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Sample Report - 4M3542 **M/W:** M

Requested 17.04.2019
APNr 4095

Height cm **Weight** kg **Body Mass Index** *0,0* **Reported** 30.04.2019

ANAMNESE

Keine Angaben

RESULTS OVERVIEW

1. Homocystein is raised, indicating functional deficiencies in B-vitamine provision, although folic acid is within normal range.
2. Vitamin D is decreased.

Further therapeutic control recommended.

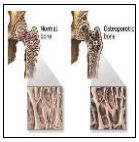
THERAPEUTIC RECOMMENDATIONS:
1. Reduction of the homocystein levesl with folic acid, B6 and B12 and where indicated Vitamin B2 and Betain supplementation. Control in 2 - 3 months.
2. Supplementation of vitamin D.

Kind Regards
Dipl.Biol. B. Knabenschuh / Dr. R. Raßhofer

METABOLISM/CHD

Homocystein	8,3	µmol/l	< 8	

OSTEOPOROSIS



Osteoporosis

Vitamin D (25-OH)	15,2	ng/ml	30 - 80	

ESSENTIAL NUTRIENTS

Selenium (WB)	164,0	µg/l	121 - 168	
Zinc (WB)	667	µg/dl	400 - 750	
Holo-Transcobalamine(B12)	123,00	pmol/l	> 35	
Folic Acid (S)	7,9	ng/ml	3,5 - 20	
Please note the new reference range from november 29th 2018.				
Ubichinon/Q10	900	µg/l	750 - 1200	

EXPLANATION

The metabolite h o m o c y s t e i n e is regarded as an independent risk factor of atherosclerosis, thrombosis and neurodegenerative diseases. Increased risk is seen already from homcysteine values of 8 µmol/l, the higher homocysteine the higher the risk of atherothrombotic complications.
The turnover of homocysteine is regulated by various enzymes and their cofactors vitamine B6, B12 and folate. Hyperhomocysteinemia is thus either caused by vitamine defieciencies, mostly folate, or by hypoactive genetic variant of one of

the enzymes, mostly the folate dependant MTHFR (methylenetetrahydrofolate reductase). In any case the apropriate treatment is the supplementation with the deficient vitamine: folate >> vit B12 >> vit B6. In some cases additional vitamine B2 and a methylene donor like betaine may be neccessary too. In cases of genetic enzyme deficiencies it may be advisible to identify the responsible enzyme variant and focus the long term supplementation on the appropriate vitamine cofactor.

V i t a m i n D is a fat-soluble vitamin and homone that is essential for maintaining normal calcium metabolism but has numerous additional effects. Vitamin D (cholecalciferol) can be synthesized in the skin/liver upon exposure to ultraviolet-B (UVB) radiation from sunlight and activated to the dihydroxy form 1,25(OH) vit D3 in the kidney.Dietary sources of vit D are rare in modern nutrition. Synthesis of adequate amounts through exposure to UVB radiation in the skin is therfore essential for optimal health.
The best known effect of vit D3 is facilitating the intestinal absorption of calcium and in addition the uptake of phosphate and magnesium ions. Vit D3 also controls the urinary calcium release and stimulates the incorporation of Ca (and Mg) ions into bone. Numerous hormonal effects of vit D3 are also known. Vit D is a potent modulator of celluar immune activity, has strong antiinflammatory effects, increases insulin sensitivity and improves the contractive power of skeletal and heart muscle. Since inappropriate activation of the renin-angiotensin system is thought to play a role in some forms of hypertension, adequate vitamin D levels may also be important in blood pressure control.
Probably the most important effect of vit D3 is the participation in the control of cellular growth and division and thus its chemopreventive potential. All peripheral organs have been proven to express specific receptors for vit D and having hydroxylase activity to intracellularly activate cholecalciferol to the active 1,25(OH)2 calcitriol.
In vitamin D3 deficiency the systemic calcium equilibrium is maintained by the mobilization of calcium from bone through parathyroid hormone. Recent research clearly suggests that less obvious states of vitamin D deficiency with corresponding higher levels of parathyroid hormone are very common in the central and northern european population. On the long term this leads to increased risk of osteoporosis, fatigue, inflammatory activity and even increased risk of up to seventeen different types of cancer, namely colon, prostate and breast.

At present a minimum of 32 ng/l (80 nmol/l) vit D (cholecalciferol) is recommended, in older adults 40 - 80 ng/l. In many cases this level can only be reached by supplementation with 1000 - 5000 IU of vit D. Up to 10.000 IU per day no risk of overdosing is to be expected.
The measurement of 1,25(OH)2 vit D3 is usually of no value since peripheral organs relay on cholecalciferol and only the vit D/cholecalciferol test mirrors the systemic situation. The dihydroxy form is often found normal to high in deficiency states due to the activation of renal hydroxylase activity at low vit D.