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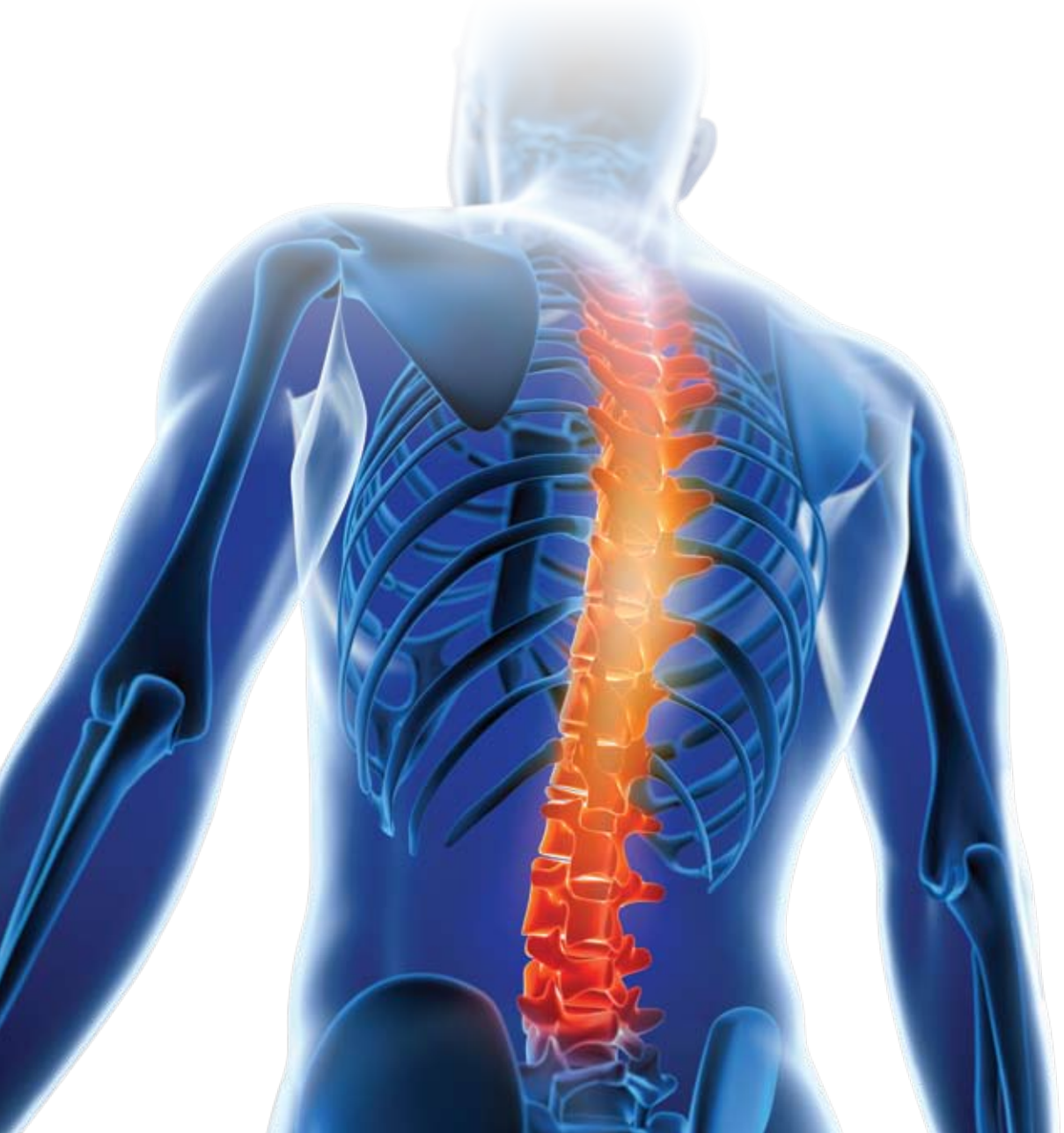

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TOO Times of Orthopedics®
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EDITORIAL

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Editor's Message

It gives us great pleasure and pride to pen the introduction of the twenty-fourth issue of Times of Orthopedics (TOO), a peer-reviewed journal.

The aim of this series is to have a mix of academic, scientific, and lifestyle-related articles to keep you up-to-date with the developments in our field. TOO is being launched with an aim to provide a rapid and reliable source of information in the mode of original articles, review articles, case reports, short communications, etc. in all areas of the field. TOO will focus predominantly on the areas of trauma, but wide and relevant aspects of bone tumors, pediatric orthopedics, foot and ankle surgery, joint replacements, metabolic bone diseases, etc. will also be covered. Although several important topics are mentioned, we believe TOO will not limit the consideration for publication of other allied topics, if found suitable to cover under the wide scope of this journal.

An enormous amount of work has gone into the development of this journal, and I believe you will see that effort reflected in this and future editions in the impact it will have on the readers. It has been an interesting start, many aspects of which our President has shared in his welcome notes. As we look at TOO, it is important to keep in mind that it represents the collective thinking of a group of innovative individuals at Science Integra with whom I am privileged to work. Firstly, we want TOO to be a premier academic journal in the engagement of our fraternity. We want it to look different, to be different, and to be one journal that, will be as dynamic as the work going on in our discipline, a rarity in academic publishing. Secondly, we want it to be a vehicle for a new type of conversation and have a place in academic review, tenure, and promotion. Lastly, we want TOO to lead the way in defining scholarship in the fraternity: scholarship in which faculty, students, and community members participate from idea to presentation. We do not know how, but we intend to get there.

Authors are encouraged to share their ideas and valuable research outcomes through this platform and to provide Indian readers with updated and most important information in this regard. We will work to make TOO not only a success in India but a platform to reckon across countries too. Those wanting to be a part of this winning team may share your papers by sending us an e-mail attachment to the editorial office at sushrutdsurgeon@gmail.com or production@scienceintegra.com

Role of Calcium Citrate Malate, Vitamin D, and Vitamin K in treating osteoporosis and associated fractures

Authors: Dr. Amit Ajgaonkar, Dr. Chetan Pradhan

Global and Indian burden of osteoporosis

Osteoporosis characterized by low bone mass and microarchitecture deterioration of bone is a systematic bone disorder, which leads to bone fragility and fractures.[1] Globally, over 200 million people are affected by osteoporosis and are diagnosed more often in women than in men. Nearly 9%–38% of women and 2%–8% of men in the developed countries are affected by osteoporosis.[2] As per the World health organization (WHO) statistics, one in eight men and one in three women are affected by osteoporosis in India.[1] Around 50 million people in India are either osteoporotic (T-score <2.5) or have low bone mass (T-score between -1.0 and -2.5).[3] Sun exposure, lack of physical activity, and deficiency of calcium, vitamin D, and vitamin K contributes to the development of osteoporosis.[1,4] However, an increase in vitamins and calcium intake may prevent the development of osteoporosis and fractures. Hence, the first step in any therapeutic strategy for osteoporosis will be the correction of calcium and vitamin insufficiency.[5]

Importance of calcium, vitamin D, and vitamin K for osteoporosis prevention and treatment

Calcium is the most required nutrient for bone health and maintenance.[4,6] It is essential for achieving optimal bone mass and preventing bone mass loss with age.[7] During growth and late menarcheal age, inadequate calcium intake can affect peak bone mass and may increase the risk of fracture in later life.[4] Therefore, obtaining the recommended doses of calcium will ensure peak bone mass development in adolescence and young adulthood.[6] Calcium supplementation has shown effectiveness in preventing osteoporotic fractures in postmenopausal women. A 30% fracture risk reduction with an intake of approximately 1000 mg/day of elemental calcium was reported in postmenopausal women with osteoporosis.[6] Moreover, long term treatment with calcium alone can lead to adverse effects like renal colic. Hence, calcium citrate is recommended along with vitamin D for the prevention or treatment of osteoporosis.[7]

Vitamin K is recognized as key nutrition in the optimization of bone health.[4] It exists in the following forms: Vitamin K_1 (phylloquinone) and vitamin K_2 (menaquinone).[8] Vitamin K_2 has an osteoprotective function such as promotes bone formation via stimulation of osteoblast differentiation increases the level of some bone formation markers (alkaline phosphate), regulates the extracellular matrix mineralization, upregulates the bone marker gene expression, and inhibits osteoclastogenesis.[9] Vitamin K_2 improves vertebral and low bone mineral density (BMD) in postmenopausal women with osteoporosis as compared to women without osteoporosis.[10] Both bone and cardiovascular health of patients with osteoporosis would benefit from vitamin K_2 intake.[8]

Vitamin D is an essential nutrient for the maintenance of bone health.[6] Stimulation of bone resorption and regulation of intestinal calcium absorption are the key functions of vitamin D.[6] It also enhances osteoclastic activity, influences differentiation of bone cell precursors, stimulates bone matrix formation, and bone maturation.[4] Vitamin D deficiency is highly prevalent in osteoporotic patients than in normal subjects. Thus, treatment with vitamin D improves bone density indices and reduced the incidence of osteoporosis.[11] In addition, vitamin D supplementation in patients with osteoporosis may improve BMD, decreases bone turnover, and reduces the risk of fragility fracture, bone resorption, and subsequent bone loss.[6,12]

Osteoporosis and risk of fracture

Osteoporotic fractures most commonly occurs in the hip, vertebrae, and wrist[13] Osteoporotic fractures impose a greater financial, medical, and socioeconomic burden on society and are associated with substantial disability, pain, and reduced quality of life.[13,14]

Vertebral fracture accounts for nearly 50% of osteoporotic fractures. Around 5% of men and 12.1% of women are affected by vertebral fracture.[14] Vertebral fractures are also associated with 4.4-fold increased risk of mortality.[13]

Hip fracture affects more than 1/3rd of individuals with osteoporosis in inpatient and outpatient care.[4] The lifetime risk of a hip fracture is estimated to be one in five in women older than 50 years.[13]

Wrist fracture affects around 41,000 of individuals among 180,000 osteoporosis-related symptomatic fractures. Patients at initial fracture are at greater risk of developing subsequent fractures. For instance, a woman with a vertebral fracture has an increased relative risk (RR) of 1.4 for a wrist fracture.[13]

Combination of calcium, vitamin D, and vitamin K: Basis of preventive and therapeutic regimens for osteoporosis

Effects of combination on fractures, bone density, and serum PTH

The effective prevention of fractures in women and men with calcium-vitamin D combination is of particular interest, as calcium and vitamin D insufficiencies inducing secondary hyperparathyroidism have extended from the elderly population to the younger population. Two controlled studies performed in elderly women and men have demonstrated a significant reduction in hip and other non-vertebral fractures with a combined supplement of calcium and vitamin D. Also, a significant increase in BMD

Role of Calcium, Vitamin D, and Vitamin K in osteoporosis and fractures

Calcium — Effective in the preventive treatment of osteoporosis (≥ 50 years of age)[6]

- Reduces the risk of non-vertebral fractures[15]
- Capable of inducing a decrease in serum parathyroid hormone (PTH) in postmenopausal women[5]
- Capable of one-third reduction in all type of fracture[5]
- Prevent bone loss and fracture in people 50 years old[11]
- In a study, calcium alone caused a positive mean percentage BMD change from baseline of 2.05% for total body bone density, 1.9% at the distal radius, 1.66% lumbar spine, and 1.6% at the hip . Also, a reduction in vertebral fractures, with relative risk reduction of 0.79 (95% Confidence interval (CI) 0.54 to 1.09) was obserbed with calcium supplement[6]

Vitamin D — Effective in the prevention of osteoporosis and recurrence of osteoporotic fractures[16]

- Reduces falls in elderly populations, as well as hip, vertebral, and nonvertebral fractures[7]
- Adequate intake contribute to bone and muscle health and thereby reduces the risk of fragility fracture[17]
- Improve BMD and decreases bone turnover in patient with osteoporosis[12]
- In a study, vitamin D contributes to 37% reduction of vertebral fractures in women with postmenopausal osteoporosis[6]
- In a study, vitamin D at a dose of 700 to 800 units per day reduced the relative risk of 26% of hip fracture and 23% of nonvertebral fracture[6]

Vitamin K — Speed fracture healing[5]

- Plays a role in gamma-carboxylation of bone-specific Gla-containing proteins mainly osteocalcin, matrix Gla protein (MGP) and protein S[5]
- In a study, 45 mg/day of vitamin K_2 contributes to 1.3% increase in metacarpal BMD in osteoporotic patients vs 3.8% decrease in control group[5]
- Vitamin K ranked 6th among 18 interventions for lumbar spine, indicating that vitamin K supplementation can increase lumbar spine BMD[10]
- Vitamin K_2 can improve vertebral BMD in postmenopausal osteoporosis[10]

Synergistic interplay of calcium, vitamin D, and vitamin K

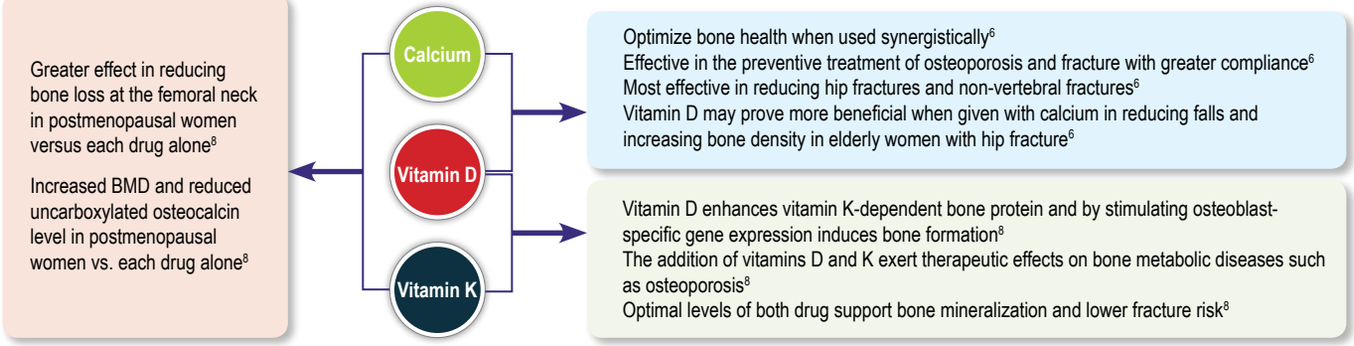

Table 1. Effects of calcium and vitamin D supplementation on fractures and serum PTH							
	Daily dose		Duration (years)	Population (mean age)	Mean Ca intake/ day (mg)	Effects on fractures	Change in sPTH (%)
	Ca (mg)	Vit D (IU)					
Chapuy	1200	800	3	3270 F (84)	512	fewer hip and other non-vertebral fractures (p <0.02)	– 47
Dawson-Hughe	500	700	3	380 F+M (71)	725	fewer non-vertebral fractures (p <0.02)	– 33 (in F)
sPTH: serum parathyroid hormone, Ca: calcium, vit: vitamin D, F: females, M: males							

and a decrease in serum parathyroid hormone (PTH) has been reported with a combined supplement of calcium and vitamin D, Table 1.[5]

Similarly, in another study patients who received a combination of calcium and vitamin D showed a reduction in the risk of hip fractures by 19% (Hazard ratio (HR), 0.81; 95% CI, 0.71 to 0.93), and non-vertebral fractures by 5% (HR, 0.95; 95% CI, 0.90 to 1.00).[15] Also, drug studies of antiresorptive and anabolic agents and strontium ranelate demonstrated a reduction in risk of osteoporotic fractures in patients taking calcium and vitamin D supplements.[7]

Also, studies showed a complementary effect of vitamin K_2 to vitamin D and calcium supplements on reducing bone loss and increasing BMD versus vitamin D and calcium alone.[8] In a study, Vitamin K_2 in combination with calcium and vitamin D is associated with a significant increase in ultra-distal BMD and bone mineral content.[4] Furthermore, in elderly patients with Alzheimer's disease, treatment with vitamins D and K with calcium increased BMD and led to the prevention of non-vertebral fracture [odds ratio: 7.5 (95% CI 5.6, 10.1)].[8] Therefore, calcium citrate combined with vitamin D and vitamin K_2 is the combination of choice for the prevention or treatment of osteoporosis and associated fractures.[4,7,8]

> **Combining Vitamin K_2 with Calcium and Vitamin D may help prevent long-term complications associated with calcium intake**

Summary

- Deficiency of calcium, vitamin D, and vitamin K contributes to the development of osteoporosis.
- Adequate intake of vitamins and calcium may prevent osteoporosis and fractures. Hence, the first step in any therapeutic strategy for osteoporosis will be the correction of calcium and vitamin insufficiency.
- A 30% fracture risk reduction with an intake of approximately 1000 mg/day of elemental calcium was reported in postmenopausal women with osteoporosis.
- Vitamin D supplementation in patients with osteoporosis improves BMD, decreases bone turnover, and reduces the risk of fragility fracture, bone resorption, and subsequent bone loss.
- Vitamin K_2 has osteoprotective functions, it promotes bone formation via stimulation of osteoblast differentiation, increases the level of bone formation markers, regulates the extracellular matrix mineralization, upregulates the bone marker gene expression, and inhibits the osteoclastogenesis.
- Studies showed a complementary effect of vitamin K_2 to vitamin D and calcium supplements on reducing bone loss and increasing BMD versus each drug alone. Hence, calcium citrate, vitamin D, and vitamin K_2 is the combination of choice for the prevention or treatment of osteoporosis and associated fractures.

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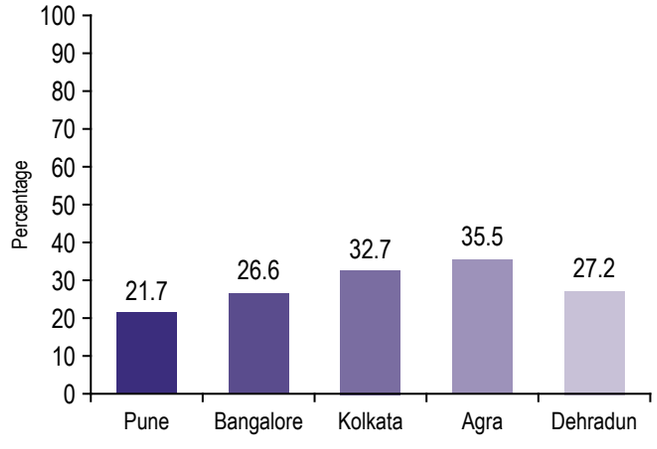
Muscle force output and movement variability: An important outcome variables of knee osteoarthritis and TKA procedure

Authors: Dr. Sushrut Babhulkar, Dr. Farheen Rana

Indian burden of knee osteoarthritis

Knee osteoarthritis (OA) associated with pain and disability is the most highly prevalent condition in India causing a significant burden on society, particularly in the elderly population. In India, the overall estimation of knee OA is reported to be 28.7%. However, the prevalence varies slightly according to individuals states of India (Pune, Bangalore, Kolkata, Agra, and Dehradun), Figure 1. Nearly, 31.6% of women and 28.1% of men are affected by Knee OA in India. According to the Indian study, the prevalence of OA knee increases with increasing body mass index (BMI), significantly more in obese patients (33%) than underweight (28%). Knee OA is also highly prevalent in patients who do not exercise vs. who regularly exercise (82.9% vs. 36%, respectively), who used western toilet vs. Indian toilet (42.1% vs. 29.7%). According to radiographic (X-ray) diagnosis of OA, 78.8% of patients with OA had pain sometime in the past 5 years, 57.9% had pain in the last 3 months, and 35.8% experienced knee stiffness at least once in the last 3 months.[1]

Figure 1 Prevalence of OA-Knee in India (City wise)



Muscle force output and movement variability in knee OA and after TKA

Muscle force output and movement variability is the important outcome variable that not only conveys the effects of pathological conditions such as knee OA but also surgical interventions such as total knee arthroplasty (TKA).[2]

Muscle force output variability

Knee OA associated with pain and loss of motion often results in restricted activity, decreased neuromuscular control, impaired proprioceptive acuity, and loss of independence during daily living activities. In knee OA and following TKA, the decreased strength in quadriceps impairs the ability to perform functional tasks, requiring adequate muscle strength and motor control.[2] Also, neuromuscular activation deficits with decrease in proprioception and kinesthetic awareness contribute to these strength deficits, slower movement patterns, as well as reduced force steadiness, both prior to and post TKA. Such adaptation reduced the ability to exert a steady force output during submaximal activities, such as those needed during daily living activities, as well as greater variability in movement patterns. These variabilities in the muscle output and the movement patterns affects the outcome of OA treatment as well as the TKA procedure, in terms of movement and performance of activities. However, understanding how these impairments are altered following TKA can modify

these variables to attain beneficial effects in post-TKA rehabilitation.[2]

In knee OA patients, different neural mechanisms can be explained by complex innervation strategies that contribute to quadriceps arthrogenic muscle inhibition (AMI) and lower extremity muscle force steadiness (MFS).

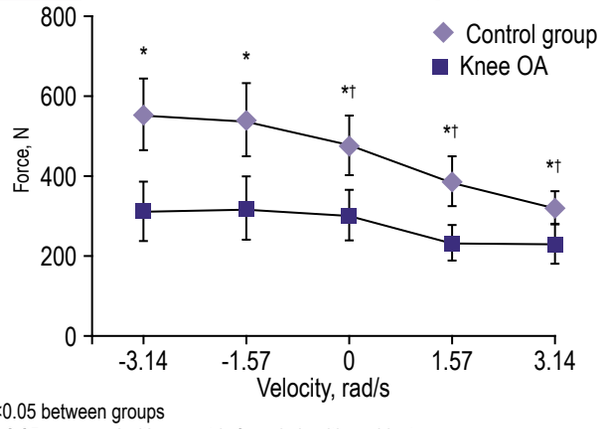
- **AMI:** Quadriceps AMI causes an altered neuromuscular control, loss of ability to control motor output, and hence inability to fully activate the quadriceps muscle.
- **Lower extremity MFS**: The lack of lower extremity MFS indicates impairment while performing functional tasks such as walking endurance, chair rising, and stair climbing.[2]

Muscle output variability in older adults with knee OA

In a study conducted by Hortobágyi et al, the distribution of errors in knee joint proprioception, quadriceps force accuracy, and steadiness and muscle strength was evaluated in patients with knee OA (n = 20) vs. control group (n = 20). The researchers assessed the quadriceps force accuracy and steadiness during a force target-tracking task and the maximal voluntary quadriceps force during eccentric, isometric, and concentric contractions.

- Knee OA subjects needed 67% more time to complete functional tasks, produced 82% more proprioception errors, and 89% more errors in accurately matching target forces during submaximal quadriceps contractions compared to control group (all p<0.05).
- Knee OA subjects had 155% more force variability than controls, with eccentric contractions (15.5N) being particularly unsteady compared with concentric contraction (8.5N; p<0.05)
- OA patients had especially weakened (76%) ability to produce maximal voluntary eccentric strength than isometric and concentric conditions (56%), Figure 2
- Overall, compared to the control group, knee OA patients produced 63% less quadriceps force (p<0.05).

Figure 2 Maximal isokinetic quadriceps force in subjects with knee OA and control group



*p<0.05 between groups
†p<0.05 compared with eccentric force in healthy subjects
‡p<0.05 compared with isometric and eccentric force in OA patients
N=Newtons; s=seconds. Error bars show SD

In conclusion, this data suggest that OA patients needed more time to complete tasks, and have an impaired ability to accurately produce maximal strength and submaximal quadriceps forces.[3]

> **In knee OA patients, more time is needed to complete functional tasks, with higher proprioception errors, more errors in accurately matching target forces, and more force variability.**

Muscle output variability in older adults following TKA

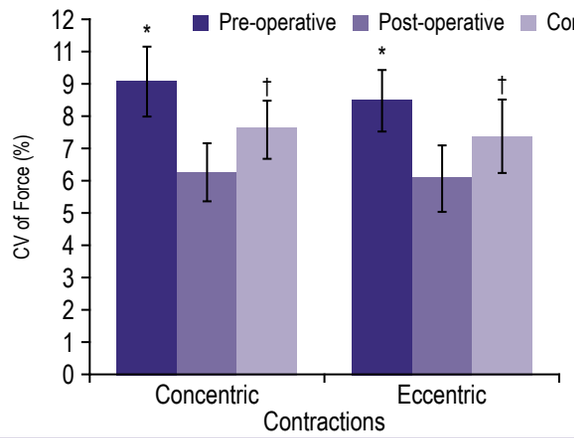
A study evaluated MFS of submaximal quadriceps force output in patients with knee OA before and at 6 months after TKA (n = 13) and compared to the group of age-matched controls with native knees (n = 11). The muscle steadiness was compared based on the muscle strength, maximal voluntary isometric contraction (MVIC), and quadriceps MFS during anisometric eccentric and concentric contractions at 50% maximum volume contraction.

- Pre-operatively, quadriceps MFS for both concentric (9.88% ± 1.13% vs. 8.29% ± 0.91%; p=0.005) and eccentric contractions (9.23% ± 1.18% vs. 8.03% ± 1.17%; p=0.045) was significantly higher in the TKA group relative to controls.
- Post-operative MFS in the TKA group for both concentric (6.82% ± 0.98% vs. 8.29% ± 0.91%; p=0.005) and eccentric contractions (6.62% ± 1.08% vs. 8.03% ± 1.17%; p=0.017) of quadriceps was significantly lower than control group.
- MVIC relative to BMI (MVIC/BMI) was significantly lower in the TKA group both preoperatively (6.64 ± 2.87 N/BMI;

p<0.001) and post-operatively (7.84 ± 2.31 N/BMI; p=0.001) compared to controls (12.57 ± 3.86 N/BMI).

- Comparison between pre- and post-operative visit showed a significant improvement in coefficient variation (CV) of MFS on the surgical leg for both concentric and eccentric contractions, figure 3.[4]

Figure 3 Comparison of CV of MFS between pre-operative and post-operative visit for concentric an eccentric contractions in TKA vs. controls groups.



In conclusion, MFS was significantly lower in the TKA-GROUP post-operatively, indicating a significant improvement between the pre- and post-operative visits.

"In older adults after TKA, the pre-operative force steadiness in concentric and eccentric contractions of the quadriceps was significantly higher than in the controls. However, post-operatively quadriceps force steadiness for both concentric and eccentric contractions was significantly lower in the OA group compared to controls."

Muscle movement variability

The movement variability in OA is suggestive of a pathological or impaired condition with reduced function and future risk of mobility deficits. The movement variability is seen clinically as variability in gait or level walking variability.[2] Lower extremity activities analysis while walking at different speeds and under difficult conditions can help in identifying specific patterns of gait associated with knee OA [5]

Movement variation in older adults with knee OA

In older adults with knee OA, the OA affected the kinematics and kinetics of the knee.[6] The severity of the OA also determines the degree of variability of gait characteristics. Ko SU et al, examined knee OA associated gait characteristics during walking conditions (usual-walking, fast-walking, and usual-walking-after-30 min) and also during various challenging gait tasks encountered in daily life among older adults. They observed that knee range of motion was lower for knee-OA patients in the fast-walking and usual-walking-after-30 min tasks compared to non-OA controls. (p<0.030). Also, lower gait speed and shorter stance period were observed in knee-OA patients in usual-walking and usual-walking-after 30-min compared to non-OA controls. In the sagittal plane, Knee-OA participants had greater absorptive mechanical work expenditures (MWEs) of the knee for the usual-walking and fast-walking tasks compared to non-OA controls. (p=0.151). While, in the frontal plane, peak moment from the knee joint of knee-OA was greater for the usual-walking and usual-walking-after-30 min than non-OA controls. Hence, the study concluded that older adults with knee-OA walked slower in usual-walking and usual-walking-after-30 min tasks and had a slower gait speed and shorter stance for all three walking tasks than non-OA controls.[5]

"Older adults with knee OA walk slower and have lower gait speed and shorter stance for all walking task (usual-walking, fast-walking, and usual-walking-after-30 min) compared to non-OA controls."

Muscle movement variation in older adults following TKA

Fallah-Yakhdani et al examined the determinant of contractions during gait in patients with knee OA before and 1 year after TKA (n = 14) compared to healthy peers (n =12) and young subjects (n = 15).

Study results:

- Pre-operatively, the variability of sagittal plane knee movements (measured in degree/second) increased with speed. Pre-operatively, the patients' affected and unaffected legs were less variable than those of the young controls and the affected leg was less variable than the healthy peers.
- Post-operatively, the variability in the knee OA group was further decreased to a level significantly below both control groups.[7]

> **Sagittal plane knee movement variability in individuals with OA after TKA suggest that patients' affected and unaffected legs, pre-operatively were less variable than those of the young controls and healthy peers. And, post-operatively, the variability in the knee OA group was decreased to a level significantly below both control groups (unaffected knee and healthy control).**

Summary

Knee OA associated with pain and disability is the most highly prevalent condition in India causing a significant burden on society, particularly in the elderly population. Knee OA often results in restricted activity, decreased neuromuscular control, impaired proprioceptive acuity, and loss of independence during daily living activities.

In knee OA, the decreased strength in quadriceps impairs the ability to perform functional tasks requiring adequate muscle strength and motor control.

In knee OA patients, the lower extremity steadiness, higher force variability, and increased time to complete functional tasks can be explained by complex innervation strategies that cause quadriceps AMI and a lack of lower extremity MFS.

Quadriceps AMI causes an altered neuromuscular control, loss of ability to control motor output, and hence inability to fully activate the quadriceps muscle.

The lack of lower extremity MFS indicates impairment while performing functional tasks such as walking endurance, chair rising, and stair climbing.

In knee OA, a decrease in proprioception and kinesthetic awareness results in neuromuscular activation deficits. This variation in the muscle output and the movement patterns affects the outcome of OA treatment as well as the TKA procedure, in terms of movement and performance of daily activities. However, understanding how the modification of the impairments can alter the outcome of TKA may benefit in post TKA rehabilitation.

Clinical studies on muscle force output in individuals with knee OA after TKA showed a higher MFS, pre-operatively, and lower MFS postoperatively, indicating a significant improvement between pre-operative and post-operative visits.

Clinical studies on muscle movement variability in individuals with OA after TKA suggest that patients' affected and unaffected legs, pre-operatively were less variable than those of the young controls and healthy peers. And, post-operatively, the variability in the knee OA group was further decreased to a level significantly below both control groups (unaffected knee and healthy control).

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Clinical risk factors and assessment of fracture risk in osteoporosis

Author: Dr. Sanskriti Babhulkar

The burden of osteoporotic fractures

Osteoporosis is a common disorder characterized by low bone mass in addition to microarchitectural disruption and skeletal fragility.[1] Osteoporosis increases the risk of fracture, predominantly at the spine, hip, humerus, pelvis, vertebra, and distal forearm (wrist).[1,2] Approximately 10 million osteoporotic fractures occur worldwide annually.[2] In developed counties, 180,000 annually are estimated to be affected with osteoporosis-related symptomatic fractures. Of these, 70,000 are associated with hip fractures, 25,000 with clinical vertebral fractures, and 41,000 with wrist fractures.[3] While in India, around 21 million osteoporotic fractures (332,000 - hip fractures) occurred annually.[2] The consequences of fractures include limited ambulation, depression, loss of independence, and chronic pain. Osteoporosis, in addition to being the major cause of fractures, also render patients bedridden with serious complications.[1]

Osteoporosis and fragility fractures: Risk assessment

The risk of osteoporosis and fragility fracture depends on several independent risk factors. The increased risk of fractures in patients with osteoporosis is either due to factors that predominantly cause a reduction in bone mineral density (BMD) or factors that are completely independent of BMD, such as bone quality (bone geometry, microstructure, and turnover) and extraskeletal factors. Subjects with multiple risk factors are more at risk of fracture than those with a single risk factor, including an isolated reduction in BMD.[4]

The risk of fractures is also known to increase with non-BMD related factors like increasing age, history of the previous fracture, falls, glucocorticoid therapy, smoking status, and many more, Table 1.[1,4] The presence of comorbidities, including chronic inflammation, impairment of bone quality, decreased mobility, increased risk of falls, and vitamin D deficiency increases the risk of fracture, and genetics exert a strong influence on BMD and bone microarchitecture.[4] The assessment of microarchitecture requires a bone biopsy, micro-computed tomography (microCT), or micro-magnetic resonance imaging (microMRI), which are not routinely practiced in clinics. Incorporation of these non-BMD risk factors increases the sensitivity of fracture risk assessment and therefore, improves intervention strategies.[1]

Table 1. Risk factors for osteoporosis and fragility fractures[4]

BMD
Age
Fragility fractures after 40 years of age
Family history of fragility fractures
Glucocorticoid therapy
Premature menopause (< 45 years)
Low body weight
Age of menopause
Reduced calcium intake
Reduced physical activity
Smoking
The propensity to fall (such as physical disability)
Alcohol consumption
Risk factors for falls
Vitamin D deficiency
Drugs (benzodiazepines or diureticsor)

Bone mineral density: BMD is the most important assessment parameter for the diagnosis of osteoporosis and fragility fracture risk.[4] BMD is influenced by genetic, nutritional, and coexisting diseases and it depends on peak bone mass and bone loss due to menopause and aging. Many studies indicate a 1.5–3.0 fold increase risk of fracture for each decrease in BMD of a standard deviation (SD).[4] World Health Organization (WHO) defined osteoporosis in postmenopausal Caucasian women as a value for BMD that lies more than 2.5 SD below the average value for young healthy women (a T-score of ≤ 2.5 SD). Severe osteoporosis i.e. established disease also fits into the same threshold, but with one or more prior fragility fractures. Hip is the preferred site for diagnostic BMD measurement. The diagnosis of osteoporosis in men utilizes the same BMD threshold as used in women, since for any given BMD, the age-adjusted fracture risk is similar in both the sexes.[5]

Dual-energy X-ray absorptiometry (DEXA) is the most widely validated technique used for the measurement of BMD.[6]

The ability of a bone mass measurement to predict fracture depends on its accuracy, which is used for the prognosis of the disease. The accuracy of BMD in predicting fracture is improved by site-specific measurements (Table 2). When the BMD is normal, there is only a reduced risk of fracture, but not complete eradication of the risk that a fracture would not occur.[5]

Table 2. Age-adjusted relative increase in risk of fracture (with 95% CI) in women for every 1 SD decrease in BMD (absorptiometry) below the mean value for age (from 19)

Site of measurement	Forearm fracture RR (95% CI)	Hip fracture RR (95% CI)	Vertebral fracture RR (95% CI)	All fractures RR (95% CI)
Distal radius	1.7 (1.4–2.0)	1.8 (1.4–2.2)	1.7 (1.4–2.1)	1.4 (1.3–1.6)
Femoral neck	1.4 (1.4–1.6)	2.6 (2.0–3.5)	1.8 (1.1–2.7)	1.6 (1.4–1.8)
Lumbar spine	1.5 (1.3–1.8)	1.6 (1.2–2.2)	2.3 (1.9–2.8)	1.5 (1.4–1.7)

CI: Confidence interval, SD: Standard deviation; RR: Relative risk; BMD: Bone mineral density

Age: Age contributes independently to the risk of fractures, irrespective of BMD.[4] The risk of fractures for any BMD measurement, is much higher in the elderly than in the young population. The association of fracture risk with age is due to a deterioration in biomechanical factors (bone architecture and bone quality), as well as age-related increase risk of multiple falls, reduction in BMD of the proximal femur, and increased comorbidities in the elderly.[4,7] Tables 3 represent the association between age, BMD, and fracture probability at the hip in both genders. At the BMD threshold for osteoporosis (T-score: -2.5 SD), the 10-year probability of hip fracture in men and women ranges from 1.4%–14.2% depending on age.[5]

Glucocorticoid therapy: Several drugs are linked to an increased risk of osteoporosis and fragility fracture. Among these, glucocorticoid therapy is the most common cause of secondary osteoporosis. Around 30%–50% of patients treated with long-term glucocorticoid therapy are associated with fragility fracture.[4]

History of fragility fracture: History of fragility fracture is an important risk factor for future fractures. The risk of fracture is approximately doubled with a history of fragility fracture. The increase in risk is marked for a vertebral fracture in the presence of a previous spine fracture. For instance, the presence of two or more prevalent vertebral fractures was associated with a twelve-fold increase in the risk of fractures for any given BMD.[5]

BMI: Low body mass index (BMI) is an important risk factor for fractures, with marked risk in lean individuals (BMI <20 kg/m^2). Increments in weight to BMI >20 kg/m^2 have a little protective effect. It is important to note that although leanness is a risk factor for fracture, conversely, obesity is not a protective factor. This association of fracture risk with lean body mass is dependent on BMD.[5]

Hormonal factors — gender differences: The peak bone mass attained in women is lower than in men. The incidences of hip fractures in women at any age, is double the incidences in men at that age. This is because of increased bone loss in women after menopause. Hormonal factors that contribute to increased fracture risk include premature menopause, primary or secondary amenorrhea (as from female athlete triad or anorexia nervosa), hyperthyroidism, hyperadrenocorticism, and primary and secondary hypogonadism in men.[7]

Risk factors for falls: Risk factors for falls such as musculoskeletal and neuromuscular impairment, impaired

Table 3. Ten-year probability of hip fracture in both men and women according to age and BMD at the femoral neck

Age (years)	Men				Women			
	Population	T-score			Population	T-score		
		−1	−2.5	≤ −2.5		−1	−2.5	≤ −2.5
45	0.5	0.7	2.2	3.4	0.4	0.4	1.4	2.2
50	0.8	1.1	3.4	5.1	0.6	0.5	1.7	2.9
55	0.8	0.9	3.1	4.9	1.2	0.7	2.9	4.9
60	1.2	1.2	3.7	6.0	2.3	1.1	4.4	7.8
65	2.1	1.9	5.3	8.8	3.9	1.5	5.9	11.3
70	3.4	2.7	8.5	14.3	7.3	2.0	8.8	18.3
75	5.9	4.1	14.2	24.2	11.7	2.3	11.1	24.6
80	7.6	4.6	13.7	24.3	15.5	2.5	11.5	27.9
85	7.1	7.6	10.5	19.9	16.1	2.1	10.0	25.8

BMD: Bone mineral density

visual acuity, hearing loss, dementia, depression, stroke-related impairment, and vitamin D deficiency plays a key role in the occurrence of fractures, especially in elderly. Over 80% of nonvertebral fractures are associated with falls.[4]

Lifestyle risk factors: Adequate calcium intake is important for the growth and maintenance of bone mass. Vitamin D is an essential nutrient for intestinal absorption of calcium. Therefore, the lifestyle risk factors for osteoporotic fractures include physical inactivity or sedentary lifestyle, low dietary calcium intake, and vitamin D deficiency, in addition to cigarette smoking, caffeine intake, and excessive consumption of alcohol.[7]

Risk assessment charts

Though low BMD is the main risk factor for fragility fractures, it should not be considered alone when defining the overall fracture risk and the single intervention threshold. For a given T-score, other factors such as age may increase the risk of fracture. To better define the risk for fracture, specific algorithms such as Garvan calculator, the QFracture, and FRAX® (incorporate several risk factors in addition to age) have been developed. FRAX® is the most extensively employed and validated tool used in defining postmenopausal osteoporosis or other types of osteoporosis.[4]

SIOT recommendation statements for osteoporotic fracture risk assessment

Italian Society for Orthopaedics and Traumatology (SIOT) recommendation statements for osteoporotic fracture risk assessment is depicted in below toolbox.[4]

Toolbox for guidance-Risk assessment

- Consider clinical risk factors when evaluating the risk of osteoporotic fractures.
- BMD (hip) as measured by DEXA can be used to refine this risk.
- FRAX® method is used in defining the individual 10-year probability of major fracture (vertebral, hip, forearm, humerus) and hip fracture in both sexes.
- Postmenopausal women with single or multiple risk factors should be evaluated for fracture risk.
- Patients with vertebral fractures or previous low-trauma fractures are identified as being at high risk of fracture regardless of the BMD measurement or the presence of other risk factors.[4]

Summary

- Osteoporosis increases the risk of fracture, predominantly at the spine, hip, wrist, humerus, and the pelvis.
- The risk of fracture increases with factors that predominantly cause a reduction in BMD or factors that are completely independent of BMD.
- Factors independent of BMD that contribute to fracture risk includes age, prior fragility fracture, premature menopause, a family history, and the use of glucocorticoid therapy. Since several of these risk factors are partly dependent on BMD, their use in conjunction with BMD improves the sensitivity of fracture prediction.
- To better define the risk for osteoporotic fractures, specific algorithms such as Garvan calculator, QFracture, and FRAX® have been developed. Among these, FRAX® is the most extensively employed and validated tool used in osteoporosis.
- According to SIOT guidelines, clinical risk factors should be considered when assessing the risk of osteoporotic fractures.

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What's New!!!

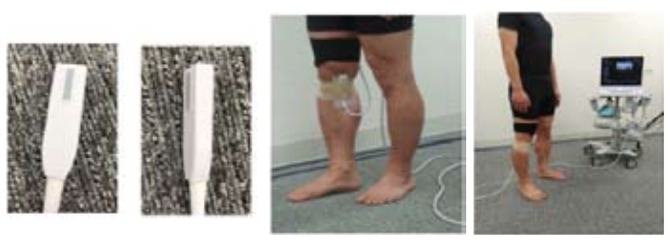
Flat-shaped ultrasound transducer: A useful tool in visualizing actual meniscal movements in knee osteoarthritis

Ishii Y, Nakashima Y, Ishikawa M, et al. Dynamic ultrasonography of the medial meniscus during walking in knee osteoarthritis. Knee. 2020; 27(4): 1256-62.

A study demonstrated flat-shaped ultrasound transducer, a useful tool in visualizing actual medial meniscal movements during walking in patients with primary unilateral or bilateral knee osteoarthritis, Figure 1. In this study, medial meniscal extrusion (MME) and ΔMME (minimum and maximum MME difference during the stance phase of the gait cycle) was evaluated in patients with knee OA (n = 6) using novel ultrasound transducer and compared with healthy volunteers (n = 6).

- In both groups, MME in the stance phase was clearly visualized using ultrasound transducer.
- The mean values of MME during early, middle, and late stance phase is reported to be 5.2 ± 2.0, 5.7 ± 2.0, and 5.1 ± 2.0 in the knee OA group vs. 1.1 ± 0.5, 1.4 ± 0.5, and 1.0 ± 0.2 in the control group, respectively, indicating a significantly greater MME in the OA group vs. control group ($p<0.01$).
- Also, the mean values of ΔMME in the stance phase were significantly greater in the knee OA group vs. control group. (1.5 ± 0.3 vs. 0.8 ± 0.1 mm, respectively; $p <0.01$).

Figure 1 Special linear-array transducer and setting.



(a). Specially designed linear-array transducer with a flat interface. (b) Medial aspect of the right knee after fixing the transducer. (c) The participant along with the transducer and long cable.

Standardized out-patient protocols for managing osteoporosis in this COVID-19 era

Zou J, Song D-W, Niu J-J, et al. Standardized out-patient diagnosis and treatment process for osteoporosis clinics during the COVID-19 pandemic. Eur Rev Med Pharmacol Sci. 2020; 24(10): 5778–82.

The novel COVID-19 pandemic is still serious in the global world. The osteoporosis clinic has now become a new hotspot for coronavirus infection. Hence, a standardized out-patient protocol is necessary to ensure that osteoporosis patients and medical staff receive safe and effective treatment. The following suggestions should be strictly followed in order to prevent and control osteoporosis in an orthopedic clinic during this pandemic.

- Use of specialized diagnosis and treatment techniques such as prevention techniques for outpatient medical staff, increasing awareness of COVID-19 prevention, and strictly screening patients with COVID-19 infection in the outpatient care.
- The outpatient medical staff should wear eye patches or goggles, isolation clothing, latex gloves, and surgical masks correctly.
- Use hand disinfection before and after wearing the mask and gloves.
- Doctors must conduct one-to-one diagnosis and treatment in the waiting area to avoid crowd gathering.
- If the patient is a suspected case of COVID 19, immediately isolate for single room treatment.
- Insistent administration of anti-osteoporosis drugs during outbreaks.
- Home prevention including keeping windows open, sunlight exposure, regular exercise, and provide enough protein and calcium supplements.
- Use online platforms for follow-up and evaluation of osteoporosis.

PRECAUTIONS FROM EXTERNAL FACTORS IN DAILY LIFE

Precautions from external factors in daily life

After your surgery, you should inform any other doctor that you visit, like your dentist, that you have an artificial joint.

These joints are at a risk of bacterial infection introduced by any invasive procedures such as surgery, dental or gum work, urological and endoscopic procedures, or from infections elsewhere in the body.

A. Driving

- Avoid driving till you recover fully. If your right knee was replaced, avoid driving for 6 to 8 weeks. Remember that your reflexes may not be as sharp as those before your surgery.

B. Metal detectors

- The sensitivity of metal detectors vary and it is unlikely that your prosthesis will cause an alarm. You should carry a medical alert card indicating you have an artificial joint.

C. Sleeping positions

- You can safely sleep on your back, on either side or on your stomach.

D. Return to work

- Depending on the type of activities you perform, it may be 6 to 8 weeks before you return to work.

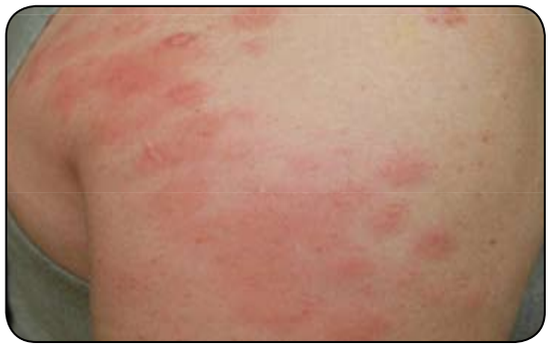
E. Other activities

- Avoid activities that put stress on the knee. These activities include playing tennis, badminton, football, baseball, jumping, squats, skiing and jogging. Do not lift anything heavy.

WHIZ

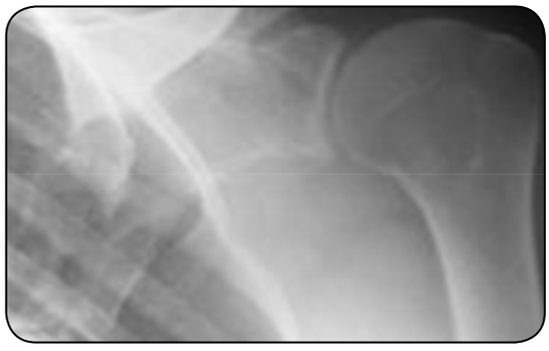
1. A 26-year-old woman after two-week of arthroscopic rotator cuff repair presented for follow-up. She complaints of mild pruritic eruption that begins after one week of surgery, which was gradually worsening. What is the most likely diagnosis?

A. Cellulitis ☐
B. Cold panniculitis ☐
C. Cutaneous lymphoma ☐
D. Sweet syndrome ☐
E. Urticaria ☐



2. A 47-year-old man presented with progressive osteolysis of the left shoulder girdle. He had a history of left clavicle and scapula fracture caused by a fall. Loss of tactile sensation of the bony structure and muscle atrophy was observed during the physical examination around the shoulder girdle. What is the most likely diagnosis?

A. Angiosarcoma ☐
B. Osteomyelitis ☐
C. Lytic phase of Paget disease ☐
D. Gorham-Stout disease ☐
E. Giant cell tumor of bone ☐



ANSWERS: 1. B, 2. D

TOO
PODCAST

Hello friends,

Welcome to the TOO podcast series of lectures.

Osteoarthritis is a public health concern of global importance. Recent Osteoarthritis Research Society International (OARSI) white paper on osteoarthritis noted that it affects 240 million people globally, with about 10% male and 18% of females > 60 years of age. It carries substantial morbidity, including disability and reduce quality of life.

My talk will focus on the following points:

- Prevalence of osteoarthritis in India
- Current treatment options
  - » Non-pharmacological treatment (exercise, weight loss, yoga, walking aid as indicated)
  - » Pharmacological treatment (Paracetamol, non-steroidal anti-inflammatory drugs [NSAIDs])
- American College of Rheumatology/ Arthritis foundation guideline 2019 recommendations on the management of osteoarthritis (hand, hip, and knee)
- Additive effect of NSAIDs and paracetamol combination on pain management

I hope you all like this combined effort of ours.

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