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Title: A young woman with hematuria and Intra-uterine fetal demise: Hemolytic transfusion reaction in a rare Bombay blood group phenotype

INTRODUCTION

The Bombay Blood Group, discovered in Mumbai in 1952, is an extremely rare blood type with the absence of A, B, and H antigens and the presence of anti-A, anti-B, and anti-H antibodies. Often misidentified as O group in routine testing, reverse grouping is essential for accurate detection. This phenotype results from a mutation in the H gene, leading to the inability to produce the H antigen. Transfusion with incompatible blood types can trigger acute hemolytic reactions, presenting as hematuria, fever, and cola-colored urine. Pregnant individuals with the Bombay Blood Group face heightened risks due to bleeding complications and limited donor availability. This case highlights a rare instance of **acute hemolytic transfusion reaction due to misidentification of Bombay Blood Group, resulting in intrauterine demise.**

CASE SUMMARY

Patient Profile:
A 25-year-old primigravida, 40 weeks + 6 days pregnant, presented with complaints of abdominal pain and cola-colored urine (hematuria) for one day. She was referred from a district hospital where she was being managed for anemia and severe preeclampsia.

On Admission:

- **Vitals:** Pallor (2+), PR 100/min, BP 162/110 mmHg, afebrile, SpO_2 99% (room air).
- **Clinical Findings:** Dark red, cola-coloured urine with reduced output (100 mL/day), uterus term-sized, no fetal heart sounds detected (confirmed by USG).
- **Provisional Diagnosis:** Severe preeclampsia, intrauterine fetal demise (IUFD), moderate anemia.
- **Lab Findings:** Acute kidney injury (AKI), hemolysis (low hematocrit, raised LDH).

Key Discovery:
Disproportionate hematuria prompted further investigation, revealing the patient's **Bombay blood group phenotype**, confirmed through reverse crossmatching. History indicated a prior transfusion with mismatched blood (O Rh-positive) at the district hospital, leading to **acute hemolytic transfusion reaction (AHTR)**.

Final Diagnosis:
Primigravida with 40 weeks + 6 days pregnancy, IUFD, severe preeclampsia, moderate anemia, Bombay blood group, and AHTR.

Management and Outcome:

1. **Medications:** Antihypertensives, magnesium sulfate prophylaxis.
2. **Obstetric Care:** Failed induction of labor led to an emergency cesarean section, delivering a 3.2 kg stillborn male.
3. **Renal Support:** Due to worsening kidney function, dialysis was initiated on Day 4 postpartum.

4. Postpartum Course: Gradual improvement in renal parameters; patient discharged in satisfactory condition.

Follow-up:
The patient remains under regular nephrology follow-up

Table: Investigations at admission

Parameters	Values at admission	Reference ranges
Hemoglobin (g/dL)	8.9	12-16
Hematocrit (%)	27.5	35-50
Mean corpuscular volume (fL)	81.6	78-96
Mean corpuscular haemoglobin (pg)	26.6	27-33
White blood cell count (x 10^9/L)	19.4	4-11
Platelet count (x 10^9/L)	153	150-450
Total bilirubin (mg/dL)	1.57	0.3-1.2
Direct bilirubin (mg/dL)	0.58	0-0.2
AST (U/L)	316	0-50
ALT (U/L)	163	0-50
ALP (U/L)	414	50-140
LDH (U/L)	2112	140-280
Creatinine (mg/dL)	1.38	0.4-1.4
Serum urea (mg/dL)	42.9	10-45

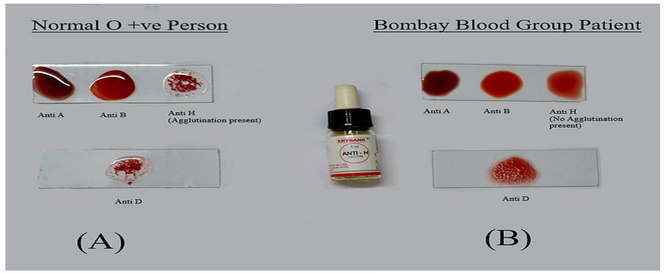
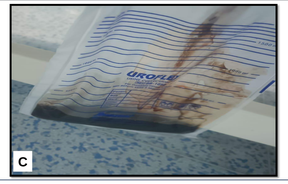


Fig A- Cola Coloured urine in Urobag



DISCUSSION

The Bombay blood group, a rare blood type lacking ABH antigens, can be misidentified as O group without proper testing [5]. Genetically inherited as an autosomal recessive trait on chromosome 19 (19q13.3), the phenotype (Oh) results from the absence of H antigen due to fructosyl transferase deficiency [6, 7]. Individuals with the Bombay phenotype can only receive blood from donors with the same phenotype, making transfusions challenging, especially during emergencies or pregnancy. The para-Bombay phenotype, a variant with weak A or B antigen expression, also lacks H antigen [8]. Prevalence is higher in western and southern India, with Maharashtra accounting for 62.6% of recorded cases, often linked to consanguineous marriages [9].

Pregnant individuals with the Bombay phenotype face unique risks, including hemolytic disease of the fetus and newborn (HDFN), postpartum hemorrhage, and transfusion reactions. Early planning for autologous blood donation is critical [10]. In this case, a misdiagnosed Bombay blood group led to acute hemolytic transfusion reaction, intrauterine fetal demise, and acute kidney injury. Similar cases emphasize the importance of precise blood typing and multidisciplinary management to prevent complications.

CONCLUSION

In summary, the Bombay blood group phenotype may appear similar to the O blood group due to the absence of A and B antigens in blood typing.

However, individuals with the Bombay blood group produce strong anti-H antibodies, which make them incompatible with standard O-type blood transfusions. They can only receive blood from other Bombay blood donors.

This case underscores the critical importance of accurate diagnosis of the Bombay blood group in pregnant females.
Additionally, family screening, genetic counselling, and the establishment of a local Bombay blood donor registry are essential steps for managing this rare condition and ensuring the availability of compatible blood for therapeutic use

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