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Cancer-Associated Osteoporosis and Treatment-Induced Bone Loss: Mechanisms, Prevention, and Therapeutic Strategies

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Abstract

Background. Cancer-associated osteoporosis and cancer treatment-induced bone loss (CTIBL) represent an increasingly important dimension of treatment-related morbidity. Skeletal fragility in cancer reflects the convergence of accelerated bone remodeling, endocrine disruption, treatment-related toxicity, and deterioration of the muscle-bone unit, generating fracture risk that may not be fully captured by bone mineral density alone.

Aim. To critically integrate current evidence on the mechanisms, risk stratification, prevention, and treatment of cancer-associated osteoporosis and CTIBL, with emphasis on clinically actionable strategies for preserving skeletal integrity.

Material and methods. A narrative review was conducted using international guidelines and consensus statements, randomized controlled trials, systematic reviews, meta-analyses, and key observational studies addressing bone health in adults with cancer.

Results. Aromatase inhibition and androgen deprivation are major drivers of CTIBL, while glucocorticoid exposure, gonadal dysfunction, sarcopenia, and physical deconditioning further amplify skeletal vulnerability. Contemporary management requires risk stratification integrating bone mineral density with clinical and treatment-specific determinants of fracture risk. Bisphosphonates and denosumab remain the principal pharmacological strategies, but treatment selection and discontinuation require consideration of renal function, calcium homeostasis, osteonecrosis risk, and rebound-associated vertebral fractures. Resistance and impact-loading exercise provide a complementary strategy by targeting muscle function, mechanical loading, and other modifiable determinants of skeletal resilience.

Conclusions. Cancer-associated bone fragility should be regarded as a dynamic, treatment-modifiable component of oncological morbidity rather than an isolated consequence of reduced bone mineral density. Integrating treatment-specific fracture risk, bone-targeted pharmacotherapy, and preservation of the muscle-bone unit provides a clinically coherent framework for protecting skeletal health across the cancer continuum.

Keywords: cancer-associated osteoporosis, cancer treatment-induced bone loss, CTIBL, bone fragility, fracture risk, bone remodeling, antiresorptive therapy, exercise

1. Introduction

Cancer survival has improved substantially, transforming treatment-related morbidity into an increasingly important determinant of long-term outcomes. Within this evolving landscape, skeletal health represents a clinically relevant but still incompletely integrated component of cancer care. Patients with malignancy are exposed to fracture risk arising not only from age and conventional osteoporosis risk factors, but also from cancer-specific and treatment-specific mechanisms that can profoundly disturb skeletal homeostasis. Cancer-associated bone fragility should therefore not be regarded simply as osteoporosis occurring in a patient with cancer, but

as the cumulative skeletal consequence of malignancy, anticancer therapy, endocrine disruption, and progressive musculoskeletal vulnerability [1-3].

Cancer treatment-induced bone loss (CTIBL) represents one of the most clinically relevant manifestations of this process. Estrogen deprivation during aromatase inhibitor therapy and profound androgen suppression during androgen deprivation therapy shift bone remodeling toward accelerated resorption, leading to loss of bone mineral density (BMD) and increased fracture susceptibility [4-10]. Long-term endocrine therapy data confirm clinically relevant BMD loss and increased fracture risk associated with aromatase inhibition, while ADT is associated with both skeletal deterioration and increased fracture susceptibility [11-15]. Skeletal deterioration may be further amplified by glucocorticoid exposure, chemotherapy-induced gonadal dysfunction, hematopoietic cell transplantation, nutritional impairment, sarcopenia, and physical deconditioning; ADT-associated loss of lean body mass further illustrates the close interaction between skeletal and muscular toxicity [6-8,16]. These factors frequently coexist, creating a dynamic and cumulative risk profile that cannot be adequately characterized by a single treatment exposure or densitometric measurement [2-5].

This complexity challenges the conventional BMD-centered model of osteoporosis assessment. In patients with cancer, fracture susceptibility emerges from the interaction between skeletal deterioration, anticancer treatment, endocrine status, previous fracture, age, comorbidities, muscle loss, impaired balance, and functional decline. Consequently, BMD remains essential but insufficient as an isolated measure of skeletal vulnerability. A more clinically meaningful approach requires integration of densitometric findings with patient-specific and treatment-specific determinants of fracture risk [2-5,8,9].

The availability of effective bone-targeted therapies makes early recognition of skeletal risk clinically actionable. Bisphosphonates, particularly zoledronic acid, can prevent or attenuate aromatase inhibitor-associated bone loss, while adjuvant bisphosphonate therapy may provide additional oncological benefit in selected postmenopausal women with early breast cancer [17-20]. Denosumab has demonstrated reduction in clinical fractures in women receiving aromatase inhibitors and reduction in new vertebral fractures in men undergoing androgen deprivation therapy [21-26]. However, skeletal efficacy should not be equated with anticancer efficacy, as illustrated by the divergent disease-outcome findings of adjuvant denosumab trials [22,23,27]. Treatment selection also requires consideration of renal function, calcium homeostasis, treatment duration, and medication-related osteonecrosis of the jaw [28-30]. Denosumab discontinuation represents a particular challenge because rebound activation of

bone turnover may result in rapid bone loss and vertebral fractures, making an appropriate exit strategy an integral component of treatment planning [31].

An important distinction must also be maintained between the prevention of osteoporosis-related fragility fractures and the prevention of skeletal complications of metastatic bone disease. Although bone-modifying agents are used in both settings, their indications, dosing, therapeutic objectives, and safety profiles differ substantially [2,28-30]. Similarly, skeletal efficacy should not be interpreted as evidence of anticancer activity [20,22,23,27]. Distinguishing these therapeutic contexts is essential for the rational use of bone-targeted agents in oncology.

Pharmacological intervention, however, addresses only one component of fracture vulnerability. Cancer and its treatment may simultaneously compromise muscle mass, strength, balance, and habitual mechanical loading, thereby increasing fracture susceptibility through pathways that extend beyond bone density [16]. Resistance and impact-loading exercise may complement pharmacological treatment by preserving skeletal loading, muscle function, balance, and physical capacity. Randomized trials in prostate and breast cancer populations, together with systematic reviews and meta-analytic evidence, support beneficial effects of structured exercise on bone and muscle-related outcomes [32–40]. This interconnected deterioration provides a strong rationale for considering the muscle–bone unit as a therapeutic target.

Taken together, current evidence supports a shift from the isolated management of low BMD toward the prevention of cancer-associated skeletal fragility. The clinically relevant endpoint is not osteoporosis as a densitometric diagnosis alone, but fracture prevention in a patient whose skeletal and functional vulnerability evolves throughout cancer treatment and survivorship [2-5]. Integrating treatment-specific risk assessment, bone-targeted pharmacotherapy, and preservation of the muscle-bone unit may therefore provide a more comprehensive framework for long-term skeletal protection across the cancer continuum [2,3,8,32-40].

Research Objective.

To critically evaluate cancer-associated osteoporosis and CTIBL as a continuum of skeletal fragility and to define an evidence-based framework integrating treatment-specific fracture risk, bone-directed therapy, and preservation of musculoskeletal function across the cancer care continuum.

Research Problems.

To what extent do conventional osteoporosis assessment strategies capture fracture risk in patients with cancer? How should cancer- and treatment-specific skeletal risk, bone status, and functional impairment inform preventive and therapeutic decisions? What is the complementary role of bone-directed pharmacotherapy and exercise in fracture prevention across treatment and survivorship?

Research Hypothesis.

Cancer-associated skeletal fragility extends beyond BMD-defined osteoporosis; integrating treatment-specific risk, clinical fracture determinants, and muscle-bone dysfunction may enable more comprehensive risk stratification and more individualized fracture prevention.

2. Methodology

Review design

This integrative narrative review critically synthesized current evidence on cancer-associated osteoporosis and cancer treatment-induced bone loss (CTIBL), integrating mechanisms, fracture-risk assessment, pharmacological management, and exercise-based prevention.

Literature search

PubMed/MEDLINE, Scopus, and Web of Science were searched using combinations of terms including *“cancer-associated osteoporosis”, “cancer treatment-induced bone loss”, “CTIBL”, “fracture risk”, “bone mineral density”, “aromatase inhibitors”, “androgen deprivation therapy”, “bisphosphonates”, “denosumab”, “sarcopenia”, “exercise”, and “cancer survivorship”*. Reference lists of key guidelines, consensus statements, and major trials were additionally screened.

Study selection and eligibility criteria

Priority was given to international guidelines and consensus statements, randomized controlled trials, systematic reviews, meta-analyses, and large observational studies. Eligible publications addressed:

- mechanisms and determinants of cancer-associated bone loss;
- skeletal effects of anticancer therapies;
- fracture-risk assessment and prevention;

- efficacy and safety of bisphosphonates and denosumab;
- exercise-based strategies targeting bone and muscle health.

Non-peer-reviewed publications, conference abstracts, editorials, letters, pediatric studies, and studies without direct relevance to CTIBL or cancer-associated skeletal fragility were excluded.

Data synthesis

Evidence was synthesized across four domains: pathophysiology, fracture-risk assessment, pharmacological management, and exercise-based preservation of the muscle-bone unit. Particular emphasis was placed on clinically actionable evidence from breast and prostate cancer. Owing to heterogeneity in populations, interventions, and outcomes, no quantitative meta-analysis was performed.

Limitations

The narrative design is susceptible to selection bias and does not provide pooled effect estimates. Evidence is also uneven across cancer types, with the strongest data derived from breast and prostate cancer. To minimize these limitations, priority was given to high-level evidence and pivotal clinical studies.

3. Cancer-Associated Osteoporosis and Treatment-Induced Bone Loss

Cancer-associated osteoporosis represents a distinct form of secondary skeletal fragility in which baseline fracture susceptibility is amplified by malignancy and anticancer treatment. Cancer treatment-induced bone loss (CTIBL) is central to this phenotype, but its clinical consequences extend beyond measurable reductions in bone mineral density (BMD). Sex-steroid deprivation, glucocorticoid exposure, treatment-induced gonadal dysfunction, hematopoietic cell transplantation, sarcopenia, and physical deconditioning may converge to accelerate skeletal deterioration and increase fracture susceptibility [2-8].

Endocrine therapy provides the clearest model of CTIBL. Aromatase inhibitors induce profound estrogen deprivation, accelerating bone turnover, reducing BMD, and increasing fracture risk [4,9-14]. Androgen deprivation therapy (ADT) similarly disrupts sex-steroid-dependent skeletal homeostasis and is associated with bone loss and fractures [5,15,24-26]. Importantly, ADT also promotes loss of lean mass, linking skeletal deterioration to progressive impairment of the muscle–bone unit [16]. CTIBL should therefore be regarded not as an

isolated adverse effect on bone, but as part of a broader treatment-induced musculoskeletal phenotype.

Skeletal vulnerability may be further amplified by chemotherapy-induced hypogonadism, glucocorticoids, and hematopoietic cell transplantation, particularly when these exposures coexist with older age, previous fragility fracture, low BMD, nutritional impairment, or reduced physical activity [2,3,6-8]. The resulting risk is cumulative and treatment-dependent. Consequently, identical baseline BMD values may carry substantially different prognostic significance according to the type, intensity, and duration of anticancer therapy [3-5,8,9].

This heterogeneity exposes the limitations of a predominantly densitometric approach. DXA remains fundamental, but BMD alone does not capture the anticipated velocity of skeletal deterioration, treatment-related muscle loss, functional decline, or falls risk [3,5,7,8,16]. Conventional fracture-risk assessment should therefore be interpreted within the oncological context rather than applied independently of treatment exposure [2-5,9].

Cancer-associated skeletal fragility is best conceptualized as a dynamic, treatment-modified fracture-risk state. Assessment should integrate baseline skeletal status and previous fracture with conventional risk factors, anticipated treatment toxicity, and musculoskeletal reserve [2-5,8,9]. This shifts the clinical focus from detecting established osteoporosis to identifying fracture vulnerability early enough for effective prevention [2,3,8].

4. Prevention and Therapeutic Strategies

The therapeutic objective in CTIBL is fracture prevention rather than preservation of bone mineral density (BMD) alone. Management should therefore be risk-adapted and combine modification of reversible skeletal risk factors with bone-directed pharmacotherapy and preservation of musculoskeletal function [2-5,8,9]. Importantly, improvement in BMD, reduction in fracture incidence, and potential anticancer effects represent distinct therapeutic outcomes and should not be interpreted interchangeably.

Bisphosphonates and denosumab constitute the principal pharmacological strategies for CTIBL [2,4,5,8,9]. Zoledronic acid has consistently prevented aromatase inhibitor-associated bone loss, with the Z-FAST and ZO-FAST trials demonstrating sustained preservation of BMD during endocrine therapy [17-19]. Beyond skeletal protection, individual-patient meta-analysis demonstrated that adjuvant bisphosphonates can reduce bone recurrence and breast cancer mortality in postmenopausal women with early breast cancer [20]. Thus, in appropriately selected patients, bisphosphonate therapy may address both skeletal and oncological objectives.

Denosumab is supported by particularly strong fracture-specific evidence. In ABCSG-18, denosumab significantly reduced clinical fractures in postmenopausal women receiving aromatase inhibitors [21]. In men undergoing androgen deprivation therapy, denosumab increased BMD and reduced the incidence of new vertebral fractures [24,25]. However, skeletal efficacy should not be extrapolated to anticancer efficacy. Although disease-related outcomes were explored in ABCSG-18 [22,23], D-CARE failed to demonstrate improvement in its primary disease-related endpoint [27]. These divergent findings emphasize that BMD preservation, fracture prevention, and oncological benefit require separate interpretation.

Selection of antiresorptive therapy should therefore be individualized according to fracture risk, renal function, calcium homeostasis, anticipated treatment duration, adherence, and feasibility of long-term treatment [2-5,8,9]. Medication-related osteonecrosis of the jaw requires appropriate risk assessment and preventive dental management [28,29]. Importantly, the risk profile associated with high-dose bone-modifying therapy for metastatic disease should not be directly extrapolated to osteoporosis-dose regimens [28-30].

Denosumab requires particular attention to therapeutic sequencing. Because its antiresorptive effect is rapidly reversible, discontinuation may result in rebound bone turnover, rapid BMD loss, and multiple vertebral fractures [31]. Denosumab should therefore not be initiated without consideration of a subsequent antiresorptive strategy. In this context, treatment discontinuation is an integral component of treatment design.

Pharmacological therapy alone does not address the full spectrum of fracture vulnerability. Resistance and impact-loading exercise may preserve BMD, muscle strength, and body composition in breast and prostate cancer populations [32-36]. Systematic reviews further support the role of structured physical activity in maintaining bone health during and after cancer treatment, although fracture-specific evidence remains limited [37-40]. Exercise should therefore be considered complementary to, rather than a substitute for, pharmacological therapy, targeting functional components of the muscle–bone unit that antiresorptive treatment does not directly address.

Overall, optimal skeletal protection requires an integrated and longitudinal strategy that aligns intervention with individual fracture risk, the specific oncological exposure, and the clinical endpoint requiring modification. The management of CTIBL should therefore extend beyond attenuation of BMD loss toward the broader objective of preventing fragility fractures while preserving musculoskeletal function throughout cancer treatment and survivorship.

5. Discussion

The evidence reviewed here supports a broader interpretation of cancer-associated osteoporosis than that provided by conventional BMD-based definitions. The principal clinical challenge is not simply accelerated bone loss, but the emergence of fracture vulnerability through the interaction of baseline skeletal status, treatment-specific toxicity, and progressive loss of musculoskeletal reserve [2-5,8]. This distinction is important because patients exposed to potent endocrine deprivation or other bone-toxic therapies may develop clinically relevant fracture risk before conventional densitometric thresholds for osteoporosis are reached [3-5,9,10].

A key finding is the heterogeneity of evidence supporting bone-directed interventions. Preservation of BMD, prevention of fragility fractures, and potential anticancer effects represent distinct outcomes and should not be interpreted interchangeably. Zoledronic acid has consistently demonstrated protection against aromatase inhibitor-associated BMD loss [17-19], whereas denosumab is supported by direct fracture-reduction evidence in both women receiving aromatase inhibitors and men undergoing ADT [21,24]. Conversely, the divergent oncological findings of ABCSG-18 and D-CARE illustrate the limitations of extrapolating skeletal efficacy to cancer-related outcomes [22,23,27]. Similarly, the EBCTCG meta-analysis indicates that the anticancer benefit of adjuvant bisphosphonates is dependent on menopausal context rather than representing a universal effect across patients with breast cancer [20].

These differences have direct implications for therapeutic selection. The clinically relevant question is not which antiresorptive agent produces the greatest increase in BMD, but which intervention best addresses the dominant risk and therapeutic objective in an individual patient. Fracture risk, oncological setting, renal function, treatment duration, toxicity, adherence, and feasibility of long-term sequencing should therefore inform treatment selection [2-5,8,9]. Denosumab provides a particularly clear example: strong antifracture efficacy [21,24] must be balanced against the consequences of unplanned discontinuation, making therapeutic sequencing part of the initial treatment strategy rather than an issue considered only at withdrawal [31].

Another important consideration is that pharmacological suppression of bone resorption addresses only part of the fracture-risk phenotype. ADT-associated sarcopenia supports a broader concept of treatment-induced musculoskeletal vulnerability [16]. Resistance and impact-loading exercise may complement pharmacotherapy by preserving BMD, muscle strength, body composition, and functional reserve in breast and prostate cancer populations [32-36]. However, systematic reviews indicate that evidence is substantially stronger for bone

and functional outcomes than for fracture reduction itself [37-39]. Exercise should therefore be regarded as a clinically relevant component of multimodal prevention without extrapolating intermediate musculoskeletal benefits to established antifracture efficacy.

Several uncertainties limit translation of the available evidence. Current recommendations and high-quality intervention data are concentrated predominantly in breast and prostate cancer, particularly in populations receiving aromatase inhibitors or ADT [4,5,9,10,21,24]. Evidence is less developed for other solid tumors, hematological malignancies, and patients exposed to complex multimodal therapy, although hematopoietic cell transplantation represents a recognized high-risk setting [7]. Treatment thresholds, monitoring intervals, optimal duration of antiresorptive therapy, and sequencing strategies also remain incompletely defined across oncological populations [2-5,8,31]. Furthermore, the frequent use of BMD as an endpoint limits conclusions regarding outcomes of greater clinical relevance, including fractures, falls, disability, and preservation of physical function.

Future research should therefore move from bone-loss prevention toward integrated fracture prevention. Trials should prioritize fracture and functional endpoints, determine how cancer-specific treatment exposure modifies conventional fracture prediction, and establish whether combined pharmacological and exercise-based interventions translate into superior clinical outcomes. Greater representation of malignancies beyond breast and prostate cancer will be essential for developing treatment-specific skeletal protection applicable across the broader cancer population.

Taken together, the available evidence supports a transition from a static osteoporosis model toward anticipatory, treatment-informed skeletal care. BMD remains fundamental, but should be interpreted alongside oncological exposure and musculoskeletal reserve. The emerging therapeutic objective is therefore broader than preservation of bone mass: it is the prevention of fragility fractures while maintaining musculoskeletal function throughout cancer treatment and survivorship.

6. Conclusions

Cancer-associated osteoporosis and CTIBL should be recognized as a dynamic, treatment-modified state of skeletal fragility rather than a BMD-defined complication of cancer therapy. Effective fracture prevention requires early identification of skeletal vulnerability, integration of oncological exposure into risk assessment, and coordinated preservation of bone and musculoskeletal function. Management should therefore shift from reactive treatment of

established bone loss toward individualized, anticipatory, and longitudinal fracture prevention throughout cancer treatment and survivorship.

Disclosure Statement

The author confirms that this manuscript represents original work that has not been published previously and is not currently under consideration for publication elsewhere. The author assumes full responsibility for the content, interpretation, and conclusions presented in this review.

Supplementary Materials

No supplementary materials are associated with this article.

Author Contributions

Paulina Patrycja Turzyńska conceived and designed the study, developed the conceptual framework, conducted the literature review, interpreted the evidence, and wrote, reviewed, and revised the manuscript. The author has read and approved the final version of the manuscript.

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Informed Consent Statement

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Conflicts of Interest

The author declares no conflicts of interest.

Declaration of Generative AI and AI-Assisted Technologies

During the preparation of this work, the author used ChatGPT-5.2 to improve grammar and language clarity. After using this tool, the author reviewed and edited the content as needed and takes full responsibility for the content of the publication.

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