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Editorial

Role of Vitamin D in Cerebral Vasospasm

K M Ziaur Rahman and Md Moshiur Rahman*

Department of Neurosurgery, Holy Family Red Crescent Medical College Hospital, Dhaka, Bangladesh

***Corresponding Author:** Md Moshiur Rahman, Department of Neurosurgery, Holy Family Red Crescent Medical College Hospital, Dhaka, Bangladesh.

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CVS is a threatening consequence of subarachnoid hemorrhage (SAH), which can lead to subsequent stroke [1]. Current CVS therapy options, such as nicardipine, a calcium channel blocker, and clazosentan, an endothelin-1 antagonist, have failed to improve patient outcomes [2]. Inflammatory activation is part of the CVS pathomechanism [3]. Because of the complicated pathophysiology involving several pathways, basic vasodilator or anti-inflammatory medication is unlikely to significantly affect CVS severity.

Vasospasm occurs a week or so following SAH. Because there is such a long interval between the commencement of ictus and the development of symptoms, it is believed that treatments can be administered throughout this period. Inflammation following SAH appears to start early in several trials. Vitamin D (VitD) is a fat-soluble ecosteroids-based chemical substance primarily responsible for calcium and phosphorus control, among other physiological tasks [4]. A clinical investigation linked higher VitD levels to a lower incidence and severity of CVS and a better prognosis in two separate clinical studies [5]. VitD insufficiency is also linked to a poor post-SAH prognosis [5]. In a mouse model, supplementing with 1,25-vitamin D reduced spasm development, mediated by SDF1 induction [5]. VitD-responsive gene induction in myeloid cells could be used as a biomarker to predict the prognosis of SAH [4]. VitD and possibly SDF1 should be clinically tested in order to avoid CVS and improve SAH patient outcomes.

Obesity, diabetes, hypertension, and cancer are linked to a lack of 25(OH)D3, promoting bone demineralization [4,6]. Levels of 25(OH)D3 have been linked to cardiac myocyte regulation, systolic blood pressure regulation, glycemic management, vascular function, high-density cholesterol, and metabolic syndrome, all of which influence cerebrovascular and cardiovascular events [4,6]. Patients who are 25(OH)D3 deficient should take at least 50,000 IU (1,250 mcg) of ergocalciferol, or VD2, once a week or more for 6 - 8 weeks, then 800-1,000 IU (20 - 25 mcg) daily after that [7]. In response to local vascular injury, 1,25-dihydroxycholecalciferol (1,25-VitD3) enhances vascular regeneration [3]. Furthermore, 1,25-VitD3 has anti-inflammatory properties, influences myeloid cell development, and has been linked to the induction of protective vascular genes [8]. As a result, VitD may help avoid the vascular changes that contribute to CVS following SAH. As a result, a lack of 1,25-VitD3 has been related to a higher risk of cardiovascular disease, stroke, and autoimmune disorders [8]. VitD deficiency is more common in patients who require treatment for cerebral aneurysms, and SAH patients have a high prevalence of VitD deficiency [9]. More research is needed to investigate the possible impacts of VitD insufficiency to establish an evidence-based conclusion.

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