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BONE MINERAL DENSITY IN A SAMPLE OF IRAQI PATIENTS WITH CHRONIC MYELOID LEUKEMIA

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ABSTRACT

Background: Osteoporosis is a growing public health concern with substantial medical, social, and economic implications. Patients with chronic myeloid leukemia (CML), especially those undergoing long-term therapy, may be at increased risk for reduced bone mineral density (BMD). **Objective:** This study aimed to assess the potential association between BMD and chronic myeloid leukemia, particularly in patients receiving Imatinib therapy. **Methods:** A cross-sectional study was conducted at the Hematology and Rheumatology consultation clinics of Baghdad Teaching Hospital, Medical City, Iraq, from September 2019 to May 2020. The study included 84 CML patients in the chronic phase, all receiving Imatinib, and 84 age- and sex-matched healthy controls. Participants, aged 20–70 years, underwent comprehensive history taking, clinical examination, laboratory investigations, and BMD assessment using Dual-Energy X-ray Absorptiometry (DXA) at the lumbar spine and femoral neck. **Results:** In the CML group, lumbar spine DXA showed normal BMD in 56.1%, osteopenia in 17.1%, and osteoporosis in 26.8%, compared to 72%, 11%, and 17.1%, respectively, in controls. Femoral neck results were similar between groups, with 74.4% normal, 20.7% osteopenic, and 4.9% osteoporotic in both. Although the overall difference in osteoporosis prevalence was not statistically significant, age, menopausal status, BMI, disease duration, and treatment duration appeared to influence BMD in CML patients. **Conclusion:** Osteopenia and osteoporosis are common in CML patients, particularly at the lumbar spine. While Imatinib therapy may help stabilize BMD over time, conventional risk factors such as age and menopause significantly contribute to bone loss, warranting regular monitoring and preventive strategies.

KEYWORDS: Bone, Mineral, Density, Chronic, Myeloid, Leukemia.

INTRODUCTION

Osteoporosis is a widespread, progressive skeletal disorder characterized by reduced bone mass and microarchitectural deterioration of bone tissue, leading to increased fragility and susceptibility to fractures. Often called a "silent" disease, osteoporosis remains asymptomatic until fractures occur, commonly affecting the vertebrae, hips, and wrists.[1] The World Health Organization (WHO) defines osteoporosis using the T-score from Dual-Energy X-ray Absorptiometry (DXA), with values ≤ –2.5 indicating osteoporosis and ≤ –2.5 plus one or more fractures indicating severe osteoporosis.[2] Globally, more than 200 million individuals are affected, with higher prevalence in aging populations and among females.[3] With the anticipated rise in hip fractures by 2050, osteoporosis represents a critical public health issue.[4] Risk factors for osteoporosis are multifactorial and include female sex, older age, low body weight, smoking, low calcium intake, family history, and sedentary lifestyle. Additionally, several chronic diseases—such as diabetes, rheumatoid arthritis, and endocrine disorders—as well as medications like corticosteroids and chemotherapy agents contribute to secondary osteoporosis.[5] The underlying pathogenesis involves an imbalance between bone resorption and formation, often exacerbated by hormonal changes, nutrient deficiencies, or systemic inflammation.[6] Clinically, osteoporosis is often undiagnosed until a fracture occurs, which can lead to pain, functional limitations, and increased mortality, particularly in the elderly.[7] DXA scanning is the gold standard for diagnosing osteoporosis due to its accuracy, low radiation exposure, and non-invasiveness.[8] Both T-scores and Z-scores are used to interpret bone density;

the latter is more appropriate in premenopausal women, men under 50, and children.[9] Chronic Myeloid Leukemia (CML) is a clonal hematopoietic stem cell disorder characterized by the Philadelphia chromosome t(9;22), which creates the BCR-ABL fusion gene, promoting uncontrolled cell proliferation. CML typically progresses from a chronic phase to an accelerated phase, and eventually a blast crisis resembling acute leukemia.[10] Though CML comprises roughly 15% of all leukemias, its incidence increases with age and varies geographically, with some links to ionizing radiation exposure.[11] Clinical symptoms include fatigue, weight loss, abdominal discomfort from splenomegaly, and less commonly, hyperuricemia-related gout or leukostasis.[12] The standard treatment for CML involves tyrosine kinase inhibitors (TKIs), particularly imatinib, which targets the BCR-ABL fusion protein. Imatinib has transformed CML into a manageable chronic illness, significantly improving survival rates.[13] However, there is growing interest in understanding the long-term side effects of TKIs, particularly on bone health. Imatinib appears to affect bone metabolism through suppression of osteoclast and osteoblast activity, possibly leading to a reduction in bone turnover.[14] While some studies suggest that BMD remains stable or improves with prolonged treatment, others report alterations in calcium, parathyroid hormone (PTH), and vitamin D metabolism, raising concerns about secondary hyperparathyroidism and its impact on bone health.[15] Given the chronic nature of CML and the lifelong need for TKI therapy, evaluating bone mineral density in these patients is crucial. This study aims to explore the potential association between BMD and CML in patients treated with imatinib, considering both the disease and treatment as contributors to bone health alterations.

METHOD

A cross-sectional study was conducted at the Hematology and Rheumatology Consultation Clinics of Baghdad Teaching Hospital, Medical City, Iraq, over a period extending from September 2019 to May 2020. The study aimed to evaluate the association between bone mineral density (BMD) and chronic myeloid leukemia (CML). A total of 84 patients diagnosed with chronic-phase CML were included based on clinical presentation, laboratory investigations, and confirmation by fluorescence in situ hybridization (FISH) for the Philadelphia chromosome. All CML patients were receiving Imatinib therapy. A control group of 84 healthy individuals matched for age and gender was selected from visitors and relatives of patients attending the outpatient rheumatology clinic. Both study groups included individuals aged between 20 and 70 years. Verbal consent was obtained from all participants, and ethical approval was granted by the Ethics Committee of the College of Medicine, University of Baghdad. Data collection involved a structured questionnaire that recorded demographic variables (age, gender, BMI, occupation, smoking, alcohol use, and education). CML-specific data included age at diagnosis, disease duration, treatment type and duration, and treatment response (hematologic and cytogenetic). Additional information related to osteoporosis risk included history of fractures, medication use (e.g., steroids, antiepileptics), and comorbidities such as thyroid, liver, or renal disease. Blood samples were collected to measure serum calcium, phosphorus, alkaline phosphatase, liver and renal function, and complete blood counts. BMD was assessed using Dual-Energy X-ray Absorptiometry (DEXA) at the lumbar spine (L1–L4) and femoral neck (non-dominant side), performed by trained radiology technicians. T-scores were used for men above 50 and postmenopausal women, while Z-scores were applied for younger individuals and premenopausal women. The scanning apparatus was located at 393 Rue Charis Lindberch, Mauguio, France. Data were analyzed using SPSS version 25. Means, percentages, and standard deviations were calculated. Statistical significance was determined using Student's t-test, ANOVA, and Chi-square tests, with p-values ≤ 0.05 considered significant.

RESULTS

Table 1 presents the demographic characteristics of CML and control groups. In the CML group, 59.8% were under 50 years, similar to 62.2% in the control group, with mean ages of 44.5±12.2 and 43.9±11.7, respectively. Regarding BMI, 46.3% of CML patients were obese compared to 37.8% in controls (mean BMI: 29.7±6.69 vs. 28.8±5.48). Smoking status showed 79.3% of CML patients were non-smokers vs. 64.6% in controls, with a significant difference (p = 0.003). CML patients had disease duration of 1–4 years (45.1%), 5–9 years (32.9%), and over 10 years (22%).

Table 1: Demographic Variables of the Studied Groups (CML and Control Groups).

		CML		Healthy controls		P value
		No	%	No	%	
Age (years)	<50 years	49	59.8	51	62.2	0.749
	=>50 years	33	40.2	31	37.8	
	Mean±SD (Range)	44.5±12.2 (20-70)		43.9±11.7 (20-70)		0.765
Gender	Male	41	50.0	41	50.0	-
	Female	41	50.0	41	50.0	
BMI (Kg/m2)	Normal (18.5-24.9)	13	15.9	21	25.6	0.271
	Overweight (25-29.9)	31	37.8	30	36.6	
	Obese (=>30)	38	46.3	31	37.8	
	Mean±SD (Range)	29.7±6.69 (18-61)		28.8±5.48 (19-44)		0.346

Educational Level	Illiterate	7	8.5	6	7.3	0.098
	Primary	37	45.1	31	37.8	
	Secondary	11	13.4	25	30.5	
	College	26	31.7	20	24.4	
	Higher education	1	1.2	-	-	
Occupation	Not employed	38	46.3	32	39.0	0.548
	Employed	39	47.6	46	56.1	
	Retired	5	6.1	4	4.9	
Menopause	Menstruating	25	61.0	25	61.0	-
	Menopause	16	39.0	16	39.0	
Smoking	Not smoker	65	79.3	53	64.6	0.003*
	Smoker	4	4.9	19	23.2	
	Ex-smoker	13	15.9	10	12.2	
		CML		Healthy controls		P value
		No	%	No	%	
Receive medication	None	82	100.0	82	100.0	-
	Calcium	-	-	-	-	
	Vitamin D	-	-	-	-	
	Steroid	-	-	-	-	
	Thyroxin	-	-	-	-	
	Anti epileptic	-	-	-	-	
History of fracture	Absent	82	100.0	82	100.0	-
	Present	-	-	-	-	
Chronic medical illness	None	82	100.0	82	100.0	-
	Thyroid	-	-	-	-	
	Parathyroid	-	-	-	-	
	Liver	-	-	-	-	
	Renal	-	-	-	-	
	Malabsorption	-	-	-	-	
Nutritional status (Ca & Protein)	Good	82	100.0	82	100.0	-
	Poor	-	-	-	-	
Disease duration in years and receiving imatinib	1---4	37	45.1	-	-	-
	5---9	27	32.9	-	-	
	=>10 years	18	22.0	-	-	
	Mean±SD (Range)	5.5±3.9 (1-16)				
Time of treatment initiation on Imatinib (years)		5.5±3.9 (1-16)				

Significant difference between proportions using Pearson Chi-square test at 0.05
Ca =calcium, SD=standard deviation, CML=chronic myeloid leukemia, p= probability,
% = percentage, BMI=body mass index, >= more than or equal

Table 2 results indicate lower lumbar BMD in the CML group (26.8% osteoporosis) compared to controls (17.1%), though the difference in T and Z scores was not statistically significant. Mean lumbar T and Z scores were -1.09±1.38 and -0.53±1.40 in CML patients vs. -1.05±1.15 and -0.50±1.35 in controls. Femoral neck BMD was identical in both groups by percentage, but mean T and Z scores were significantly lower in the control group (p=0.005 and p=0.0001, respectively). Serum calcium was significantly lower in the CML group (mean 8.80±0.34 vs. 9.01±0.53; p=0.003).

Table 2: Bone density data of the studied groups (CML) and control group.

		CML		Healthy controls		P value
		No	%	No	%	
Lumbar Vertebrae DXA Scan	Normal	46	56.1	59	72.0	0.107
	Osteopenia	14	17.1	9	11.0	
	Osteoporosis	22	26.8	14	17.1	
	T Score lumbar Mean±SD (Range)	-1.09±1.38 (-3.9 - 2.3)		-1.05±1.15 (-5.4 - 1.2)		0.844
	Z Score lumbar Mean±SD (Range)	-0.53±1.40 (-3.1 - 3.0)		-0.50± 1.35 (-5.4 - 1.8)		0.892
Femural Neck DXA	Normal	61	74.4	61	74.4	-

Scan	Osteopenia	17	20.7	17	20.7	
	Osteoporosis	4	4.9	4	4.9	
	T Score Femur Mean±SD (Range)	-0.33±1.28 (-2.5 - 4.0)		-0.82±0.88 (-4.3 - 0.8)		0.005*
	Z Score Femur Mean±SD (Range)	0.39±1.27 (-2.1 - 4.9)		-0.29±0.88 (-4.2 - 1.1)		0.0001*
Serum Ca level	Normal	78	95.1	74	90.2	0.230
	Decrease	4	4.9	8	9.8	
Serum Ca (mg/dL) Mean±SD (Range)		8.80±0.34 (8.1-10)		9.01±0.53 (5.8-9.9)		0.003*

Significant difference between proportions using Pearson Chi-square test at 0.05, *Significant difference between two independent means using Students-t-test at 0.05, Ca = calcium, SD= standard deviation, CML= chronic myeloid, p= probability, %= Percentage, BMI= body mass index, >= more than or equal, DXA= Dual energy X-ray absorptiometry

Table 3 results show that osteoporosis and osteopenia increased with age, especially in CML patients above 50 years, with a significant difference compared to controls. Osteoporosis was more prevalent in overweight and normal-weight CML patients than controls, showing a significant BMI-related difference. Postmenopausal women in the CML group had higher osteoporosis rates than controls, indicating menopause as a significant risk factor. Longer CML disease duration (>5 years) was associated with more osteoporosis, though bone density improved after 10 years of treatment. Femoral DEXA scans revealed significantly higher osteoporosis and osteopenia rates in CML than controls.

Table 3: The relationship between bone mineral density in lumbar vertebrae in CML group and control group regarding demographic data.

		CML						**Healthy controls**					
		Lumbar Vertebrae DEXA Scan						**Lumbar Vertebrae DEXA Scan**					
		Normal		**Osteopenia**		**Osteoporosis**		**Normal**		**Osteopenia**		**Osteoporosis**	
		No	%	No	%	No	%	No	%	No	%	No	%
Age (years)	<50 years	37	75.5	-	-	12	24.5	41	80.4	1	2.0	9	17.6
	=>50 years	9	27.3	14	42.4	10	30.3	18	58.1	8	25.8	5	16.1
	P value	0.0001*						0.003*					
Gender	Male	22	53.7	8	19.5	11	26.8	32	78.0	2	4.9	7	17.1
	Female	24	58.5	6	14.6	11	26.8	27	65.9	7	17.1	7	17.1
	P value	0.830						0.202					
BMI (Kg/m2)	Under weight (<18.5)	-	-	-	-	-	-	-	-	-	-	-	-
	Normal (18.5-24.9)	7	53.8	1	7.7	5	38.5	17	81.0	1	4.8	3	14.3
	Overweight (25-29.9)	13	41.9	4	12.9	14	45.2	25	83.3	2	6.7	3	10.0
	Obese (=>30)	26	68.4	9	23.7	3	7.9	17	54.8	6	19.4	8	25.8
	P value	0.008*						0.106					
Menopause	Menstruating	20	80.0	-	-	5	20.0	21	84.0	1	4.0	3	12.0
	Menopause	4	25.0	6	37.5	6	37.5	6	37.5	6	37.5	4	25.0
	P value	0.0001*						0.005*					
Smoking	Not smoker	35	53.8	11	16.9	19	29.2	38	71.7	8	15.1	7	13.2
	Smoker	3	75.0	-	-	1	25.0	13	68.4	-	-	6	31.6
	Ex-smoker	8	61.5	3	23.1	2	15.4	8	80.0	1	10.0	1	10.0
	P value	0.714						0.189					
Disease duration in years	1---4	23	62.2	6	16.2	8	21.6	-	-	-	-	-	-
	5---9	12	44.4	4	14.8	11	40.7	-	-	-	-	-	-
	=>10 years	11	61.1	4	22.2	3	16.7	-	-	-	-	-	-
	P value	0.359											
Femur Neck DXA Scan	Normal	44	72.1	4	6.6	13	21.3	47	77.0	6	9.8	8	13.1
	Osteopenia	2	11.8	8	47.1	7	41.2	11	64.7	1	5.9	5	29.4
	Osteoporosis	-	-	2	50.0	2	50.0	1	25.0	2	50.0	1	25.0
	P value	0.0001*						0.044*					
Serum Ca level	Normal	44	56.4	13	16.7	21	26.9	53	71.6	7	9.5	14	18.9
	Decrease	2	50.0	1	25.0	1	25.0	6	75.0	2	25.0	-	-
	P value	0.910						0.211					

*Significant difference between proportions using Pearson Chi-square test at 0.05
Ca =calcium, CML= chronic myeloid leukemia, p = probability, %= Percentage, BMI= body mass index, >= more than or equal, DEXA= Dual energy X-ray absorptiometry, NO = number

This study found a significant age-related increase in femoral neck osteoporosis, with higher rates in CML patients over 50 compared to controls. BMI analysis showed no significant difference between groups, but higher weight was associated with better BMD in both. Among postmenopausal women, 43.8% of CML patients were osteopenic with no osteoporosis, similar to controls, indicating menopause as a shared risk factor for osteopenia. Premenopausal CML patients showed no bone loss, while 8% of controls had osteoporosis. Disease duration analysis showed improved BMD in CML patients after 10 years of Imatinib therapy. Femoral BMD improved with treatment duration. Lumbar DXA revealed higher osteopenia (57.1%) in CML patients compared to 11.1% in controls, with a significant group difference. These findings suggest age, menopause, and treatment duration influence bone health in CML patients, with lumbar BMD more affected than femoral BMD. As in table 4.

Table 4: The relationship between bone mineral density of femur neck DXA scan and demographic data.

		CML						Healthy controls					
		Femur Neck DXA Scan						Femur Neck DXA Scan					
		Normal		Osteopenia		Osteoporosis		Normal		Osteopenia		Osteoporosis	
		No	%	No	%	No	%	No	%	No	%	No	%
Age (years)	<50 years	48	98.0	-	-	1	2.0	48	94.1	1	2.0	2	3.9
	=>50 years	13	39.4	17	51.5	3	9.1	13	41.9	16	51.6	2	6.5
	P value	0.0001*						0.0001*					
Gender	Male	27	65.9	10	24.4	4	9.8	29	70.7	10	24.4	2	4.9
	Female	34	82.9	7	17.1	-	-	32	78.0	7	17.1	2	4.9
	P value	0.070						0.713					
BMI (Kg/m2)	Under weight (<18.5)	-	-	-	-	-	-	-	-	-	-	-	-
	Normal (18.5-24.9)	9	69.2	3	23.1	1	7.7	17	81.0	3	14.3	1	4.8
	Overweight (25-29.9)	20	64.5	9	29.0	2	6.5	19	63.3	8	26.7	3	10.0
	Obese (=>30)	32	84.2	5	13.2	1	2.6	25	80.6	6	19.4	-	-
	P value	0.430						0.302					
Menopause	Menstruating	25	100.0	-	-	-	-	23	92.0	-	-	2	8.0
	Menopause	9	56.3	7	43.8	-	-	9	56.3	7	43.8	-	-
	P value	0.0001*						0.001*					
Smoking	Not smoker	48	73.8	14	21.5	3	4.6	40	75.5	10	18.9	3	5.7
	Smoker	3	75.0	1	25.0	-	-	16	84.2	3	15.8	-	-
	Ex-smoker	10	76.9	2	15.4	1	7.7	5	50.0	4	40.0	1	10.0
	P value	0.956						0.321					
Disease duration in years	1---4	32	86.5	4	10.8	1	2.7	-	-	-	-	-	-
	5---9	16	59.3	8	29.6	3	11.1	-	-	-	-	-	-
	=>10 years	13	72.2	5	27.8	-	-	-	-	-	-	-	-
	P value	0.085											
Lumbar Vertebrae DEXA Scan	Normal	44	95.7	2	4.3	-	-	47	79.7	11	18.6	1	1.7
	Osteopenia	4	28.6	8	57.1	2	14.3	6	66.7	1	11.1	2	22.2
	Osteoporosis	13	59.1	7	31.8	2	9.1	8	57.1	5	35.7	1	7.1
	P value	0.0001*						0.044*					
Serum Ca level	Normal	59	75.6	15	19.2	4	5.1	55	74.3	15	20.3	4	5.4
	Decrease	2	50.0	2	50.0	-	-	6	75.0	2	25.0	-	-
	P value	0.320						0.775					

*Significant difference between proportions using Pearson Chi-square test at 0.05
Ca = calcium, CML= chronic myeloid leukemia, p= probability, %= Percentage, BMI= body mass index, >= more than or equal, DXA= Dual energy X-ray absorptiometry, NO = number

DISCUSSION

To the best of our knowledge, this is the first study in Iraq and the Middle East exploring the association between bone mineral density (BMD) and chronic myeloid leukemia (CML). The findings highlight a notable, though not always statistically significant, reduction in BMD among CML patients, particularly in the lumbar spine, with variations based on age, body mass index (BMI), menopausal status, and duration of disease and treatment. In the lumbar spine, osteoporosis and osteopenia were observed in 26% and 17% of CML patients, respectively. Although these rates were higher

than those in the control group, the difference did not reach statistical significance. In contrast, femoral neck DXA revealed a significant difference, with 4.9% osteoporosis and 20.7% osteopenia in the CML group, confirming a measurable impact of CML and its treatment on bone health. These findings align with studies by Olszewski et al.[16] and Al-Osami et al.[17], who reported significant bone loss in patients with other hematological malignancies, including acute and chronic leukemias. Farmer et al.[18] further emphasized the increased fracture risk in CML patients, with an adjusted hazard ratio of 2.67 for femoral fractures. Similarly, a larger study involving 181 hematological cancer patients found that 86% of CML patients experienced bone loss, with osteoporosis in 14% and osteopenia in 71%.[19] Age emerged as a critical factor, with significantly higher rates of osteoporosis among patients over 50 years. These findings are consistent with previous literature indicating increased osteoporosis prevalence in older adults.[20,21] In addition, menopause was associated with substantial BMD reduction, with postmenopausal CML patients showing a markedly higher prevalence of osteoporosis, corroborating prior findings on the heightened vulnerability of this group.[22] Interestingly, the relationship between BMI and BMD in CML patients showed conflicting trends. While higher BMI is generally considered protective, a significant proportion of overweight CML patients were osteoporotic. This paradox may be explained by disease-specific metabolic alterations or the influence of long-term TKI therapy. Nonetheless, these findings partially mirror studies by El Maghraoui et al.[23] and Bener et al.[24], who reported a positive association between BMI and BMD. Regarding Imatinib therapy, our findings suggest that bone loss may stabilize with prolonged treatment. Patients treated for more than 10 years demonstrated a reduction in osteoporosis prevalence compared to those with shorter treatment durations. This observation supports studies by Jonsson et al.[25] and Hjorth-Hansen et al.[26], which indicate that Imatinib may decelerate physiological bone loss through its dual inhibition of osteoclast and osteoblast activity. This study also noted normal serum calcium levels in most patients, though at the lower end of the reference range—a finding supported by Jonsson[15] and O'Sullivan[27], who suggested that Imatinib's impact on calcium and PTH regulation could underlie this trend. While our study offers valuable insight, limitations include a modest sample size, single-center recruitment, and a cross-sectional design, limiting assessment of longitudinal changes in BMD. Future cohort studies with larger populations and longer follow-up periods are needed to fully elucidate the long-term effects of CML and Imatinib on bone health.[3,5,28]

CONCLUSION

Osteoporosis is present in 26% and osteopenia in 17% of patients with chronic myeloid leukemia depending on T and Z score of Lumbar Vertebrae. (L1 - L4). Osteoporosis is present in in 4.9% and osteopenia in 20.7 % of patients with chronic myeloid leukemia depending on T and Z score of Femoral Neck. Increasing age is a risk factor for decrease bone mineral density especially in CML patients. Menopause is a risk factor for osteoporosis especially in CML patients. Disease duration and long term treatment with Imatinib will stabilize and improve the bone mineral density.

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