D3 for 8 weeks, followed by daily maintenance doses between 1500 and 2000 IU. Both loading and maintenance doses may be folds higher in those with increasing risks for the development or recurrence of vitamin D deficiency. Concurrent calcium supplementation is a key component of effective therapy, and a preventative strategy should always address underlying causes, if possible.117,118

In addition to maintaining sufficient serum 25-OH D levels, patients with end-stage renal and/or hepatic disease impairing vitamin D activation and resulting in hypocalcaemia, in addition to those with secondary hyperparathyroidism or hypoparathyroidism require activated vitamin D therapy (e.g. 1, 25-OH D$_2$: 0.25–0.5 μg/day).119,120 Patients with granulomatous disorders and dysregulated 1, 25-OH D$_2$ activity may require vitamin D replacement, but 25-OH D levels $>30$ ng/mL can worsen the associated hypercalcaemia. Therefore, careful monitoring of vitamin D status, serum, and urinary calcium is necessary in these patients.117,121

**Conclusion**

Vitamin D deficiency is a highly prevalent condition and is independently associated with most CVD risk factors and to CVD morbidity and mortality. Despite a large body of experimental, cross-sectional, and prospective evidence that implicate vitamin D deficiency in the pathogenesis of CVD, the causality of this relationship remains to be established. Most importantly, randomized trials of vitamin D therapy with CVD endpoint are needed to support a role for vitamin D therapy in cardiovascular protection.

**Conflict of interest:** none declared.

**References**

The list of references is available in the online version of this paper.

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**CARDIOVASCULAR FLASHLIGHT**

**An adult with right aortic arch and dysphagia**

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A 38-year-old male presented with history of atypical chest pain and dysphagia. Cardiovascular examination was unremarkable. Electrocardiography and transthoracic echocardiography were normal. Chest radiograph showed right-sided aortic arch (Panel A). Barium swallow was performed, which showed extrinsic compression from right side (Panel B) and from posterior aspect (Panel C) at the level of T4 vertebra. Computed tomography aortogram confirmed right-sided aortic arch with left-sided large aortic diverticulum extending posterior to the oesophagus with marked compression (Panel D and E). There was no compression of the trachea. The volume rendered reconstruction image showed anomalous origin of left common carotid from the ascending aorta and left subclavian artery originating from the diverticulum. The patient underwent successful surgical resection of the diverticulum with reimplantation of the left subclavian artery to descending thoracic aorta. He made prompt recovery with dramatic improvement in dysphagia. Aortic arch anomalies should be ruled out in patients with dysphagia and right aortic arch irrespective of age at presentation. In individuals with suspected aortic arch anomalies, computed tomography imaging by providing an accurate anatomical diagnosis guides surgical decision-making.

Panel A Chest radiograph showing right-sided aortic arch (arrow). Panels B and C Barium swallow imaging showing extrinsic compression on right side (B) and on posterior aspect (C) of oesophagus (arrows). Panels D and E Computed tomography aortogram showing large aortic diverticula (asterisk) with retrooesophageal extension causing marked compression on the oesophagus (arrow). (F) Computed tomography volume rendered aortography showing right-sided aortic arch with anomalous origin of left common carotid from ascending aorta and left subclavian artery arising from aortic diverticulum.

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