International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Gaikwad P et al. Int J Reprod Contracept Obstet Gynecol. 2022 Oct;11(10):2872-2873
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20222495

Case Report

A rare case report of haemolytic uremic syndrome in post caesarean section

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Received: 29 July 2022
Revised: 13 September 2022
Accepted: 14 September 2022

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ABSTRACT

Haemolytic uremic syndrome (HUS) is a clinical syndrome characterised by progressive kidney failure associated with microangiopathic haemolytic anemia and thrombocytopenia. Women may be genetically predisposed to develop HUS which gets aggravated by pregnancy. We presented a case of 25 year old G2p1l1 with h/o 9 months amenorrhoea was admitted in active labour, underwent emergency caesarean section for non-progression of labour. Postoperatively patient had decreased urine output with unexplained thrombocytopenia with deranged renal function tests and increased serum LDH with normal coagulation profile and no any evidence of foci of infection for which HUS was suspected. Patient was treated with hemodialysis, plasmapheresis and other supportive medical treatment.

Keywords: Haemolytic uremic syndrome, Postoperative oliguria, Microangiopathic haemolytic anemia and thrombocytopenia

INTRODUCTION

HUS is characterised by microangiopathic haemolytic anemia, thrombocytopenia, renal failure, fever and altered sensorium. It is a disastrous haemolytic disease characterized by diffuse endothelial damage and platelet consumption and affects one out of every 25,000 pregnancies.[1] Early and correct diagnosis and timely management are crucial to improve outcome.

CASE REPORT

A 25 year old G2p1l1 with previous ptvd was admitted on 20 March 2022 with h/o 9 months ammenorrohea in active labour to labour room. On per abdominal examination uterus was full term, cephalic, head engaged, adequate contraction. On per vaginal examination cervix dilatation was 4-5 cm, 50-60% effaced, artificial rupture of membrane done, liquor was clear. Patient was shifted for emergency LSCS for non progression of labour. On post operative day 3 patient had decreased urine output (200 ml since 12 hours), injection lasix 20 mg was given but there was no increase in urine output. She developed hypotension and tachycardia and blood investigation suggestive of thrombocytopenia (41000), creatinine deranged (4.4). Patient was shifted to ICU and started on injection noradrenaline with urine output in tube and BP-80/50 mmhg.

Other investigations

Ultrasonography (abdomen+pelvis): Bilateral increased renal cortical echogenicity suggestive of acute renal parenchymal changes; urine routine microscopy: pus cells: 2-3, red cells: 15-20, budding yeast+, mobile bacteria+; blood culture report was suggestive of presence of *E. coli* organism; LDH-1506 (U/l).

After multidisciplinary consultation, the patient was treated with a combination of plasmapheresis (2 times) and intermittent haemodialysis (3 times), also received intermittent infusion of type B Rh positive 1 packed cell

volume and 8 units of fresh frozen plasma was given injection meropenem, furosemide and sodamint. The patient's condition was improved eventually and discharged on 12 and followed up every 2 weeks.

Table 1: Preoperative versus postoperative blood investigations.

Investigations	Preoperative values	Postoperative values (day 3)
Hb (g%)	11.0	8.3
Platelet count (mcl)	180,000	41,000
White blood cells (per cmm)	10,000	26,000
Urea (mg/dl)	64	155
Creatinine (mg/dl)	0.8	4.4

DISCUSSION

HUS is loosely defined by the presence of microangiopathic haemolytic anemia and renal impairment, most common variant is shiga-toxin producing *E. coli*. Pregnancy associated atypical HUS is a systemic disease associated with uncontrolled alternative complement pathway activation, the complement regulation can be congenital or acquired. The affected patients often have a low C3 and a normal C4 levels characteristic of alternative pathway activation. Factor H deficiency, the most common defect, has been linked to families with aHUS. Factor H competes with factor B to prevent the formation of C3bBb and acts as a factor I, which proteolytically degrades C3b. More than 70 mutations of the factor H gene have been identified. Most are missense mutations that produce abnormalities in the C-terminus region, affecting its binding to C3b but not its concentration, other mutations result in low levels or the complete absence of the protein. Deficiencies in other complement-regulatory proteins, such as factor I, factor B, membrane cofactor protein (CD46), C3, complement factor H-related protein 1 (CFHR1), CFHR3, CFHR5 and thrombomodulin, have also been reported. Finally, an autoimmune variant of a HUS has been discovered. DEAP HUS is often associated with a deletion of an 84-kb fragment of the chromosome that encodes for CFHR1 and CFHR3. The autoantibody blocks the binding of factor H to C3b and surface-bound C3 convertase.

Damage to endothelial cells is the primary event in the pathogenesis of haemolytic uremic syndrome. The cardinal lesion is composed of arteriolar and capillary microthrombi and red blood cell fragmentation. HUS is classified into 2 categories, depending on whether it is associated with shiga-like toxin or not that is typical and atypical type. Atypical HUS can be sporadic or familial and the disease may occur overall without a gastrointestinal prodrome and these patients have a poor outcome among them 50% may progress to end stage renal disease or irreversible brain damage.[2] Most cases of pregnancy-associated HUS occur during postpartum period. Alexandra et al found 76% cases presenting during post-partum period and Fakhouri et al observed 79% incidence in postpartum period.[3,4]

Management includes plasmapheresis, high dose steroids, packed cell transfusions and complement inhibitors. Twice daily plasma exchange with rituximab may be effective in refractory cases. Plasma exchange is effective in certain types of atypical HUS as it replaces complement-regulatory proteins. Eculizumab is a monoclonal antibody to C5 that is approved for use in atypical HUS which has been shown to abort MAHA and improve renal function.[5]

CONCLUSION

A typical HUS in pregnancy is a challenge to diagnose it at early stage. Timely diagnosis and multidisciplinary management is always lifesaving. One may suspect HUS in postpartum period if a patient suddenly develop thrombocytopenia with raised LDH and deranged KFT and can be managed by plasmapheresis mainly along with other supportive measures.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Saad AF, Roman J, Wyble A, Pacheco LD. Pregnancy-associated atypical hemolytic- uremic syndrome. Asia J Pharmaceut Report. 2016;6(1):125.
2. Sangani MA, Airao BB, Pandya MR. A rare case presentation of haemolytic uremic syndrome in post caesarean section. IJOGR. 2015;2(3):176-8.
3. Fakhouri F, Roumenina L, Provot F, Sallee M, Caillard S, Couzi L, et al. Pregnancy-associated haemolytic uremic syndrome revisited in the era of complement gene mutations. Am Soc Nephrol. 2010;21(5):859-67.
4. Khatod N, Mukharjee S, Malviya V. Postpartum atypical haemolytic uremic syndrome:a rare case report. Int J Reprod Contracept Obstet Gynecol. 2020;9(11):4710-2.
5. Siegler RL. Management of haemolytic-uremic syndrome. J Pediatr. 1988;112(6):1014-20.

Cite this article as: Gaikwad P, Deshmukh J, Godbole V, Pandey S. A rare case report of haemolytic uremic syndrome in post caesarean section. Int J Reprod Contracept Obstet Gynecol 2022;11:2872-3.