A2B Blood Group Without Anti-A1 Lectin Antibodies in a Child With an Enzymopathy Hemolytic Disease

Fadi Busaleh 1, Dunya Bu-Izran 2, Zainab Alhajji 3, Rawya Qahtan 4, Abdulatif Alnaaim 5, Haider Alnofaily 6, Salah Almohammed 3

1. Pediatrics, Maternity and Children Hospital (MCH), Al-Ahsa, SAU 2. Medicine, King Faisal University, Al Hofuf, SAU 3. Pediatric Medicine, Maternity and Children Hospital (MCH), Al-Ahsa, SAU 4. Family Medicine, Ministry of Health, Al-Hasa, SAU 5. Medicine, Medical School of Olsztyn, Olsztyn, POL 6. Pediatrics, Ministry of Health Holdings, Al Hufof, SAU

Corresponding author: Fadi Busaleh, fadi.busaleh@gmail.com

Abstract

Generally, within the ABO blood group system, the AB group is subdivided into two subtypes, A1B and A2B, with the A2B subtype considered to be the rarest and the A1B subtype the most common. Given that the A2B subtype is the rarest one, its presence is associated with many challenges. In this report, we present the case of a child with a chronic hemolytic disease with the A2B blood group but without anti-A1 lectin antibodies, as well as the challenges encountered.

Introduction

Packed red blood cell (PRBC) transfusion is a modality used in the management of severe anemia to restore the body's oxygen-carrying capacity to meet the body's metabolic demands [1]. In general, the compatibility of a transfusion is determined by blood group systems. These blood group systems are numerous, reaching up to 36 systems, among which there are a major system and a minor system [1]. The ABO blood group system is a primary system that determines the initial compatibility in blood transfusion medicine. It consists of four main groups and other minor groups. In this report, we present the case of a child with an A2B blood group and acute hemolytic anemia, as well as the challenges encountered [2-3].

Case Presentation

A two-year-old boy was admitted to the Maternity and Children Hospital, Al-Ahsa, Saudi Arabia, with acute hemolytic anemia secondary to glucose-6-phosphate dehydrogenase (G6PD) deficiency. The patient presented with a progressive pallor and red-colored urine associated with a decrease in activity for one day. This episode was preceded by the ingestion of fava beans two days prior to presentation. On examination, the patient was pale and lethargic but not jaundiced. He was also tachycardic (145 bpm) and had a mid-systolic murmur at the upper-left sternal border (grade 3) with no radiation or thrill and with an unremarkable systemic examination. Initial laboratory investigations revealed a decrease in the levels of hemoglobin (6.0 g/dL) with hemoglobinuria (Table 1). Therefore, the patient required a red blood cell transfusion because of his symptomatic anemia and critical levels of hemoglobin. Crossmatching prior to transfusion revealed that the child has an A2B blood group with positive RhD. Therefore, he was transfused with 15 mL/kg compatible type B RhD+ blood group PRBCs to avoid developing anti-A antibodies. After the transfusion, the patient stabilized hemodynamically and his hemoglobin levels increased back to 8 g/dL. His urine also became clear within a day of admission, indicating the cessation of hemolysis. Therefore, he was discharged on the second day on 1 mg of folic acid once daily for a month, to be followed up by a pediatric hematologist.

| Complete Blood Count | Test          | Result       | Reference range |
|----------------------|---------------|--------------|-----------------|
| White blood cell count | 18.6 × 10^3 mg/dL | 3–14 × 10^3 mg/dL |
| Hemoglobin           | 6.0 g/dL      | 11.1–12.6 g/dL |
| Platelets            | 316 × 10^3    | 150–350 × 10^3 |

How to cite this article

Busaleh F, Bu-Izran D, Alhajji Z, et al. (December 29, 2021) A2B Blood Group Without Anti-A1 Lectin Antibodies in a Child With an Enzymopathy Hemolytic Disease. Cureus 13(12): e20815. DOI: 10.7759/cureus.20815
**Mean corpuscular volume** 82 fL 77–87 fL

**Reticulocyte count** 7.94% 0.5%–1.5%

**Blood Chemistry Tests**

| Test                  | Result  | Reference range     |
|-----------------------|---------|---------------------|
| Serum sodium          | 135 mmol/L | 133–152 mmol/L    |
| Serum potassium        | 4.8 mmol/L | 4.1–5.3 mmol/L    |
| Serum calcium          | 2.02 mmol/L | 2.12–2.52 mmol/L  |
| Serum magnesium        | 0.95 mmol/L | 0.74–0.99 mmol/L  |
| Serum chloride         | 98 mmol/L | 98–115 mmol/L      |
| Serum phosphate        | 1.05 mmol/L | 0.8–1.5 mmol/L   |
| Blood glucose          | 91 mg/dL | 74–106 mg/dL       |
| Blood urea nitrogen    | 4.87 mmol/L | 1.70–8.30 mmol/L  |
| Creatinine             | 23.46 μmol/L | 49–115 μmol/L    |
| Total bilirubin        | 1.4 mg/dL | 0.2–1.0 mg/dL     |
| Direct bilirubin       | 0.34 mg/dL | 0.00–0.02 mg/dL  |
| Alkaline phosphatase   | 229 units/L | 46–116 units/L   |

**Hematological work up**

| Test                  | Result    | Reference range    |
|-----------------------|-----------|--------------------|
| ABO blood group       | A2B       | A,B and O          |
| RhD blood group       | Positive  | Positive or Negative |
| Direct Combs test     | Negative  | Negative           |
| G6PD enzyme activity  | Deficient | Normal             |
| HbA1                  | 97.3%     | 97.8%–97.8%        |
| HbA2                  | 2.7%      | 2.2%–3.2%          |
| Hemoglobin H preparation | Negative | Negative           |
| Anti-A antibodies     | Negative  | Negative           |
| Anti-B antibodies     | Negative  | Negative           |
| Urine analysis        |           |                    |
| Red blood cells       | 0         | <5 RBC/hpf         |
| White blood cells     | 0         | <5 WBC/hpf         |
| Hemoglobinuria        | +2        | Negative           |

**TABLE 1: Laboratory findings observed in the patient**

G6PD: glucose-6-phosphate dehydrogenase; HbA1: hemoglobin A1; HbA2: hemoglobin A2

**Discussion**

Since the discovery of the ABO blood group system early in the 20th century by Karl Landsteiner, its antigen compatibility has been considered central for decisions regarding transfusion adaptability. In general, according to the oligosaccharide antigens present on the cell membrane of red blood cells, the ABO blood group system is divided into four main groups: A, B, AB, and O. Two other subgroups are also rarely observed: A2 and A2B. The rarest of these subtypes is A2B, which is present in only 1% of the world’s population [3]. Moreover, around one-third of A2B blood group carriers have spontaneously formed anti-A
antibodies [4]. However, even though our patient is an A2B blood group carrier, he did not have anti-A antibodies.

In blood transfusion medicine, the AB blood group is considered a universal recipient, whereas the O blood group is considered a universal donor [5]. According to Saboor et al., transfusion between individuals with an identical blood group is the best choice. However, because of the rarity of the A2B blood group and the difficulty of finding a compatible match, the O blood group can be used instead [3]. Since our patient had a blood group mimicking the AB blood group but had no anti-A or anti-B antibodies, this allowed us to use either type A or B blood for transfusion. However, we chose type B blood because he may develop a risk of forming anti-A antibodies (anti-A1 lectin antibodies) later on [4].

Generally, hemolytic anemia secondary to a G6PD deficiency is a benign disease that can be controlled by avoiding triggering factors, such as oxidative stress. Special dietary habits should be followed, including the avoidance of fava beans and their products, as they are considered the main triggering factor for hemolysis in G6PD-deficient patients. It is also important to avoid oxidative agents, such as sulfa drugs, or some types of toilet deodorants (e.g., naphthalene balls), which may induce hemolysis [6]. Since our patient was G6PD deficient, we extended our blood group antigen matching to include minor blood groups. This was done to avoid subsequent alloimmunization with recurrent PRBC transfusions [7].

Conclusions
Blood transfusion is a life-saving procedure with a high risk of complications. Its compatibility is determined by multiple antigens, most importantly the ABO blood group system, with its four main types. Therefore, it is important to have better knowledge of the compatibility and complications of transfusion that may be encountered, especially for patients with chronic hemolytic diseases who require recurring transfusions.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References
1. Storry JR, Castilho L, Chen Q, et al.: International society of blood transfusion working party on red cell immunogenetics and terminology: report of the Seoul and London meetings. ISBT Sci Ser. 2016, 11:118-22. 10.1111/voxs.12280
2. Cooling L: ABO, H, and Lewis blood groups and structurally related antigens . Technical Manual. 18th Edn. Fung M, Grossman B, Hillyer C (ed): AABB, Bethesda (MD); 2014. 290-315.
3. Saboor M, Zehra A, Hamali HA, Halawani AJ, Moharki AA, Madkhali AM, Abdullah S: Prevalence of A2 and A2B subgroups and anti-A1 antibody in blood donors in Jazan, Saudi Arabia. Int J Gen Med. 2020, 13:787-90. 10.2147/ijgm.S272698
4. Shastry S, Bhat S: Imbalance in A2 and A2B phenotype frequency of ABO group in South India . Blood Transfus. 2010, 8:267-70. 10.2450/2010.0147-09
5. Harmening DM: Modern Blood Banking & Transfusion Practices. 7th edn. Davis (ed): 2019, Philadelphia, PA; 119-148.
6. Richardson SR, O’Malley GF: Glucose 6 Phosphate Dehydrogenase Deficiency . StatPearls [Internet], Treasure Island (FL); 2021.
7. Heinrichs KF, Howk N, Mazel DS, et al.: Providing ABO-identical platelets and cryoprecipitate to (almost) all patients: approach, logistics, and associated decreases in transfusion reaction and red blood cell alloimmunization incidence. Transfusion. 2012, 52:635-40. 10.1111/j.1537-2995.2011.03529.x