Vitamin D

- Exploring the Link: Vitamin D Levels and Its Clinical Implications in Guillain-Barre Syndrome Patients
- Triglyceride-glucose index and periodontitis: evidence from two population-based surveys
- Supplement and nutraceutical therapy in traumatic brain injury
- Comprehensive guidelines for prehabilitation in spine surgery
- Development of animal models with chronic kidney disease-mineral and bone disorder based on clinical characteristics and pathogenesis
- Vitamin D supplementation during intensive care unit stay is associated with improved outcomes in critically Ill patients with sepsis: a cohort study
- The Association of Vitamin D, Nerve Growth Factor (NGF), Brain-Derived Neurotrophic Factor (BDNF), and Glial Cell-Derived Neurotrophic Factor (GDNF) with Development in Children
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Vitamin D refers to a group of fat-soluble secosteroids responsible for enhancing intestinal absorption of calcium, iron, magnesium, phosphate, and zinc. In humans, the most important compounds in this group are vitamin D3 (also known as cholecalciferol) and vitamin D2 (ergocalciferol). Cholecalciferol and ergocalciferol can be ingested from the diet and from supplements.

Very few foods contain vitamin D; synthesis of vitamin D (specifically cholecalciferol) in the skin is the major natural source of the vitamin. Dermal synthesis of vitamin D from cholesterol is dependent on sun exposure (specifically UVB radiation).

Epidemiological studies show a strong association between decreased vitamin D levels and an increase in aneurysm rupture. However, the causality and mechanism remain largely unknown. Kimura et al. tested whether vitamin D deficiency promotes aneurysm rupture and examined the underlying mechanism for the protective role of vitamin D against the development of aneurysm rupture utilizing a mouse model of intracranial aneurysm. Mice consuming a vitamin D-deficient diet had a higher rupture rate than mice with a regular diet. Vitamin D deficiency increased proinflammatory cytokines in the cerebral arteries. Concurrently, vitamin D receptor knockout mice had a higher rupture rate than the corresponding wild-type littermates. The vitamin D receptors on endothelial and vascular smooth muscle cells, but not on hematopoietic cells, mediated the effect of aneurysm rupture. The results establish that vitamin D protects against the development of aneurysmal rupture through the vitamin D receptors on vascular endothelial and smooth muscle cells. Vitamin D supplementation may be a viable pharmacologic therapy for preventing aneurysm rupture [1)]

The evidence linking vitamin D (VitD) levels and Spontaneous Intracerebral Hemorrhage Risk Factors remains inconclusive. Szejko et al. tested the hypothesis that lower genetically determined VitD levels are associated with a higher risk of ICH. They conducted a 2-sample Mendelian Randomization (MR) study using publicly available summary statistics from published genome-wide association study of

VitD levels (417 580 study participants) and ICH (1545 ICH cases and 1481 matched controls). They used the inverse variance-weighted average method to generate causal estimates and the MR Pleiotropy Residual Sum and Outlier and MR-Egger approaches to assess for horizontal pleiotropy. To account for known differences in their underlying mechanism, we implemented stratified analysis based on the location of the hemorrhage within the brain (lobar or nonlobar). Our primary analysis indicated that each SD decrease in genetically instrumented VitD levels was associated with a 60% increased risk of ICH (odds ratio [OR], 1.60; [95% CI, 1.05-2.43]; P=0.029). They found no evidence of horizontal pleiotropy (MR-Egger intercept and MR Pleiotropy Residual Sum and Outlier global test with P>0.05). Stratified analyses indicated that the association was stronger for nonlobar ICH (OR, 1.87; [95% CI, 1.18-2.97]; P=0.007) compared with lobar ICH (OR, 1.43; [95% CI, 0.86-2.38]; P=0.17). Lower levels of genetically proxied VitD levels are associated with higher ICH risk. These results provide evidence for a causal role of VitD metabolism in ICH [2)].

The hippocampus is susceptible to damage in patients with epilepsy and in animals with seizures caused by excitotoxic agents. The effect of vitamin D on hippocampal apoptosis related with seizures has not been reported. However, epileptic patients have an increased risk of hypovitaminosis D which is most likely due to the effects of antiepileptic drugs. Therefore, in a study of Şahin et al., from Trabzon, it was aimed to evaluate the effects of vitamin D on hippocampal apoptosis related with seizures by using pentylenetetrazol (PTZ) and kainic acid (KA) in rats.

Male Sprague Dawley rats, aged 5.5 weeks, were randomly divided into six groups: control, vitamin D, PTZ, KA, PTZ + vitamin D and KA + vitamin D groups. The groups that received vitamin D were given 500 IU/kg of vitamin D daily for two weeks in addition to a standard diet. At the end of this period, PTZ and KA were applied to trigger seizures in the rats in the seizure groups. 24 h after the administration of PTZ and KA, the rats were decapitated. In the hippocampal region, apoptosis was assessed by TUNEL and brain-derived neurotrophic factor (BDNF), Bax, caspase-3 and c-fos activation were evaluated by immunohistochemical method.

BDNF level increased while c-fos, Bax and caspase-3 levels decreased ($p < 0.0001$, in all) in the hippocampal neurons of the groups that were pre-treated with vitamin D before the administration of PTZ and KA, in comparison with the PTZ and KA groups. Vitamin D significantly decreased the number of apoptotic cells in these rats in comparison with the PTZ and KA groups ($p < 0.0001$).

This study indicates that vitamin D has neuroprotective effects on hippocampal apoptosis induced by PTZ and KA in rats. With this study it is suggested that keeping vitamin D levels within normal limits may be beneficial for patients with epilepsy, especially children [3)].

A significant association appears to exist between lack of VitD supplementation and venous thromboembolism (VTE) occurrence in persons with acute spinal cord injury (SCI) and low VitD (LVitD) levels [4)].

Few studies have examined the relationship between diet and Modic changes. Johansen et al. studied the relationship between vitamin D and MC and surprisingly found that MC were more common in individuals with normal levels of vitamin D than in those with low levels. However, the mechanisms underlying the development of MC remain unclear at present. Findings suggest that the link between

vitamin D and MC is perhaps related to inflammation, though further confirmatory studies are needed [5].

Individuals with MC are expected to have low levels of vitamin D because of an increased susceptibility to inflammation and/or because microfractures occur in the vertebrae because of increased levels of parathyroid hormone [6] [7].

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