G antigen was first described as a part of Rhesus (Rh) blood group system and is dependent on the expression of both the RHCE*Ce and RHD alleles [1]. In fact, the accurate differentiation of anti-D, anti-C, and anti-G is essential to decide whether Rh(D) prophylaxis should be given or not in alloimmunized pregnancies [7,8], to diagnose the accurate type of HDFN and to avoid the occurrence of potential Rh alloimmunization or transfusion reaction in D negative patients. The basis of reactivity for G antigen is Ser103, which is encoded by RHD and by the C allele of RHCE genes [5]. Anti-D is the most common antibody responsible for severe hemolytic disease of the fetus and newborn (HDFN), but other Rh antibodies, such as anti-C, anti-E, anti-e, and anti-G, can also cause HDFN [2,5]. Most individuals who do not carry an RHD or RHCE*Ce allele would be expected to be negative for G antigen [[2], [3], [4]], but there are some exceptions, such as lack of G antigen in a very small number of D + C- people or the presence of G antigen in a very small number of D-C- people [3].