MAYO CLINIC LABORATORIES

Test Definition: PTFIB

PT-Fibrinogen, P

Overview

Useful For

Detecting increased or decreased fibrinogen (factor 1) concentration of acquired or congenital origin
Differentiating hypofibrinogenemia from dysfibrinogenemia

Method Name

Only orderable as part of a coagulation reflex. For more information see:
ALUPP / Lupus Anticoagulant Profile, Plasma
ALBLD / Bleeding Diathesis Profile, Limited, Plasma
AATHR / Thrombophilia Profile, Plasma
APROL / Prolonged Clot Time Profile, Plasma
ADIC / Disseminated Intravascular Coagulation/Intravascular Coagulation and Fibrinolysis (DIC/ICF) Profile, Plasma

NY State Available

Yes

Specimen

Specimen Type

Plasma Na Cit

Specimen Required

Only orderable as part of a coagulation reflex. For more information see:
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AATHR / Thrombophilia Profile, Plasma
APROL / Prolonged Clot Time Profile, Plasma
ADIC / Disseminated Intravascular Coagulation/Intravascular Coagulation and Fibrinolysis (DIC/ICF) Profile, Plasma

Reject Due To

Gross hemolysis	Reject
Gross lipemia	Reject
Gross icterus	Reject

Specimen Minimum Volume

1 mL

Specimen Stability Information

Specimen Type	Temperature	Time	Special Container
Plasma Na Cit	Frozen (preferred)	14 days	

Clinical & Interpretive

Clinical Information

Fibrinogen, also known as factor 1, is a plasma protein that can be transformed by thrombin into a fibrin gel ("the clot"). Fibrinogen is synthesized in the liver and circulates in the plasma as a disulfide-bonded dimer of 3 subunit chains. The biological half-life of plasma fibrinogen is 3 to 5 days.

An isolated deficiency of fibrinogen may be inherited as an autosomal recessive trait (afibrinogenemia or hypofibrinogenemia) and is one of the rarest of the inherited coagulation factor deficiencies.

Acquired causes of decreased fibrinogen levels include acute or decompensated intravascular coagulation and fibrinolysis (disseminated intravascular coagulation), advanced liver disease, L-asparaginase therapy, and therapy with fibrinolytic agents (eg, streptokinase, urokinase, tissue plasminogen activator).

Fibrinogen function abnormalities, dysfibrinogenemias, may be inherited (congenital) or acquired. Patients with dysfibrinogenemia are generally asymptomatic. However, the congenital dysfibrinogenemias are more likely than the acquired to be associated with bleeding or thrombotic disorders. While the dysfibrinogenemias are generally not associated with clinically significant hemostasis problems, they characteristically produce a prolonged thrombin time clotting test. Congenital dysfibrinogenemias usually are inherited as autosomal codominant traits.

Acquired dysfibrinogenemias mainly occur in association with liver disease (eg, chronic hepatitis, hepatoma) or renal diseases associated with elevated fibrinogen levels.

Fibrinogen is an acute-phase reactant, so a number of acquired conditions can result in an increase in its plasma level:

-Acute or chronic inflammatory illnesses

-Nephrotic syndrome

-Liver disease and cirrhosis

-Pregnancy or estrogen therapy

-Compensated intravascular coagulation

The finding of an increased level of fibrinogen in a patient with obscure symptoms suggests an organic rather than a functional condition. Chronically increased fibrinogen has been recognized as a risk factor for development of arterial and venous thromboembolism.

Reference Values

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APROL / Prolonged Clot Time Profile, Plasma

ADIC / Disseminated Intravascular Coagulation/Intravascular Coagulation and Fibrinolysis (DIC/ICF) Profile, Plasma

261-595 mg/dL

In normal full-term newborns and in healthy pre-mature infants (30-36 weeks gestation) fibrinogen is near adult levels (>150) and remains at adult levels throughout childhood.

Interpretation

This test assesses the level of total clottable fibrinogen (see Cautions).

Cautions

Fibrinogen assay results may be affected by excess heparin (>1 U/mL), hemoglobin (>100 mg/dL), triglycerides (>700 mg/dL), bilirubin (>15 mg/dL), and by degradation products (fibrin or fibrinogen) in the plasma assayed.

Clinical Reference

1. Rossi E, Mondonico P, Lombardi A, Preda L: Method for the determination of functional (clottable) fibrinogen by the new family of ACL coagulometers. Thromb Res 1988 Dec;52(5):453-468
2. Palareti G, Maccaferri M, Manotti C, et al: Fibrinogen assays: A collaborative study of six different methods. Clin Chem 1991 May;37(5):714-719

Performance

Method Description

The PT Fibrinogen assay is performed using the HemosIL PT-Fibrinogen kit on the Instrumentation Laboratory ACL TOP. Prothrombin Time (PT) thromboplastin reagent is added to patient plasma; endogenous thrombin from the patient's plasma is generated during the reaction and converts fibrinogen to fibrin. This change from fibrinogen to fibrin is monitored by the instrument through reading the light absorbance over a set amount of time. At the end of the allotted time, the instrument uses a set algorithm to determine the delta and plot it against the calibration curve. The delta value is directly proportional to the amount of fibrinogen in the sample.(Package insert: HemosIL PT Fibrinogen. Instrumentation Laboratory Company, Lexington, MA01/2016)

PDF Report

No

Specimen Retention Time

7 days

Performing Laboratory Location

Rochester

Fees & Codes

Test Classification

This test has been cleared, approved, or is exempt by the US Food and Drug Administration and is used per manufacturer's instructions. Performance characteristics were verified by Mayo Clinic in a manner consistent with CLIA requirements.

CPT Code Information

85385