

Selección de Resúmenes de Menopausia

Semana del 15 al 21 de Febrero de 2017 Juan Enrique Blümel. Departamento Medicina Sur. Universidad de Chile

J Clin Diagn Res. 2016 Dec;10(12):CC13-CC16. doi: 10.7860/JCDR/2016/23433.9004. Epub 2016 Dec 1. Motor Nerve Conduction Velocity In Postmenopausal Women with Peripheral Neuropathy.

Singh A, Asif N, Singh PN, Hossain MM.

INTRODUCTION: The post-menopausal phase is characterized by a decline in the serum oestrogen and progesterone levels. This phase is also associated with higher incidence of peripheral neuropathy. AIM: To explore the relationship between the peripheral motor nerve status and serum oestrogen and progesterone levels through assessment of Motor Nerve Conduction Velocity (MNCV) in post-menopausal women with peripheral neuropathy. MATERIALS AND METHODS: This cross-sectional study was conducted at Jawaharlal Nehru Medical College during 2011-2013. The study included 30 post-menopausal women with peripheral neuropathy (age: 51.4±7.9) and 30 post-menopausal women without peripheral neuropathy (control) (age: 52.5±4.9). They were compared for MNCV in median, ulnar and common peroneal nerves and serum levels of oestrogen and progesterone estimated through enzyme immunoassays. To study the relationship between hormone levels and MNCV, a stepwise linear regression analysis was done. RESULTS: The post-menopausal women with peripheral neuropathy had significantly lower MNCV and serum oestrogen and progesterone levels as compared to control subjects. Stepwise linear regression analysis showed oestrogen with main effect on MNCV. CONCLUSION: The findings of the present study suggest that while the post-menopausal age group is at a greater risk of peripheral neuropathy, it is the decline in the serum estrogen levels which is critical in the development of peripheral neuropathy.

Clin Auton Res. 2017 Feb 16. doi: 10.1007/s10286-017-0403-0. [Epub ahead of print] Neural control of blood pressure in women: differences according to age.

Peinado AB, Harvey RE, Hart EC, Charkoudian N, Curry TB, Nicholson WT, Wallin BG, Joyner MJ, Barnes JN. PURPOSE: The blood pressure "error signal" represents the difference between an individual's mean diastolic blood pressure and the diastolic blood pressure at which 50% of cardiac cycles are associated with a muscle sympathetic nerve activity burst (the "T50"). In this study we evaluated whether T50 and the error signal related to the extent of change in blood pressure during autonomic blockade in young and older women, to study potential differences in sympathetic neural mechanisms regulating blood pressure before and after menopause. METHODS: We measured muscle sympathetic nerve activity and blood pressure in 12 premenopausal (25 \pm 1 years) and 12 postmenopausal women (61 ± 2 years) before and during complete autonomic blockade with trimethaphan camsylate. RESULTS: At baseline, young women had a negative error signal (-8 \pm 1 versus 2 \pm 1 mmHg, p < 0.001; respectively) and lower muscle sympathetic nerve activity (15 \pm 1 versus 33 \pm 3 bursts/min, p < 0.001; respectively) than older women. The change in diastolic blood pressure after autonomic blockade was associated with baseline T50 in older women (r = -0.725, p = 0.008) but not in young women (r = -0.337, p = 0.29). Women with the most negative error signal had the lowest muscle sympathetic nerve activity in both groups (young: r = 0.886, p < 0.001; older: r = 0.870, p < 0.001). CONCLUSIONS: Our results suggest that there are differences in baroreflex control of muscle sympathetic nerve activity between young and older women, using the T50 and error signal analysis. This approach provides further information on autonomic control of blood pressure in women.

Case Rep Gastroenterol. 2017 Jan 27;11(1):23-28. doi: 10.1159/000452735.

Black Cohosh Hepatotoxicity with Autoimmune Hepatitis Presentation.

Franco DL, Kale S, Lam-Himlin DM, Harrison ME.

Herbal medicines have been used for the treatment of various ailments since time immemorial. Black cohosh (BC) is well known for the treatment of postmenopausal symptoms, with conflicting evidence supporting its safety and benefits. We present a rare case of BC-induced autoimmune hepatitis (AIH) with hepatotoxicity in a 69-year-old female. To our knowledge, this represents the third case of BC-induced AIH.

Neurology. 2017 Feb 15. pii: 10.1212/WNL. doi: 10.1212/WNL.0000000000003696. [Epub ahead of print] Postmenopausal hormone therapy and Alzheimer disease: A prospective cohort study.

Imtiaz B, Tuppurainen M, Rikkonen T, Kivipelto M2 Soininen H, Kröger H, Tolppanen AM.

OBJECTIVE: To explore the association between postmenopausal hormone therapy (HT) and Alzheimer disease (AD). METHODS: Twenty-year follow-up data from the Kuopio Osteoporosis Risk Factor and Prevention study cohort were used. Self-administered questionnaires were sent to all women aged 47-56 years, residing in Kuopio Province starting in 1989 until 2009, every 5th year. Register-based information on HT prescriptions was available since 1995. Probable AD cases, based on DSM-IV and National Institute of Neurological and Communicative Disorders and Stroke-Alzheimer's Disease and Related Disorders Association criteria, were identified from the special reimbursement register (1999-2009). The study population included 8,195 women (227 cases of incident AD). RESULTS: Postmenopausal estrogen use was not associated with AD risk in register-based or self-reported data (hazard ratio/95% confidence interval 0.92/0.68-1.2, 0.99/0.75-1.3, respectively). Long-term self-reported postmenopausal HT was associated with reduced AD risk (0.53/0.31-0.91). Similar results were obtained with any dementia diagnosis in the hospital discharge register as an outcome. CONCLUSIONS: Our results do not provide strong evidence for a protective association between postmenopausal HT use and AD or dementia, although we observed a reduced AD risk among those with long-term self-reported HT use.

Am J Clin Nutr. 2017 Feb 15. pii: ajcn141531. doi: 10.3945/ajcn.116.141531. [Epub ahead of print] Vitamin D is not associated with incident dementia or cognitive impairment: an 18-y follow-up study in community-living old men.

Olsson E, Byberg L, Karlström B, Cederholm T, Melhus H, Sjögren P, Kilander L.

Background: Vitamin D has been implicated as being important for maintaining cognitive function in old age. Results from longitudinal studies examining the association of vitamin D with incident dementia and cognitive impairment have been inconsistent. Objective: We investigated the relation between vitamin D, assessed in 3 different ways, and the risk of dementia. Design: We measured plasma 25-hydroxyvitamin D [25(OH)D] with the use of high-performance liquid chromatography-mass spectrometry, assessed dietary vitamin D intake with the use of 7-d dietary records, and created a vitamin D-synthesis genetic risk score (GRS) at baseline (1991-1995) in a cohort of 1182 Swedish men (mean age: 71 y). In a maximum of 18 y (median: 12 y) of follow-up, 116 men developed Alzheimer disease, 64 men developed vascular dementia, and 250 men developed all-cause dementia. An additional 80 men declined in cognitive function as assessed with the use of the Mini-Mental State Examination. Adjusted HRs and ORs were calculated with the use of Cox and logistic regressions. Results: The mean \pm SD plasma 25(OH)D concentration was 68.7 \pm 19.1 nmol/L. Plasma 25(OH)D, dietary vitamin D intake, and vitamin D-synthesis GRS were not associated with any cognitive outcomes (crude and adjusted HRs and ORs were ~1.0 for all continuous exposures). The adjusted HR for all-cause dementia was 0.88 (95% CI: 0.59, 1.31) in men with plasma 25(OH)D concentrations ≤50 compared with >75 nmol/L. The adjusted HR for all-cause dementia was 0.92 (95% CI: 0.63, 1.32) for the lowest compared with highest tertiles of vitamin D intake. The adjusted HR for the continuous GRS for all-cause dementia was 1.04 (95% CI: 0.91, 1.19). Conclusion: In this cohort study, we show that there is no association between baseline vitamin D status and long-term risk of dementia or cognitive impairment over an 18-y period of time.

J Hum Lact. 2017 Feb 1:890334416683676. doi: 10.1177/0890334416683676. [Epub ahead of print] Breastfeeding Mode and Risk of Breast Cancer.

Unar-Munguía M, Torres-Mejía G, Colchero MA, González de Cosío T.

BACKGROUND: Breastfeeding reduces women's risk of breast cancer. Since exclusive breastfeeding has a stronger hormonal effect, it could theoretically result in a greater reduction in breast cancer risk than any breastfeeding mode. No meta-analysis has examined breast cancer risk by breastfeeding mode. Research aim: The authors conducted a meta-analysis for breast cancer risk in parous women who breastfed exclusively or in any mode versus parous women who formula fed their infants, and they estimated the summary dose-response association by the accumulated duration of any breastfeeding mode. METHODS: A systematic review of studies published between 2005 and 2015 analyzing breastfeeding and breast cancer risk in women was conducted in PubMed and EBSCOhost. A meta-analysis (n = 65 studies) with fixed effects (or random effects, if heterogeneity existed) was carried out stratified by breastfeeding mode and menopausal and parity status. A summary dose-response association was estimated using the generalized least-

squares method. RESULTS: The summary relative risk (SRR) for breast cancer in parous women who breastfed exclusively was 0.72, 95% confidence interval (CI) [0.58, 0.90], versus parous women who had never breastfed. For parous women who breastfed in any mode, the SRR was lower in both premenopausal women (0.86, 95% CI [0.80, 0.93]) and postmenopausal women (0.89, 95% CI [0.83, 0.95]). There was no heterogeneity or publication bias. There is weak evidence of a difference between exclusive and any breastfeeding mode (p = .08). The summary dose-response curve was nonlinear (p < .001). CONCLUSION: Exclusive breastfeeding among parous women reduces the risk of breast cancer compared with parous women who do not breastfeed exclusively.

Park YM, Pereira RI, Erickson CB, Swibas TA, Kang C, Van Pelt RE.

OBJECTIVE: Short-term administration of estradiol (E2) improves insulin-stimulated glucose disposal rate in early postmenopausal (EPM) women compared with a reduction in late postmenopausal (LPM) women. The underlying mechanisms by which E2 action on glucose disposal rate reversed from beneficial early to harmful late in menopause is unknown, but might include adverse changes in estrogen receptors (ERs) or other biomarkers of cellular energy metabolism with age or duration of estrogen deficiency. METHODS: We retrospectively analyzed skeletal muscle samples from 27 postmenopausal women who were 6 years or less past menopause (EPM; n=13) or at least 10 years past menopause (LPM; n=14). Fasted skeletal muscle (vastus lateralis) samples were collected after 1 week administration of transdermal E2 or placebo, in random cross-over design. RESULTS: Compared with EPM, LPM had reduced skeletal muscle ER α and ER β nuclear protein. Short-term E2 treatment did not change nuclear ER α or ER β , but decreased cytosolic ERα, so the proportion of ERα in the nucleus compared with the cytosol tended to increase. There was a group-by-treatment interaction (P<0.05) for nuclear proliferator-activated receptor γ co-activator 1- α and phosphorylated adenosine monophosphate-activated protein kinase, such that E2 increased these proteins in EPM, but decreased these proteins in LPM. CONCLUSIONS: These preliminary studies of skeletal muscle from early and late postmenopausal women treated with E2 suggest there may be declines in skeletal muscle ER and changes in the E2mediated regulation of cellular energy homeostasis with increasing time since menopause. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited.