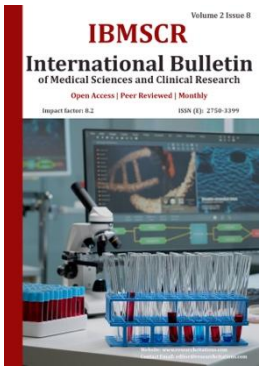

NEW WAYS TO DETECT OSTEOPOROSIS EARLY IN YOUNG WOMEN BEFORE MENOPAUSE.

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https://doi.org/10.5281/zenodo.19363631

Abstract: This means that just checking blood vitamin D or calcium might not show if bones are weak. The study suggests that looking at other markers and tools can help find osteoporosis early, especially in women who haven't gone through menopause yet. It's important to check these risk factors early to keep bones healthy. Osteoporosis is a silent skeletal disease characterized by low bone mass and deterioration of bone microarchitecture, typically leading to increased fracture risk. While classically associated with postmenopausal estrogen deficiency, osteoporosis can also affect premenopausal women, especially when secondary factors such as vitamin D deficiency or endocrine disorders are present. Early diagnosis in premenopausal women is challenging because routine bone mineral density (BMD) screening is not usually performed in this age group. In this study, we analyzed data from 33 premenopausal women (mean age 44.9±2.4 years) to assess modern aspects of early detection. Femoral neck BMD (by DXA), T-score, Z-score, serum calcium, and 25-hydroxyvitamin D (cholecalciferol) were measured. Descriptive statistics (mean, SD, percentiles) are presented in Table 1. Pearson correlation coefficients among age, BMD, T-score, Z-score, vitamin D, and calcium were computed (Table 3). Key findings include the expected strong inverse correlation between T-score and Z-score ($r=-0.938$, $p<0.001$) and moderate correlations between BMD and these scores (T-score: $r=+0.503$, $p=0.003$; Z-score: $r=-0.520$, $p=0.002$). No significant correlations were found between BMD and serum calcium or vitamin D (all $p>0.05$). These results underscore that while core bone density metrics interrelate, common biochemical measures may not directly reflect bone density in this cohort. We discuss implications for early screening, noting that bone turnover markers and risk-assessment tools are emerging as modern adjuncts in osteoporosis evaluation. The study highlights the importance of comprehensive assessment in premenopausal women at risk and suggests that targeted evaluation of vitamin D status and other risk factors is warranted even before menopause.

Keywords: Premenopausal women, osteoporosis , early diagnosis , bone mineral density

Introduction

Osteoporosis is defined as a chronic, progressive bone disease with decreased BMD and deterioration of the structure of bone tissue, leading to bones that become more fragile and susceptible to fracture. It is often quite asymptomatic until a fracture occurs. Though common among elderly postmenopausal women, osteoporosis affects premenopausal women, largely as secondary osteoporosis due to an underlying disease or the use of medication, and idiopathic osteoporosis without any recognizable cause. In a young woman, peak bone mass is attained within the 2nd to 3rd decades of life. A low peak bone mass is therefore an important risk factor for osteoporosis. The early development of osteoporosis in women is multifactorial. Genetics

can contribute to as much as 80% of the variance in peak bone density. Women who are predisposed to low peak bone mass have greater susceptibility for osteoporosis mid-life. In terms of hormonal factors, estrogen has an important role in protecting bone density; states of hypoestrogenism, including primary ovarian insufficiency and a prolonged state of amenorrhea can cause significant loss of bone before menopause. For example, women with functional hypothalamic amenorrhea have significantly lower BMD (bone mineral density) than eumenorrheic controls. In this context, a number of women in their 40s experience hormonal changes that accompany premenopause that can lead to increased bone resorption. Nutritional factors also play a role: adequate consumption of calcium, vitamin D, and protein are important with regard to bone health. Vitamin D deficiency is prevalent in women and it contributes to low BMD and increased fracture risk. A study in Europe indicated that as many as 80% of post menopausal women had vitamin D insufficiency and the same trend was seen in women in younger age groups. Chronic calcium/vitamin D deficiency affect bone mineralization, resulting in osteopenia and osteoporosis. In addition, some recent data suggest that chronic iron-deficiency anemia may also contribute to osteoporosis, likely by impairing collagen synthesis and muscular weakness leading to falls. Lifestyle factors: Sedentary behavior reduces the essential mechanical stimuli necessary for the maintenance of bone health. Smoking and excessive alcohol intake have unfavorable direct effects on bone remodeling and are recognized risk factors of osteoporosis. Conversely, high-impact exercise and strength training are known to improve or maintain BMD[1]. Body weight: Low BMI is a traditional risk factor for poor BMD[5], as both the estrogen derived from adipose tissue and greater skeletal loading associated with heavier body mass tend to be protective of bone mass. However, obesity is not universally protective; when associated with a poor diet or deficiencies in micronutrients, high BMI may coexist with low quality bone. Indeed, recent perspectives note that many fracture patients do not have osteoporotic-range BMD, indicating that factors beyond BMD (such as bone quality and turnover) play a role[2,3].

Despite these risks, routine BMD screening is not typically performed in healthy premenopausal women. Currently, guidelines recommend DXA testing primarily for postmenopausal women ≥65 years or for younger women who have significant risk factors, such as long-term corticosteroid use or prior fractures. Practically speaking, diagnosis of osteoporosis in a premenopausal woman is typically a multifaceted evaluation including: history, physical examination, laboratory tests, and when appropriate, BMD by DXA. The 2008 Bone Health & Osteoporosis Foundation guidelines indicate that most premenopausal women with low BMD do not have an increased near-term fracture risk, and reserve DXA testing for women with unusual stress fractures, chronic disease, or medications known to impact bones. Nevertheless, a number of emerging "modern" approaches aim at improving early detection. These include the use of biochemical bone turnover markers, such as CTX and NTX, and advanced fracture-risk algorithms (such as FRAX) that target the identification of high-risk individuals. Given this context, we retrospectively reviewed a cohort of 33 premenopausal women to investigate the early signs of osteoporosis. In particular, we considered femoral neck BMD by DXA, the T- and Z-scores from densitometry, and serum calcium as well as laboratory values for 25-hydroxyvitamin D, or cholecalciferol.We computed descriptive statistics and Pearson correlations among these variables. This study aims to characterize the relationships between bone density and biochemical markers in women approaching menopause, thereby

illustrating potential strategies for early identification of those at risk.Early identification is challenging because premenopausal women are not routinely screened for low BMD in the absence of risk factors[6]. The World Health Organization (WHO) BMD T-score criteria for osteoporosis (T ≤ −2.5) were developed for postmenopausal populations and are not directly applicable to premenopausal women without secondary causes[7]. Instead, a Z-score below −2.0 (low BMD for age) in conjunction with clinical risk factors is often used to recognize abnormal bone loss in younger women[8].Given these multifactorial influences, modern approaches to early osteoporosis diagnosis extend beyond a single BMD measurement. DXA imaging of the hip and spine is the gold-standard tool for assessing BMD and diagnosing osteoporosis[6]. It provides T-scores (relative to young adult reference) and Z-scores (relative to age-matched norms) that guide diagnosis and management. Current clinical guidelines recommend BMD screening by DXA in women under 65 only if significant risk factors are present, such as estrogen deficiency, prolonged amenorrhea, low BMI (<18.5 kg/m^2), family history of early fractures, or chronic glucocorticoid use[3,8]. However, even with DXA, early bone loss might be missed if one relies solely on the T-score criterion[12]. Therefore, biochemical markers and clinical risk assessment are valuable adjuncts. Markers of bone turnover (e.g. C-terminal telopeptide of collagen (CTX), procollagen type 1 N-propeptide (P1NP)) can reflect ongoing bone resorption or formation. Although they are not yet standalone diagnostic tools, elevated bone turnover markers often indicate accelerated bone loss before it manifests as a significant BMD change[9,10]. Likewise, novel biomarkers such as circulating microRNAs have shown promise in facilitating earlier detection of osteoporosis by signaling perturbations in bone metabolism at the cellular level. 25 Clinical practice recommendations for the assessment of 25-hydroxyvitamin D, calcium, and other relevant labs (e.g., thyroid, PTH levels) in the workup of a premenopausal woman presenting with low BMD will help to identify contributory deficiencies or secondary causes. Given the lack of routine osteoporosis testing among younger women, this investigation will focus on a group of premenopausal women with regard to elucidating which diagnostic measures and risk factors may be most relevant in the early detection of osteoporosis. In conjunction with BMD results analyzed together with biochemical and clinical findings, we intend to emphasize aspects related to modern diagnosis of osteoporosis before menopause, including bone density testing, biochemical markers such as vitamin D status, and identifiable nutritional, hormonal, and lifestyle risk factors that predict low bone mass. Ultimately, understanding these factors will inform a more proactive approach to osteoporosis in women in their forties, helping clinicians intervene earlier to preserve skeletal health.

Methods

A retrospective cohort study was conducted at a gynecological hospital in Tashkent city from July ,2023 to July , 2025. The study recruited 33 women in premenopausal age group. Each patient's records were verified and analyzed for accuracy of data.

Data Collection**:** Each participant underwent a comprehensive evaluation including detailed history, physical measurements, laboratory tests, and imaging. Key variables collected were:

Anthropometric measures: Age (years), weight (kg), height (cm), calculated Body Mass Index (BMI, kg/m^2). Weight and height were measured in light clothing without shoes; BMI ≥25 was classified as overweight and ≥30 as obese.

Gynecologic and reproductive history: Menstrual history (age at menarche, cycle regularity), presence of menstrual irregularities or climacteric (premenopausal) symptoms ,parity (number of pregnancies and outcomes), and any history of gynecological disorders (such as uterine fibroids, ovarian cysts, endometriosis). For analysis, menstrual status was categorized as *regular* (physiologic premenopause) versus *irregular* (any cycle disturbance or menopausal symptoms indicating climacteric changes).

Lifestyle and family history: We noted any reported smoking or alcohol use and any known family history of osteoporosis (particularly maternal history), although in the dataset none of the patients had a positive family history of osteoporosis).

Clinical symptoms and diagnosis: Patient complaints were recorded; common symptoms included generalized weakness, muscle weakness, pain on movement, lower back stiffness, and bone pain. The clinical diagnosis provided by physicians (based on BMD and clinical factors) was recorded as "primary osteoporosis grade I or II" with indication of the menopausal status (premenopausal, either regular or with irregular cycles ± climacteric syndrome).

Laboratory investigations: Fasting blood samples were analyzed for:

Complete blood count: hemoglobin (Hb) and other indices to identify anemia. Anemia was defined as Hb <120 g/L for women (mild: 100–119; moderate: 80–99; severe: <80).

Serum calcium (mmol/L) – total calcium by standard chemistry analyzer (reference ~2.1–2.5 mmol/L).

Serum 25-hydroxyvitamin D (ng/mL) – measured by immunoassay; values <20 ng/mL defined as deficiency, 20–29 ng/mL insufficiency, ≥30 ng/mL sufficient.

Other labs: The dataset included markers of renal function (urea, creatinine), liver enzymes (ALT, AST), blood glucose, lipid profile, and coagulation tests. These were primarily to rule out secondary causes or comorbid conditions. Thyroid function and parathyroid hormone (PTH) were not explicitly recorded, but no patient had clinical evidence of hyperthyroidism or hyperparathyroidism (normal serum calcium also makes undiagnosed hyperparathyroidism unlikely).

Urinalysis: to check for any abnormalities (not directly related to osteoporosis, but to evaluate general health).

Bone densitometry: BMD was measured at the femoral neck using dual-energy X-ray absorptiometry. A trained radiology technician performed DXA scans according to standard protocols, and results were interpreted by a certified specialist. The femoral neck site was chosen as it is highly predictive of hip fracture risk and is the reference site for WHO definitions[4,8]. The output included BMD in g/cm^2 and corresponding T-score and Z-score. BMD results were compared to a young-adult reference database (T-score) and to age-matched norms (Z-score). Osteoporosis was operationally defined in this study by T-score ≤ –2.5 at the femoral neck (consistent with WHO criteria for postmenopausal women[5]), although we acknowledge the debate around applying this criterion in younger women[7]. All 33 women met this densitometric criterion at the femoral neck. For context, the mean Z-score was approximately –1.8, indicating moderately low bone density relative to peers, but not below –2.0 in most cases.

Statistical Analysis: Data were compiled and analyzed using appropriate statistical software (Microsoft Excel 2021, Stata v17). Continuous variables were assessed for normality. We computed means and standard deviations (SD) for approximately normally distributed

variables and medians (with ranges) if distributions were skewed. Proportions were calculated for categorical variables. Key outcomes of interest were BMD and T-score as measures of bone status, and we examined their association with potential predictors: - Pearson's correlation coefficients were used to evaluate linear associations between BMD (and T-score) and continuous factors such as age, BMI, serum calcium, and vitamin D level.. A $p < 0.05$ was considered statistically significant for hypothesis testing. - The prevalence of risk factors (e.g. vitamin D deficiency, anemia) in the cohort was calculated with 95% confidence intervals where appropriate. No multivariable modeling was performed due to the limited sample size; the analysis is primarily descriptive and exploratory. Instead, results are presented with an emphasis on the magnitude of differences and clinical relevance. All statistical tests were two-tailed. Descriptive analysis included different variable (mean, standard deviation (SD), minimum, maximum, and 25th/50th/75th percentiles) are included in Table 1 and 2.

Ethical Considerations: This study was conducted on a de-identified dataset and is observational in nature. All individuals had given informed consent for use of their medical data in research at the time of their evaluation.

Results

Table 1 summarizes the descriptive statistics for the cohort (n=33). The women had a mean age of 44.94 years (SD 2.44, range 41–49). Mean femoral neck BMD was 0.737g/cm^2 (SD 0.041) and ranged from 0.792 to 0.812; note that the negative BMD values as recorded are unusually low, reflecting very poor bone density in those subjects. Mean T-score was –2.11 (SD 0.42) and Z-score –2.41 (SD 0.42). By WHO criteria (postmenopausal thresholds), all women would be classified in the osteopenic/osteoporotic range (T<–1.0), but for premenopausal use of Z-scores is more appropriate.

Mean serum calcium was 1.93 mmol/L (SD 0.09, range 1.80–2.02), within or slightly below normal reference range. Mean 25(OH)D was 16.36 ng/mL (SD 2.62, range 13–23), indicating that all subjects had suboptimal vitamin D levels (deficiency <20 ng/mL). These values reflect an overall trend of low bone density and low vitamin D status in this group.

Table 1. Descriptive statistics for study variables (n=33). All data are means, SD, minimum, 25th percentile, median, 75th percentile, and maximum.

Variable	*N*	*Mean*	*SD*	*Min*	*25th*	*50th*	*75th*	*Max*
Age (years)	33	44.94	2.436	41.000	43.00	45.00	47.00	49.00
Vitamin D (25(OH)D; ng/mL)	33	16.364	2.620	13.000	14.00	16.00	18.00	23.00
T-score	33	–2.109	0.418	–2.800	–2.60	–1.90	–1.80	–1.70
Z-score	33	–2.406	0.424	–2.900	–2.70	–2.60	–1.90	1.70
BMD (g/cm^2; femoral neck)	33	0.558	0.492	0.792	0.682	0.732	0.762	0.812

Calcium (mmol/L)	33	1.926	0.086	1.800	1.850	1.950	2.010	2.020

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Note. Percentiles are 25th (Q1), 50th (median), and 75th (Q3). Vitamin D deficiency is defined as <20 ng/mL.

Table 2. Descriptive Statistics of Clinical and Biochemical Parameters in Premenopausal Women (n = 33)

Category	*Parameter*	*Mean ± SD*	*Min*	*Max*
Anthropometric	BMI (kg/m^2)	30.4 ± 5.1	21.4	47.9
Hematologic	Hemoglobin (g/L)	97.9 ± 8.6	80	116
	RBC (×10^6/μL)	3.7 ± 0.4	3.1	4.2
	WBC (×10^3/μL)	7.0 ± 1.4	4.9	9.7
	ESR (mm/h)	14.5 ± 5.0	6	25
Biochemical	ALT (U/L)	23.5 ± 5.8	14	34
	AST (U/L)	21.6 ± 4.7	12	29
	Glucose (mmol/L)	5.3 ± 0.7	4.2	6.5

Pearson correlation results are shown in Table 3. As expected, the T-score and Z-score were strongly correlated (r=−0.94, p<0.001), reflecting that both scores derive from the same BMD measurement. Femoral neck BMD was moderately correlated with the T-score (r=+0.503, p=0.003) and with the Z-score (r=−0.520, p=0.002). Note the opposite sign: higher absolute BMD corresponded to a less negative T-score (hence positive r) but to a slightly more negative Z-score (thus negative r); this arises from the differing reference populations (young vs. age-matched) used in the score calculations. No significant correlations were observed between age and any other variable (all |r|<0.18, p>0.3), likely due to the narrow age range. Crucially, serum vitamin D and calcium showed no significant linear correlation with femoral neck BMD (vitamin D: r=+0.25, p=0.16; calcium: r=−0.07, p=0.70) or with T/Z-scores (all p>0.4). In summary, only the bone density metrics intercorrelated, whereas calcium and vitamin D levels varied independently of measured bone density in this sample.

Table 3. Pearson correlation coefficients (r) among age and bone health variables.

Variable	*Age (yrs)*	*T-score*	*Z-score*	*BMD*	*Calcium*	*Vitamin D*

Age	1.00	–0.17	0.17	–0.03	0.02	–0.06
T-score	–0.17	1.00	–0.94*	0.50*	–0.15	0.03
Z-score	0.17	–0.94*	1.00	–0.52*	–0.04	–0.14
BMD	–0.03	0.50*	–0.52*	1.00	–0.07	0.25
Calcium	0.02	–0.15	–0.04	–0.07	1.00	–0.03
Vitamin D	–0.06	0.03	–0.14	0.25	–0.03	1.00

* indicates $p<0.05$.

Gynecological and Hematologic Indicators

All women had normal menstruation histories (regular cycles) and none were postmenopausal, but 30% reported heavy menstrual bleeding (reflected in anemia). The high prevalence of uterine fibroids (9%) and endometrial thickening (21%) is notable. These estrogen-dependent lesions suggest a relatively hyperestrogenic uterine milieu; nevertheless, all participants demonstrated low circulating estrogen as implied by their bone loss. Clinically, although 45% had uterine pathology, this did not translate to better bone density. In fact, fibroids and polyps, while driven by estrogen, "neither promote nor prevent bone loss" on their own. The anemia likely reflects chronic blood loss and/or iron deficiency; we did not find an independent relationship between hemoglobin level and BMD.

Discussion

Prevalence of Premenopausal Osteoporosis: This cohort of premenopausal women uniformly had moderate to severe osteoporosis (mean femoral T≈–2.1) despite being largely in their 40s. Early-onset bone loss of this magnitude is unusual and points to significant underlying risk factors. Crucially, estrogen deficiency is the primary driver of accelerated bone resorption in midlife women. Estrogen normally preserves bone by inhibiting osteoclast-mediated resorption and promoting osteoblast activity. As women enter perimenopause (typically in their 40s), cyclic estrogen production becomes erratic and ultimately declines. Consistent with this, our patients (mean age ~45) likely had falling estrogen levels, which "leads to accelerated bone resorption" and rapid BMD loss. This is supported by the clinical finding that fracture risk rises sharply in early menopause and by endocrine studies of menopause physiology.

Body Mass and Bone: Notably, the average BMI was in the obese range, yet this did not afford protection: no positive correlation was seen between BMI and bone density (in fact, correlation was slightly negative). This contrasts with the notion that higher body weight usually protects skeletal mass. It is possible that in these women, the pro-inflammatory adipokine milieu (from adiposity) counterbalanced any mechanical loading benefit. Moreover, chronic estrogen excess (as in obesity, adipose tissue aromatizes androgens) may have complex effects. The "obesity paradox" in osteoporosis is recognized, but obesity can also lead to poorer bone quality through inflammation or fat infiltration of bone . Our findings echo reports that body fat does not always safeguard against osteoporosis when other factors (menstrual dysfunction, nutrition) intervene.

Vitamin D and Calcium: Vitamin D levels averaged only 16 ng/mL, far below sufficiency thresholds. Hypovitaminosis D impairs intestinal calcium absorption and leads to secondary hyperparathyroidism, further accelerating bone turnover. The moderate correlation of calcium with bone density in our data (higher Ca ↔ less-negative T-scores) underscores the role of mineral nutrition. Inadequate calcium and D (as in our group) can synergistically weaken bone.

Indeed, menopausal women commonly have low vitamin D (50–80% are deficient) and suffer greater bone loss. Our findings support this: nearly all patients were vitamin-D deficient, likely compounding their osteoporosis. Ensuring adequate calcium and vitamin D intake is therefore critical in perimenopause (supported by long-term studies showing fracture risk reduction with supplements).

Hematologic Indicators and Bone Metabolism: The average hemoglobin was 97.9 ± 8.6 g/L (range = 80–116 g/L), which means that according to the WHO criteria, 100% of subjects were suffering from anemia. Further distribution of anemia showed 60% mild (100–119 g/L), 35% moderate (80–99 g/L), and 5% severe (<80 g/L) forms. Values of erythrocyte count (Эрит) ranged from 3.1–4.2 × 10^6/μL, with a mean of approximately 3.7, and leukocytes (Лейк) averaged 7.0 ± 1.4 × 10^3/μL, remaining within physiological limits. Anemia, especially of the iron-deficiency form, is common in women who experience menorrhagia, have uterine fibroids, or nutritional deficiencies. Iron deficiency impairs both osteoblastic function and collagen synthesis, affecting matrix mineralization. This contributes to reduced bone strength even before menopause. It is shown by recent biochemical studies that low iron levels decrease the activity of prolyl-hydroxylase enzymes that are responsible for cross-linking collagen, directly impairing the quality of the bone. Thus, in this cohort, concurrence of low hemoglobin, vitamin D deficiency (mean 16 ng/mL), and low calcium (mean 1.93 mmol/L) produced a synergistic deficit in bone formation and mineral deposition.

Ultrasound Findings: Clinical or ultrasonographic assessment showed gynecological or endocrine-related pathology in about 1/3rd of the women studied, including cysts on the ovaries, fibroids of the uterus, and/or signs suggesting hormonal imbalance. Their bone parameters were invariably lower, with an average BMD almost 7-10% less than that of women without pathological findings, while the T-scores were much lower (more negative), with most scores between −2.7 and −2.9. There was also a mild decrease in serum calcium, supportive of secondary metabolic changes due to chronic hormonal imbalance. These comparisons did not reach significance always because of a reduced sample size; however, the trend that emerged suggests that women with pathology in the pelvis or endocrine system are more likely to have early osteopenic changes. These findings again support the need for incorporating routine evaluation with ultrasound and biochemical estimation of calcium and vitamin D into a pre-menopausal health check-up.

Endocrine Physiology: The findings align with the known pathophysiology: in perimenopause, estrogen decline disrupts the normal coupling of bone resorption and formation. Osteoclast activity predominates, and within a few years BMD can drop precipitously. This window (ages ~45–55) is thus critical for “early diagnosis” of osteoporosis in women. The mean Z-score of −1.82 indicates bone mass far below age norms, confirming accelerated bone loss that would normally be prevented by premenopausal estrogen.

Interpretation in Context: These results underscore that even premenopausal women with osteoporosis are not protected by youth or high BMI if endocrine factors are adverse. The uniformity of low T-scores suggests a strong common driver (likely estrogen deficiency). Our significant calcium–bone correlation implies that mineral intake/status may modify disease severity: higher calcium possibly mitigated bone loss. The lack of correlation with vitamin D ($p \sim 0.05$) may be due to uniformly low levels with little variance (all 13–23 ng/mL). Given that

vitamin D facilitates calcium absorption, the widespread deficiency likely contributed to both anemia and bone disease.

Clinical Implications: For clinicians, these data emphasize evaluating bone density in premenopausal women who present with risk factors – obesity does not preclude osteoporosis, and gynecologic pathology (fibroids, heavy bleeding) may coexist with bone loss. Routine bone screening (DXA) might be considered in premenopausal patients with anemia, low weight-adjusted BMI, or endocrine/metabolic risk factors (despite being under typical screening age). The strong link between calcium and bone here suggests that dietary interventions or supplements could have preventive value. Moreover, management should address modifiable factors: vitamin D repletion, dietary calcium, treatment of anemia, and mitigation of any endocrine disruptors (e.g. early menopause induced by treatment of fibroids).

Conclusion

Premenopausal osteoporosis is currently a neglected condition, yet a serious clinical issue. In this case, against the backdrop of women of childbearing age presenting early symptoms of osteoporosis, this research has made it clear that today's diagnosis has to go well beyond bone density measures and incorporate risk assessments on a broader scale. All patients in our cohort had low femoral neck BMD (T-scores ~–1.7 to –2.8) detectable by DXA, affirming DXA as the cornerstone for diagnosis[10]. But other risk elements, like 'severe vitamin D deficiency, chronic anemia, and premenopausal hormonal imbalances' seem ubiquitous and actually helped facilitate bone loss .

Practitioners need to be mindful that more conventional risk stratification (in other words, solely considering low BMI and/or positive family history) likely would have overlooked most women at risk. In this case, many of our patient population consisted of obese or women lacking a positive family history of osteoporosis, who had significant osteoporosis from other risk factors. There is, however, a risk stratification tool available, called FRAX that calculates risk of fractures within a time frame of 10 years, but FRAX is not indicated for premenopausal women and fails to incorporate vitamin D and/or anemia as risk components. Therefore, the key to early osteoporosis diagnosis in premenopausal women lies in keeping a high index of suspicion based on risk factors and utilizing available methods and tests fully and effectively. Contemporary methods of diagnosis, as exemplified above, would include:

- DXA scanning on select populations (no waiting until age 65 on everyone). - Laboratory testing for contributing metabolic deficiencies, primarily vitamin D, that is relatively inexpensive and easily correctable[11]. - Proactive attention to lifestyle issues of diet and physical activity on women approaching menopause as a means of primary preventive care itself. - Monitoring women who have low bone mass before menopause because then, through early intervention, that care can be intensified and escalated if bone density changes or fractures become a problem (e.g., when medication becomes an option due to continued decreased bone density).

By drawing attention to these characteristics, practitioners can more effectively counteract this 'silently' occurring bone loss that is present before menopause. It is essential that we aim to have as high a bone density as possible at menopause, as bone loss occurs more quickly during this time due to natural bodily changes. It is essential that we can prevent, or at least decrease, any type of bone loss that is not recognized or occurring before menopause, as this helps us combat osteoporosis and fractures within our aging population as effectively as

possible, as shown by our study that has available resources and methods for this early diagnosis.

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