H·O·D
HOUSE of DIAGNOSTICS

Patient Name :	Demo Patient Name	**Lab No :**	Demo Visit No
Age / Sex :	28 Y / M	**Registration On :**	31-Dec-24 14:37
Referred By :	DEMO HOSPITAL	**Patient ID :**	UHID.DEMO.001
Centre :	HOD Head Office		

Protein S

Sodium Citrate 3.2% Sample

Accession No: DEMO_BARCODE **Collected On:** 31-Dec-24 14:37 **Received On:** 31-Dec-24 17:39 **Approved On:** 31-Dec-24 19:32

Observation	Result	Unit	Biological Ref. Interval	Method
Free Protein S Activity	**28.2**	%	74.1 - 146.1	Optical (Coagulation)

Clinical Significance
Protein S is a vitamin K-dependent plasma glycoprotein synthesized predominantly within the liver. Protein S is also synthesized in endothelial cells and present in platelets. As a part of the plasma anticoagulant system, protein S acts as a necessary cofactor to activated protein C (APC) in the proteolytic inactivation of procoagulant factors Va and VIIIa. About 60% of the total plasma protein S antigen circulates bound to C4b binding protein (C4b-BP), while the remainder circulates as "free" protein S. Only free protein S has anticoagulant activity. Congenital protein S deficiency is an autosomal codominant disorder that is present in 1% to 3% of patients with venous thromboembolism. Heterozygous protein S deficiency carriers have approximately a 10-fold increased risk of venous thromboembolism. Other phenotypic expressions of heterozygous congenital protein S deficiency include recurrent miscarriage, complications of pregnancy (preeclampsia, abruptio placentae, intrauterine growth restriction, and stillbirth) and possibly arterial thrombosis. Three types of heterozygous congenital protein S deficiency have been described according to the levels of total protein S antigen, free protein S antigen, and protein S (APC cofactor) activity in plasma.
Types of Heterozygous Protein S Deficiency

Type	Protein S Antigen	Free Protein S Antigen	Total Protein Activity
I	Decreased	Decreased	Decreased
II	Normal	Normal	Decreased
III	Decreased	Normal	Decreased

Type I and III protein S deficiency are much more common than Type II (dysfunctional) protein S deficiency. Type III protein S deficiency appears to be partly due to mutations within the protein S binding region for C4b-BP. Homozygous protein S deficiency is rare, but can present as neonatal purpura fulminans, reflecting severe intravascular coagulation and fibrinolysis/disseminated intravascular coagulation (ICF/DIC) caused by the absence or near absence of plasma protein S. Acquired deficiency of protein S is much more common than hereditary protein S deficiency and is generally of unknown hemostatic significance (ie,uncertain thrombosis risk). Among the many causes of acquired protein S deficiency are:
-Vitamin K deficiency, Oral anticoagulant therapy, Acute illness (eg, acute thrombosis, recent surgery, or other disorder associated with acute inflammation), Liver disease, ICF/DIC, Thrombotic thrombocytopenic purpura, Pregnancy, oral contraceptive, or estrogen therapy, Nephrotic syndrome and Sickle cell anemia

Note: Newborn infants have normal or near-normal free protein S antigen (>=50%), although total protein S antigen is usually below the adult reference range. There are insufficient data concerning protein S activity in normal neonates, infants, and children; but normal or near-normal activity (> =50%) probably is present by age 3 to 6 months.

Cautions:
Spuriously low protein S activity may be observed in conditions with
- Very high factor VIII (>250%) activity or Activated protein C resistance (eg, heterozygosity or homozygosity for the factor V Leiden mutation)
- Very high factor VIII (>250%) activity or Activated protein C resistance (eg, heterozygosity or homozygosity for the factor V Leiden mutation) may cause a spuriously low protein S activity result.
- Heparin level >1 U/mL may cause a false-high result.
- Activated protein C resistance (eg, heterozygosity or homozygosity for the factor V Leiden mutation) may cause a spuriously low protein S activity result.
- The presence of a lupus anticoagulant or a specific factor V inhibitor may cause the protein S activity to appear spuriously normal (or elevated), when protein S activity is truly decreased (or normal).
- Coumadin therapy may result in decreased protein S activity (and free protein S antigen).
- Acute or chronic inflammation can result in decreased protein S activity (and free protein S antigen).
Interpret protein S activity results with caution when any of the above patient conditions are present. Protein S antigen assay (free protein S antigen, with concomitant or reflexive total protein S antigen assay), rather than protein S activity (functional) assay, is recommended as the initial testing approach for detecting congenital protein S deficiency, because of the greater variety of patient conditions that can interfere with the accuracy of functional testing as compared to antigen testing.
In general, it is preferable not to test for protein S deficiency during acute illness, pregnancy, or postpartum. Elective testing for protein S deficiency should be delayed for at least 30 days after cessation of warfarin therapy.

Remarks: Please correlate results with clinical conditions.

This is a Demo Signature
and the doctor's signature should appear here

In case of any unexpected or alarming results, please contact us immediately for re-confirmation, clarifications, and rectifications, if needed.

Scan to Validate