

Patient Name : Mr MR.DUMMY

DOB/Age/Gender : 23 Y/Male

Patient ID / UHID : 8052575/RCL7248767

Referred By : Dr. Dr. X

Sample Type : Citrate Plasma

Sample Collected : Apr 26, 2024, 01:00 PM

Report Date : May 25, 2024, 06:02 PM.

Barcode No : HY585715

Report Status : Final Report

Test Description	Value(s)	Unit(s)	Reference Range
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Thrombophilia Profile

Activated Partial Thromboplastin Time (APTT)

APTT <i>Viscosity-based clot detection</i>	33.9	Sec	23.8-34.8
Control (MNAPTT)	28.0		

Interpretation:

The APTT is a one-stage test. This is used for the diagnosis of bleeding disorders. APTT may be used in the patient to check treatment for those who are taking Heparin or other blood-thinning medicines. APTT measures the intrinsic system and common pathways. APTT used in the diagnosis of Hemophilia and Christmas disease.

Abnormal High results of APTT are due to:

1. All congenital deficiencies of Intrinsic system coagulation factors.
2. Cirrhosis, Drugs, Heparin therapy, Warfarin therapy.
3. Disseminated intravascular coagulopathy (DIC), Fibrin breakdown products.
4. Factor XII deficiency, Hemophilia A and B, Hypofibrinogenemia, Von Willebrand's disease.
5. Malabsorption, Vit K deficiency, Fibrin breakdown products, Leukemia.
6. In the case of streptokinase and urokinase.
7. Circulating anticoagulant inhibitors.

These may be specific for factor VIII.

1. These are seen as anti-factor VIII and anti-factor IX in 5% to 10% of hemophilic patients.
2. These are also in multiple plasma transfusions.
3. Drug reactions.
4. In the case of tuberculosis.
5. In autoimmune diseases like SLE and rheumatoid arthritis.

**Dr. Dummy**

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Processing Lab :-

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Anti Thrombin III Activity/Functional

Anti Thrombin III Activity <i>Viscosity-based clot detection</i>	81	%	Non Pregnant : 80-120 First Trimester : 89-114 Second Trimester : 88-112 Third Trimester : 82-116
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Interpretation:**Interpretation :**

1. Anti Thrombin III is a protein molecule that inactivates several enzymes of the coagulation system.
2. Congenital ATIII deficiency leads to increased risk of venous and arterial thrombosis, with an onset of clinical manifestations typically appearing in young adulthood.
3. At present, the Anti-thrombin congenital deficiencies are classified into four categories as below:-

Type of Deficiency	Protein Amount	Biological Activity	Remarks
I	Decreased	Decreased	More frequent case
II RS (reactive site)	Normal	Decreased	Anomaly of the reactive site
II HBS (heparin binding site)	Normal	Normal when heparin is absent	Anomaly of the AT-heparin binding
II PE (pleiotropic effect)	Decreased	Decreased	Dysfunctional protein and at reduced quality

4. Acquired ATIII deficiency occurs in DIC, hemolytic uremic syndrome and venoocclusive disease (VOD) in patients undergoing bone marrow transplant, Nephrotic syndrome , Liver disease and L-asparaginase treatments

Increased levels of antithrombin occur in :

1. Acute hepatitis/cholestasis .
2. Kidney transplant .
3. Vitamin K deficiency .
4. Warfarin (coumadin) anticoagulation therapy (sometimes) Testing for antithrombin deficiency is not recommended unless the patients condition is stable.

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Lupus Anticoagulant

Patient Value	42.1	sec	33.1-45.1
Control value	39.1	sec	
Screen Ratio	1.08		<1.20
DRVVT Screening	Negative		

Interpretation:**Method:** Dilute Russell viper venom method (dRVV), electromechanical clot detection.**Remarks:**

1. This is only a screening test.

2. If Screening test is positive, then a confirmatory test is necessary.

3. The presence of LA in the sample is confirmed when the Normalized Ratio (calculated as ratio of dRVV screen ratio to dRVV confirmatory ratio) value is greater or equal to reference value.

Test description: Diluted RVV Screen test is performed with reagent containing a low concentration of phospholipids. If lupus anticoagulant (LA) is present, the clotting time will be lengthened. dRVV confirmatory testing is done with reagent containing higher concentration of phospholipids, which neutralizes the LA (when present in the sample) and corrects the clotting time to normal thereby confirming the presence of LA.**Notes:**

1. As per ISTH(International society on thrombosis and hemostasis) guidelines , Lupus Anticoagulant detection must be done by using at least two clot based assays employing separate clotting principles like Lupus sensitive APTT & dRVVT.

2. Results of this test should always be interpreted in conjunction with the patient's medical history, clinical presentation and other findings.

3. A positive LA can be seen in otherwise normal individuals and in certain viral or other infections.

4. Once a patient has been tested positive for LA, it is imperative that testing be repeated on a second occasion > 12 weeks after the initial testing.

5. Anticoagulation therapy effects such as Warfarin (especially when the effect is supratherapeutic), excess Heparin, direct thrombin inhibitors (DTI) (eg, Dabigatran [Pradaxa]), Argatroban [Ancova], Bivalirudin [Angiomax]), direct factor Xa inhibitors (eg, Rivaroxaban [Xarelto], Apixaban [Eliquis], Edoxaban [Savaysa]) may result in a false-positive assay performance for LA. Clinical correlation and repeat testing after discontinuation (>1 week) of anticoagulation therapy is suggested.

6. Although the dilute Russell viper venom time (dRVVT) reagents contain a heparin inhibitor (Polybrene) that is sufficient for neutralization of heparin (up to 1-2 U/mL), the results may not necessarily represent what would occur if no heparin were present in the specimen. Therefore, DRVVT results from heparinized plasma should be interpreted with caution.

7. dRVVT assays, when performed in isolation, will not distinguish LA from heparin or inhibitors of factors V or VIII, which may cause false-positive results of LA testing.

Comments: Lupus Anticoagulants are heterogenous IgG or IgM autoantibodies which interfere with phospholipid dependent in vitro coagulation tests, particularly activated partial thromboplastin time (APTT). These antibodies are associated with thrombosis (arterial & venous), recurrent abortions, neurological & neuropsychiatric disorders. Various methods for testing Lupus Anticoagulants include Lupus sensitive APTT (PTT-LA), activated kaolin clotting time and dilute Russell Viper Venom time. Out of these the dRVVT assay is the most robust & specific because dRVVT is not influenced by deficiencies of intrinsic pathway or antibodies to factors VIII, IX or XI.a**Protein C Activity / Functional**

Protein C Activity Viscosity-based clot detection	90	%	70-130
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Interpretation:**Interpretation :**

Protein C is a major physiological anticoagulant The activated form (with protein S as a cofactor) degrades Factor Va and Factor VIIIa. An inherited decrease in protein C increases the patients risk for venous thromboembolism.

1. Artificially decreased functional protein C values may be seen in patients with abnormally elevated levels of factor VIII.

2. Artificially increased levels of functional protein C values may be seen in patients on heparin therapy.

3. Patients on oral anticoagulants will have decreased functional protein C values. Patients should be off oral anticoagulant therapy for two weeks for accurate measurement of functional protein C levels.

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PROTEIN S, (FREE) ANTIGEN.

PROTEIN S, (FREE) ANTIGEN <i>Viscosity-based clot detection</i>	81	%	71-113 For Pregnancy : 1st Trimester : 34 - 133 2nd Trimester : 29 - 113 3rd Trimester : 23 - 65
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Interpretation:**Note**

1. Determination of both Free & Bound Protein S are recommended to identify hereditary or acquired protein S deficiency.
2. Very high factor VIII (>400%) activity, Activated protein C resistance, Coumadin therapy & acute or chronic inflammation may cause spuriously low results.
3. Mutation in factor V at its protein Ca cleavage site may lead to diminished recovery of protein S.
4. Test conducted on citrated plasma.

Comment

Protein S is a vitamin K dependent plasma glycoprotein, exists in plasma as 40% free and 60% bound to C4b Binding protein. Free protein S possesses both ACP - dependent & independent anticoagulant properties. Its deficiency is associated with increased risk of thrombosis. The prevalence of protein S deficiency in thrombophilic population is 7 - 12%. The estimated thrombotic risk is 10-15 folds in patient with this deficiency. There is characteristic decrease of free protein S during pregnancy. At birth protein S activity is close to that of adult with total & free protein S being low.

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Activated Protein C Resistance (APCR)

Activated Protein C Resistance (APCR) <i>Viscosity-based clot detection</i>	121	Sec	<= 120 SECONDS ARE REGARDED AS APCR POSITIVE
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Interpretation:

Test done on Fully Automated Coagulometer (Clotting)

Interpretation :

1. Activated protein C resistance is a hemostatic disorder characterized by a poor anticoagulant response to activated protein C (APC). It is the inability of protein C to cleave Factor Va and/or Factor VIIIa, which allows for longer duration of thrombin generation and may lead to a hypercoagulable state. This may be hereditary or acquired.
2. The ratio of the clot time in the presence of activated protein C (aPC) to the clot time in the absence of aPC is used to determine aPC resistance.
3. Certain interference factors may cause ratio values which cannot clearly be attributed to a particular genotype, or may lead to clotting times that exceed the maximum admitted detection time of the instrument. In these cases, further investigation by PCR and the determination of individual factors are recommended.

False Negative :

1. Low levels of Factor V (<50%) .
2. Thrombin inhibitors (hirudin, argatroban) .

False Positive :

1. Aprotinin.
2. Protamine.

Note :

All borderline results should be confirmed by Factor V Leiden mutant detection test by PCR.

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✉ ccsupport@redcliffelabs.com

🌐 www.redcliffelabs.com

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Referred By : Dr. Dr. X

Sample Type : Serum

Sample Collected : Apr 26, 2024, 01:00 PM

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Barcode No : SI484087

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Test Description	Value(s)	Unit(s)	Reference Range
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Anti Cardiolipin IgG Antibodies

Cardiolipin Antibody ACL- IgG (Serum, EIA)	4.32	GPLU/ml	< 12.0 GPLU/ml
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Interpretation:

RESULT IN GPLU/ml	REMARKS
< 11.9	Negative
≥ 12.0-17.9	Equivocal
≥ 18.0	Positive

Comments

Antibodies against cardiolipin belong to the group of anti-phospholipid antibodies specific for negatively charged phospholipids, components of biological membranes. Cardiolipin is an acidic phospholipid derived from glycerol. Antiphospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus (SLE) and related diseases. The prevalence of anti-cardiolipin antibodies in SLE is 24-50%. The occurrence of anti-cardiolipin antibodies in patients with SLE and related diseases is typical of a secondary anti-phospholipid syndrome (APS). In contrast, anti-cardiolipin antibodies in patients with no other autoimmune diseases characterize the primary anti-phospholipid syndrome (APS). Many studies have shown a correlation between these autoantibodies and an enhanced incidence of thrombosis, thrombocytopenia and habitual abortions (as a consequence of placental infarct). The exact mechanism by which pathogenic anti-phospholipid antibodies induce thrombosis is not yet fully revealed.

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Anti Cardiolipin IgM Antibodies

Cardiolipin Antibody ACL- IgM (Serum, EIA)	4.61	MPLU/ml	< 12.0 MPLU/ml
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Interpretation:

RESULT IN MPLU/ml	REMARKS
< 11.9	Negative
≥ 12.0-17.9	Equivocal
≥ 18.0	Positive

Comments

Antibodies against cardiolipin belong to the group of anti-phospholipid antibodies specific for negatively charged phospholipids, components of biological membranes. Cardiolipin is an acidic phospholipid derived from glycerol. Antiphospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus (SLE) and related diseases. The prevalence of anti-cardiolipin antibodies in SLE is 24-50%. The occurrence of anti-cardiolipin antibodies in patients with SLE and related diseases is typical of a secondary anti-phospholipid syndrome (APS). In contrast, anti-cardiolipin antibodies in patients with no other autoimmune diseases characterize the primary anti-phospholipid syndrome (APS). Many studies have shown a correlation between these autoantibodies and an enhanced incidence of thrombosis, thrombocytopenia and habitual abortions (as a consequence of placental infarct). The exact mechanism by which pathogenic anti-phospholipid antibodies induce thrombosis is not yet fully revealed.

*** End Of Report ***

Disclaimer: Method given in report are only indicative and can be changed depending upon type of machine and kit available at time of testing.

Not all tests at all locations are under NABL scope. Availability of tests under NABL scope varies from lab to lab.

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