitivity, specificity, positive predictive value, and negative predictive value of CT were 89%, 35%, 74%, and 60%, respectively; and the corresponding values for MRI were 87%, 37%, 74%, and 58%, respectively. There were no significant differences in accuracy of diagnosing of between imaging techniques."[72]

William J. Jeffcoate in 2017 [73]found "in study of 166 patients that the sensitivity, specificity, PPV and NPV for CT were 87%, 37%, 74% and 60%. These data indicate that CT is little better than MRI for routine purposes; both have high

sensitivity but limited specificity leading to appreciable numbers of false-positive results."

MAGNETIC RESONANCE IMAGING

"MRI allows early detection of osteomyelitis and assessment of the extent of involvement and the activity of the disease in cases of chronic bone infection. MRI is highly sensitive for detecting osteomyelitis as early as 3 to 5 days after the onset of infection.(kocher) MRI advantages go far beyond diagnosis only, helping the surgeon to plan the optimal surgical management and to assess the extent of devitalized tissue, which contributes to the definition of the critical adjacent structures involved that would require modified management to avoid morbidity and complications. Metallic implants, however, may produce local artifacts that decrease image quality."[74]

In 1985 Michael T. Modic et al studied "multi modality radiology in patients suspected of having vertebral osteomyelitis. The sensitivity and specificity on MRI was 96% and 92%, bone scans it was 90% and 78% and plain radiographs 82% and 57% respectively."[57]

Evan Unger et al [75] in the year 1987 conducted "a study to evaluate the use of MRI in detection of osteomyelitis on 35 patients. The sensitivities of MRI and bone scintigraphy was 92% and 82% respectively while specificities were 96% and 65%."

In the year 1990 M. D . Cohen et al [76]studied "the differences in MRI findings of acute and chronic osteomyelitis in 17 patients. Abnormal MR signals were noted in all the patients. Chronic osteomyelitis was suggested by a good interface between normal and abnormal bone marrow (60%), cortical thickening +/- sequestrum (100%) and well defined boundaries to soft tissue abnormality (66%).

Poor interface between normal and abnormal marrow (75%), absent cortical thickening (100%) and poorly defined soft tissue abnormality (87.5%) suggested acute disease."

William B. Morrison et al "in their evaluation of 51 cases of suspected osteomyelitis in the year 1993 concluded that scintigraphy demonstrated a sensitivity of 61% and specificity of 33%. For non enhanced MR imaging sensitivity was 79% and specificity was 53%. For fat suppressed contrast enhanced imaging, sensitivity was 88% and specificity of 93%."[77]

In 1995 Diego Jaramillo et al concluded "from their study of 84 patients that MRI has a positive prediction value of 85% for osteomyelitis while scintigraphy had positive prediction value of 83%."[58]

In the year 2003 Michael Karchevsky et al [78] "studied 50 cases and described the MRI findings - was as follows: synovial enhancement (98%), perisynovial edema (84%), joint effusions (70%), fluid outpouching (53%), fluid enhancement (30%), and synovial thickening (22%). The marrow showed bare area changes (86%), abnormal T2 signal (84%), abnormal gadolinium enhancement (81%), and abnormal T1 signal (66%)."

U. Rozzanigo et al[79] concluded "in a study of sixteen diabetic patients who underwent foot MRI between January 2006 and September 2007 for suspected unilateral osteomyelitis that final diagnosis of osteomyelitis was made based on clinical, imaging, microbiological and histological findings, in 13/16 cases. Foot MRI allowed a correct diagnosis in 15/16 patients, with 1 false positive result demonstrated by computed tomography (CT)-guided bone biopsy."

Michael Karchevsky et al[80] concluded in his "study of 50 consecutive cases of bone infections evaluated by two observers for synovial enhancement, peri synovial edema, joint effusion, fluid out pouching, fluid enhancement, and synovial thickening. The marrow was assessed for abnormal signal on T1-and T2-weighted images or after contrast enhancement. MRI findings were compared with microbiologic, clinical, and surgical data and diagnoses. The frequency of MRI findings in septic joints was as follows: synovial enhancement (98%), perisynovial edema (84%), joint effusions (70%), fluid outpouching (53%), fluid enhancement (30%), and synovial thickening (22%). The marrow showed bare area changes (86%), abnormal T2 signal (84%), abnormal gadolinium enhancement (81%), and abnormal T1 signal (66%). Associated osteomyelitis more often showed T1 signal abnormalities and was diffuse. Abnormal marrow signal—particularly if it was diffuse and seen on T1-weighted images—had the highest association with concomitant osteomyelitis."

Asad Nawaz et al[81] concluded "in their study of 110 patients that FDG-PET ([18 f]-2-fluoro-2-deoxy-D-glucose (FDG)-positron emission tomography) correctly diagnosed osteomyelitis in 21 of 26 patients and correctly excluded it in 74 of 80, with sensitivity, specificity and accuracy of 81%, 93% and 90%, respectively. MRI correctly diagnosed osteomyelitis in 20 of 22 and correctly excluded it in 56 of 72, with sensitivity, specificity, and accuracy of 91%, 78% and 81% respectively. PFR (plain film radiography) correctly diagnosed osteomyelitis in 15 of 24 and correctly excluded it in 65 of 75, with sensitivity, specificity and accuracy of 63%, 87% and 81% respectively. Hence stating that MRI is very specific for diagnosing osteomyelitis in foot and can be complimented if study is done along with FDG-PET scan."

A Guha et al[82] concluded after their study of 34 were paediatric patients up to the age of 18 years that in the absence of specific diagnostic criteria, “Whole Body MRI in combination with clinical assessment can aid in the diagnosis of chronic recurrent multifocal osteomyelitis and Whole Body MRI has almost entirely replaced bone biopsy in the diagnosis of chronic recurrent multifocal osteomyelitis in their institution.”

Marie T. Dinh et al[83] conducted “meta-analysis for the accuracy of diagnostic tests for osteomyelitis in diabetic patients with foot ulcers. Pooled sensitivity and specificity, the summary measure of accuracy (Q*), and diagnostic odds ratio were calculated. Exposed bone or probe-to-bone test had a sensitivity of 60% and a specificity of 91%. Plain radiography had a sensitivity of 54% and a specificity of 68%. MRI had a sensitivity of 90% and a specificity of 79%. Bone scan was found to have a sensitivity of 81% and a specificity of 28%. Leukocyte scan was found to have a sensitivity of 74% and a specificity of 68%. The presence of exposed bone or a positive probe-to-bone test result is moderately predictive of osteomyelitis. MRI is the most accurate imaging test for diagnosis of osteomyelitis.”

Lauren W. Averill et al[84] conducted MRI studies of 78 skeletally immature children and adolescents (median age, 3.6 years) with suspected nonspinal osteomyelitis. “There was no significant difference between the sensitivity and specificity of unenhanced MRI ($p = 1.0$) and those of contrast-enhanced MRI ($p = 0.77$) for the diagnosis of osteomyelitis. Nonetheless, there was a significant ($p < 0.001$) increase in confidence in the diagnosis of osteomyelitis and its complications. This increase in confidence was most pronounced for the diagnosis of abscess (46%). Although it does not increase the sensitivity or specificity of the diagnosis, use of

contrast-enhanced MRI does increase reader confidence in the diagnosis of osteomyelitis and its complications in cases in which bone or soft-tissue edema is found on unenhanced images."

Jan Fritz et al[85] concluded in their study of 13 children with chronic recurrent multifocal osteomyelitis. "MR imaging depicted 101 ill-defined edemalike osseous lesions. Contiguous physeal relationship (89%, 66 of 74), periosteal reaction (48%, 48 of 101), and symmetric involvement (85%, 11 of 13) were present. MR imaging demonstrated multifocality in all patients. Sensitivity for radiography was 0.13 (70 of 119); physical examination, 0.31 (52 of 299); and serum inflammatory markers, 0.15 (two of 13).Whole-body MR imaging is useful for detection of CRMO, particularly in indeterminate cases, because it is more likely to show abnormalities."

In 2007 Susan A. Connolly et al [86] "advocated the use of MRI as the imaging technique of choice for any child suspected of having pelvic osteomyelitis through her study of 38 children as MRI had diagnosed more positive cases as compared to ultrasound and skeletal scintigraphy."

In the year 2007 Alok Kapoor et al studied Sixteen patients. "In all studies combined, the Diagnosing Foot Osteomyelitis (DOR) for MRI was 42.1 (95% confidence interval, 14.8-119.9), and the specificity at a 90% sensitivity cut point was 82.5%. The DOR did not vary greatly among subsets of studies. In studies in which a direct comparison could be made with other technologies, the DOR for MRI was consistently better than that for bone scanning (7 studies—149.9 vs 3.6), plain radiography (9 studies—81.5 vs 3.3), and white blood cell studies (3 studies—120.3 vs 3.4). Conclusion: found that MRI performs well in the diagnosis of osteomyelitis

of the foot and ankle and can be used to rule in or rule out the diagnosis. Magnetic resonance imaging performance was markedly superior to that of technetium Tc 99m bone scanning, plain radiography, and white blood cell studies."[87]

Erika McPhee et al in 2007 concluded from their study of 23 children suspected of having osteomyelitis that "Magnetic resonance imaging is a sensitive technique for evaluation of pyogenic infections involving the pelvis. MRI with gadolinium enhancement should be performed as an early study. Magnetic resonance imaging is also effective in identifying other conditions that may resemble pelvic osteomyelitis clinically."[88]

Lauren W. Averill in the year 2007 [84] evaluated "whether the use of fat-suppressed contrast-enhanced MRI, compared with unenhanced MRI alone, increases reader confidence in the diagnosis of osteomyelitis and its complications in 78 skeletally immature children and adolescents (median age, 3.6 years) with suspected nonspinal osteomyelitis. Osteomyelitis was clinically diagnosed in 40 cases (51%). There was no significant difference between the sensitivity and specificity of unenhanced MRI (p = 1.0) and those of contrast-enhanced MRI (p = 0.77) for the diagnosis of osteomyelitis. Nonetheless, there was a significant ($p < 0.001$) increase in confidence in the diagnosis of osteomyelitis and its complications. This increase in confidence was most pronounced for the diagnosis of abscess (46%)."

In the year 2009 Dalius Malcius et al [89] conducted a study involving 183 patients with the aim "to establish and compare diagnostic accuracy (sensitivity, specificity, and diagnostic odds ratio) of plain x-ray, ultrasonography, bone scintigraphy, computed tomography (CT), and magnetic resonance imaging (MRI) in pediatric acute hematogenous osteomyelitis. Acute hematogenous osteomyelitis was

diagnosed in 156 (85%) patients, and 27 (15%) had other diseases. A total of 169 early plain x-rays (median on the first day of hospital stay), 142 late x-rays (15th day of hospital stay), 82 ultrasonographies (second day), 76 bone scintigraphy (third day), 38 MRI scans (seventh day), and 17 CT (15th day) were performed. The sensitivity of ultrasonography was 0.55 (95% CI, 0.43–0.67); specificity, 0.47 (95% CI, 0.24–0.7); and diagnostic odds ratio, 1.08 (95% CI, 0.3–3.84). The sensitivity of CT was 0.67 (95% CI, 0.38–0.88); specificity, 0.5 (95% CI, 0.01– 0.98); and diagnostic odds ratio, 2.0 (95% CI, 0.02–172.4). The sensitivity of early x-ray was 0.16 (95% CI 0.1–0.23); specificity, 0.96 (95% CI, 0.78–1.0); and diagnostic odds ratio, 4.34 (95% CI, 0.63–186.3). The sensitivity of MRI was 0.81 (95% CI, 0.64–0.93); specificity, 0.67 (95% CI, 0.22–0.96); and diagnostic odds ratio, 8.67 (95% CI, 0.91–108.5). The sensitivity of late x-ray was 0.82 (95% CI, 0.75–0.88); specificity, 0.92 (95% CI, 0.62–1.0); and diagnostic odds ratio, 51.17 (95% CI, 6.61–2222.0). The sensitivity of bone scintigraphy was 0.81 (95% CI, 0.68–0.90); specificity, 0.84 (95% CI, 0.60–0.97); and diagnostic odds ratio, 22.30 (95% CI, 4.9–132.7). They concluded that late x-ray is the most valuable radiologic method in the diagnosis of acute hematogenous osteomyelitis, but bone scintigraphy and magnetic resonance imaging are the most valuable tests at the onset of the disease.”

Carlos Pineda et al in 2009 [(1)] stated from his research which was titled “"Radiographic Imaging in Osteomyelitis: The Role of Plain Radiography, Computed Tomography, Ultrasonography, Magnetic Resonance Imaging, and Scintigraphy" and concluded that conventional radiography should always be the first imaging modality to start with, as it provides an overview of the anatomy and the pathologic conditions of the bone and soft tissues of the region of interest. Sonography is most useful in the

diagnosis of fluid collections, periosteal involvement, and surrounding soft tissue abnormalities and may provide guidance for diagnostic or therapeutic aspiration, drainage, or tissue biopsy. Computed tomography scan can be a useful method to detect early osseous erosion and to document the presence of sequestrum, foreign body, or gas formation but generally is less sensitive than other modalities for the detection of bone infection. Magnetic resonance imaging is the most sensitive and most specific imaging modality for the detection of osteomyelitis and provides superb anatomic detail and more accurate information of the extent of the infectious process and soft tissues involved. Nuclear medicine imaging is particularly useful in identifying multifocal osseous involvement."

In 2013 Benjamin Matthew Howe et al studied "the T1-weighted magnetic resonance imaging (MRI) features associated with diabetic pedal osteomyelitis are present in histopathologically proven cases of non-pedal osteomyelitis in 75 patients and demonstrated T1-weighted imaging features typical of pedal osteomyelitis with a confluent region of decreased signal intensity, hypointense, or isointense relative to skeletal muscle in a geographic pattern with medullary distribution. Of the 5 cases that did not demonstrate the typical T1 features associated with pedal osteomyelitis, 4 were considered to have a hematologic mechanism of infection given the absence of surgery, skin ulceration, or a penetrating injury."[(90)]

C. Thévenin-Lemoin et al in 2016 did a pathophysiology study "to describe the MRI features of acute osteomyelitis. MRI revealed metaphyseal involved in all cases, characterised by an inflammatory signal with increased uptake after injection. The diaphysis was involved in 6 cases (29%), metaphysis in 13 cases (62%) and epiphysis in 10 cases (48%). A devascularized area was identified in 14 cases (67%)

and joint involvement in 10 cases (48%). A subperiosteal abscess was found in nine cases (43%) and a metaphyseal abscess in two cases (10%). Soft tissue extension was found in 20 cases (95%) with an abscess in two cases (10%).MRI has excellent sensitivity (98%) and specificity (92%) to detect acute osteomyelitis."[91]

In the year 2017 O¨ mer Kasalak et al tried "to evaluate the value of magnetic resonance imaging (MRI) signs in differentiating Ewing sarcoma from osteomyelitis and concluded that diagnostic accuracies were 82.4% and 79.4% for the presence of a soft-tissue mass, and 64.7% and 58.8% for a sharp transition zone of the bone lesion, for readers 1 and 2 respectively. Diagnostic accuracies of all other MRI signs were all < 50%."[74]

RADIONUCLIDE IMAGING

"Nuclear medicine imaging can detect osteomyelitis 10 to 14 days before changes are visible on plain radiographs. Several agents have been studied, which includ technetium-99m–labeled methylene diphosphonate (^{99m}Tc-MDP), gallium-67 citrate, and indium-111–labeled white blood cells. These are highly sensitive but have the inconvenience of low specificity. Consequently, it is difficult to differentiate osteomyelitis from other conditions such as crystal arthropathies, arthritis, fractures, neoplasia, or cellulites. Nuclear medicine scans may be a useful adjunctive study when x-rays are altered by pathologic or postsurgical changes." [92]

David L. Gilday et al in 1975 [93] observed in a study of 134 patients suspected of having Osteomyelitis "radionuclide diagnosis was correct in 70 out of 71 patients and no false positive in all of 43 normal patients. The typical appearance of osteomyelitis was a well-defined focus of increased radioactivity in the bone image.

The " blood pool" images were less valuable in the spine due to the underlying organs (liver and gut), but the bone image demonstrated the abnormal vertebral bodies involved in the diskitis-osteomyelitis, usually confirming diagnosis. The bone images were positive as early as 24 hours after the onset of symptoms, well before changes were evident in the radiographs. “

Littenberg B. et al in 1992 [(4)] “in a meta-analysis test performance of technetium bone scanning found that false-positive rate of the bone scan is at best in the range of 10 to 20%. This occurs at sensitivities between 70 and 80%. The studies with increased sensitivity also reported sizable increases in the false-positive rate ranging from 20 to over 90%. Even small increases in sensitivity have necessitated large sacrifices in specificity. Seven of the ten studies reported specificities under 70%. They concluded in many clinical situations, the specificity of the bone scan will not be high enough to confirm the diagnosis of osteomyelitis.”

Rubello D et al in 2004 performed scintigraphy in a series of 220 consecutive patients with suspected bone infection. “The sensitivity is ~91% with a specificity of 70%.” [(94)]

In 2007 Emilios E. Pakos et al performed “a meta-analysis of the sensitivity and specificity of antigranulocyte scintigraphy with monoclonal antibodies (MoAbs) in the diagnosis of osteomyelitis across different patient groups and clinical settings and concluded that Antigranulocyte scintigraphy with MoAbs has a sensitivity of 81% and a specificity of 77% in the diagnosis of osteomyelitis.”[(95)]

In 2012 Morbach H. et al [(96)] in a comparative study “to analyse sensitivity of bone scintigraphy using 99mTechnetium-labelled methylene diphosphonate (Tc-99m MDP) and magnetic resonance imaging (MRI) in the detection of chronic non-

bacterial osteomyelitis. Sensitivity of MRI compared to bone scintigraphy was superior in detecting lesions in the long bones of the thigh and the lower legs (100% vs. 78.4%, respectively). Therefore, depending on clinical relevance, MRI rather than planar bone scintigraphy should be considered for the detection of CNO lesions at diagnosis."

Shabana Saeed et al in 2013 [97] performed a study in 65 patients and stated that "the sensitivity, specificity and accuracy of (99m)Tc-UBI 29-41 scan in combination with three-phase bone scan for the diagnosis of osteomyelitis in diabetic foot was 100 %. Accuracy for soft-tissue infection was also 100 %."

In the year 2017 Petteri Lankinen et al concluded in his research on 40 patients that "F-FDG PET may help to confirm the presence of metabolically active infection in patients and guide their appropriate treatment which are culture negative on microbiology. Culture-negative cases may represent low-grade infections with a lower metabolic activity than culture-positive cases. F-FDG PET could potentially detect such a difference." [98]

Narjess Ayati et al in 2017 [99]performed a study to assess the diagnostic value of 99mTc-ubiquicidin scintigraphy in differentiating between osteomyelitis and bone tumors on 30 patients. They concluded that "99mTc-UBI scintigraphy in the dynamic imaging format was very useful with high accuracy in differentiating between infectious and tumoral lesions and showed the high accuracy of this noninvasive modality in acute osteomyelitis with low diagnostic value in chronic infectious processes. The sensitivity, specificity, negative and positive predictive value, and accuracy of the time–activity curve for osteomyelitis were 73.6 (54–93), 100, 66.6 (43–91), 100, and 82%, respectively."

RESULTS AND ANALYSIS

TABLE 3: DISTRIBUTION OF CASES ACCORDING TO AGE

AGE (YRS)	N	%
≤10	6	13.3
11-20	8	17.8
21-30	8	17.8
31-40	8	17.8
41-50	7	15.6
51-60	2	4.4
>60	6	13.3
Total	45	100

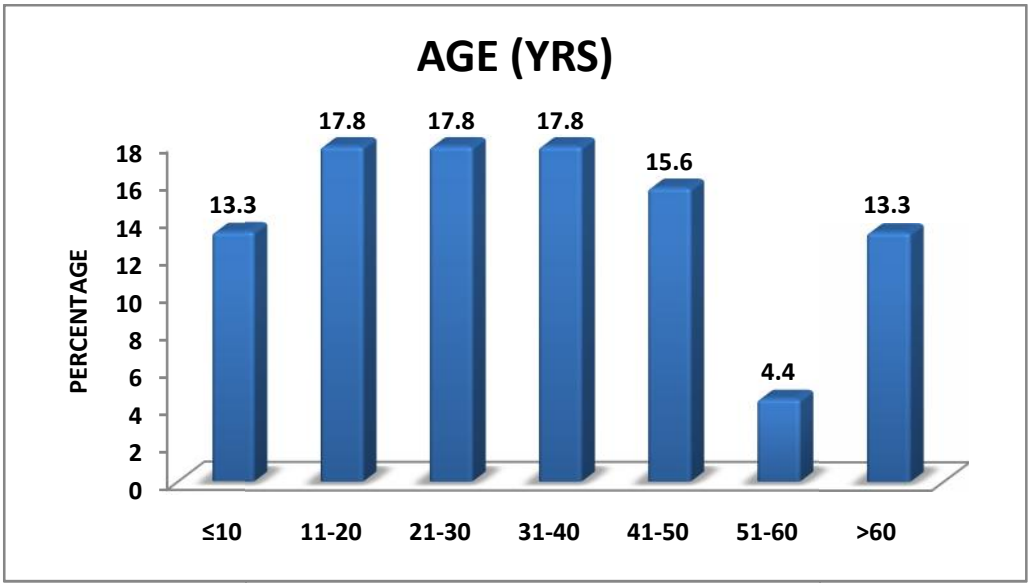
AGE (YRS)	RANGE	MEAN	SD
	3-80	34.0	21.0

The total number of cases studied were 45 in a time period of approximately two years.

Age distribution:

The age distribution of the patients in our study ranged from 3 to 80 years with the mean age being 34 ± 9.81 years.

FIGURE 6: DISTRIBUTION OF CASES ACCORDING TO AGE



Gender distribution:

All of the 45 cases studied 29 were males and 16 female subjects who came with clinical suspicion of osteomyelitis.

TABLE 4: DISTRIBUTION OF CASES ACCORDING TO GENDER

GENDER	N	%
MALE	29	64.4
FEMALE	16	35.6
Total	45	100

FIGURE 7: DISTRIBUTION OF CASES ACCORDING TO GENDER

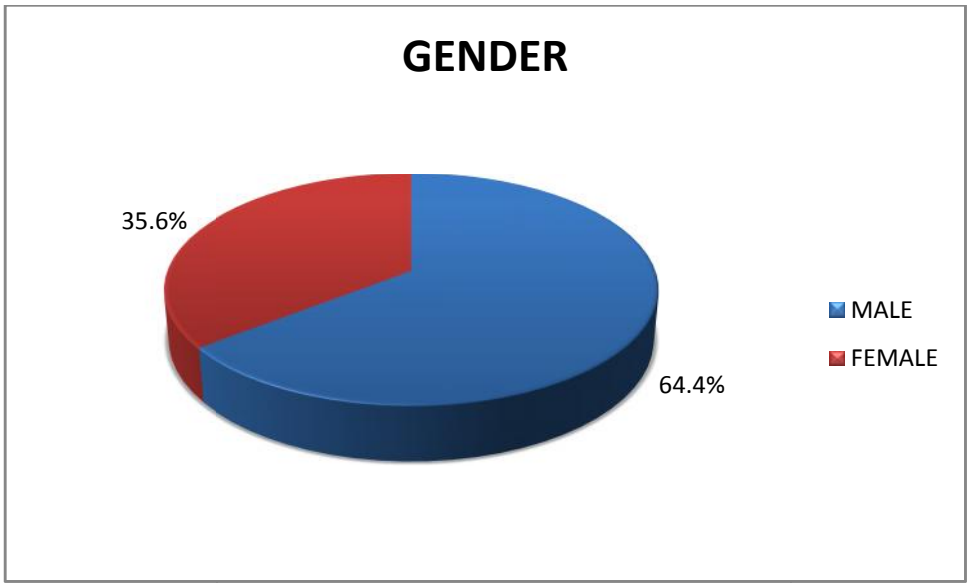


TABLE 5: ASSOCIATION OF AGE AND GENDER

AGE (YRS)	Male		Female		p value
	N	%	N	%	
≤10	5	17.2	1	6.3	0.247
11-20	7	24.1	1	6.3	
21-30	5	17.2	3	18.8	
31-40	5	17.2	3	18.8	
41-50	3	10.3	4	25.0	
51-60	0	0.0	2	12.5	
>60	4	13.8	2	12.5	
Total	29	100.0	16	100.0	

FIGURE 8: ASSOCIATION OF AGE AND GENDER

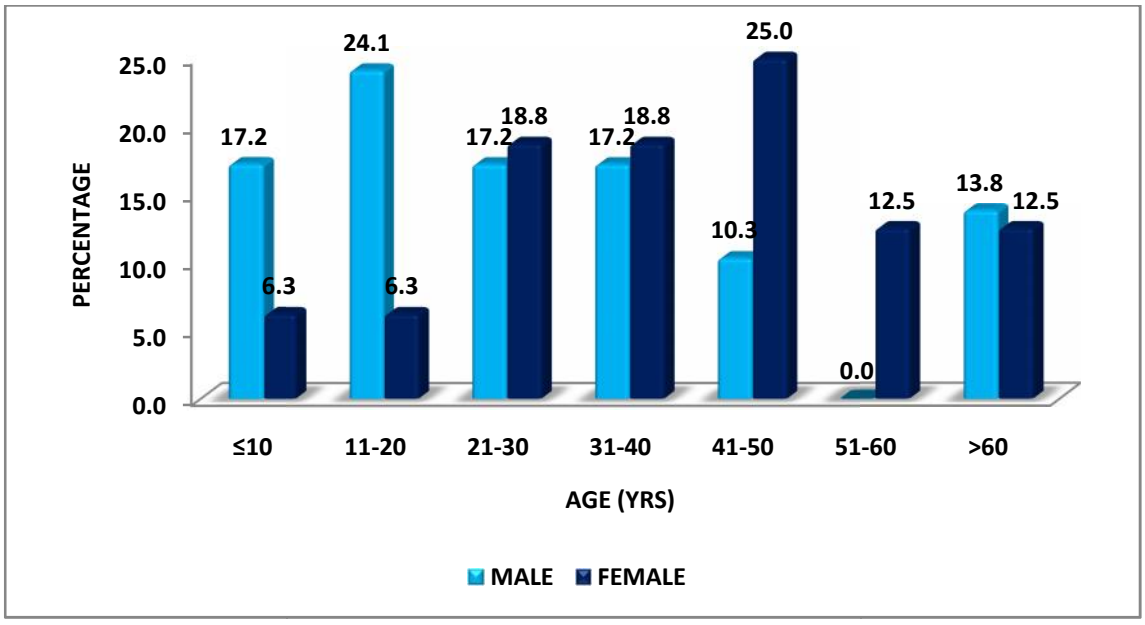
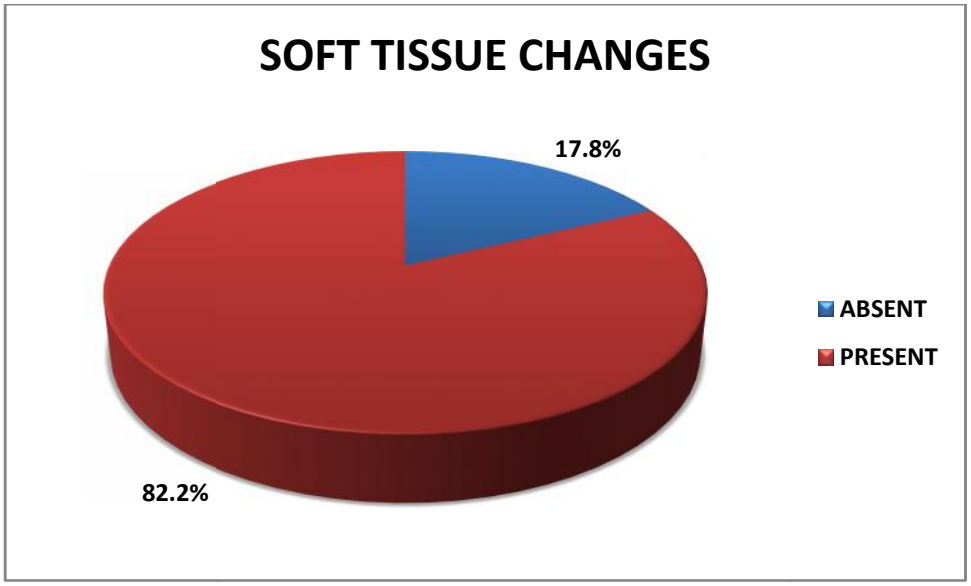


TABLE 6: DISTRIBUTION OF CASES ACCORDING TO SOFT TISSUE CHANGES

SOFT TISSUE CHANGES	N	%
ABSENT	8	17.8
PRESENT	37	82.2
Total	45	100

FIGURE 9: DISTRIBUTION OF CASES ACCORDING TO SOFT TISSUE CHANGES

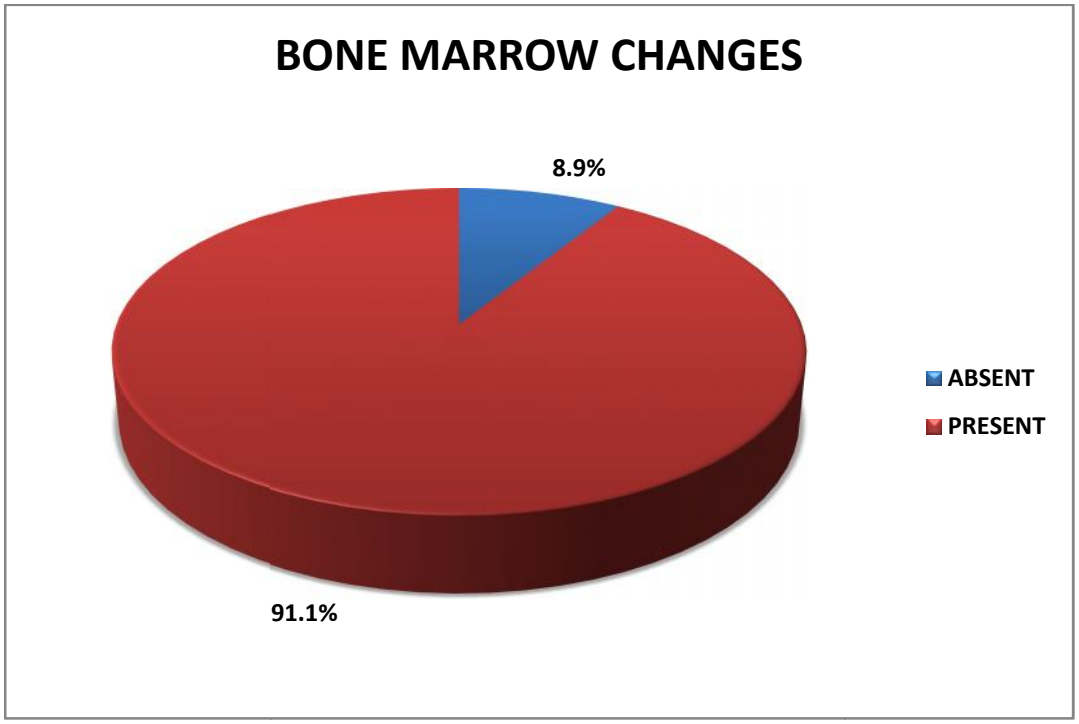


Among the 45 cases in our study 37 patients showed soft tissue changes in cross sectional MRI scans.

TABLE 7: DISTRIBUTION OF CASES ACCORDING TO BONE MARROW CHANGES

BONE MARROW CHANGES	N	%
ABSENT	4	8.9
PRESENT	41	91.1
Total	45	100

FIGURE 10: DISTRIBUTION OF CASES ACCORDING TO BONE MARROW CHANGES



Bone marrow changes were noted in 41 patients which amounts to 91.1% of the group as depicted in the pie chart and table above.

TABLE 8: DISTRIBUTION OF CASES ACCORDING TO FLUID

FLUID	N	%
ABSENT	6	13.3
PRESENT	39	86.7
Total	45	100

FIGURE 11: DISTRIBUTION OF CASES ACCORDING TO FLUID

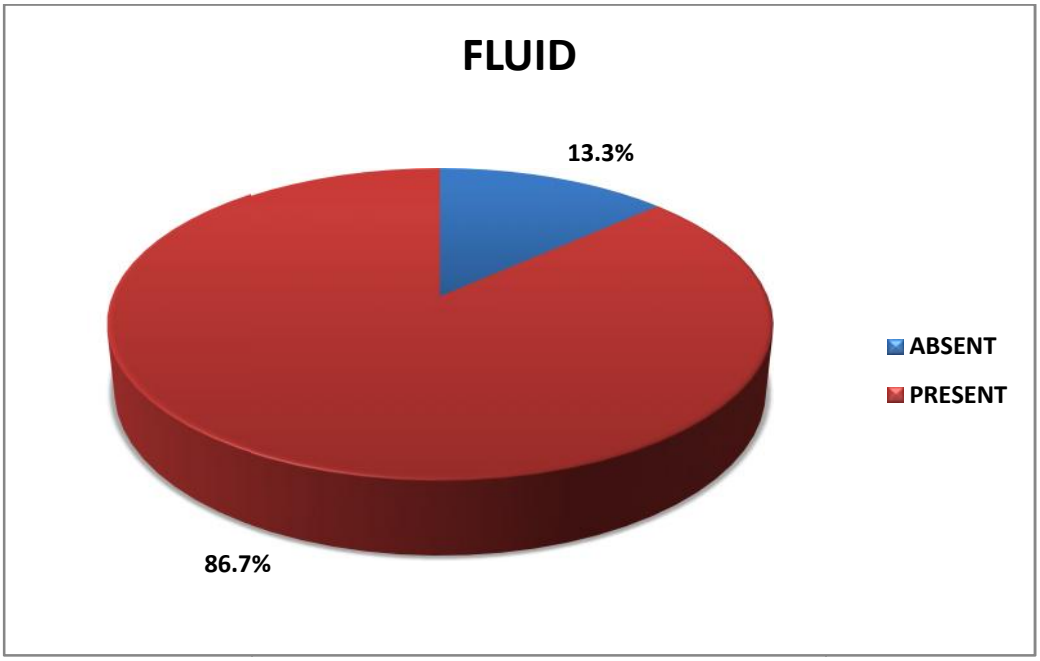


Figure showing the percentage of patients having fluid collection in the pathologically infected part.

TABLE 9: DISTRIBUTION OF CASES ACCORDING TO SURROUNDING BONE MARROW OEDEMA

SURROUNDING BONE MARROW OEDEMA	N	%
ABSENT	10	22.2
PRESENT	35	77.8
Total	45	100

FIGURE 12: DISTRIBUTION OF CASES ACCORDING TO SURROUNDING BONE MARROW OEDEMA

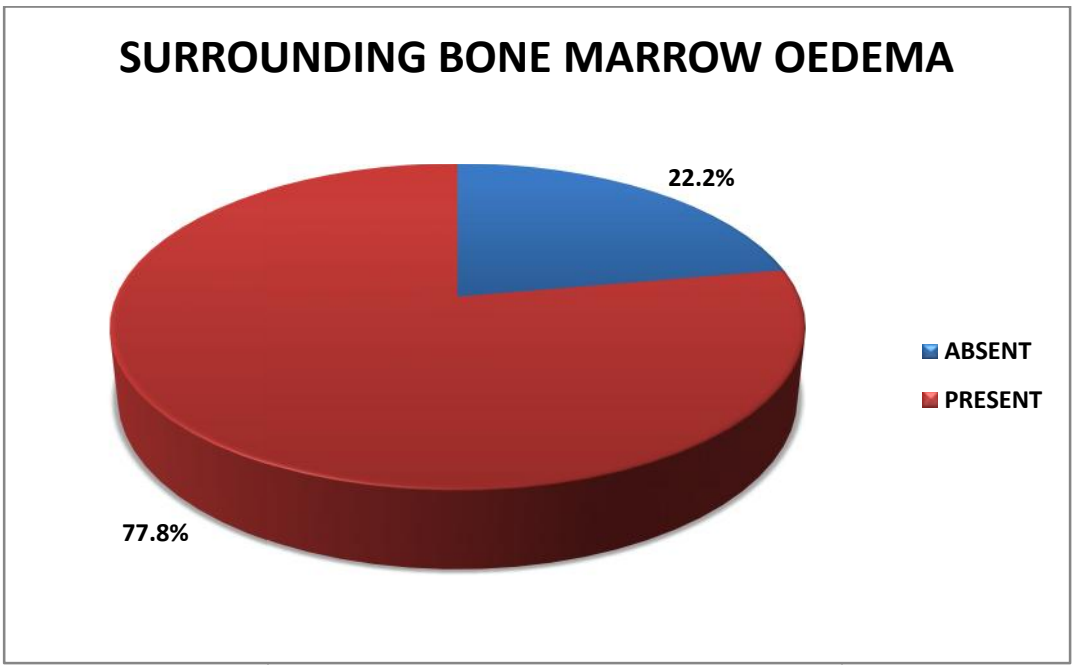


Figure and table showing the percentage of patients having bone marrow edema in the affected bone. 77.8 % (35) cases had positive findings while 22.2 % (10) showed normal bone marrow.

TABLE 10: DISTRIBUTION OF CASES ACCORDING TO INTRAOSSEOUS FINDINGS

INTRAOSSEOUS FINDINGS	N	%
ABSENT	20	44.4
PRESENT	25	55.6
Total	45	100

FIGURE 13: DISTRIBUTION OF CASES ACCORDING TO INTRAOSSEOUS FINDINGS

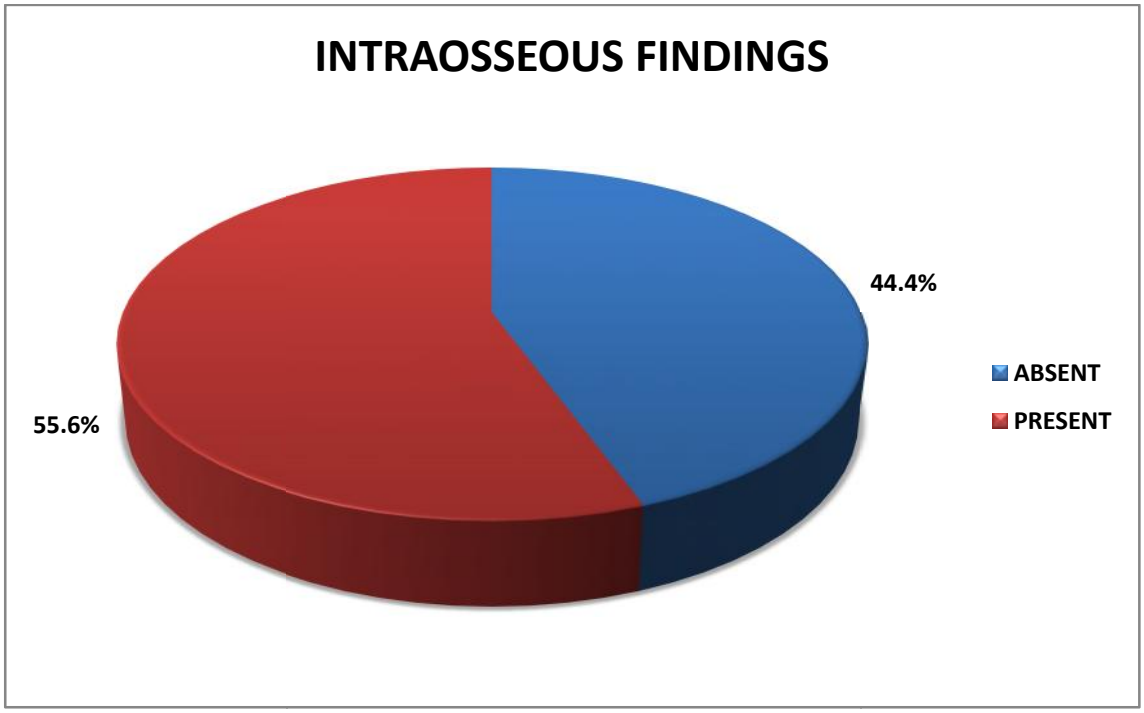
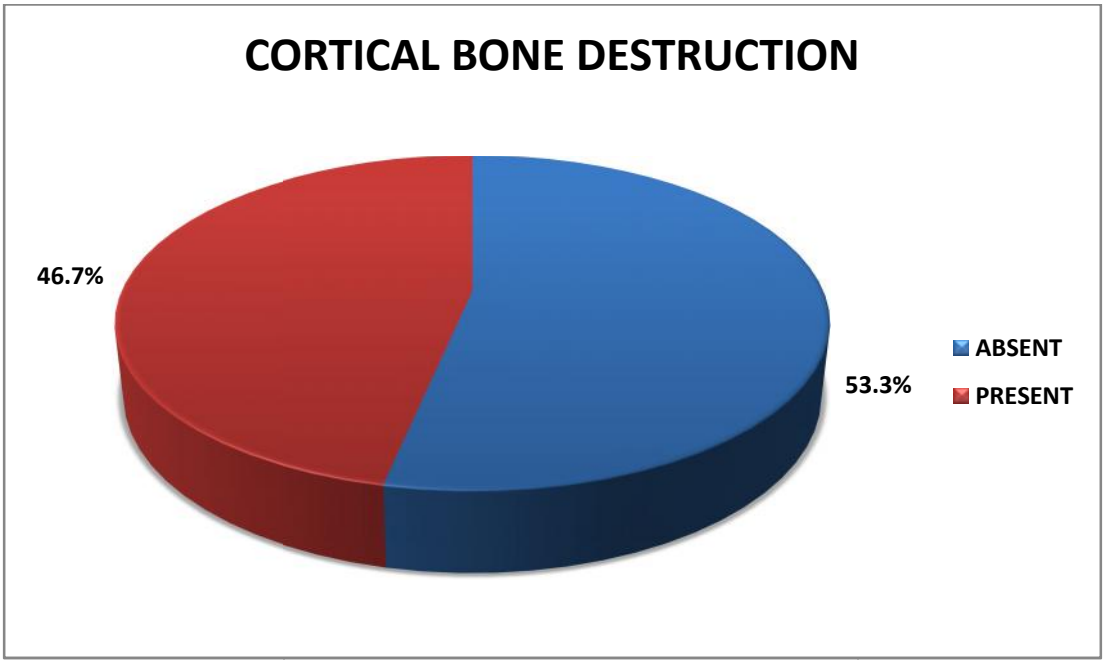


Figure and table showing the percentage of patients having intraosseous in the affected bone.

TABLE 11: DISTRIBUTION OF CASES ACCORDING TO CORTICAL BONE DESTRUCTION

CORTICAL BONE DESTRUCTION	N	%
ABSENT	24	53.3
PRESENT	21	46.7
Total	45	100

FIGURE 14: DISTRIBUTION OF CASES ACCORDING TO CORTICAL BONE DESTRUCTION



In our study cortical bone destruction was seen in 21 patients.

TABLE 12: DISTRIBUTION OF CASES ACCORDING TO POST CONTRAST ENHANCEMENT

POST CONTRAST ENHANCEMENT	N	%
ABSENT	9	20
PRESENT	36	80
Total	45	100

FIGURE 15: DISTRIBUTION OF CASES ACCORDING TO POST CONTRAST ENHANCEMENT

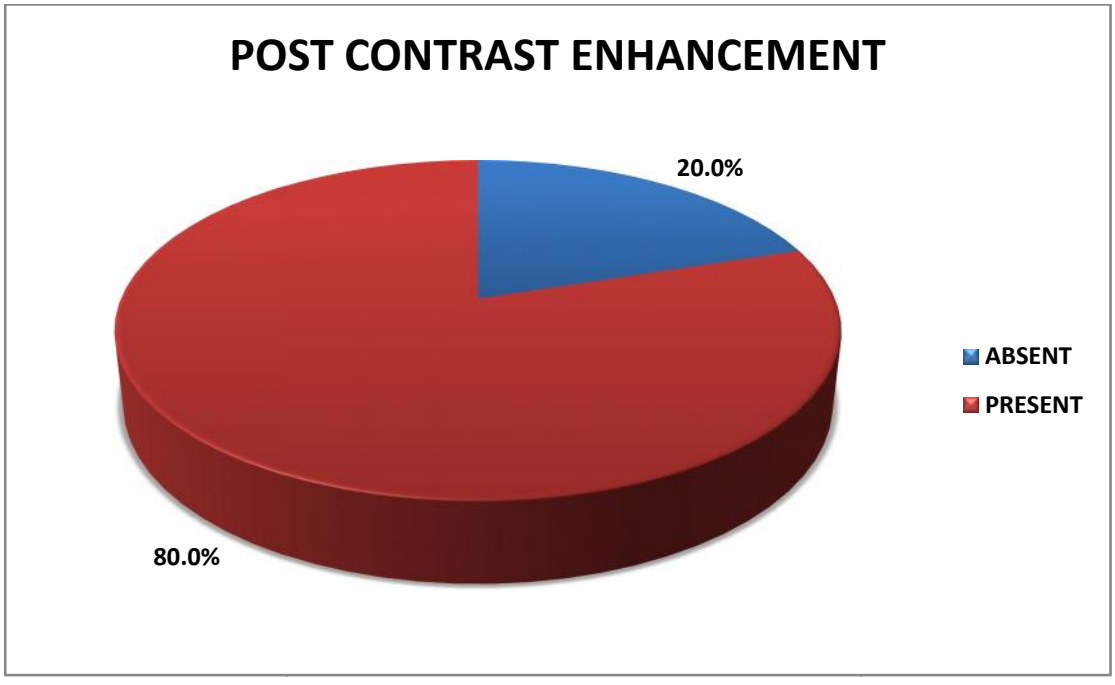
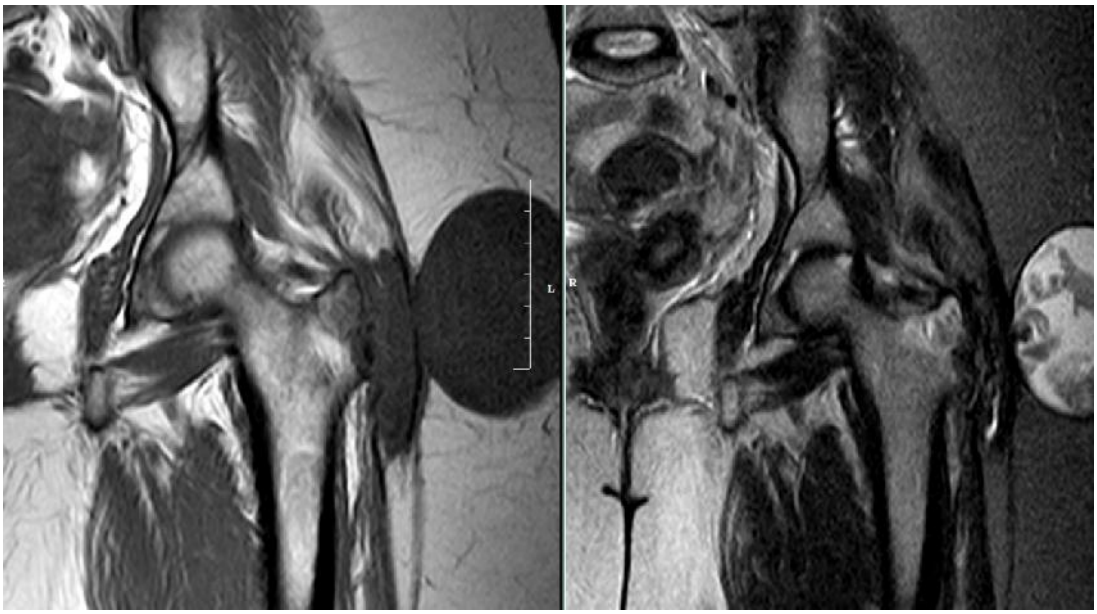


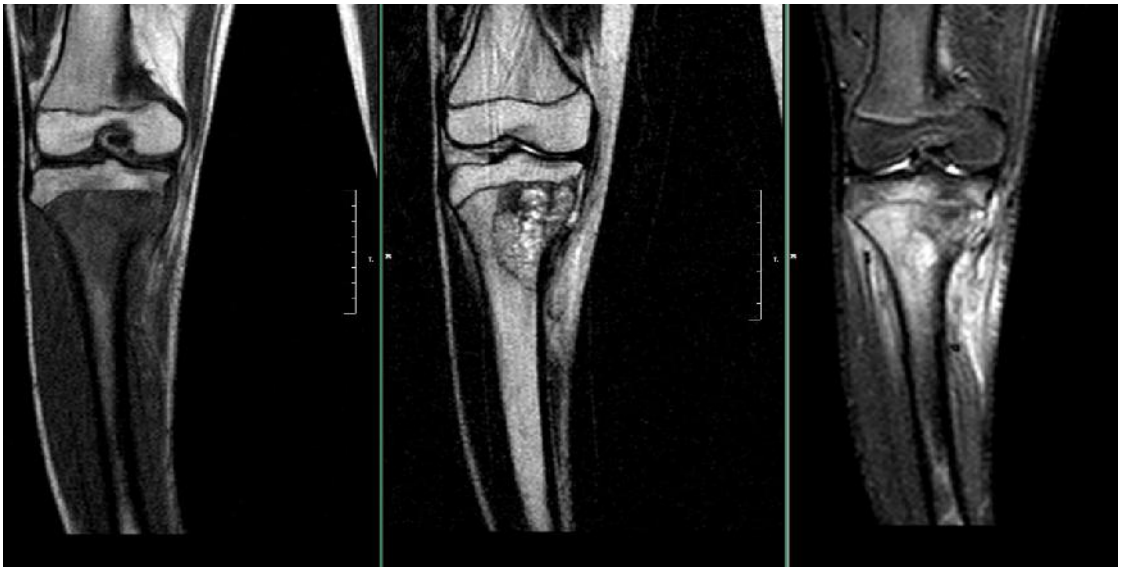
Figure and table depicting the number of patients showing subsequent enhancement after the administration of contrast.

IMAGING GALLERY

SOFT TISSUE CHANGES

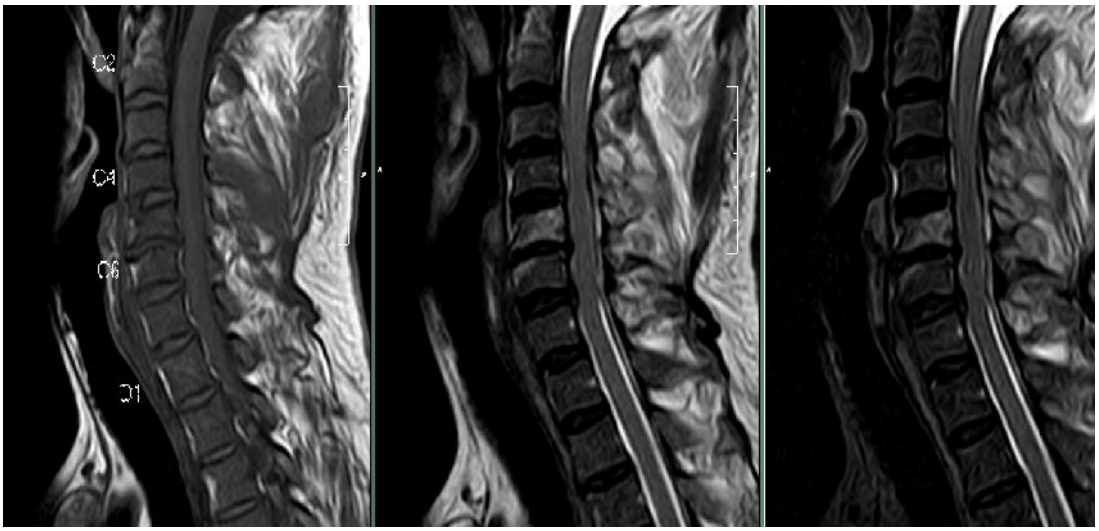


Above images shows ill-defined altered signal intensity (hyperintense on T2W and hypointense on T1W sequences) in the intramuscular compartment adjacent to greater trochanter of left femur – soft tissue changes.

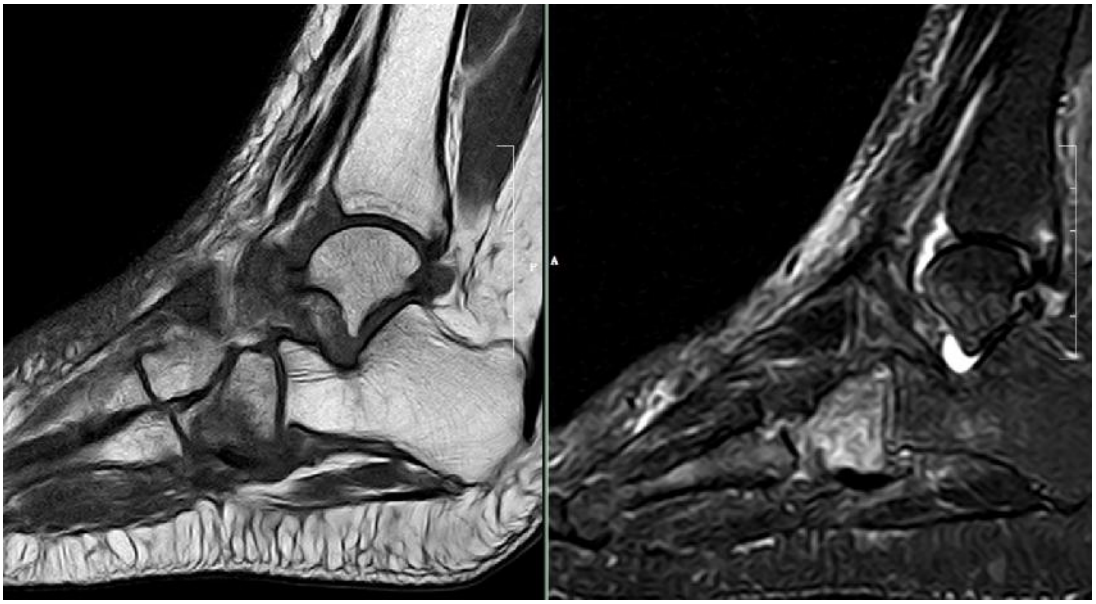


Above images shows ill-defined altered signal intensity (hyperintense on T2W and hypointense on T1W sequences) in the intramuscular compartment adjacent to proximal end of right tibia – soft tissue changes.

BONE MARROW CHANGES

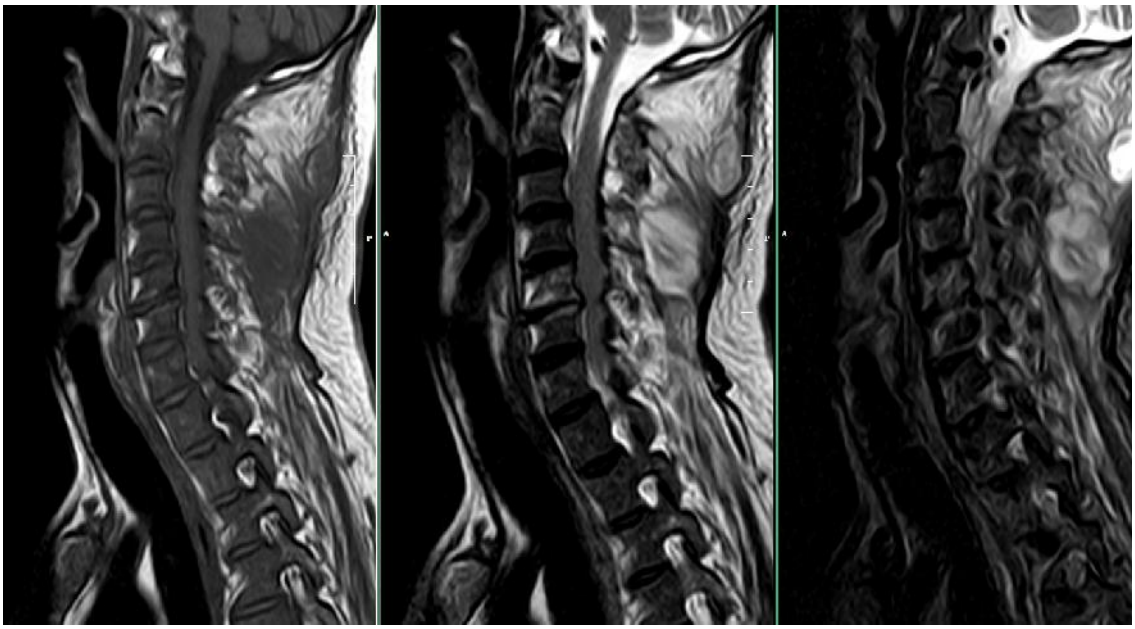


Above images shows altered signal intensity (hyperintense on T2W/STIR and hypointense on T1W sequences) in the C-5 vertebral body – bone marrow changes.

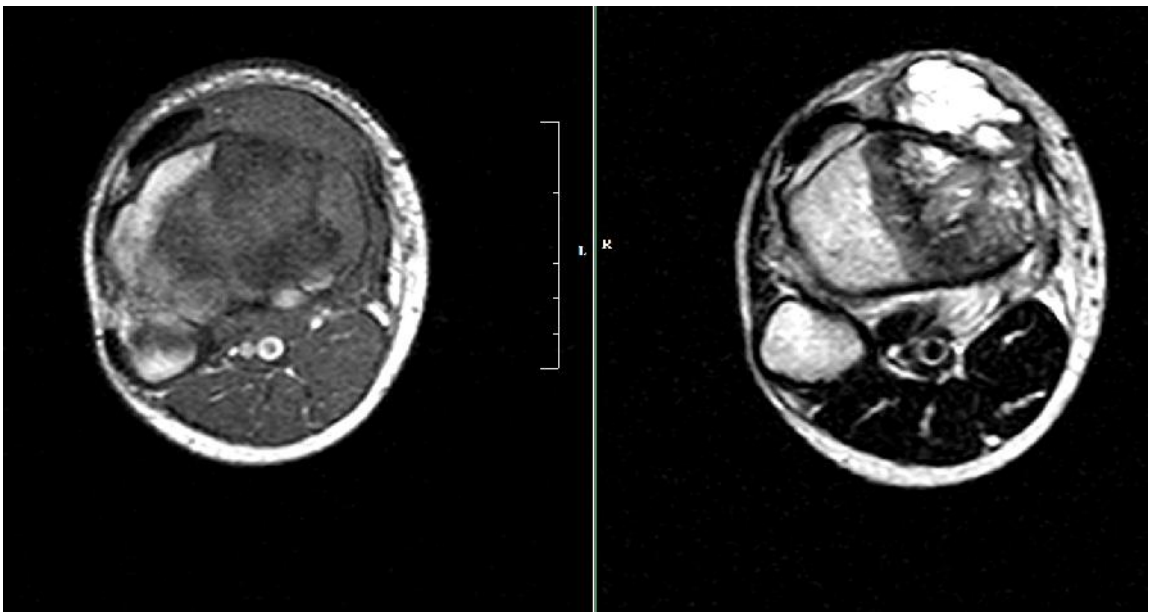


Above images shows altered signal intensity (hyperintense on STIR and hypointense on T1W sequences) in the cuboid – bone marrow changes.

PRESENCE OF FLUID

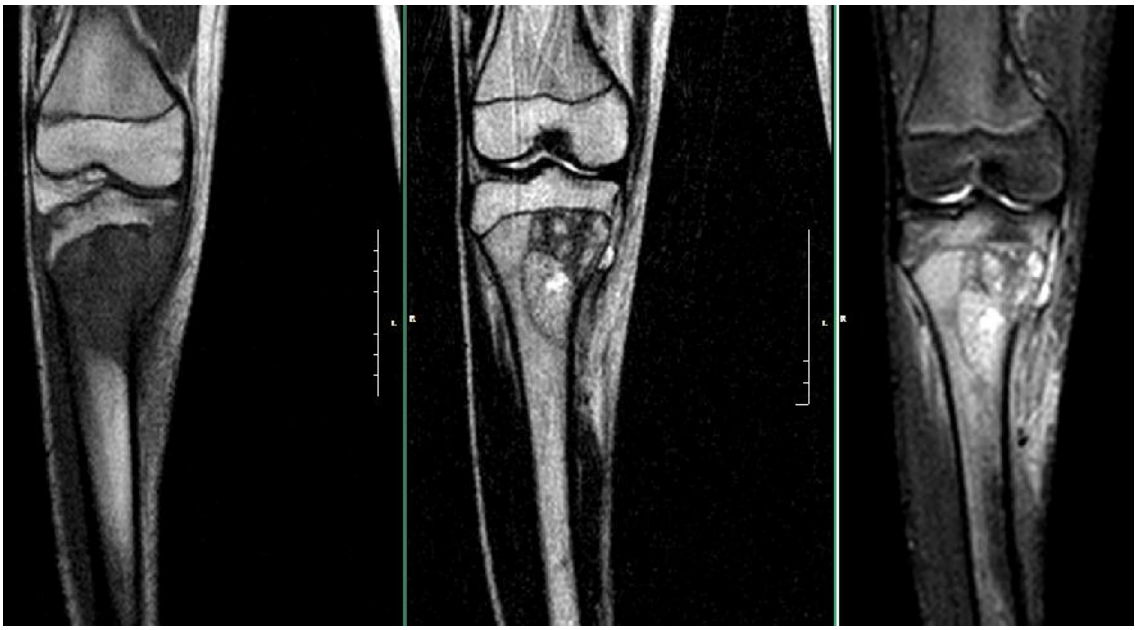


Above images shows ill-defined altered signal intensity (hyperintense on T2W/STIR and hypointense on T1W sequences) in the intramuscular compartment posterior to the cervical spine – likely fluid collection.

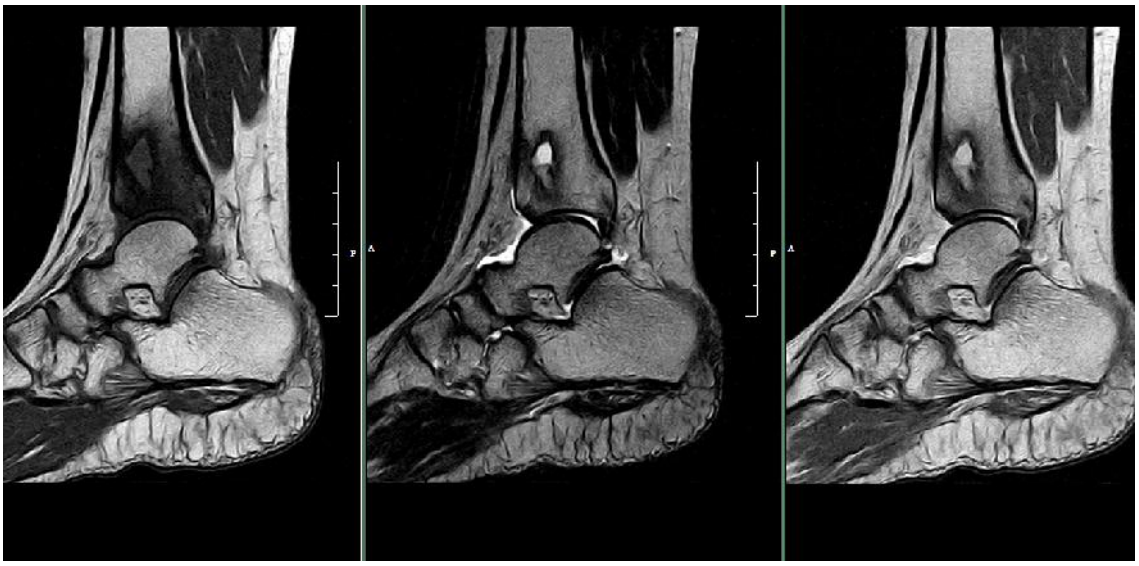


Above images shows ill-defined altered signal intensity (hyperintense on T2W and hypointense on T1W sequences) in the intramuscular compartment anterior to the right knee – likely fluid collection.

SURROUNDING BONE MARROW OEDEMA

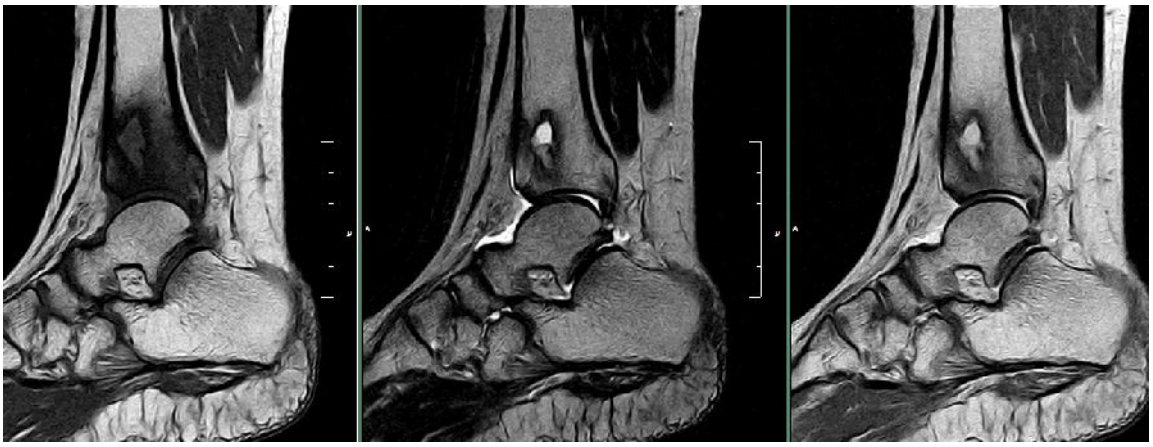


Above images shows ill-defined altered signal intensity (hyperintense on T2W/STIR and hypointense on T1W sequences) in the proximal end of right tibia – likely bone marrow edema.

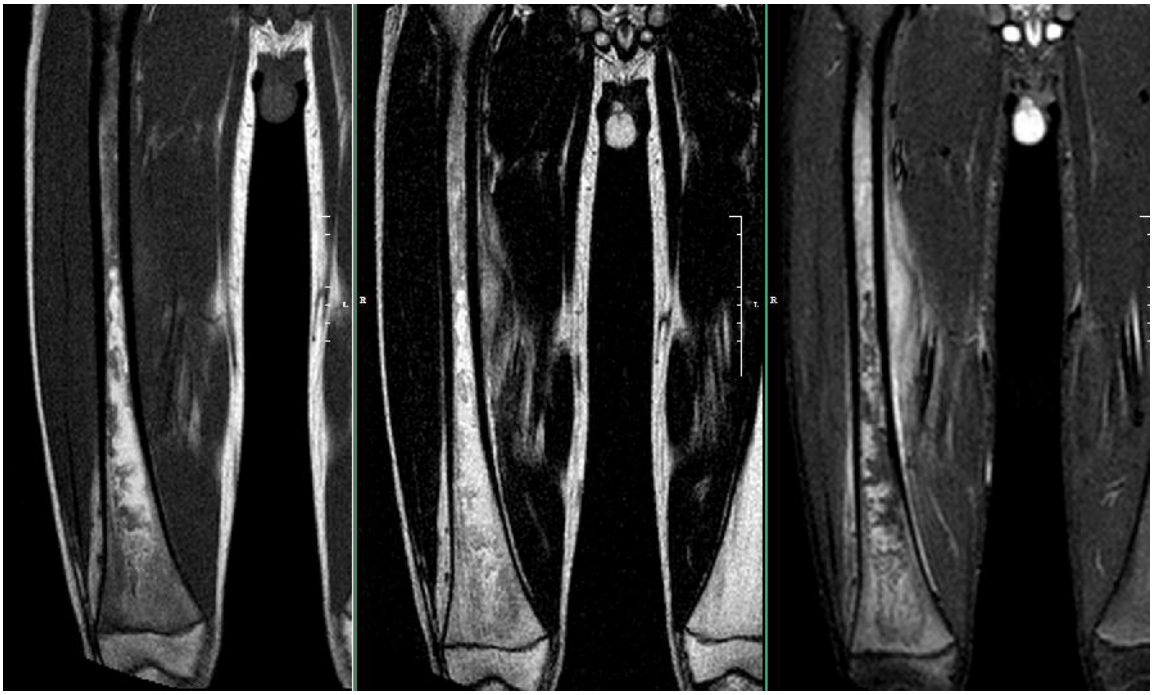


Above images shows ill-defined altered signal intensity (hyperintense on T2W/STIR and hypointense on T1W sequences) in the distal end of right tibia – likely bone marrow edema.

INTRAOSSEOUS FINDINGS

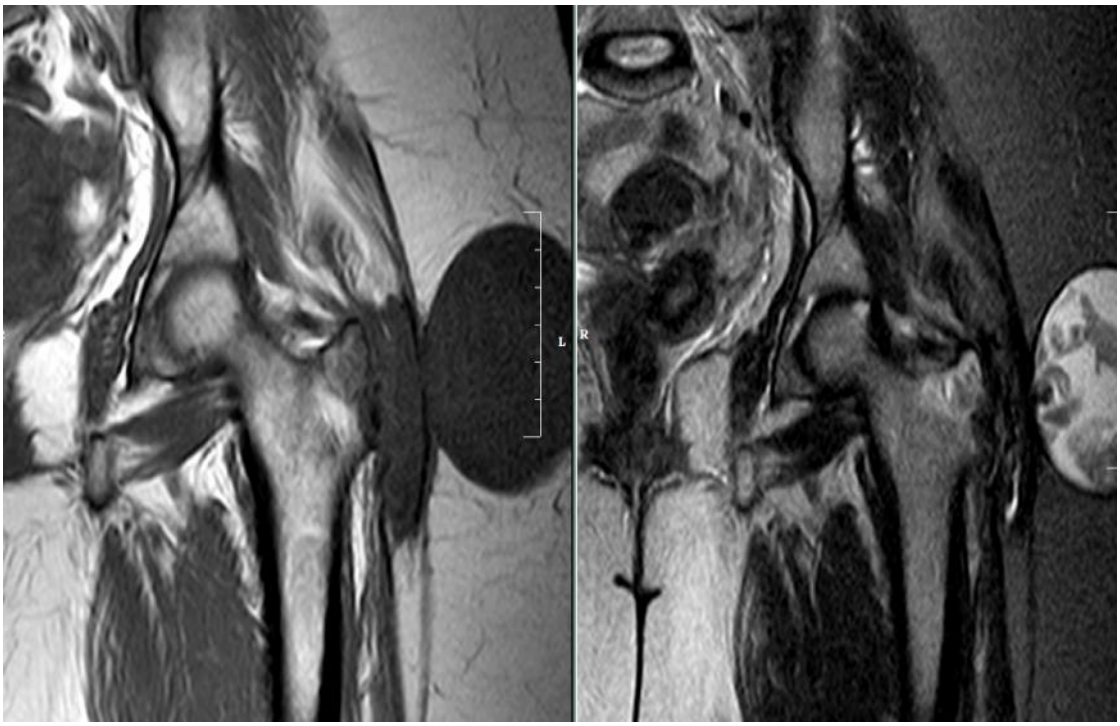


Above images shows well-defined altered signal intensity (hyperintense on T2W/STIR and hypointense on T1W sequences) in the distal end of right tibia – likely sequestrum.

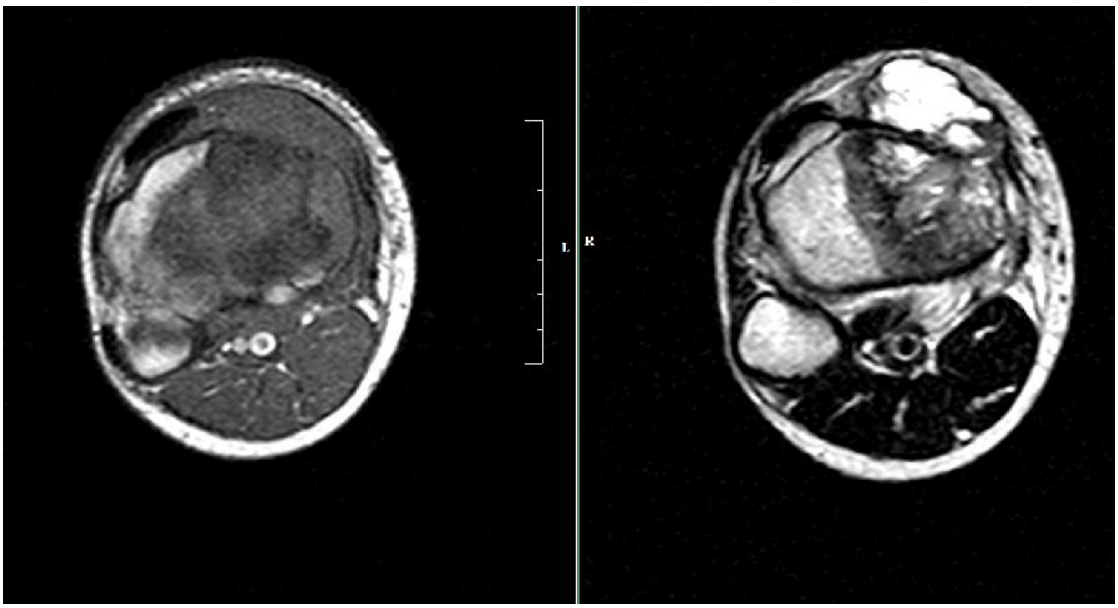


Above images shows well-defined altered signal intensity (hyperintense on T2W/STIR and hypointense on T1W sequences) in the shaft of right femur – likely sequestrum.

CORTICAL BONE DESTRUCTION

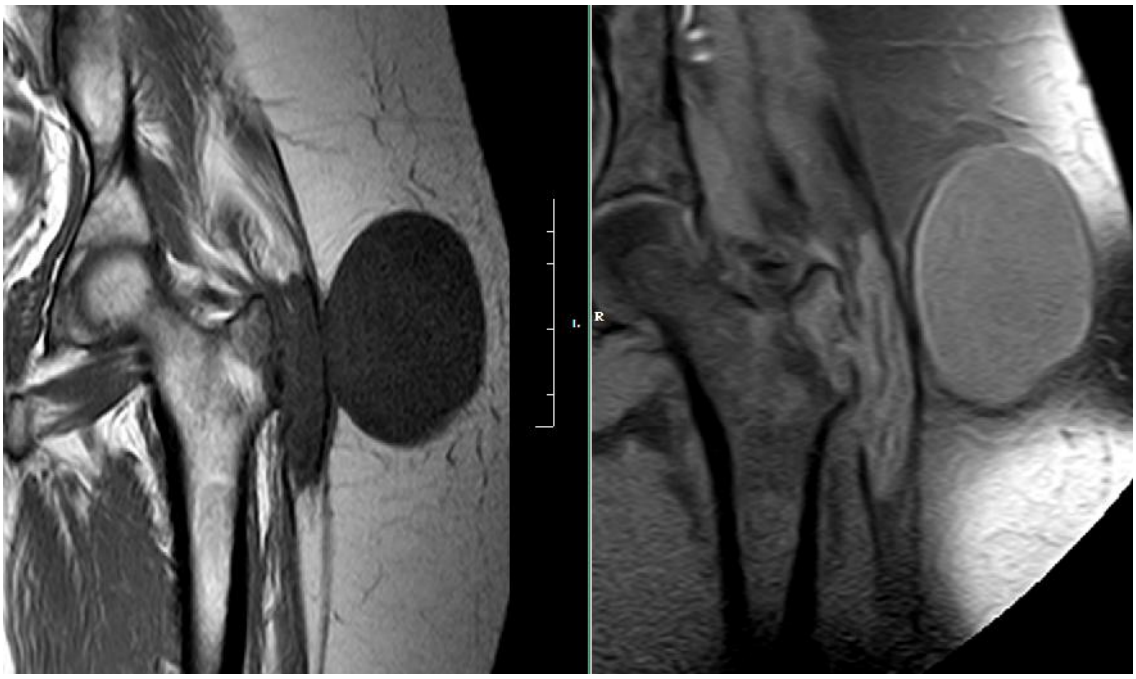


Above images shows ill-defined altered signal intensity (hyperintense on T2W and hypointense on T1W sequences) with discontinuity at the lateral margins of greater trochanter of left femur – likely cortical destruction.

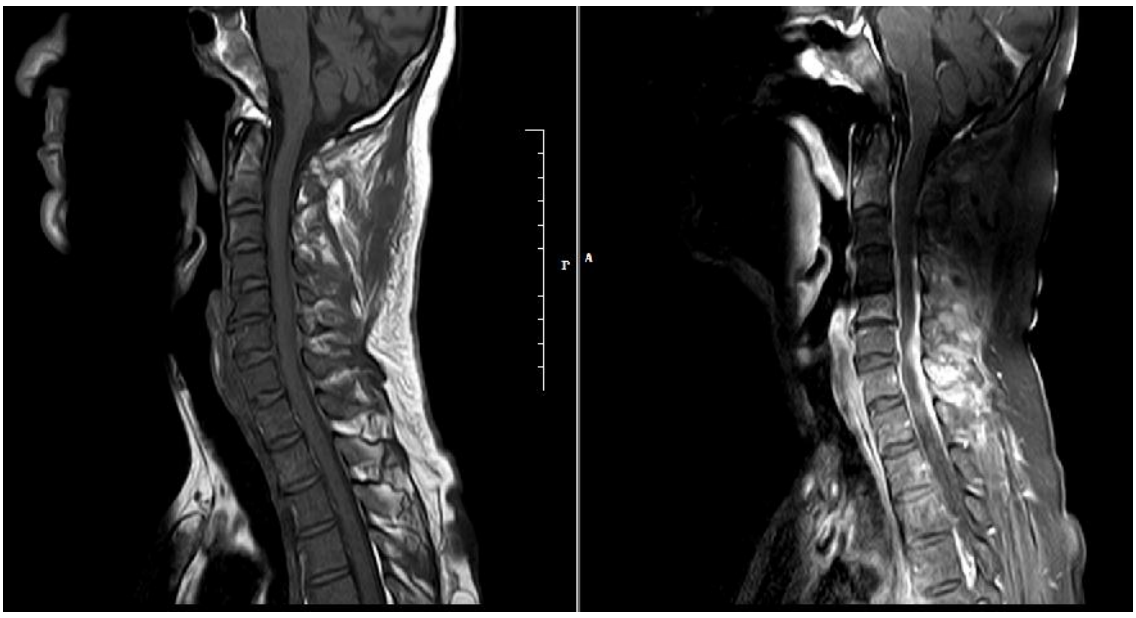


Above images shows ill-defined altered signal intensity (hyperintense on T2W and hypointense on T1W sequences) with discontinuity at the anterior margins of trochanter of right tibia– likely cortical destruction.

POST CONTRAST ENHANCEMENT



Above images shows ill-defined signal intensity (hypointense on T1W sequences) in the left greater trochanter and intramuscular compartment adjacent to it with hyperintensity on T1W after post contrast administration.



Above images shows ill-defined signal intensity (hypointense on T1W sequences) in the vertebral body and spinous process of C-5 and intramuscular compartment adjacent to it with hyperintensity on T1W after post contrast administration.

DISCUSSION

In this study 45 patients were evaluated by MRI scans who came to B.L.D.E. (Deemed to be university) Shri B.M. Patil medical college hospital and research centre with a clinical suspicion of Osteomyelitis using Philips Achieva 1.5 Tesla.

Magnetic resonance imaging (MRI) protocols used:

In suspected osteomyelitis, the affected area is imaged in axial, sagittal and coronal planes using multiple pulse sequences.

The typical sequences used in the evaluation of osteomyelitis are as follows:

- T1-weighted (T1W) sequences provide good anatomical detail and enable delineation of the medulla, cortex, periosteum and soft tissues.

- Fluid-sensitive sequences include T2-weighted (T2W), fat-suppressed (FS) and short-tau inversion recovery (STIR) sequences.

- Proton density-weighted (PD) sequences are intermediately weighted between T1 and T2.

T1-weighted post contrast Fat suppressed sequences to analyse the enhancement pattern.

C. Thévenin-Lemoine et al [91] in the year 2016 conducted a similar study involving 20 patients and using same MRI protocols as stated above to visualize osteomyelitis characteristics on MRI.

DEMOGRAPHICS:

Incidence of osteomyelitis with age and sex

In our study of 45 cases osteomyelitis was almost evenly distributed in patients of all age groups.

M. Beth McCarville et al [(59)] in their study have described a similar pattern with no age group predominance.

Øystein Rolandsen Riise et al[(100)] also concluded that there was no age predeliction in cases of osteomyelitis which matches the interpretation of our research.

In our study there was more predominance noted in males as 64.4% and females were only 35.6%.

In a similar study by L Grammatico-Guillon et al[(101)] stated that positive cases of osteomyelitis do have a male predominance (24.5 per 100000 vs 18.7 per 100000 in favor of boys).

Presence of soft tissue changes in cases of osteomyelitis:

In our study soft tissue changes were detected in 82.2% cases.

Edward T. Mah et al [(63)] in their study concluded the presence of soft tissue edema in around 91% of the subjects.

In a similar study C. Thévenin-Lemoine et al described the presence of soft tissue changes which appear hyperintense on T2W/STIR and hypointense on T1W sequences. They had 95% of cases which showed soft tissue changes.

Presence of bone marrow changes in affected bones in cases of osteomyelitis:

In our study bone marrow changes (hyperintense on T2W/STIR and hypointense on T1W sequences) were detected in 91.1% cases.

Erika McPhee et al[88] in their research stated the presence of bone marrow Altered signal intensities in almost 86% of subjects which is almost similar to our research.

Presence of adjacent fluid in cases of osteomyelitis:

In our study adjacent fluid collection to affected bones was detected in 86.7% subjects.

In a similar study conducted by Quamar AZAM et al[102] they stated the presence of fluid in cases of infective etiology in almost 76.3% cases.

Both of these studies show a similar pattern.

Presence of intraosseous findings in cases of osteomyelitis:

In our study of 45 cases intraosseous findings in affected bones were detected in 55.6% subjects.

Charalampos G. Zalavras et al[103] described the presence of intraosseous findings as an important finding in positive cases of osteomyelitis in around 60% of cases they studied.

Our study shows a similar pattern of occurrence.

Presence of cortical bone destruction in cases of osteomyelitis:

In our study of 45 cases cortical bone destruction in affected bones was noted in 46.7% subjects.

In a similar study by E.C. Orpe et al[69] 64% of their research subjects showed the presence of sequestra, which is an important characteristic of osteomyelitis and its chronicity.

Presence of post contrast enhancement in bones and adjacent soft tissue in cases of osteomyelitis:

Our study showed positive enhancement after post contrast administration in almost 80% of cases.

Hilary Umans et al[104] in their study of 11 cases presence of contrast enhancement was noted in 90% of cases.

Post contrast enhancement with presence of adjacent fluid, bone marrow changes, cortical bone destruction are highly indicative of osteomyelitis and could very well indicate the stage and chronicity of the disease.

SUMMARY

In our study series of 45 cases, we got 32 cases of infective etiology (osteomyelitis), 37 cases showed soft tissue changes, 41 cases had bone marrow changes, 39 cases in which fluid was present, 25 cases had intraosseous findings, 21 cases showed cortical bone destruction and 36 cases were showing contrast enhancement after administration.

The typical clinical presentation of osteomyelitis with pain, erythema and oedema of the affected part is nonspecific and can be caused by a multitude of other diseases.

There was overall male preponderance (64.4 %) with mean age 34 years in our study.

MRI is a revolutionary imaging modality that helps in evaluating the normal anatomical structures, normal variants, distribution features, localization and assessing the extent of various pathologies affecting the bone.

MRI is ideal for evaluation of Osteomyelitis.

CONCLUSION

Osteomyelitis often needs quite one imaging technique for associate correct diagnosing. The variable imaging look of osteomyelitis is also explained by the various morbid mechanisms concerned within the unfold of the infection and by the age-related organic process of bone.

Conventional radiography the primary imaging modality. It provides an outline of the anatomy and therefore the pathologic conditions of the bone and soft tissues of the region of interest.

Ultrasound represents a non-invasive technique to judge the concerned soft tissues and animal tissue bone and should offer steerage for diagnostic or therapeutic interventional, evacuation or tissue diagnostic test.

CT is also helpful within the analysis of chronic osteomyelitis, significantly in areas with a posh anatomy. CT could offer data concerning the presence of sequestra, cloaca, animal tissue destruction and reactive involucrum formation. Additionally, it's used for imaging-guided diagnostic test and aspiration of infectious material for microbiological examination.

MRI and Nuclear Medicine the foremost sensitive and specific strategies for the detection of osteomyelitis. MRI is that the most popular modality for early detection of osteomyelitis. The fat orb register T1–WI is pathognomonic for acute osteomyelitis while the shadow sign is pathognomonic for a Brodie symptom in acute osteitis. a mixture of T1– and Fat–Sat T2–WI and metallic element increased imaging is obligatory.

Advantages of MRI includes multiplanar capabilities and soft tissue contrast resolution which is superior to that of CT. Absence of ionizing radiation, and all compartments in and around the spine are displayed with MRI and unlike other studies. MRI also provides direct visualization of the disc and the spinal cord. Inflammatory changes can be detected early enough to affect patient outcome. Patient follow up is easy and accurate. Regression of abnormalities caused by surgical and/or medical therapy can be well documented. The routine sagittal and axial sequences employed are usually T1- and T2-weighted fast spin echo sequences. In addition, gadoliniumDTPA, T1 images are useful in differentiating inflammation from abscess, necrosis and bone sequestrae as a result of osteomyelitis and also in helping to localize and detect subtle areas of inflammation within the vertebral bodies as well which has proven to be difficult in conventional radiography.

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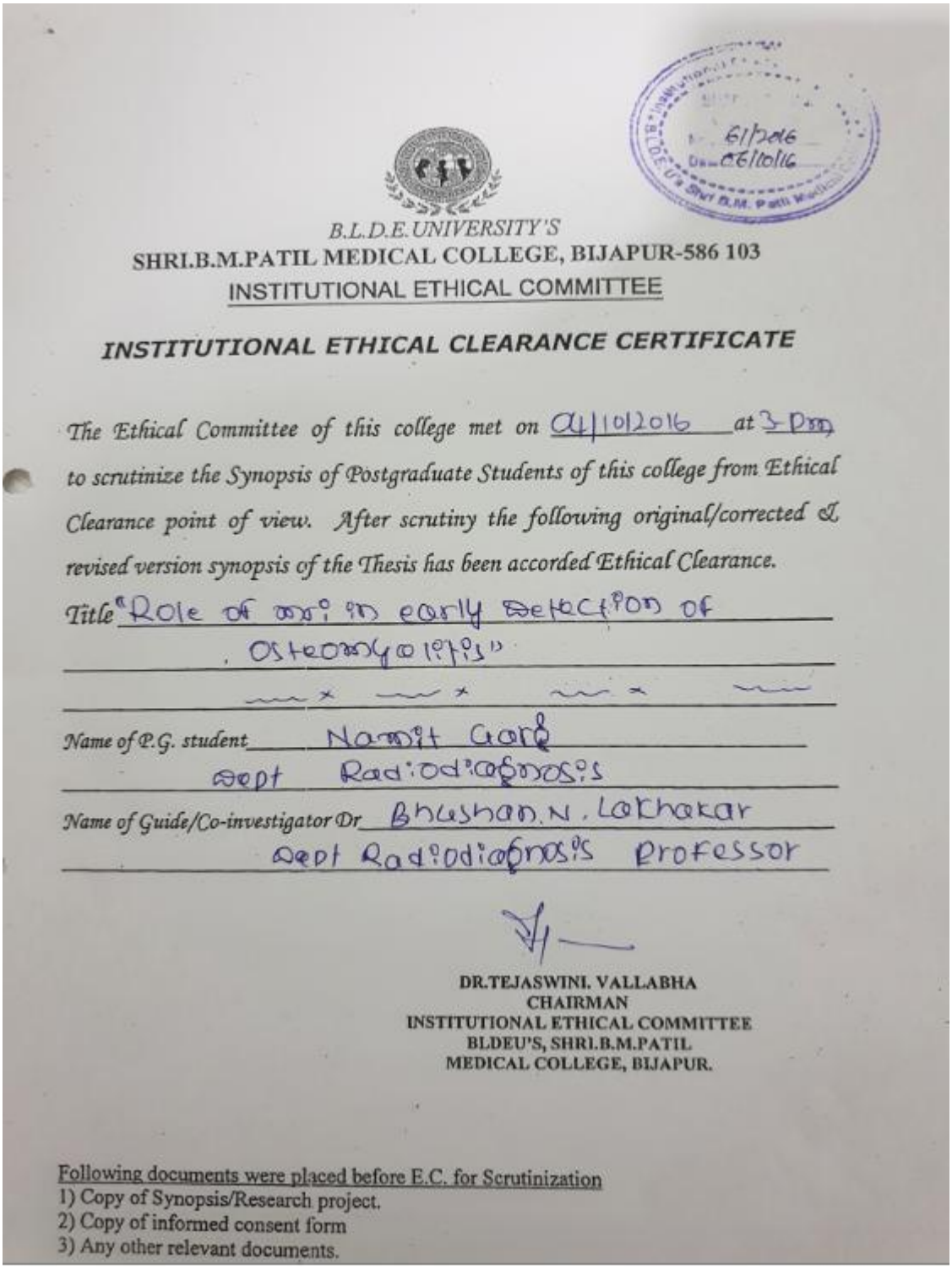
ANNEXURES

ETHICAL COMMITTEE CLEARANCE CERTIFICATE



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SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04/10/2016 *at* 3 PM *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "Role of MRI in early detection of Osteomyelitis"

Name of P.G. student Namit Garg

Dept Radiodiagnosis

Name of Guide/Co-investigator Dr Bhushan N. Lakhakar

Dept Radiodiagnosis Professor

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

CASE SHEET PROFORMA

1. Name:

2. Age:

3. Hospital No.:

4. Relevant complaints &history:

5. MRI findings:

- **Soft tissue changes-**
- **Bone marrow changes on MRI-**
- **Central high signal (fluid)-**
- **Surrounding bone marrow oedema-**
- **Intraosseous findings-**
- **Cortical bone destruction-**
- **Post contrast enhancement of bone marrow, abscess margins, periosteum and adjacent soft tissue collection-**

6. Post diagnosis follow up(if any):

CONSENT FORM

TITLE OF RESEARCH:

“Role of MRI in Osteomyelitis”

GUIDE : DR. BHUSHAN N. LAKHKAR

P.G. STUDENT : DR. NAMIT GARG

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to assess the role of MRI in the evaluation of Osteomyelitis.

PROCEDURE:

I understand that I will undergo history, clinical examination, MRI scan and follow up.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved and I may experience mild pain during the above mentioned procedures.

BENEFITS:

I understand that my participation in this study will help to assess the role of MRI in the evaluation of Osteomyelitis.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of hospital. If the data is used for publications the identity of the patient will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time

INJURY STATEMENT:

I understand in the unlikely event of injury to me during the study I will get medical treatment but no further compensations. I will not hold the hospital and its staff responsible for any untoward incidence during the course of study.

I have read the foregoing information, or it has been explained to me in my own language. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.

Name of Participant_____

Signature of Participant *Thumb print of participant*

Dr. Bhushan N Lakhar **Dr. Namit Garg**

(Guide) **(Investigator)**

KEY TO MASTER CHART

MRI	-	Magnetic Resonance Imaging
+	-	Present
-	-	Absent

MASTER CHART

SERIAL NO.	NAME	AGE (YEARS)	GENDER	HOSPITAL NO.	MRI FINDINGS						
					SOFT TISSUE CHANGES	BONE MARROW CHANGES	FLUID	SURROUNDING BONE MARROW OEDEMA	INTRAOSSEOUS FINDINGS	CORTICAL BONE DESTRUCTION	POST CONTRAST ENHANCEMENT
1.	A A PATIL	50	MALE	263879	-	+	+	+	+	-	+
2.	ADITYA	15	MALE	277320	+	+	+	+	+	+	+
3.	AKKUTAI	48	FEMALE	260480	+	+	+	+	+	-	+
4.	ANNAPURNA	53	FEMALE	240845	+	+	+	+	+	+	+
5.	ANUSHA	14	FEMALE	37676	+	+	+	+	+	+	+
6.	ARIFA	48	FEMALE	271319	+	+	+	+	-	+	+
7.	BABU	79	MALE	243020	+	+	+	+	+	+	+
8.	BISMILLA	33	FEMALE	866	-	+	+	+	-	-	+
9.	CHANDRAWWA	70	FEMALE	24948	+	+	+	+	+	+	+
10.	CHAYA	45	FEMALE	148299	-	+	-	-	-	-	-
11.	HANAMANTH	13	MALE	342086	+	+	+	+	-	-	+
12.	HANAMAVVA	45	FEMALE	16232	-	+	+	+	-	-	+
13.	HASANSAB	50	MALE	25092	+	+	+	+	+	+	+
14.	JAHANGEER	40	MALE	32251	+	+	+	+	+	+	+
15.	JYOTI	22	FEMALE	314790	+	+	+	+	+	+	+
16.	KHAJASAB	15	MALE	354404	+	+	+	+	+	+	+
17.	LAXMI	38	FEMALE	213273	+	+	+	+	-	-	+
18.	MALAPPA KADARI	70	MALE	135994	+	+	+	+	+	+	+
19.	MALLAMMA	80	FEMALE	148879	+	+	+	-	-	-	-
20.	MALLAPPA	70	MALE	231900	+	+	+	+	-	-	+
21.	MALLIKARJUN	13	MALE	248244	+	+	+	+	+	+	+

22.	MAREPPA	10	MALE	203181	+	+	+	+	-	-	+
23.	MD ISAK	34	MALE	40007	-	+	-	-	-	-	-
24.	MONESH	11	MALE	43798	+	+	+	+	-	-	+
25.	PARSHURAM	20	MALE	43082	+	-	-	-	-	-	-
26.	PRAKASH	30	MALE	13207	+	+	+	+	+	+	+
27.	RAFEEQ INAMADAR	28	MALE	220726	+	+	+	+	+	+	+
28.	RAHUL	31	MALE	145650	-	+	+	-	-	-	-
29.	RAJU	14	MALE	215302	+	+	+	+	+	+	+
30.	REVANSIDDA	24	MALE	124969	+	+	+	+	+	+	+
31.	SALEEMA	58	FEMALE	146130	+	+	+	+	-	+	+
32.	SAMARTH	3	MALE	173367	+	+	+	+	+	-	+
33.	SAMIK	3	MALE	197169	+	+	+	+	-	-	+
34.	SANDEEP	28	MALE	145443	+	-	+	-	-	-	-
35.	SHAKEERA	22	FEMALE	263953	+	+	+	+	+	+	+
36.	SHIVAKUMAR	10	MALE	382430	+	+	+	+	+	+	+
37.	SHREEDEVI	28	FEMALE	18050	+	+	+	-	-	-	+
38.	SHRIDHAR	7	MALE	34221	+	+	+	+	+	-	+
39.	SHRISHAIL	48	MALE	145733	-	-	-	-	-	-	-
40.	SHRISHAIL ALAGUR	40	MALE	146944	-	+	-	-	-	-	-
41.	SHRUTI	9	FEMALE	131630	+	+	+	+	+	-	+
42.	SIDDAPPA	37	MALE	26049	+	+	+	+	+	-	+
43.	SURESH	64	MALE	77245	+	+	+	+	+	+	+
44.	VISHWANATH	25	MALE	146582	+	-	-	-	-	-	-
45.	YALLAWWA	35	FEMALE	169716	+	+	+	+	+	+	+