Do not confuse anti-LW autoantibodies with anti-D

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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 has three currently known antigens: the highly frequent LW and the less frequent LW and LW. 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. 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. The LW antigens 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, and 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 has three currently known antigens: the highly frequent LW and the less frequent LW and LW. 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. 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.

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 (R0); KEL:-1; JK:1-2; absorption and elutions control: positive; direct antiglobulin test (DAT - polyspecific and monospecific) positive; complement test: negative; red cell R0R0 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- may or may not react 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). 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).

We considered acceptable to conclude that anti-LW autoantibodies may not be identified and are probably being falsely defined as autoantibodies of the Rh system.

References


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