

Specimen Collected: 12-Mar-24 12:39

AB Ident Panel-PEG (IRL) Procedure	Received: 13-Mar-24 13:15 Result	Units	Report/Verified: 13-Mar-24 13:25 Reference Interval
PEG Panel Identification	Done		
Antigen Testing, RBC Phenotype Extended	Received: 13-Mar-24 13:15		Report/Verified: 13-Mar-24 13:25
Procedure	Result	Units	Reference Interval
RBC Phenotype Extended	See Below ^{t1}		
Elution And Antibody Identification, RBC	Received: 13-Mar-24 13:15		Report/Verified: 13-Mar-24 13:25
Procedure	Result	Units	Reference Interval
Interp Elution	See Below ^{t2}		
RBC Antibody ID Package (IRL) Procedure	Received: 13-Mar-24 13:14		Report/Verified: 13-Mar-24 13:25
Procedure	Result	Units	Reference Interval
ABORh	O Positive		
Direct Coombs	IgG+C3- * f1		[Negative]
Antibody Identification PKG	See Below ^{t3}		
Selected Liq Nitro 2 Red Cell Panel	Received: 13-Mar-24 13:15		Report/Verified: 13-Mar-24 13:25
Procedure	Result	Units	Reference Interval
Selected Liq Nitro 2 Panel ID	Done		
Selected Liq Nitro 2 Panel ID	Done		
Selected Liquid Red Cell Panel Procedure	Received: 13-Mar-24 13:15		Report/Verified: 13-Mar-24 13:25
Procedure	Result	Units	Reference Interval
Selected Red Cell Panel	Done		
Warm Triple Adsorption Procedure	Received: 13-Mar-24 13:15		Report/Verified: 13-Mar-24 13:25
Procedure	Result	Units	Reference Interval
Warm Triple Adsorption, Nbr. Performed	3		

Interpretive Text

t1: 12-Mar-24 12:39 (RBC Phenotype Extended)

This patient appears to have the following red cell extended phenotype:

DCE/DcE K- Fy(a+b-) Jk(a-b+) S+s-

(ISBT) RH:1,2,3,4,5 KEL:-1 FY:1,-2 JK:-3,4 MNS:3,-4

The patient's red cells were EDTA glycine-acid treated to remove the coating IgG, allowing for the use of antisera requiring antiglobulin reagents.

t2: 12-Mar-24 12:39 (Interp Elution)

The eluate was reactive with all cells tested showing no apparent specificity. This reactivity pattern is consistent with a warm autoantibody.

t3: 12-Mar-24 12:39 (Antibody Identification PKG)

Anti-s(MNS4) and an apparent warm autoantibody were identified in this patient's serum. No additional red cell antibodies were apparent at this time.

*=Abnormal, #=Corrected, C=Critical, f=Result Footnote, H-High, i-Test Information, L-Low, t-Interpretive Text, @=Performing lab

Unless otherwise indicated, testing performed at:

ARUP Laboratories

500 Chipeta Way, Salt Lake City, UT 84108

Laboratory Director: Jonathan R. Genzen, MD, PhD

ARUP Accession: 24-072-900125

Report Request ID: 19129241

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Interpretive Text

t3: 12-Mar-24 12:39 (Antibody Identification PKG)

A warm adsorption procedure using selected R1R1, R2R2, and rr cells was used to remove the autoantibody reactivity revealing the underlying anti-s. The use of this method cannot exclude the possible presence of an alloantibody directed against a high-frequency antigen which could present similar results.

Anti-s is a clinically significant antibody capable of causing transfusion reactions.

Warm autoantibodies may be related to the patient's condition, drug induced (most commonly alpha-methyl dopa, levodopa, mefenamic acid, procainamide, piperacillin, and some cephalosporins, ie cefotetan, ceftriaxone), or may occur in association with a disease process such as infections, neoplasms, and autoimmune disorders. Clinical significance of warm autoantibodies may vary. Increased bilirubin, elevated reticulocyte values and decreased haptoglobin may indicate immune hemolysis.

If red cell transfusion is required for this patient, donor units selected, shall be negative for the s (MNS4) antigen. It is recommended that the institution's policy for selecting and crossmatching units be followed.

Result Footnote

f1: Direct Coombs

IgG: Positive

Complement (C3): Negative

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