

Postmenopausal Osteoporosis is an Age Related Physiological Change and not A Disease

Review Article

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Abstract

For the past few decades it is taught at medical schools that hormonal and calcium deficiencies are the main culprit in causation of Postmenopausal Osteoporosis. Because of this myth, hormone replacement therapy (HRT) has been used increasingly to prevent and cure PMO by the medical graduates at global level. The very definition of “normality” is flawed seriously. The statistical mean plus/minus two standard deviations automatically brings in five per cent of normal people into the fold of patients-the false positives. This goes up to 25% when disease statistics are used to measure healthy people. If we extend this definition further, almost all will come under the umbrella of patients! Age related osteoporosis falls under this label.

Key words: Osteoporosis, hormone, calcium, *Asthisoushrya*, Bone density, disease mongering

Introduction

A renowned American professor of medicine, Leon Eisenberg, with thirty five years of practical experience in her field, asks a fresh intern in her firm two important questions and surprisingly gets answers that baffle her! The first of it being “When does a healthy individual become a patient?” and the intern answers: “after consulting a physician”. Second question being “when does a patient become healthy individual? The answer was “rarely ever, if ever”. Hi-tech modern medicine follows the linear laws in a non-linear dynamic human system. Doctors have been predicting the unpredictable future of their patients for a long time in this system. The very definition of “normality” is flawed seriously. The statistical mean plus/minus two standard deviations automatically brings in five per cent of normal people into the fold of patients-the false positives. This goes up to 25% when disease statistics are used to measure healthy people. If we extend this definition further, almost all will come under the umbrella of patients! Age related osteoporosis falls under this label.

Osteoporosis [Osteo-bone (Asthi), Poros-holes (Soushrya- Sarandratwam), Osis-condition] is the most common metabolic bone disease which is defined as reduction of bone mass (or density) or the presence of a fragility fracture. The reduction in bone tissue is accompanied by deterioration in the architecture of the skeleton, leading to markedly increased risk of fracture. In 1994 a World Health organization study group recommended a clinical definition of osteoporosis

based on bone mineral density (BMD) measurements expressed in standard deviation (SD) units called T-scores which are calculated by taking the difference between a patient’s measured BMD and the mean BMD of healthy young adults matched for gender and ethnic group and expressing the difference relative to the young adult population SD.

There is some space for a doubt to consider this clinical definition of osteoporosis for postmenopausal women because decrease in bone density is a universal feature of ageing. How then could one compare their bone mass density with that of young healthy adults? Similarly then grey hair will be a disease compared to the young dark hair. How ever now days, irrespective of age related changes in the bone remodeling process a patient with T- score less than – 2.5 at spine, hip or forearm is diagnosed with postmenopausal osteoporosis. Routine screening of apparently healthy people could seriously damage their health says an editorial in a recent issue of the British Medical Journal (1). This brings to my mind saying of Mark Twain which goes thus: “For a man with a hammer in the hand, and wanting to use it badly, everything in the world looks a nail needing hammering.”

Epidemiological studies in the United States of America have estimated that more than one million Americans experience a significant fragility fracture each year at a cost of over \$ 14 billion and in the United Kingdom osteoporotic fractures occur in 150,000 individuals annually at a cost of over £750 million. High incidence of PMO could be because of “disease mongering” by the pharmaceutical industries, disease mongering medicalises human life turning ordinary ailments into medical problems, seeing mild symptoms as serious, treating personal problems as medical, seeing risks as diseases , and framing prevalence estimates to maximize potential market(2). They see what they want to see in their research (3)!

From physiological point of view, once peak skeletal mass is attained, the process of remodeling becomes the principle metabolic activity of the skeleton.

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This process has three primary functions: to repair micro damage within the skeleton, to maintain skeletal strength and, to supply calcium from the skeleton to maintain serum calcium. Bone remodeling is regulated by several circulating hormones, including estrogens, androgens, vitamin D, and PTH, as well as locally produced growth factors such as IGF-1and2, TGF, PTHrP, prostaglandins, TNF. Additional influences include nutrition, physical activity, genetic influences, race etc. The end result of this bone remodeling process (BRP) is that the resorbed bone is replaced by an equal amount of new bone tissue. After age 30 to 45, the resorption and formation process become imbalanced, and RESORPTION EXCEEDS FORMATION which can be taken as increased catabolic asthidhatwagni in Ayurveda.

PMO results from, bone loss due to normal age related changes which is swabhavika (4,5) in bone remodeling. Now it is very clear that PMO is age related, and diagnosis of a disease there needs the comparison with those age group normals and not with young adults bone density score (6). Many times in these elderly women there is no deficiency of serum calcium either (7). About 20% of postmenopausal women with osteoporosis have hyper-calciurea, here hormone deficiency is not the only culprit, but resorption rate is higher than formation, so physician should think about increased resorption not about formation rate by giving calcium supplements etc.

But over the past few decades it is taught at medical schools that hormonal and calcium deficiencies are the main causes. Because of this preaching, hormone replacement therapy (HRT) has been used increasingly to prevent and cure PMO and other chronic diseases by the medical graduates at global level. But based on pathophysiology of PMO there are no scientific convincing clinical evidences to use HRT and calcium. This view is supported by recent conclusion of many clinical trials of HRT that is “do not use estrogen / progesterone to prevent chronic diseases” including PMO, risks are higher than benefits; those being ovarian cancer, breast cancer, stroke, pulmonary embolism, coronary heart disease etc (8,9). Present day management of PMO brings to my mind a saying of Nin Anais: “We do not see things as they are; we see them as we are”

Looking at clinical features of PMO, most individuals diagnosed on the basis of bone densitometry are asymptomatic; many patients with quite advanced osteoporosis remain asymptomatic until a fracture occurs, because low bone density alone does not cause any symptoms, osteoporotic fractures, lifting a heavy weight precipitates pain in the vertebral fracture. The clinical presentation of vertebral fracture is highly variable, in some cases the onset is sudden with acute back pain, where as in others the presentation is insidious with gradual loss of height and chronic back pain, the pain of osteoporotic fracture often radiates anteriorly with nerve root distribution and may be made worse by back movement.

Asymptomatic postmenopausal women are treated with HRT without knowing through what mechanism hormones act (10) for prevention and cure

of chronic diseases including osteoporosis and osteoporotic fractures based on very poor evidence of benefits. Use of HRT has increased among postmenopausal women in western countries. As estimated 20 million women worldwide were using it in the late 1990's (11).

JAMA of July, 2002-VOL 288, No. 3, 321-333 had good news for asymptomatic postmenopausal women but bad news for medi-business that overall health risks of hormone replacement outweighs the good effects. Many other clinical trials also echoed the same sentiments. All the killer diseases that HRT was supposed to prevent are in fact brought on by HRT (12,13, 9,14,15,16,17,18,19,20,21)!

As long as women are asymptomatic it is better not to predict unpredictable things and interfere with HRT. Sir William Osler, a great brain in modern medicine had warned years ago: “patient doing well-do not interfere.” One example is a study on insulin therapy on diabetics, only symptomatic patients got benefits but asymptomatic patients did not get any benefits (22). But proper traditional diet, lifestyle and seasonal purification procedures which are essential part of life can be followed for healthy a life.

Ayurvedic texts do not explain asymptomatic diseases and their management. Acharya's have explained kriyakala (different stages of disease manifestation) with chikitsa (treatment) to check progress of the samprapti (pathogenesis) but not for asymptomatic disease.

Ayurvedic diagnosis of PMO is also not possible until and unless there occurs a fracture. The question of management does not therefore arise. The osteoporotic changes detected by DEXA can be diagnosed as Asthisoushrya. Arunadatta and Hemadri commenting on symptoms caused by aggravated vata dosha(23) opine that Asthisoushrya means Asthi (Bone) and Soushrya (with pores). Comparison of PMO with Asthigata vata does not hold well because the latter is characterized by “Bhedho asthiparvanam, sandhishoolam, mamsabala kshayam, aswapnah santataruk cha (24)”

Asthi Soushrya is swabhavika (age related changes etc) so curative approach by any system of health delivery may not be acceptable and successful (Swabhavo Nispratikiyaya – natural changes can not be treated) but surely the rate of resorption can be slowed down by diet, life style changes and exercise according to dinacharya (daily regimen), rutucharya (seasonal regimen), and asthi pradoshaja chikitsa that is basti in grishma rutu. The principles and efficacy of Indian system of medicine are demonstrated truth established after several examinations and reasoning thousands of years ago which is known as Siddhant (25).

All women, irrespective of race and nationality, attain menopause and have deficiency of hormones but incidence of PMO is high in western countries compared to ours. There could be many reasons-increased salt intake in the preserved food is a very important reason in addition to the weather, smoking, alcohol, contraceptives, refined oils etc (26). Indian foods, in moderation, could be having some preventive effect (27).

If postmenopausal women have signs and symptoms of Asthi kshaya, Asthi pradoshaja chikitsa may be adopted. “Bastaya Ksheera Sarpishi tiktakopahitani cha”(28) snehapana and ksheera basti with Panchatikta Guggulaghrata, Mahatiktaghrata, Patoladighrta, Panchatiktaghrata, Tiktaka ghrata etc can be tried to slow down the process of Asthikshaya.

Conclusion

Estrogen deficiency is not the only culprit in postmenopausal osteoporosis. The latter is definitely age related. For diagnosis of asthishuoshirya bone densitometry test is must. Curative approach for an age related disease by any system of medicine looks preposterous, but the rate of resorption can be slowed down by diet and lifestyle. (dinacharya, rutucharya and, by basti in every grishma rutu as explained in Asthi Pradoshaja Chikitsa)

References:

1. Sarah Stewart-Brown, Andrew Farmer, Screening could seriously damage your health. *British Medical Journal*. 22 February, 1997; 314; 533
2. Ray Moynihan, Richard Smith, Too much medicine? *British Medical Journal*. 13 April, 2002; 324; 886-890
3. James McCormack, Trisha Greenhalgh, Seeing what you want to see in randomized controlled trials: versions and perversions of UKPDS data. *British Medical Journal*. 24 June, 2000; 320; 1720-1723
4. Vinayakumar M, Abul k. Abbas, Nelson Fausto. *Robbins and Corton Pathologic Basis of Disease*, 7ed. Philadelphia; Elsevier Inc; 2005. 1283p.
5. Vinayakumar, Ramzis Cortan, Stanly L. Robbins. *Robins Basic pathology*, 7ed. Philadelphia; Elsevier Inc; 2003. 758pp
6. Lawrence M, Tierney.Jr, Stephen J. MacPhee, Maxine A. Papodkis. *Current Medical Diagnosis and Treatment*. 39ed. United States of America; McGraw-Hill; 2000. 1110p
7. Lawrence M, Tierney.Jr, Stephen J. MacPhee, Maxine A. Papodkis. *Current Medical Diagnosis and Treatment*. 39ed. United States of America; McGraw-Hill; 2000. 1110p
8. Janice Hopkins Tanne, Hormone trial for disease prevention stopped early, *British Medical Journal*. 13 July, 2002; 325; 61
9. Writing Group for the Women's Health Initiative Investigators, Risks and Benefits of Estrogen plus Progestin in Healthy Postmenopausal Women: Principal Results from the Women's Health Initiative Randomized Clinical Trial. *Journal of American Medical Association*. 17 July, 2002; 288 (3); 321-33
10. Stephen R. Rapp, Mark A. Espeland, Sally A. Shumaker, Victor W. Henderson, Robert L. Brunner, JoAnn E. Manson, Margery L. S. Gass, Marcia L. Stefanick, Dorothy S. Lane, Jennifer Hays, Karen C. Johnson, Laura H. Coker, Maggie Dailey, Deborah Bowen, for the WHIMS Investigators, Effect of Estrogen Plus Progestin on Global Cognitive Function in Postmenopausal Women. *Journal of American Medical Association*. 28 May, 2003; 289 (20); 2673-2684
11. Valerie Beral, Emily Banks, Gillian Reeves, Evidence from randomized trials on the long – term effects of hormone replacement therapy. *The Lancet*. 21 September, 2002; 360 (9337); 942-944
12. Janice Hopkins Tanne, Hormone trial for disease prevention stopped early. *British Medical Journal*. 13 July, 2002; 325; 61
13. Suzanne W. Fletcher, Graham A. Colditz, Failure of Estrogen Plus Progestin Therapy for Prevention. *Journal of American Medical Association*. 17 July, 2002; 288 (3); 366-368
14. Beral V, Banks E, Reeves G, Evidence from randomized trials on the long-term effects of hormone replacement therapy. *The Lancet*. 21 September, 2002; 360 (9337); 942-944
15. Shumaker SA, Legault C, Rapp SR, Thal L, Wallace RB, Ockene JK, Hendrix L, Jones BN 3rd, Assaf AR, Jackson RD, Kotchen JM, Wassertheil-Smoller S, Wactawski-Wende J; WHIMS Investigators, Estrogen plus progestin and the incidence of dementia and mild cognitive impairment in postmenopausal women: the Women's Health Initiative Memory Study: a randomized controlled trial. *Journal of American Medical Association*. 28 May, 2003; 289 (20); 2651-62
16. Zandi PP, Carlson MC, Plassman BL, Welsh-Bohmer KA, Mayer LS, Steffens C, Breitner JC; Cache County Memory Study Investigators, Hormone replacement therapy and incidence of Alzheimer disease in older women: the Cache County Study. *Journal of American Medical Association*. 6 November, 2002; 288 (17); 2123-2129
17. Susan M. Resnick, Victor W. Henderson, Hormone Therapy and Risk of Alzheimer Disease. *Journal of American Medical Association*. 6 November, 2002; 288 (17); 2170-2171
18. Christopher I. Li, Kathleen E. Malone, Peggy L. Porter, Noel S. Weiss, Mei-Tzu C. Tang, Kara L. Cushing-Haugen, Janet R. Daling, Relationship Between Long Durations and Different Regimens of Hormone Therapy and Risk of Breast Cancer. *Journal of American Medical Association*. 5 June, 2003; 289 (24); 3254-3263
19. Rowan T. Chlebowski, Susan L. Hendrix, Robert D. Langer, Marcia L. Stefanick, Margery Gass, Dorothy Lane, Rebecca J. Rodabough, Mary Ann Gilligan, Michele G. Cyr, Cynthia A. Thomson, Janardan Khandekar, Helen Petrovitch, Anne McTiernan, for the WHI Investigators, Influence of Estrogen Plus Progestin on Breast Cancer and Mammography in Healthy Postmenopausal Women. *Journal of American Medical Association*. 5 June, 2003; 289 (24); 3243-3253
20. Garnet L. Anderson, Howard L. Judd, Andrew M. Kaunitz, David H. Barad, MS; Shirley A. A. Beresford, Mary Pettinger, James Liu, S. Gene McNeeley, Ana Maria Lopez, for the Women's Health Initiative Investigators, Effects of Estrogen Plus Progestin on Gynecologic Cancers and

- Associated Diagnostic Procedures: The Women's Health Initiative Randomized Trail. Journal of American Medical Association. 1 October, 2003; 290 (13); 1739-1748
21. James V. Lacey, Pamela J. Mink, Jay H. Lubin, Mark E. Sherman, Rebecca Troisi, Patricia Hartge, Arthur Schatzkin, Catherine Schairer, Menopausal Hormone Replacement Therapy and Risk of Ovarian Cancer. Journal of American Medical Association. 17 July, 2002; 288 (3); 334-341
22. P. P. M. Goddijn, H. J. G. Bilo, E. J. M. Feskens, K. H. Groenier, K. I. van der Zee, B. Meyboom- de Jong, Longitudinal study on glycemic control and quality of life in patients with Type 2 diabetes mellitus referred for intensified control. Diabetic Medicine. 30 January, 1999; 16 (1); 23-30
23. Vaagbhata. Ashtanga Hridayam. 8ed. Varanasi; Chaukhambha Orientalia; 1998. 201p.
24. Agnivesh. Charaka Samhita. 4ed. Varanasi; Chaukhambha Sanskrit Sansthan; 1994. 617p.
25. Basavaraj S Hadpad, History of Indian Medicine. Aryavaidyan. August – October, 2006; 20 (1); 49-52
26. Sarah L Booth, Alice H Lichtenstein, Maureen O'Brien-Morse, Nicola M McKeown, Richard J Wood, Edward Saltzman and Caren M Gundberg, Effects of a hydrogenated form of vitamin K on bone formation and resorption. Am J Clin Nutr. 1 December, 2001; 74(6); 783-790
27. Adlercreutz H, Western diet and Western diseases: some hormonal and biochemical mechanisms and associations. Scand J Clin Lab Invest. 1990; 50 (201); 3-23 (Published online 29 March 2011).
28. Agnivesh. Charaka Samhita. 4ed. Varanasi; Chaukhambha Sanskrit Sansthan; 1994. 180p
