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Review Article **Published Date:- 2020-05-04**

The benefits of biochemical bone markers

Clinical benefits

1. To improve efficiency in Osteoporosis treatment

2. To adjust the dosage of medication for osteoporosis with BMK

3. To adjust bone life cycle as needed

4. To prevent bone necrosis which dentists have been worried.

5. To reduce cost of treatment

Research Article **Published Date:- 2020-04-13**

The alterations effects in phosphorus of erythropoietin and U-74389G

Aim: This study calculated the effects on serum phosphorus (P) levels, after treatment with either of 2 drugs: the erythropoietin (Epo) and the antioxidant lazaroid (L) drug U-74389G. The calculation was based on the results of 2 preliminary studies, each one of which estimated the certain influence, after the respective drug usage in an induced ischemia reperfusion (IR) animal experiment.

Materials and methods: The 2 main experimental endpoints at which the serum P levels were evaluated was the 60th reperfusion min (for the groups A, C and E) and the 120th reperfusion min (for the groups B, D and F). Specially, the groups A and B were processed without drugs, the groups C and D after Epo administration; whereas the groups E and F after the L administration.

Results: The first preliminary study of Epo presented a non significant hyperphosphoremic effect by 2.46% + 2.02% (p - value = 0.2168). However, the second preliminary study of U-74389G presented a non significant hypophosphoremic effect by 1.09% + 2.01% (p - value = 0.5771). These 2 studies were co-evaluated since they came from the same experimental setting. The outcome of the co-evaluation was that L is at least 0.4455128-fold [0.4445589 - 0.4464687] more hypophosphoremic than Epo (p - value = 0.0000).

Conclusions: The anti-oxidant capacities of U-74389G ascribe at least 0.4455128-fold [0.4445589 - 0.4464687] more effects than Epo (p - value = 0.0000).

Research Article **Published Date:- 2020-04-03**

Novel European Asiatic Clinical, Laboratory, Molecular and Pathobiological (2015-2020 CLMP) criteria for JAK2V617F trilinear polycythemia vera (PV), JAK2exon12 PV and JAK2V617F, CALR and MPL515 thrombocythemias: From Dameshek to Constantinescu-Vainchenker, Kralovics and Michiels

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The Myeloproliferative Neoplasms (MPN) of trilinear polycythemia vera (PV) and megakaryocytic leukemia (ML = primary megakaryocytic granulocytic myeloproliferation: PMGM) and Essential Thrombocythemia (ET) in the studies of Dameshek and Michiels are caused by the MPN driver mutations JAK2V617F, JAK2exon12, CALR and MPL515 discovered by Constantinescu-Vainchenker, Green and Kralovics. The JAK2V617F mutated trilinear myeloproliferative neoplasms (MPN) include a broad spectrum of clinical laboratory and bone marrow features in essential thrombocythemia (ET), prodromal PV and erythrocythemic PV, classical PV and advanced stages of masked PV and PV complicated by splenomegaly and secondary myelofibrosis (MF). Heterozygous JAK2V617F mutated ET is associated with low JAK2 allele and MPN disease burden and normal life expectance. In combined heterozygous and homozygous or homozygous JAK2V617F mutated trilinear PV, the JAK2 mutation load increases from less than 50% in prodromal PV and classical PV to above 50% up to 100% in hypercellular PV, advanced PV and PV with MF. Bone marrow histology show diagnostic features of eryhrocytic, megakaryocytic and granulocytic (EMG) myeloproliferation in JAK2V617F mutated trilinear MPN, which clearly differs from monolinear megakaryocytic (M) myelproliferation in MPL and CALR thrombocythemia and dual megakaryocytic granulocytic (MG) myeloproliferation in CALR mutated thrombocythemia. The morphology of clustered large pleomorphic megakaryocytes with hyperlobulated nuclei are similar in JAK2V617F thrombocythemia, prodromal PV and classical PV patients. Monolinear megakaryocytic (M) myeloproliferation of large to giant megakaryocytes with hyperlobulated staghorn-like nuclei is the hallmark of MPL515 mutated normocellular thrombocythemia. CALR mutated thrombocythemia usually presents with high platelet count around 1000x109/l and normocellular megakaryocytic (M) proliferation of immature megakaryocytes with cloud-like hyperchromatic nuclei followed by dual megakaryocytic granulocytic (MG) myeloproliferation followed by various degrees of bone marrow fibrosis. Natural history and life expectancy of MPN patients are related to the response to treatment and the degree of anemia, splenomegaly, myelofibrosis and constitutional symptoms. The acquisition of epigenetic mutations at increasing age on top of MPN disease burden independently predict unfavorable outcome in JAK2V617F, MPL515 and CALR mutated myeloproliferative neoplasms (MPNs, which mutually exclude each other).