QUE, respectively. Through a cell adhesion assay we found a 45% decrease in cellular adhesion after 10 – 50 μM of QUE treatment, with a major effect at 100 μM (83.60% ± 3.24). In addition, AE affects considerably the cell adhesion capacity, with higher levels of inhibition than QUE at all doses probed (78.24% ± 3.6 years 95% ± 8.36 vs ctrl. at 10 years 100 μg/ml, respectively). Altogether, these findings indicate that polyphenols-rich diets would be useful therapies to prevent breast cancer, due to their regulatory effects on processes involved in metastasis, such as cell proliferation, migration and adhesion.

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Código: 22
Beneficial role of alendronate on cellular and molecular processes involved in calcification/vascular remodeling

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The genesis of the atheromatous lesion involves the interaction of monocytes and platelets with the endothelium. Finally, vascular calcification takes place through vascular smooth muscle cells (VSMC) transdifferentiation to bone lineage. The impact of bisphosphonates (BP) on cardiovascular diseases remains unknown. The aim of this study was to investigate the role of the BP alendronate (ALN) in cellular and molecular processes involved in vascular disease. Murine cultures cells were used: a) endothelial cells (EC); b) VSMC; c) VSMC induced to osteoblastic transdifferentiation (VSMC-OB) in osteogenic medium (β-glicerolfostato 5 mM; $CaCl_2$ and 4 mM).The effect of ALN on platelet adhesion (PAd) and aggregation (PAg) was investigated. Under basal conditions ALN partially reduced PAd (20–35%; ALN 1–10 μM; $p < 0.01$). The stimulus induced by LPS was partially reduced in the presence of the BP (22–33%; ALN 1–10 μM; $p < 0.02$). We found that ALN markedly inhibited PAg compared with control (56%; ALN 10 μM; $p < 0.05$). Both antiplatelet effects evoked by ALN were reversed in the presence of L-NAME, an inhibitor of nitric oxide synthase. Monocyte adhesion to EC depends on the expression of adhesion molecules. ALN treatment significantly prevents the enhancement of ICAM-1 and VCAM-1 mRNA levels induced by LPS. On VSMC-OB, ALN markedly reduce the expression of RUNX2 and TNAP, showed a significant diminution in FAL activity (9–29%; ALN 1–10 μM; $p < 0.02$), as well as in extracellular calcium deposition (341 ± 33 vs 225 ± 24 μg/mg prot.; control vs ALN 5 μM; $p < 0.05$). In summary, the results suggest that ALN is an active drug at vascular level with potential beneficial effects on processes that compromise the vascular architecture.

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Código: 23
Regulation of bone-vascular axis by nutraceuticals agents

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Currently, the nutraceutical agent Genistein (Gen) is proposed as a natural therapeutic alternative given its potential biological actions. We studied the effect of Gen on bone-vascular axis and, its relationship with osteoblastic (OB) and osteoclastic (OC) differentiation. To that end murine monocytes (Mo) isolated from peripheral blood, endothelial cell (EC) and calvaria OB cultures were used. OB monolayers were incubated (24 h) with conditioned medium (medium C) obtained from EC. Medium C stimulated OB proliferation ($p < 0.01$), mitogenic action that was enhanced (0,5 fold above control, $p < 0.01$) when EC were exposed to Gen (10 nM–5 uM). Medium C obtained from EC cultures pre-incubated with L-NAME (nitric oxide synthase inhibitor) diminished OB proliferation. When OB was directly exposed to sodium nitroprusside, an exogenous nitric oxide (NO) donor, a stimulation of OB proliferation (48% above control, $p < 0.01$) was detected. The effect of Gen on OB differentiation was studied using two markers: alkaline phosphatase activity (AP) and extracellular calcium deposition. Both markers were enhanced after 12–15 days of culture (0.42 ± 0.1 vs 0.25 ± 0.08IU/mg prot. AP activity, $p < 0.05$; 133 ± 11 vs 90 ± 12 ugCa/mg prot., $p < 0.001$). Using red Alizarin staining, an increase in calcification nodules size and number were also observed. OC differentiation was studied by co-incubation of Mo with OB (15 days). Under basal conditions, the number of multinucleated cells enhanced. Indeed positive staining for tartrate-resistant acid phosphatase (TRAP) was detected. Gen decreased the amount of TRAP positive Mo. These results suggest that Gen regulates bone cells growth and differentiation, through a close link between bone and vascular systems, being NO a potential chemical messenger involved.

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Código: 24
Bone involvement in Gaucher disease: Importance of early diagnosis and treatment

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Introduction: Gaucher disease (GD) is the most prevalent lysosomal storage disease. Even asymptomatic patients may have bone complications that impair quality of life.

Objectives: Evaluating bone involvement in type 1 GD patients receiving enzymatic replacement treatment (ERT) with velaglucerase.

Methods: Observational, prospective, transversal analysis of data from X-rays, MRI, BMD and laboratory tests, including chitotriosidase (Cht), angiotensin converting enzyme (ACE), ferritin (FT).

Results: 27 patients were included (11 males; age: 24.7 ± 14 years; 33% younger than 20 years-old). Age at diagnosis was ≤20 in 74% of patients. Reason for consultation included bone symptoms in 5 cases. 11 patients had not received previous ERT.

100%, 44% and 28% had elevated Cht, ACE and FT, respectively. All subjects met therapeutic goals (TG) for visceromegalies. Ten patients reported lower limbs osteonecrosis (ON; 5 in ≥1 joint). X-ray alterations were detected in 63% of patients, including fractures: 4 clinical (long bones), 3 radiological (spine).

MRI showed femoral (52%) and spine (15%) bone infiltration. Other infiltration areas were found in 12 patients. MRI suggested also ON ($n = 10$), bone infarction ($n = 8$) and edema ($n = 6$).

Spine BMD Z-score was <-1 in 6 patients. Insufficient ($n = 11$) or deficient ($n = 5$) 25OH-D levels were found. In 7 adults with bone infiltration, CTX level was elevated. Time from diagnosis correlated with GD activity biomarkers.

Conclusions: 23% of subjects initially described bone symptoms. Even after reaching TG, 41% had irreversible bone lesions. On the contrary, bone lesions were absent in subjects with early ERT. Importance of early detection of bone involvement in GD patients is highlighted.

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Código: 29
Mediastinum tumor such as cause of hyperparathyroidism primary persistent: Diagnostic challenge

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The ectopic parathyroid tissue found in 9–20% of patients with primary hyperparathyroidism. Abnormalities in the number and anatomical position are common. The differentiation of benign and malignant parathyroid tumors is difficult. **Report:** Female,63-year-old, with nephrolithiasis, osteoporosis. Primary hyperparathyroidism by adenomas diagnosed by surgery. LAB POSTQX: Ca: 14 mg/dl, PTH: 597 pg/ml, P: 2 mg/dl, Mg: 1.70 mg/dl, VitD: 16 ng/ml. It is referred, with symptoms of severe symptomatic hypercalcemia, cardiovascular disorders, refractory to treatment. Parathyroid scintigraphy: large hyperintense focus below the thyroid gland entering the mediastinum. MRI: In the mediastinum behind the trachea and esophagus an elongated formation of 29.4 mm. Videothoracoscopy: mediastinal mass is extracted. solid tumor,5 × 4 cm. Weight 25 g. Intraoperative PTH: Pre incision: 843 pg/ml, 10 min: 198 pg/ml, 15 min: 133 pg/ml. POSTQX: Ca: 9.6 mg/dl, P: 1.5 mg/dl, Mg: 1.7 mg/ml, PTH 101 pg/ml. Screening negative family to hereditary syndromes. PATHOLOGIC ANATOMY: primary parathyroid hyperplasia. Immunostaining: CD34 (positive) D240 positive. S100: (positive) Ki67: less than 5%. Control: Ca: 10 mg/dl, P: 2 mg/dl, Mg: 2 mg/dl, PTH: 72 pg/ml, Vit D: 31 ng/ml. Patient in good general condition. **Commentary:** Patients diagnosed with ectopic parathyroid hyperplasia have very high