Clinical Policy: Measurement of Serum 1,25-dihydroxyvitamin D

Reference Number: PA.CP.MP.152
Effective Date: 05/18
Last Review Date: 12/18

Coding Implications
Revision Log

Description

Vitamin D is metabolized in the liver to 25-hydroxyvitamin D [25(OH)D], (also known as calcidiol), and then in the kidney to 1,25-dihydroxyvitamin D [1,25(OH)2D], also known as calcitriol. 25(OH)D is the major circulating form of vitamin D while 1,25(OH)2D is the active form of vitamin D. In individuals at risk for vitamin D deficiency, the best method for determining a person's vitamin D status is to measure a 25(OH)D concentration. Measurement of 1,25(OH)2D is not useful for monitoring the vitamin D status, as it does not reflect vitamin D reserves.[1] This policy addresses when measurement of 1,25(OH)2D is appropriate and medically necessary.

Policy/Criteria

I. It is the policy of PA Health & Wellness (PHW), that measurement of serum 1,25(OH)2D (CPT 82652) is **medically necessary** for monitoring certain conditions, such as acquired and inherited disorders of vitamin D and phosphate metabolism, including any of the following indications:

A. Chronic kidney disease;
B. Hereditary phosphate-losing disorders;
C. Oncogenic osteomalacia;
D. Pseudovitamin D-deficiency rickets;
E. Vitamin D-resistant rickets;
F. Chronic granuloma-forming disorders (e.g., sarcoidosis and some lymphomas).

II. It is the policy of PHW that measurement of serum 1,25(OH)2D for routine screening of average risk, asymptomatic individuals is **not medically necessary.**

Background

Vitamin D or calciferol, is a fat-soluble vitamin that plays an important role in calcium homeostasis and bone health. Vitamin D comes in two forms, D_2 and D_3. It is unique among hormones because the major source of vitamin D is exposure to natural sunlight. Very few foods naturally contain, or are fortified with, vitamin D, thus, the major cause of vitamin D deficiency is inadequate exposure to sunlight.

Screening for Vitamin D deficiency is recommended for individuals at risk, such as those with osteomalacia, osteoporosis, chronic kidney disease, hepatic failure, malabsoprtion syndromes, hyperparathyroidism, African-American and Hispanic children and adults, pregnant or lactating women, older adults with history of falls or non-traumatic fractures, obese children or adults (BMI greater than 30 kg/m^2), granuloma-forming disorders, and some lymphomas.[1]

CLINICAL POLICY

Measurement of Serum 1,25-dihydroxyvitamin D

Circulating 25(OH)D is the best indicator to monitor for vitamin D status as it is the main circulating form of vitamin D, and has a half-life of two to three weeks. In contrast, 1,25(OH)2D, has a much shorter half-life of about four hours, circulates in much lower concentrations than 25(OH)D, and is susceptible to fluctuations induced by PTH in response to subtle changes in calcium levels. Serum 1,25(OH)2D is frequently either normal or even elevated in those with vitamin D deficiency, due to secondary hyperparathyroidism.[1]

The Endocrine Society

The Endocrine Society recommends using the serum circulating 25-hydroxyvitamin D [25(OH)D] level, measured by a reliable assay, to evaluate vitamin D status in patients who are at risk for vitamin D deficiency and in whom a prompt response to optimization of vitamin D status could be expected. They note further, 1,25(OH)2D measurement does not reflect vitamin D status as levels are tightly regulated by serum levels of PTH, calcium, and phosphate. Serum 1,25(OH)2D does not reflect vitamin D reserves, and measurement of 1,25(OH)2D is not useful for monitoring the vitamin D status of patients. Serum 1,25(OH)2D is frequently either normal or even elevated in those with vitamin D deficiency, due to secondary hyperparathyroidism. Measurement of 1,25(OH)2D is useful in acquired and inherited disorders in the metabolism of 25(OH)D and phosphate, including chronic kidney disease, hereditary phosphate-losing disorders, oncogenic osteomalacia, pseudovitamin D-deficiency rickets, vitamin D-resistant rickets, as well as chronic granuloma-forming disorders such as sarcoidosis and some lymphomas.

United States Preventive Services Task Force (USPSTF)

The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening for vitamin D deficiency in asymptomatic adults.

American Congress of Obstetricians and Gynecologists

At this time, there is insufficient evidence to support a recommendation for screening all pregnant women for vitamin D deficiency. For pregnant women thought to be at increased risk of vitamin D deficiency, maternal serum 25-hydroxyvitamin D levels can be considered and should be interpreted in the context of the individual clinical circumstance.[3]

Coding Implications

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CPT® Codes	Description
82652	Vitamin D; 1, 25 dihydroxy, includes fraction(s), if performed

CLINICAL POLICY
Measurement of Serum 1,25-dihydroxyvitamin D

HCPCS Codes	Description
N/A	

ICD-10-CM Diagnosis Codes that Support Coverage Criteria

ICD-10-CM Code	Description
A15.0-A15.9	Respiratory tuberculosis
C81.00-C81.99	Hodgkin lymphoma
C82.00-C82.99	Follicular lymphoma
C83.00-C83.99	Non-follicular lymphoma
C84.00-C84.99	Mature T/NK-cell lymphomas
C88.0-C88.9	Malignant immunoproliferative diseases and certain other B-cell lymphomas
D86.0-D86.9	Sarcoidosis
E20.00	Idiopathic hypoparathyroidism
E20.8	Other hypoparathyroidism
E21.0-E21.5	Hyperparathyroidism and other disorders of parathyroid gland
E55.0	Rickets, active
E83.30-E83.39	Disorder of phosphorus metabolism and phoshatases
E83.50-E83.59	Disorders of calcium metabolism
N18.1-N18.9	Chronic kidney disease (CKD)
N25.0	Renal osteodystrophy
P37.0	Congenital tuberculosis

Reviews, Revisions, and Approvals	Date	Approval Date
Policy developed	04/18	09/24/18
Removed CPT code 82306 as the policy does not apply to this test. References reviewed and updated.	12/18	

References

1. Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, Treatment, and Prevention of Vitamin D Deficiency: an Endocrine Society Clinical Practice Guideline. The Journal of Clinical Endocrinology & Metabolism, Volume 96, Issue 7, 1 July 2011, Pages 1911–1930. Accessed Oct 24, 2018. Available at: https://academic.oup.com/jcem/article-lookup/doi/10.1210/jc.2011-0385
2. United States Preventive Services Task Force. Vitamin D Deficiency: Screening November 2014

3. American College of Obstetricians and Gynecologists. Vitamin D: Screening and Supplementation During Pregnancy. Committee Opinion Number 495. July 2011. Reaffirmed 2017.
4. Pazirandeh S, Burns DL. Overview of vitamin D. Jan 2016. In: UpToDate, Waltham, MA. Motil KJ, Drezner, MK (Eds). . Accessed 10/24/17
5. Dawson-Hughes B. Vitamin D deficiency in adults: Definition, clinical manifestations, and treatment. In: UpToDate, Waltham, MA. Drezner MK, Rosen CJ (Eds).. Accessed 10/24/18.
6. Misra M. Vitamin D insufficiency and deficiency in children and adolescents. In: UpToDate, Waltham, MA, Motil KJ, Drezner MK (Eds). Accessed 10/15/17
7. Drezner MK. Causes of vitamin D deficiency and resistance. In: UpToDate, Waltham, MA. Rosen CJ SA (Ed). Accessed 10/24/18.
8. Tebben PJ, Singh RJ, Kumar R. Vitamin D-Mediated Hypercalcemia: Mechanisms, Diagnosis, and Treatment. Endocr Rev. 2016 Oct;37(5):521-547. Epub 2016 Sep 2.
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10. Ruppe MD. X-Linked Hypophosphatemia. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2017. 2012 Feb 9 [updated 2017 Apr 13].