Alkaline Phosphatase; ALP

Pseudonyms: Basic Phosphatase

General information

Collection container:
Adults - serum (with gel separator, 4.9mL Sarstedt brown top).
Paediatrics - lithium heparin plasma (1.2mL Sarstedt orange top tube).

Type and volume of sample: The tubes should be thoroughly mixed before transport to the lab. 1mL whole blood is required as a minimum volume if only a bone profile or liver profile is requested. ALP is not available as a single test request.

Laboratory information

Method principle: ALP is analysed on the automated instruments by a colourimetric assay using p-nitrophenyl phosphate as the indicator.

The assay is standardised against a manual spectrophotometric method where results are compared to the calculated absorbance for a given amount of product generated.

Biological reference ranges:

0-7 days	75-300 U/L
8-28 days	90-477 U/L
29-90 days	90-540 U/L
91-180 days	77-540 U/L
181-360 days	87-382 U/L
361-540 days	69-434 U/L
>540 days - 2 years	60-370 U/L
2-10 years	60-320 U/L (300 U/L in males)
10-17 years	60-400 U/L
>17 years	30-130 U/L

Turnaround times: Results are typically available within 1 hour if urgent, 4 hours non urgent and next day for primary care.

Clinical information

ALP exists in various isoforms, some being true isoenzymes, that are encoded by different genes. It is widely distributed in the body, but is particularly associated with bone (osteoblasts), small intestine (mucosal cells), liver (cells of the biliary system), placenta and kidney (proximal convoluted tubules).
The forms of ALP usually present in circulation are equally of liver and bone origin.

Physiological elevations are associated with pregnancy, particularly in the third trimester and pubertal growth spurts. ALP is frequently elevated in Rickets, osteomalacia and Pagets disease and reflects the healing progress of fractures. In a jaundiced patient, a high serum ALP (up to 5 times the upper reference limit) suggests predominant cholestasis. This may be intrahepatic (e.g. cirrhosis) or extrahepatic (e.g. carcinoma of the pancreas obstructing the common bile duct); ALP does not help to distinguish between these causes. ALP is often elevated in hepatocellular causes of jaundice (because there is frequently an element of cholestasis) but typically to less than 3 times the upper reference limit. ALP activity is of prognostic value in primary biliary cirrhosis and primary sclerosing cholangitis.

Benign Transient Hyperphospatasaemia: This phenomenon characterised by very high ALP activity is usually observed in children < 5y old with no evidence of liver or bone disease on examination and no other abnormalities in biochemistry (LFT, bone or renal profiles). The levels can be in the thousands and are likely to return to within the reference range by 3 months later but it is advised to take a repeat sample 6 months later.

Hypophosphatasia: This is a rare disorder presenting with very low ALP activity, bone demineralisation, tooth loss and calcium crystal arthropathies and has 5 forms characterised by the age of presentation and severity of symptoms.

Further information: http://www.sciencedirect.com/science/article/pii/S8756328215000678

Factors known to significantly affect the results: Contamination of the sample with EDTA can reduce the apparent activity as metal co-factors essential for enzyme activity are chelated, although the effect is variable as the assay has zinc and magnesium added.

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