CORRESPONDENCE

Importance of Indigenous Lectin Panel in Immunohematology

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Dear editor,

Polyagglutination is the agglutination of altered red blood cells (RBCs) by a large proportion of ABO-compatible adult human sera, regardless of blood group and are usually nonreactive with their autologous serum or serum from cord blood samples [1]. Polyagglutination may be acquired by the exposure of cryptic carbohydrate structures in RBC membranes due to the action of microbial enzymes (TF, Tk, Th, and others) or may be persistently acquired, e.g., Tn syndrome, or may be related to inherited genetic alterations [2]. Lectins are carbohydrate binding proteins which are usually obtained from plant seeds. Some of them can agglutinate RBCs and have been used as alternative to human sera for blood typing. They have a very important role in the investigation of RBC polyagglutination [3]. These carbohydrate antigens responsible for polyagglutination are characterized by specific patterns of interaction with lectins and thus help to classify the type of polyagglutination [1]. Recently we encountered a case of polyagglutination in a patient of uterine adenomyosis requiring hysterectomy and used in-house lectin panels to determine and confirm the type of polyagglutination.

A 36 year old female, multipara, was admitted in a tertiary care hospital of West Bengal with per-vaginal bleeding, abdominal pain due to uterine adenomyosis. She had a hemoglobin (Hb) of 8 g/dl. The consulting

gynaecologist ordered two units of packed red blood cell (PRBC) for transfusion during hysterectomy. She had no previous history of transfusion. Youngest child was of age 3 years. As per previous report from outside her blood group was “O” RhD positive. But during confirmation of her blood grouping report the red cells reacted with anti-AB (2+). Subsequently, anti-A1 lectin showed 4+ agglutination (Table 1). Thus, there was a blood group discrepancy in this case. Direct anti-globulin test (DAT), auto-control and antibody screening by 3-cell panel were negative. Reaction with lectins gave the suspicion of polyagglutination. Thus, both major and minor cross match were done with O RhD positive unit. While major cross match was compatible, minor showed incompatibility. Two units of compatible, buffy coat reduced “O” RhD positive PRBCs were issued to the patient for transfusion. Transfusion was uneventful. Further investigations were done to confirm polyagglutination [1]. Six samples of fresh normal “AB” group adult sera were tested with the patient red cells at both 22 and 4 °C. Three samples of group “O” red cells were used as controls. Since the primary hospital lacked the lectin panel and cord blood at that time, the sample was sent to a super-speciality hospital of Kolkata and further work up was done there. The patient RBC was tested with three samples of “AB” group cord sera in the same manner at 22 and 4 °C. The sera study from the two institutes confirmed the presence of polyagglutination (Table 2). The indigenously prepared “in-house” lectin panel was used to detect the type of polyagglutination [4]. The agglutination pattern with the various lectins was suggestive of Tn polyagglutination [1, 3] (Table 3). Papain treated polyagglutinable cells of the patient showed reduced agglutination strength (weak + to +) with the adult sera used. This finding also supported the presence of Tn polyagglutination [1].

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Table 1 ABO discrepancy of patient sample

Red cells	Forward typing					Reverse typing			Interpretation
	Anti-A	Anti-B	Anti-A, B	Anti-A ₁	Anti-H	A ₁ cell	A ₂ cell	B cell	
Normal sample	0	0	0	0	++++	++++	++++	++++	Group “O”
Patient sample	0	0	++ (mf)	++++	++++	++++	++++	++++	Discrepancy

mf mixed field

Table 2 Confirmation of polyagglutination

Red cells	Adult sera (AB group) (6 samples)						Cord sera (AB Group) (3 samples)		
	1	2	3	4	5	6	1	2	3
Normal “O” sample 1 (22 and 4 °C)	0	0	0	0	0	0	0	0	0
Normal “O” sample 2 (22 and 4 °C)	0	0	0	0	0	0	0	0	0
Normal “O” sample 3 (22 and 4 °C)	0	0	0	0	0	0	0	0	0
Patient (22 °C)	++	++	++	++	+	++	0	0	0
Patient (4 °C)	+++	+++	+++	++	++	+++	0	0	0

Table 3 Detection of polyagglutination type using in-house lectin panel

Red cells	Dolichos biflorus	Arachis hypogaea	Glycine max	Salvia sclarea
Normal “O” sample 1	0	0	0	0
Normal “O” sample 2	0	0	0	0
Normal “O” sample 3	0	0	0	0
Patient ^a	+++	0	++	++

^a Agglutination pattern suggests Tn polyagglutination

Tn polyagglutination is caused by a mutation in the hematopoietic tissue and is considered permanent and irreversible [4]. Here also, it was probably a permanent mutation.

With the advent of monoclonal anti-sera for blood grouping and introduction of component therapy, thus abolishing minor cross-matching, the detection of polyagglutination has almost stopped. Polyagglutination itself is a very rare condition. However, routine use of anti-AB reagent in group “O” patients had led to identification of polyagglutination in this patient. The patient was anemic due to per-vaginal bleeding from adenomyosis. Except for the Hb and hematocrit, the other hematological and biochemical parameters of the lady were normal. She was instantly managed with compatible buffy-coat reduced PRBCs. Lectin study was suggestive of Tn polyagglutination, which was further supported by the enzyme test. Most blood banks in India lack commercial lectin panels because of cost and procurement difficulty. It can be developed indigenously using standard operating protocols as per the study by Das SS [5].

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Walker PS (1998) Polyagglutination. In: Harmening DM (ed) Modern blood banking and transfusion practices, 3rd edn. Jaypee Brothers Medical Publishers (P) Ltd., New Delhi, pp 433–444
2. Beck ML (2000) Red blood cell polyagglutination: clinical aspects. Semin Hematol 37:186–196
3. Bercher ME (2005) Methods section 2: red cell typing. In: American Association of Blood Banks edition. Technical manual, 15th edn, AABB, Bethesda, pp 743–744
4. Dahr W, Uhlenbruck G, Bird GW (1974) Cryptic A-like receptor sites in human erythrocyte glycoproteins: proposed nature of Tn-antigen. Vox Sang 27:29–42
5. Das SS (2015) In-house preparation of lectin panel and detection of Tn polyagglutination. Asian J Transfus Sci 9:102–103