CMMB
https://doi.org/10.31689/rmm.2025.32.3.221
REVIEWS

Clinical-Paraclinical Features of Multiple Myeloma with Bone Affection

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Abstract

Bone lesions are present in approximately 80–85% of patients with multiple myeloma at diagnosis. The most common sites of osteolysis include the spine (49–70%), ribs (45–50%), skull (35–50%), shoulder (20–35%), pelvis (30–40%), and long bones (13–35%). Bone destruction results from asynchronous bone turnover, characterized by increased osteoclastic resorption without proportional osteoblastic activity. A specific feature is the rare healing of lesions, even in complete remission.

Low-dose whole-body computed tomography is currently the gold standard for bone disease assessment in multiple myeloma, offering superior sensitivity and image quality compared to conventional radiography, with a 4–33% higher detection rate. PET-CT shows 90% sensitivity and 70–100% specificity and remains essential for identifying active lesions, monitoring bone disease progression, and evaluating response to therapy, including residual disease detection. MRI allows differentiation between healthy marrow and infiltrated tissue, identifies infiltration patterns and lesion morphology, detects early bone marrow involvement, and surpasses bone scintigraphy in identifying spinal lesions, particularly in unexplained vertebral compression fractures.

Keywords: multiple myeloma, bone disease, MRI, PET-CT

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INTRODUCTION

Multiple myeloma (MM) is a plasma cell malignancy that develops almost exclusively within the bone marrow and causes extensive skeletal destruction due to increased osteoclastic bone resorption coupled with suppressed bone formation[1].

According to the 2014 criteria of the International Myeloma Working Group (IMWG), bone lesions are considered pathognomonic for MM and are defined by the presence of one or more osteolytic lesions (≥5 mm), detected by computed tomography (CT), including low-dose whole-body CT, positron emission tomography combined with CT (PET-CT), or magnetic resonance imaging (MRI)[2,3].

Bone destruction increases the risk of pathological fractures and spinal cord compression, significantly reduces quality of life, mainly due to impaired functional capacity and bone pain and is associated with reduced survival and increased mortality. As the disease progresses, skeletal involvement becomes more severe, ranging from generalized osteoporosis to focal lytic lesions with fractures[4,5,6,7,8].

In this context, the aim of the present study is to provide a narrative synthesis of the most recent clinical and paraclinical data regarding bone lesions in patients with multiple myeloma.

MATERIALS AND METHODS

To achieve the stated objective, an initial search was conducted in the following scientific databases: PubMed, Hinari (Health Internet Access to Research Initiative), SpringerLink, NCBI (National Center for Biotechnology Information), and Medline. Article selection criteria focused on recent data regarding the etiology, pathogenesis, epidemiology, classification, and diagnosis of bone lesions in patients with multiple myeloma. The following keywords were used in various combinations: "bone disease in multiple myeloma," along with "etiology," "pathophysiology," "epidemiology," "symptomatology," and "diagnosis" to optimize the search output.

Advanced filtering was applied to refine the selection: full-text availability, English language, and publication date between 2000 and 2024. After a preliminary review of titles, original research articles, editorials, narrative reviews, systematic reviews, and meta-analyses were selected, provided they contained relevant information and up-to-date concepts on the etiology, pathogenesis, epidemiology, symptomatology, and diagnosis of bone lesions in multiple myeloma. Additionally, reference lists of selected sources were manually reviewed to identify further relevant publications not retrieved in the initial database search.

The information from the included publications was collected, categorized, assessed, and synthesized to highlight the main contemporary perspectives on the clinical and pathophysiological aspects of bone disease in MM.

To minimize the risk of systematic bias, extensive database searches were performed to identify the maximum number of relevant sources. Only studies meeting established validity criteria were included, and reliable exclusion criteria were applied.

When necessary, additional resources were consulted to clarify specific terms. Duplicate entries, articles unrelated to the study's objective, and sources without full text access were excluded from the final list of included publications.

RESULTS

Following the data processing from PubMed, Hinari, SpringerLink, NCBI, and Medline databases, and based on the defined search criteria, a total of 234 articles addressing the etiology, pathogenesis, epidemiology, symptomatology, and diagnosis of bone lesions in patients with multiple myeloma (MM) were identified. After an initial title screening, 38 articles were considered potentially relevant. Following a detailed review, 30 publications were ultimately selected as representative and included in the final bibliography of this narrative synthesis.

Articles that did not reflect the scope of the study, despite being retrieved by the search algorithm, as well as those inaccessible via Hinari or the scientific medical library of the "Nicolae Testemițanu" State University of Medicine and Pharmacy, were excluded.

Bone disease, one of the major complications of MM, is characterized by extensive bone loss and the development of osteolytic lesions, frequently resulting in pathological fractures. Bone lesions are present in approximately 80% of newly diagnosed symptomatic MM patients and in over 90% during the course of the disease. These complications are associated with significant morbidity and mortality. The axial skeleton, particularly the spine and proximal long bones, is

most commonly affected, although any bone may be involved. The spine is the most frequent site of osteoporosis, osteolysis, or vertebral compression fractures (VCFs) caused by MM. Patients face an increased risk of skeletal-related events (SREs), including pain, VCFs, spinal instability and cord compression, hypercalcemia, and pathological fractures requiring radiotherapy or surgical intervention.

Skeletal lesions impair mobility and daily independence, reduce survival and quality of life mainly due to limited physical function and bone pain, and contribute to increased treatment costs. Radiotherapy and surgery are often required. These factors collectively have a negative impact on quality of life and significantly reduce overall survival[4,5,6,7,8,9,10,11,12,13,14,15,16,33].

A vertebral fracture is defined as a reduction of more than 20% in vertebral body height from its original dimensions. Primary osteoporosis accounts for approximately 85% of VCFs, while secondary osteoporosis and malignancies account for the remaining 15%[17].

Nearly half of patients with bone involvement in MM will experience at least one skeletal-related event, which increases mortality risk by 20-40%. This highlights the clinical importance of bone fractures from diagnosis and throughout the disease course[15].

Despite continuous improvement in MM patient outcomes due to more effective therapies, bone complications remain a major issue. A substantial proportion of patients will develop bone lesions during disease progression. Given the significant burden these complications place on both patients and healthcare systems and their serious impact on quality of life prevention remains a clinical priority[7,8,14].

Unlike other metastatic tumors, the lytic process and bone destruction observed in multiple myeloma (MM) are not followed by new bone formation. Even when MM patients respond well to therapy, skeletal-related events may continue to progress without osteolytic lesion repair[15,18].

Vertebral compression fractures (VCFs) are frequent in MM and, according to several studies, occur in 34-64% of patients at the time of diagnosis. Spinal cord compression due to focal extramedullary localization develops in 5-10% of MM patients during disease progression. Significant predictors of VCFs include male sex, stage II or III according to the International Staging System (ISS), and the presence of back pain. Vertebral-specific factors such as lower Hounsfield Unit scores, lytic lesions, abnormal spinal alignment, and a high Spinal Instability Neoplastic Score (SINS) have also been shown to significantly increase the risk of VCFs[19].

The clinical presentation depends on the nature of spinal cord compression. Vertebral involvement may lead to spinal cord compression with severe pain, deformity, neurological deficits, spinal instability, and an increased risk of new fractures. Even in the absence of VCFs, MM can directly invade the spinal canal and cause spinal cord compression in up to 10% of patients. These changes have a major impact on both patients' quality of life and prognosis[8,20].

Epidural spinal cord compression occurs in up to 20% of MM patients. Pathogenetic mechanisms involve displacement and compression of the spinal cord, caused either by epidural invasion of neoplastic tissue from vertebral masses or by bone fragments from fractured vertebral bodies. Early diagnosis and treatment of spinal MM are essential to prevent permanent sensory or motor impairment[20].

Advances in spinal surgical techniques, radiotherapy, and medical oncology have significantly expanded therapeutic options and improved outcomes for MM patients experiencing spinal cord compression[19,32].

Epidemiology. Isolated bone lesions in multiple myeloma (MM) are observed in 5-10% of cases; however, the disease usually evolves into a systemic condition characterized by multiple lytic lesions. MM demonstrates a pronounced tendency to induce bone degradation in close proximity to malignant plasma cells, making osteolytic lesions a hallmark of the disease. Approximately 80-85% of MM patients develop bone lesions at diagnosis, which often persist throughout the disease course and may remain unresolved even in cases of complete treatment response. Osteolytic lesions may affect the entire skeleton, although their incidence varies by anatomical site.

According to the literature, the descending frequency of osteolysis localization is as follows: spine (49-70%), ribs (45-50%), skull (35-50%), shoulder (20-35%), pelvis (30-40%), and long bones (13-35%). Long bone fractures most commonly involve the proximal humerus and femur[2,4,5,12,21,22,23].

Fracture rates during the first year after diagnosis are up to 18 times higher than in the general population and remain elevated for up to 10 years[24]. In the absence of adequate therapy, over 50% of patients with stage III MM (Salmon-Durie classification) develop at least one skeletal complication within two years.

Osteolytic complications include long bone fractures most frequently affecting the proximal humerus and femur often following minor or atraumatic injury, and vertebral compression fractures (VCFs), which are associated with paraplegia in 11-24% of cases[12].

Due to the distribution of hematopoietic marrow, the spine is one of the most common sites of skeletal involvement[2,21]. Estimates of spinal involvement at diagnosis range from 70% to 100% of patients. VCFs are found in 55-70% of cases, particularly in the lumbar vertebral bodies, and they represent initial clinical signs in 34-64% of patients at disease onset. Furthermore, new vertebral fractures occur annually in approximately 15-30% of MM patients[5,11,25]. Although most myeloma lesions involve the vertebral body, they may also be identified in posterior spinal elements, such as the facets, pedicles, transverse processes, and spinous processes[26].

PATHOGENESIS AND PATHOPHYSIOLOGY

Normal bone homeostasis is maintained by a balanced and continuous remodeling process involving the coordinated activity of osteoclasts and osteoblasts. Bone formation is initiated by osteoblasts, while bone resorption is carried out by osteoclasts. As new bone is formed, osteoblasts differentiate into osteocytes. The hallmark of bone disease in multiple myeloma (MM) is the uncoupling of the bone remodeling process. The pathophysiology of MM-related bone lesions involves complex interactions between myeloma cells and the bone microenvironment. These interactions lead to increased osteoclast proliferation and activity, along with impaired maturation and suppressed function of osteoblasts, resulting in the inhibition of bone repair and enhanced resorption. This imbalance causes extensive bone resorption, diffuse osteopenia, focal osteolytic lesions, epidural mass formation, and pathological fractures with spinal cord compression, without evidence of typical bone repair or regeneration, thereby compromising patient health and quality of life[2,5,7,8,9,10,12,13,14,16,20,25,27,28].

MM plasma cells are believed to secrete factors that stimulate osteoclastogenesis while inhibiting osteoblastogenesis, promoting osteoclast mediated osteolysis and blocking osteoblast mediated bone repair. In addition to direct cell to cell interactions, various soluble factors have been identified in the MM microenvironment that promote osteoclast differentiation, further exacerbating bone destruction. Moreover, stromal and osteoclast-derived factors support MM cell proliferation by directly acting on malignant plasma cells and enhancing angiogenesis. Pathological fractures in MM patients result from lytic lesions, generalized bone loss, and/or increased bone turnover[16,27,28].

MM cells may also indirectly influence bone remodeling by stimulating other bone marrow microenvironment cells to release bone-modulating factors[16,27,28]. Thus, bone destruction in MM stems from asynchronous remodeling, where increased osteoclastic resorption is not balanced by adequate osteoblastic bone formation. A unique feature of MM bone lesions is their very limited healing potential, even in patients who achieve complete remission[16,18,27,28].

In addition to signaling abnormalities regulating osteoclast and osteoblast activity, direct invasion of bone remodeling compartments by MM cells has also been suggested to contribute to the disease pathophysiology. The process of bone destruction releases factors that enhance myeloma cell survival, creating a symbiotic relationship between bone resorption and tumor proliferation[14,27,28].

As our understanding of the pathophysiology of MM bone disease advances, therapeutic approaches will continue to evolve, offering more effective options for the management of MM associated skeletal lesions[18,28].

SYMPTOMATOLOGY

Patients with bone disease in multiple myeloma (MM) often experience diffuse bone pain, particularly in the sternum and pelvic regions. In 70% of cases, bone pain is the first reported symptom at disease onset. The osteopenic condition commonly culminates in pathological fractures over 50% of MM patients will develop fractures during the disease course, predominantly involving the vertebrae, ribs, pelvis, skull, and proximal segments of the humerus and femur[6,7,8,9,20]. In some cases, a pathological fracture may be the initial manifestation of MM[7].

Clinically, vertebral plasmacytoma presents with symptoms ranging from nonspecific back pain (47%), with or without relation to weight-bearing, to acute back pain associated with structural instability due to pathological fracture, and even symptoms of neurological compression. Rapid loss of vertebral height and fracture development occur. Skeletal lesions progress

through consecutive phases: bone infiltration, skeletal erosion due to activated osteoclasts, and eventually the formation of neoplastic lesions[2,7,8,11,13,21].

Clinically, 58% of MM patients report bone pain. Uncontrolled pain at the fracture site is considered the cardinal symptom of lytic bone lesions[12]. In addition to low back pain (46.8%), other general signs and symptoms include neurological disorders (compressive myelopathy, radiculopathy,etc.) (26.6%), weakness (82%), weight loss (24%), and recurrent bleeding or infections (13%)[11]. According to several authors, up to 70% of primary complaints at the initial presentation of MM involve lumbar pain[25,34].

Spinal cord compression occurs in up to 5% of MM cases, with common symptoms including back pain (83%), motor weakness, sensory disturbances, and lower limb paresis. Bladder and bowel dysfunction are late-stage findings and rarely occur in isolation[9,29]. Spinal cord compression affects two-thirds of patients with solitary plasmacytoma involving the spine, mainly due to vertebral fracture and collapse, or less commonly, direct tumor compression of neural structures. However, this incidence decreases to 7-16% in MM cases[21,29]. Epidural mass formation may compress the spinal cord and lead to paralysis in approximately 10% of MM patients[25].

DIAGNOSIS

The clinical heterogeneity and disease burden of multiple myeloma (MM), with potentially severe consequences on patients' quality of life, require prompt diagnosis and appropriate treatment through a multidisciplinary assessment involving hematologist-oncologists, radiotherapists, and anesthesiologists[2,21].

Initially, the lesion may be solitary (plasmacytoma), but as the disease progresses, it often evolves into multiple lytic bone lesions[6,25].

Physical examination should include range-of-motion tests to assess the impact of bone lesions on mobility and function, as well as identification of pain points or suspected fractures. Evaluation of spinal alignment and posture is also important, as vertebral compression fractures (VCFs) are frequent and may lead to spinal deformities or symptoms of neural compression (paresthesia or weakness)[2].

Laboratory tests for bone evaluation in MM patients include measurements of calcium, vitamin D, fractionated alkaline phosphatase, and creatinine[5].

Imaging plays a critical role in diagnosing MM-related bone disease. Conventional radiography, computed tomography (CT), magnetic resonance imaging (MRI), and positron emission tomography-computed tomography (PET-CT) are used to comprehensively assess bone and soft tissue involvement in MM[26,30].

Conventional skeletal survey is no longer recommended due to its low sensitivity, which can result in missed detection of up to 25-50% of lytic lesions compared to low-dose whole body CT (LDWBCT). Nevertheless, it remains the "gold standard" for initial staging in newly diagnosed or relapsed MM patients in resource-limited settings. Typical findings on radiographs include areas of osteolysis without marginal sclerosis, a "punched-out" or "moth-eaten" skull, "spindle-shaped" ribs, pathological fractures, vertebral compressions, and collapse[5,6,7,8,10,12,13,14,22,23,26,30].

Low-dose whole body CT is currently the "gold standard" for the diagnosis of MM related bone disease. CT provides higher sensitivity, superior image quality, and a 4-33% higher detection rate for lytic lesions compared to standard radiographs[5,6,7,8,10,12,13,14,22,23,26,30]. It offers high resolution images with excellent tissue contrast, allowing effective identification of bone destruction, sclerosis, and soft tissue extension. However, its inability to distinguish metabolically active from inactive lesions limits its use in evaluating treatment response[2,5,6,7,8,12,14,23,26,30].

PET-CT is particularly effective in monitoring asymptomatic myeloma or solitary plasmacytoma and can distinguish between active and inactive disease, assess residual disease activity, and identify the risk of skeletal complications. It is especially useful in patients with contraindications to MRI, although it fails to detect lesions smaller than 0.5 cm. While PET-CT has high sensitivity, its specificity is relatively low[2,5,6,7,8,12,14,22,23,26].

Unlike CT or conventional radiography, which are best suited for detecting lytic lesions, MRI distinguishes myeloma-infiltrated bone marrow from healthy marrow, reveals the infiltration pattern, delineates lesion morphology, enables early detection of marrow involvement, and is particularly useful for assessing unexplained VCFs. MRI is more sensitive than bone scintigraphy in detecting spinal lesions and is currently regarded as the most sensitive and specific imaging modality for evaluating spinal involvement. It permits both morphological assessment of VCFs

and spatial evaluation of neural damage or tumor masses. Therefore, urgent MRI is essential in assessing spinal cord compression and guiding treatment decisions in patients with advanced skeletal involvement. MRI findings also change with treatment response[2,5,6,7,8,11,12,14,20,21,23,26,29,30].

MRI sensitivity for detecting malignant spinal cord compression is approximately 93%, with 97% specificity and 95% overall diagnostic accuracy[29].

Thus, imaging is essential for diagnosing MM patients, as bone lesions have prognostic significance and are part of the criteria for initiating therapy. Although conventional radiography was long considered the "gold standard," it has been largely replaced by newer modalities including LDWBCT, PET-CT, and MRI due to its limitations[14,23].

LDWBCT provides superior image quality, is widely available, highly sensitive, relatively inexpensive, and offers rapid scan times. PET-CT outperforms CT and is comparable to MRI in detecting focal lesions, while MRI is better suited for identifying diffuse bone marrow involvement. A particular advantage of MRI is its ability to differentiate between uncomplicated osteoporotic and pathological fractures[14,23].

At diagnosis, imaging assists in accurate staging and identifying suitable biopsy targets. Throughout the disease course, it plays a critical role in monitoring progression, assessing treatment response, and investigating clinical deterioration. Imaging also guides management decisions, such as initiating systemic therapy upon disease progression, or radiotherapy and surgery for critical lesions with neurological compromise[3].

CONCLUSIONS

1. Approximately 80-85% of patients with multiple myeloma (MM) present with bone lesions at the time of diagnosis. The frequency of osteolytic lesion locations is as follows: spine (49-70%), ribs (45-50%), skull (35-50%), shoulder (20-35%), pelvis (30-40%), and long bones (13-35%).
2. Bone destruction in MM results from asynchronous bone turnover, where increased osteoclastic resorption is not matched by a corresponding increase in bone formation. A hallmark of MM-related bone lesions is their extremely rare healing, even in patients with complete remission.
3. Low-dose whole-body computed tomography (LDWBCT) is currently the "gold standard" for diagnosing MM-related bone disease. CT provides higher sensitivity, better image quality, and a 4-33% greater detection rate of lytic lesions compared to conventional radiography.[31]
4. Positron emission tomography-computed tomography (PET-CT), with a sensitivity of 90% and specificity between 70-100%, remains the "gold standard" for detecting myeloma lesions, monitoring skeletal disease, differentiating active from inactive lesions, and assessing treatment response, including the detection of residual MM.
5. Magnetic resonance imaging (MRI) differentiates myeloma-infiltrated bone marrow from healthy tissue, identifies the infiltration pattern, accurately visualizes lesion morphology, allows early detection of marrow involvement, and is especially valuable for evaluating unexplained vertebral compression fractures. MRI is more sensitive than bone scintigraphy in detecting spinal lesions.
6. MRI is currently regarded as the most sensitive and specific diagnostic imaging modality for evaluating spinal lesions, as it allows morphological assessment of vertebral compression fractures alongside spatial evaluation of neural structures and tumor masses.

Conflict of Interest

The author declares no conflicts of interest.

The use of generative AI and AI-assisted technologies: The authors did not use in this article generative AI and AI-assisted technologies.

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