# Do not confuse anti-LW autoantibodies with anti-D

Marcos Paulo Miola
Sandra Valéria Bolonhesi Cervo
Roberta Maria Fachini
Octávio Ricci Júnior

Fundação Faculdade Regional de Medicina – FUNFARME, São José do Rio Preto, SP, Brazil

Even though the anti-LW antibody has little clinical importance, the present report aims at demonstrating that one must be careful about its possible identification. The weak anti-LW may be confused with anti-D auto- or allo-antibodies and differentiating them is important particularly in women during the fertile period, and in $RhD^-$ pregnant women, due to the possibility of the need for anti-RhD prophylaxis. Independently of its complexity and ambiguity, the anti-LW can be identified in transfusional agencies. The LW system[1] has three currently known antigens: the highly frequent $LW^a$ and the less frequent $LW^b$ and $LW^{ab}$. The gene that encodes the LW antigens is independent of Rh genes, but it seems that the LW glycoprotein, in order to express itself, requires an interaction with Rh proteins[2,3]. Even being different, the LW and Rh antigens are phenotypically related in such a way that $RhD^+$ adult individuals express the anti-LW much more strongly than $RhD^-$ individuals.

The expression of LW antigens may be depressed without any apparent reason during pregnancy and in some hematologic diseases (Hodgkin's, leukemias, lymphomas, sarcomas), regaining the normal or almost normal expression after pregnancy and with remission of the diseases[4]. The LW antibodies are not uncommon; they may be produced without apparent exposure during the suppression periods of antigens, generally associated with other antibodies. They are generally IgG, do not cause hemolysis, do not activate the complement system, react to anti-human globulin (AHG) and present strong reactions with $RhD^+$ red cells and may or may not react with $RhD^-$. The LW antigens are resistant to papain and chloroquine treatment but are denatured by treatment with 0.2m dithiothreitol (DTT)[5,6]. To distinguish anti-LW from anti-D, red blood cells must be treated with DTT (it denatures LW antigens but not Rh) and/or test with red cells from umbilical cord blood (as umbilical cord blood presents a high expression of LW antigens, the anti-LW reacts well with $RhD^+$ and $RhD^-$ cells)[6,7].

In our service we investigated a 71-year-old female, diabetic, Caucasian patient, who had had four gestations and suffering from high blood pressure, kidney failure, uterus neoplasia and in an acute pneumonic state. Laboratorial exams showed: pre-transfusion hemoglobin 8.4g/dL; BG: O; $RhD^+$; auto control: positive; direct antiglobulin test (DAT - polyspecific and monospecific) positive; complement test: negative; red cell panel (to study and identify antibodies): positive, anti-E, -LW identified; eluate: auto anti-LW; indirect antiglobulin test (IAT) with cord blood red cells: positive (serum and eluate); phenotyping (post-treatment with chloroquine): Rh: 1, 2, -3, 4, 5, -8 ($R_1R_0$); KEL:-1; JK:1-2; absorption and elutions with red cells $R_0R_0$ and r'r: negative for anti-G[8]; selected panel of red cells [Rh ($D^-$, $E^+$)] and treated with DTT: positive; red cell $R_0R_0$ treated with DTT: negative, due to denaturation of the LW antigens.

We concluded a diagnosis of anti-LW associated with anti-E. In the transfusion, as the LW phenotype of the donated blood was unknown, we opted for transfusion of O $RhD^-$ packed red cells as the LW antigens present weak expression in the absence of the RhD antigen[7]. We consider acceptable to conclude that anti-LW autoantibodies may not be identified and are probably being falsely defined as autoantibodies of the Rh system.

## References

1. International Society of Blood Transfusion. Names for LW (ISBT 016) Blood Group Alleles [Internet]. London: ISBT: [cited 2012 Jun 21]. Available from: http://ibgrl.blood.co.uk/ISBTPages/AlleleTerminology/016%20LW%20Alleles%20final%20Oct%2009.pdf
2. Brecher ME. Technical Manual. 15th ed. Bethesda, MD: American Association of Blood Banks; 2005.
3. Winters JL, Howard DS. Red blood cell antigen changes in malignancy: case report and review. Immunohematology. 2001;17(1):1-9.
4. Reid ME, Lomas-Francis C. The blood group antigen facts boob. 2nd ed. New York: Academic Press; 2004.
5. Vos GH, Petz LD, Garrarry G, Fudenberg HH. Autoantibodies in Acquired Hemolytic Anemia With Special Reference to the LW System. Blood. 1973;42(3):445-53.
6. Byrne KM, Byrne PC. Review: other blood group systems - Diego, Yt, Xg, Scianna, Dombrock, Colton, Landsteiner-Wiener, and Indian. Immunohematology. 2004;20(1):50-8.
7. Daniels G. Human Blood Groups. 2nd ed. Oxford, England: Blackwell Science; 1995.
8. International Society of Blood Transfusion. Names for XG (ISBT 012) Blood Group Alleles [Internet]. London: ISBT: [cited 2012 Jun 21]. Available from: http://www.isbtweb.org/fileadmin/user_upload/WP_on_Red_Cell_Immunogenetics_and/012_XG_Alleles_v2.0_110914.pdf

xxx

Conflict-of-interest disclosure:
The authors declare no competing financial interest

Submitted: 9/13/2012
Accepted: 4/4/2013

**Corresponding author:**
Marcos Paulo Miola
Hemocenter de São José do Rio Preto
Av. Jamil Feres Kfouri, nº 80 - Jardim Panorama
15091-240 São José do Rio Preto, SP, Brazil
Phone: 55 17 3201-5027
mpmiola@gmail.com

www.rbhh.org or www.scielo.br/rbhh

DOI: 10.5581/1516-8484.20130042