AUTO-ANTI-LW^a MAY MIMIC ANTI-D AT FIRST GLANCE

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Introduction

Although LW and D antigens are located on different proteins, LW glycoprotein requires an interaction with Rh proteins to exhibit its expression. RhD positive red blood cells express LW^a more strongly than RhD negative cells.
Hence, weak anti-LW may be mistaken for auto- or alloanti-D. Fortunately, and in contrast to Rh antigens, LW antigens are denatured when treated with dithiothreitol (DTT).

We report a case of a 61-year-old female patient with autoanti-LW^a-antibodies, in need of several peri- and postoperative transfusions, due to a septical infection after implantation and multiple revisions of a knee replacement surgery.
Previously, she had received multiple RhD positive transfusions and already formed an anti-E.

Methods

Standard serological techniques for antibody detection and differentiation were applied (gel-card and tube test; BioRad, Cressier, CH). Rhesus pheno- and genotype were analyzed serologically (Erytra®, Grifols, Duedingen, CH) and molecularly using PCR-SSP (inno-train GmbH, Kronberg i. T., D).
Additionally, molecular LW was confirmed by PCR-SSP (in-house).
Amplicons for *RHD* sequencing were prepared in-house and analyzed externally (Microsynth, Balgach, CH).

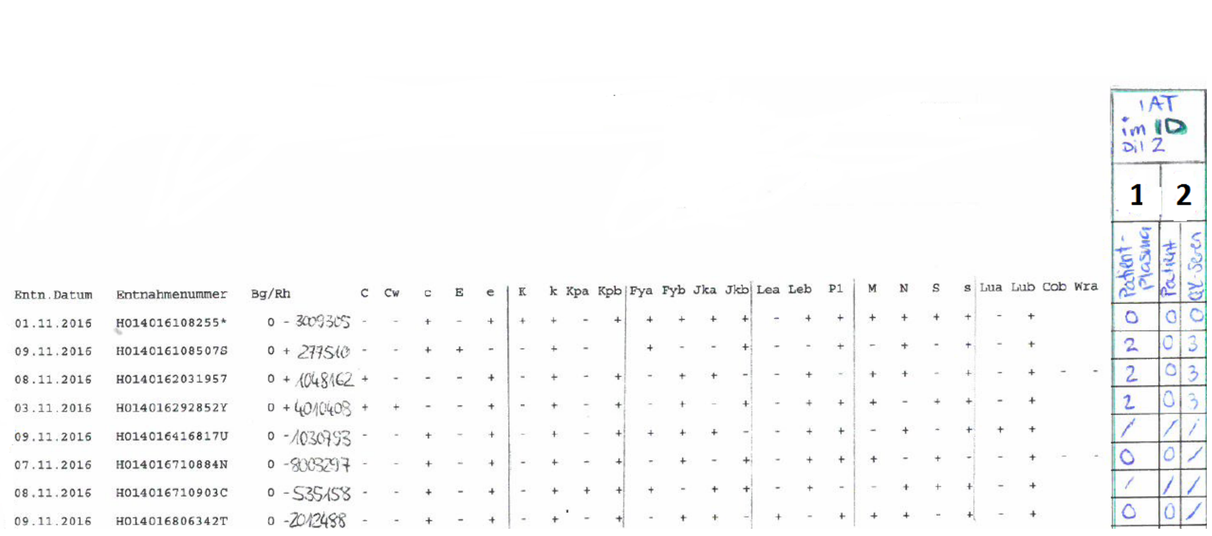

Figure 1: Serological results of antibody differentiation in indirect antiglobulin test with DTT-untreated (**1**) and with 0.2M DTT-treated (2) red blood cells.
In (2) the right column represents the positive controls (Anti-K, Anti-D)

Results

The serological analysis presented an OR$_1$r, K- phenotype.
Except the already known anti-E, the antibody differentiation revealed an antibody, weakly reactive with all RhD positive red blood cells in indirect antiglobulin test and on papainized cells.
The direct antiglobulin test and autocontrol were positive.
Sequencing of the *RHD* gene affirmed the absence of any mutation possibly explaining the presence of an alloanti-D.
However, there were no reactions with DTT-treated RhD positive and RhD negative red blood cells which confirmed the specificity of an anti-LW.
In addition, the genotyping showed the predicted phenotype LW(a+b-), suggesting the diagnosis of an autoanti-LW^a.

month/year	antibody detection	transfused red blood cells	transfusion reactions
08/2000	anti-E	2x O RhD positive, E negative	no
05/2002		3x O RhD positive, E negative	no
12/2007		4x O RhD positive, E negative	no
11/2016	anti-D ?!	4x O RhD **negative**, E negative	no
12/2016		12x O RhD **negative**, E negative	no
03/2017	autoanti-LW^a	7x O RhD positive, E negative	no
04/2017		3x O RhD positive, E negative	no
06/2017		4x O RhD positive, E negative	No

Figure 2: This table shows the transfusion course due to antibody detection . Especially the fact that there were no transfusion reactions by giving RhD positive red blood cells seems to be relevant.

Summary

Before all diagnostic procedures were finalized, the patient was transfused with 16 Orr red blood cell products.
Due to the overall outcome, still presenting autoanti-LW^a, but not showing any signs of hemolysis, we recommended to continue transfusions with RhD positive blood of compatible phenotype (R$_1$r or R$_1$R$_1$). This specifically, since autoantibodies are considered irrelevant for transfusion management.
Our approach was proven right, upon resurgery after 3 months and transfusion of 9 OR$_1$r RBC products, followed by 4 OR$_1$r RBC products another two month later, both times without any hemolytic transfusion reactions. Besides, in our case all crossmatches were consistently negative, but it has to be kept in mind, that autoanti-LW^a may cause positive crossmatches with RhD positive red blood cells.

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