

Division of Laboratory Medicine

Biochemistry

1,25-dihydroxy vitamin D (DHVD)

For diagnosis of abnormalities in calcium and phosphate metabolism. Can be used for monitoring replacement therapy in chronic kidney disease (CKD).

NOTE: The DHVD test must NOT be used in the monitoring of vitamin D status. The appropriate test for monitoring of vitamin D status, is 25OH vitamin D profile as these metabolites have a longer half-life and are better indicators of total body vitamin D stores.

Pseudonyms: Calcitriol, 1,25-dihydroxycholecalciferol, 1alpha,25-dihydroxyvitamin D3

General information

Collection container: Sarstedt serum tubes - with or without gel

Type and volume of sample: serum, collect 5mL whole blood where possible as a minimum of 0.5mL serum is required.

Specimen transport/special precautions: samples can be sent in the pneumatic transport or with a porter.

External users: DEQAS data demonstrated the samples are stable for up to 5 days at room temperature therefore first class post transport is adequate. If sent frozen at least 2 freeze-thaw cycles do not affect the results.

Laboratory information

Method principle:

DHVD is extracted from samples using immunoprecipitation before derivatisation and analysis by LC-MS/MS.

Biological reference range - 43 to 144pmol/L. It is more important to interpret the result in light of the bone chemistry (calcium, phosphate and ALP) along with PTH and 25OH vitamin D levels than to look at an absolute value.

Turnaround times: Analysed as a batch once per month.

Clinical information

Factors known to significantly affect the results: Haemolysis, lipaemia, and icterus do NOT significantly affect the results however levels in Li heparin (orange top) and EDTA (red top) plasma may be up to 10% higher.

Clinical decision points: There are a number of conditions in which DHVD measurement is useful, summarised in the table below.

Division of Laboratory Medicine

Biochemistry

For any disease, the biochemical abnormalities can be variable at presentation, multifactorial and not mutually exclusive e.g. infection with co-existing 25OHD deficiency.

Cause of altered [DHVD]	Calcium	Phosphate	PTH	25OHD	DHVD	Notes
Severe 25OHD deficiency	↓	↓	↑	↓	N/↓	Unusual. Due to lack of substrate
CKD	↓	↑	N/↑	↓	↓	eGFR<60mL/min are effectively 1αOHase deficient (inhibited by acidosis, phosphate retention, raised FGF23 and depleted 25OHD).
Vitamin D dependent Rickets(VDDR) I = 1αOHase deficiency	↓	↓	↑	N	↓	Will respond to DHVD treatment.
VDDR II (receptor mutation)	↓	↓	↑	N	↑	Often have alopecia. Do not respond to DHVD treatment
Familial hypophosphataemia	N	↓	N	N	↓	Several gene mutations/forms possible (PHEX, DMP1, SLC34A3). FGF23 elevated
Primary Hyperparathyroidism (PHPT)	↑	↓	↑	N	N/↑	Elevations in DHVD may not be very high.
Granulomatous Disease (e.g. sarcoidosis, IBD)	N/↑	N	N/↓	N	↑	Unregulated extra-renal 1αOHase activity
Infectious causes (e.g. TB, pneumonia, HIV)	↑	N	↓	N	↑	Unregulated extra-renal 1αOHase activity
Neoplastic Disease (e.g lymphoma)	↑	N	↓	N	↑	Unregulated extra-renal 1αOHase activity - associated with macrophages rather than tumour itself
Tumour-induced osteomalacia	↑	N	↓	N	↓	Tumour production of FGF23 prevents DHVD synthesis

(Last updated March 2018)