CASE REPORT: AUTOANTI-D CAUSING ACUTE WARM AUTOIMMUNE HEMOLYSIS
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Introduction
We report a case of a 68 year-old woman who developed a warm autoimmune hemolytic anemia (AIHA) caused by an autoantibody with anti-D specificity requiring transfusions.

Two weeks after suffering from gastroenteritis of unknown cause, the patient was hospitalized because of symptomatic hemolytic anemia. Laboratory tests revealed hemoglobin of 6.2g/dl, elevated lactate dehydrogenase, reticulocytes and bilirubin as well as decreased haptoglobin. Differential diagnostics excluded any primary hematological disorder such as MDS, CLL, NHL or PNH. The patient denied having taken any medications or nutrient additives in the recent past.

Methods
Standard serological methods for antibody detection and specification were applied (gel-card and tube test; BioRad, Cressier, Switzerland). The Rhesus (Rh) pheno- and genotype were assessed by serotyping (Wadiana®, Grifols, Duedingen, Switzerland) and by molecular typing using PCR-SSP (inno-train GmbH, Kronberg im Taunus, Germany and in-house).

Results
The Rh phenotype was D+, C+/c+, E+/e+ (R1R2) and K-. The direct antiglobulin test (DAT) was positive for IgG1.

The patient’s serum reacted with all RhD positive panel cells in indirect antiglobulin test (IAT) and on papain treated cells. The eluate of patient’s red blood cells reacted the same way as her serum. The autocontrol was positive. Maintained reactions with DTT(dithiothreitol)-treated RhD positive RBC ruled out the specificity of an anti-LW mimicking anti-D specificity. Initial anti-D-titer was very high (1:16384).

Additional genotyping revealed RhD homozygosity (RHD*01/RHD*01) and excluded the most prevalent RhD variants, ensuring the presence of an autoanti-D. At last follow-up, DAT and antibody detection were negative by routine techniques. However, DAT became weakly positive for IgG after incubation with patient’s serum and there were weakly reactions with some papain treated R2R2 cells in IAT. Autocontrol was positive, but the eluate was entirely negative.

Conclusion
Only few cases of autoimmune hemolytic anemia caused by autoanti-D have been published so far. [1,2,3]

In our laboratory, whenever autoanti-D is suspected certain investigations must be performed not to overlook an alloantibody. The overall results confirmed autoanti-D causing the hemolysis. Autoantibodies are generally considered irrelevant for transfusion management but become crucial when causing acute hemolysis. Fortunately, autoanti-D can easily be taken into account by choosing RhD negative red blood cells for transfusion.

In our case, the patient was initially transfused with three RhD negative red blood cell products showing an increase of the hemoglobin value up to 9.8g/dl. Shortly thereafter, when steroid therapy was started and performed over five months, no more blood transfusion was required and the patient improved completely.

References