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RESEARCH ARTICLE

DETECTION OF BOMBAY PHENOTYPE AND TRANSFUSION CHALLENGES IN BLOOD BANKING

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ABSTRACT

Background: Blood transfusion is an essential part of modern health care. Used correctly, it can save life and improve health. However, the detection and transfusion challenges in Bombay phenotype have focused particular attention.
Objective: To investigate meticulously the serological characteristics and secretor status of Bombay phenotype and transfusion management in patients.
Setting and Design: This was a descriptive study carried outat a tertiary care hospital in Belgavi, Karnataka over a period of 6 years.
Methods: A total of 27, 4361 samples including donors and patientswere tested for blood grouping. To confirm the Bombay phenotype, anti-H lectin was used in forward grouping followed by Adsorption Elution Technique.
Results: Eleven Bombay phenotypes in 27, 4361 (0.004%) were detected in the entire study, and out of them six were patients who required blood transfusion and remaining five were donors.
Conclusion: This study shows the prevalence of Bombay phenotypeassociated with consanguineous marriage, an important risk factor in southern India. The therapeutic challenges arethe arrangement of compatible blood units and management by alternate techniques.

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INTRODUCTION

Karl Landsteiner truly opened the doors of blood banking with his discovery of the first human blood group system, ABO in 1901. This marked the beginning of the concept of individual uniqueness defined by the RBC antigens present on the RBC membranes. In 1952 a rare blood group was discovered by Dr Y. M.Bhende *et al*, the Bombay phenotype in Bombay (now Mumbai), India. It represents the inheritance of a double dose of the h gene, producing the very rare genotypehh. As a result, the ABO genes cannot be expressed, and ABH antigens cannot be formed, since there is no H antigen made in the Bombay phenotype (Bhende *et al*., 1952). The RBCs of the Bombay phenotype (Oh) do not react with the anti-H lectin (Ulexeuropaeus), unlike those of the normal group O individual, which react strongly with anti-H lectin. Bombay serum contains anti-A, anti-B, anti-A,B, and anti-H. Unlike the anti-H found occasionally in the serum of A1 and A1B individuals, the Bombay anti-H can often be potent and reacts strongly at 37°C. It is an IgM antibody that can bind complement and cause RBC lysis. Transfusing normal group O blood (with the highest concentration of H antigen) to a Bombay recipient (anti-H in the serum) would cause immediatecell lysis. Therefore, only blood from another Bombay individual will be compatible and can be transfused to a Bombay recipient. ABH substance is also absent in saliva.When family studies demonstrate which *ABO* genes are inherited in the Bombay phenotype, the genes are written as superscripts (OhA, OhB, OhAB) (Denise M. Harmening, 2008). The present study, aimed at to studyand manage the rare Bombay (Oh) blood group in patients and donors visiting our institution.

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MATERIALS AND METHODS

This is a descriptive study carried out at a tertiary care hospital in Belgavi, covering its rural and urban population, regarded as a top notch hospital of North Karnataka. The study was carried out in department of Transfusion medicine (Blood Bank); both the recipients and donors were included in the study over a period of six years (2012-2017). Anti-A, anti-B, anti-AB,and anti-H was used for forward reaction and agglutination of plasma with A, B and O (H) red cells (reverse reaction) were also tested for the presence or absence of antibodies in the serum. Confirmation of Bombay phenotype: To confirm the Bombay phenotype, anti-H lectin was used in forward groupingfollowed byadsorption elution technique. To check the secretory status of the Bombay blood group, inhibition test was

performed. Fresh saliva of these patients were collected, boiled, centrifuged, and the supernatant used for testing. In two tubes marked one and two, two drops each of anti-H lectin was taken. Saliva and normal saline (each 100 µl) were taken in each tube, mixed well and incubated. To this, 50 µl of 5% cell suspension of known 'O' red cells were added, mixed, incubated, and centrifuged. All the procedures were done according to AABB and DGHS Technical Manual (Brecher, 2005; Saran, 2003). All relatives who volunteered to participate in the study were checked for the presence of Bombay phenotype.

RESULTS

A total of 27, 4361 blood grouping were carried out in our blood bank, over a period of six years (2010 -2016). Only 11 Bombay phenotypes in 27, 4361 (0.004%) were detected in the entire study, and out of them six were patients who required blood transfusion and remaining five were donors. In Absorption Elution Technique, there was no agglutination with A or B cells confirming it to be Bombay phenotype. Saliva testing of these cases by inhibition assay showed agglutination and proved them to be nonsecretor of H antigen. History of consanguineous marriage was observed in all the cases. Bombay blood group could not be detected in any other relatives who participated in our study.

DISCUSSION

The prevalence of Bombay phenotype in our donors accounted 0.001 % (5 of 27, 4361), and 0.002 % (5 of 27, 4361) among patient population, with an overall prevalence of 0.004 % (11 out of 27, 4361). All cases were hospital based samples; random population screening was not done. The prevalence is also related to high rate of consanguineous marriage, this holds evident in many published literatures as our hospital is situated in Karnataka, the southern part of India where consanguineous marriages are more prevalent and in the neighboring states like Andhra Pradesh, Tamil Nadu, and Maharashtra (Balgir, 2005). In the general population the prevalence of Bombay blood group is about 1 in 10,000 individuals in India and 1 per 1,00,000 individuals in Europe, although in some places of Bombay the prevalence of this phenotype is as high as 0.01 %. The Bombay phenotypes were also detected in Japan, Malaysia, Thailand and Sri Lanka (Talukder *et al*., 2014). Bombay phenotype is an inherited autosomal recessive trait. Underlying molecular defect shows Fucosyltransferase, an enzyme responsible for expression of H antigen, encodes two different genes known as FUT 1 (H gene) and FUT 2 (Se gene). Homozygosity defective mutation of both FUT 1 and FUT 2 genes causes lack of H(hh) antigen and non-secretor (se,se) expression leading to Bombay phenotype. Due to this recessive genetic homozygous expression, consanguineous marriages result in Bombay phenotype (Mallick *et al*., 2015). In our study all the patients and donors gave the history of consanguineous marriages.

Hence the custom of consanguineous marriages should be avoided and awareness regarding the same should be strictly emphasized. Transfusion management of patients with Bombay phenotype is a challenge; this can be achieved by avoiding unnecessary blood transfusion to the patients and treat the underlying cause. It must be emphasized that rare blood group registries must be made mandatory in all the transfusion centers. At our center a separate register is maintained for all the rare blood group donors.

A case of acute hemolytic transfusion reactions due to mismatch blood unit was noted in our study who had received O Rh D positive blood from other hospital. This is common in hospitals where most of the centers do not imply anti-H sera to detect Bombay phenotype.M Sheetal *et al* reported a case acute hemolytic transfusion reaction due to lack of proper blood grouping methods (Malhotra *et al*., 2014). There is need of uniform testing standards and include both forward grouping, reverse grouping with O cells and anti-H sera routinely, so that group matched blood is transfused safely. However Bombay phenotype can safely receive fresh frozen plasma, platelets and cryoprecipitate from any group (Bhar (Kundu) *et al*., 2015). Bombay phenotype in pregnant females leads to production of anti-H which can cause hemolytic disease of newborn (Jain *et al*., 2012). In the present study, a 26- year old female patient with Bombay phenotype had normal delivery without any complication. Three of our patients were managed by autologous transfusion. Autologous (self) transfusion is the donation of blood by the intended recipient. Autologous blood transfusion may reduce the transfusion-associated mortality by 70% and another advantage is that there is increase in erythropoiesis. Immunologic and viral infectious complications have not been reported with transfusion of autologous blood (Dos Santos *et al*., 2016). Nirmala *et al* have reported a case of CABG procedure which had 1000ml blood loss and was immediately transfused with one unit of autologous blood intraoperative (Jonnavithula *et al*., 2013). Postoperatively, the patient received one more unit of autologous blood and other from his brother with similar blood group. B Sudeshna *et al* and P Shio *et al* have also documented reports on autologous transfusion in the management of Bombay phenotype patients successfully (Bhar (Kundu) *et al*., 2015; Priye *et al*., 2015).

Similar to one of our case , three cases reported by S Manisha *et al* have been managed to operate successfully by acute normovolemichemodilution (ANH), post-operative was uneventful till their discharge. Acute normovolemiche modilution is a blood conservation technique that entails the removal of blood from a patient shortly after induction of anesthesia, with maintenance of normovolemia using crystalloid and/or colloid replacement. The blood is infused into the patient during or shortly after the surgical procedure. ANH is available, cost effective for suitable patients and reduces blood viscosity (Shrivastava *et al*., 2015). Bombay phenotype individuals are prone to leukocyte adhesion deficiency II; these patients have high leucocyte count and recurrent infections (Balgir, 2005). M Sujata *et al* had a patient in their study with history of recurrent but infrequent respiratory infections, which was reported as insignificant (Mallick *et al*., 2015). Cryopreservation can be recommended in all the Bombay phenotypes. If the blood banks can borrow or exchange rare blood units in times of need, a lot of problems related to rare blood groups can be sorted. This is only possible if each blood bank has a large number of committed regular voluntary donor's (Jonnavithula *et al*., 2013). Newer research with stem cells are coming up to produce a universal blood group in vitro, thus enabling cellular replacement therapies, this is still in trial and need approval (Priye *et al*., 2015).

Conclusion

It is important to identify Bombay phenotype in emergent situations, because any othertype of blood can have lethal effects on the recipient. It is also recommendedthat individuals with Bombay phenotype should get all their family members

andrelatives tested for the blood group, and also register themselves withleading blood banks so that in case of emergency they can be contacted. In the absence of blood donor registry, transfusion management of patients needing immediate surgery can be challenging, hence rare blood group donor register should be mandated in all the leading blood banks.

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