International Journal of Reproduction, Contraception, Obstetrics and Gynecology
*Joshi S et al. Int J Reprod Contracept Obstet Gynecol. 2026 Jan;15(1):338-341*
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20254308

**Case Report**

# Against all odds: successful maternal outcome after massive abruption with intrauterine fetal demise and severe coagulopathy

**Surbhi Joshi*, Manisha Gupta**

Department of Obstetrics and Gynaecology, Santosh Medical College Deemed to be University, Ghaziabad, Uttar Pradesh, India

**Received:** 12 November 2025
**Revised:** 10 December 2025
**Accepted:** 11 December 2025

***Correspondence:**
Dr. Surbhi Joshi,
E-mail: surbhijoshi374@gmail.com



## ABSTRACT

A 31-year-old multiparous woman presented with antepartum hemorrhage and intrauterine fetal demise (IUFD), who developed severe DIC (INR 6.0) and acute kidney injury (AKI). Despite multi-organ involvement, the patient made a complete recovery. Early multidisciplinary intervention and aggressive supportive management were the key to survival.

**Keywords:** Abruptio placentae, Disseminated intravascular coagulation, Acute kidney injury, Maternal morbidity, Multi-organ dysfunction

## INTRODUCTION

Abruptio placentae is defined as premature separation of a normally implanted placenta after 20 weeks of gestation and before delivery of the fetus, leading to significant maternal and fetal morbidity and mortality.[1] Severe cases may be complicated by disseminated intravascular coagulation (DIC), acute renal injury, and multi-organ dysfunction.[2] Prompt diagnosis, resuscitation, and correction of coagulopathy are essential to improve survival outcomes.[3] This report highlights a rare instance of successful maternal recovery following massive abruption complicated by DIC and acute kidney injury (AKI) requiring dialysis.

## CASE REPORT

A 31-year-old female, gravida 4 para 3 with one living issue, presented to the emergency department with bleeding per vaginam and decreased fetal movements.

Initial evaluation revealed pallor, restlessness, hypovolemic shock (BP 90/60 mmHg, thready pulse, low $SpO_2$). Per abdomen: uterus term-size, tender, increased tone, fetal parts not palpable. Per vaginam: cervix uneffaced, os closed, bleeding present. Fetal heart sounds absent. The patient was resuscitated with crystalloids and started on noradrenaline infusion. Cross-matched blood was arranged.

Past obstetric history included two spontaneous abortions and a previous term IUFD following caesarean section. The only live issue was through VBAC five years ago.

### *Investigations*

Ultrasound confirmed IUFD with features suggestive of abruptio placentae. Pre-operative labs: Hb 4.5 g/dl, Platelets 47,000/µl, PT 36.5 s, INR 6.0, Urea 49.2 mg/dl, Creatinine 2.28 mg/dl.

**Table 1: Trend of laboratory investigations during hospitalization.**

| | Normal range | Unit | 23/7 | 24/7 | 25/7 | 26/7 | 27/7 | 28/7 | 29/7 | 30/7 | 31/7 | 8/1 | 8/2 | 8/3 | 8/4 | 8/5 | 8/8 | 8/10 | 8/11 | 8/12 | 13/8 | 14/8 | 15/8 | 16/8 | 17/8 | 19/8 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Hb** | 11.5-15 | g/dl | 5.2 | 4.5 | 6.4 | 8.3 | 9.2 | 8.8 | 9.8 | 10.9 | 9.5 | 8.5 | 8.8 | 9.3 | 9 | 8 | 7.7 | 8.4 | 8 | 8.4 | 7.7 | 7.9 | 7.7 | 7.6 | 8.5 | 8 |
| **PC** | 150-450 | x10^3/ microl | 101 | 47 | 50 | 53 | 68 | 117 | 109 | 95 | 176 | 167 | 194 | 293 | 378 | 407 | 539 | 491 | 441 | 437 | 439 | 475 | 562 | 489 | 528 | 485 |
| **TLC** | 4-11 | x10^3/ dl | 28.62 | 11.22 | 20.01 | 21.63 | 20.41 | 18.33 | 21.76 | 18.23 | 25.16 | 26.21 | 21.49 | 25.51 | 27.57 | 21.66 | 9.41 | 7.84 | 5.45 | 3 | 2.1 | 2.46 | 2.6 | 3.19 | 4.93 | 6.49 |
| **Urea** | 15-45 | mg/dl | 29.8 | 49.2 | 53.9 | 42.3 | 51.5 | 102.1 | 80.1 | 126.3 | 95.8 | 72.7 | 131.6 | 83.9 | 128.4 | 94.5 | 180 | 112.6 | 133 | 69.2 | 82.5 | 94.2 | 106.2 | 117.5 | 52.4 | 40 |
| **Creat** | 0.57-1.11 | mg/dl | 1.4 | 2.26 | 3.64 | 2.82 | 2.08 | 4.26 | 3.29 | 4.28 | 3.94 | 3.12 | 4.13 | 3.24 | 4.34 | 3.7 | 6.37 | 4.5 | 5.21 | 3.43 | 4.25 | 5.24 | 5.85 | 6.28 | 3.35 | 2.2 |
| **BUN** | 5-20 | mg/dl | 13.9 | 22.99 | 25.19 | 19.77 | 24.07 | 47.71 | 37.43 | 59.02 | 44.77 | 33.97 | 61.5 | 39.21 | 60 | 44.16 | 84.11 | 52.6 | 62.43 | 32.3 | 38.55 | 44.02 | 49.63 | 54.91 | 24.5 | 35.4 |
| **PT** | 11.7-15.3 | sec | 78.6 | 36.5 | 16 | | 14.4 | 16.5 | | | | | | | | | | | | | | | | | | |
| **INR** | 0.9-1.2 | sec | 5.82 | 2.7 | 1.19 | | 1.07 | 1.22 | | | | | | | | | | | | | | | | | | |
| **Bil T** | 0.2-1.2 | mg/dl | 2.68 | 0.93 | 0.91 | 0.74 | 0.86 | 0.82 | 0.82 | 0.74 | 1.35 | 1.01 | 0.59 | 0.62 | 0.63 | 0.52 | 0.34 | | | | | | | | | |
| **Bil D** | 0-0.9 | mg/dl | 0.54 | 0.38 | 0.36 | 0.25 | 0.32 | 0.31 | 0.35 | 0.22 | 0.35 | 0.29 | 0.2 | 0.26 | 0.2 | 0.18 | 0.08 | | | | | | | | | |
| **Bil ID** | 0-0.5 | mg/dl | 2.14 | 0.55 | 0.55 | 0.49 | 0.54 | 0.51 | 0.47 | 0.52 | 1 | 0.72 | 0.39 | 0.36 | 0.43 | 0.34 | 0.26 | | | | | | | | | |
| **SGOT** | 5-34 | U/l | 41.5 | 92.8 | 171.1 | 71.7 | 34.7 | 21.8 | 26.6 | 21.4 | 31.5 | 35.7 | 31.8 | 45.2 | 58.7 | 65.1 | 67 | | | | | | | | | |
| **SGPT** | 0-55 | U/l | 21.7 | 42.9 | 119.8 | 88.8 | 49.1 | 28.4 | 18 | 11.7 | 11.5 | 14.1 | 17.5 | 24.3 | 33.1 | 41 | 50 | | | | | | | | | |

***Intra-operative findings***

Emergency LSCS was performed under general anesthesia. A stillborn baby in cephalic presentation was delivered. Peritoneum was adherent to anterior uterine wall and cervix; bladder was pushed up. A retroplacental clot (~500 ml) and couvelaire uterus were noted (Figure-1).

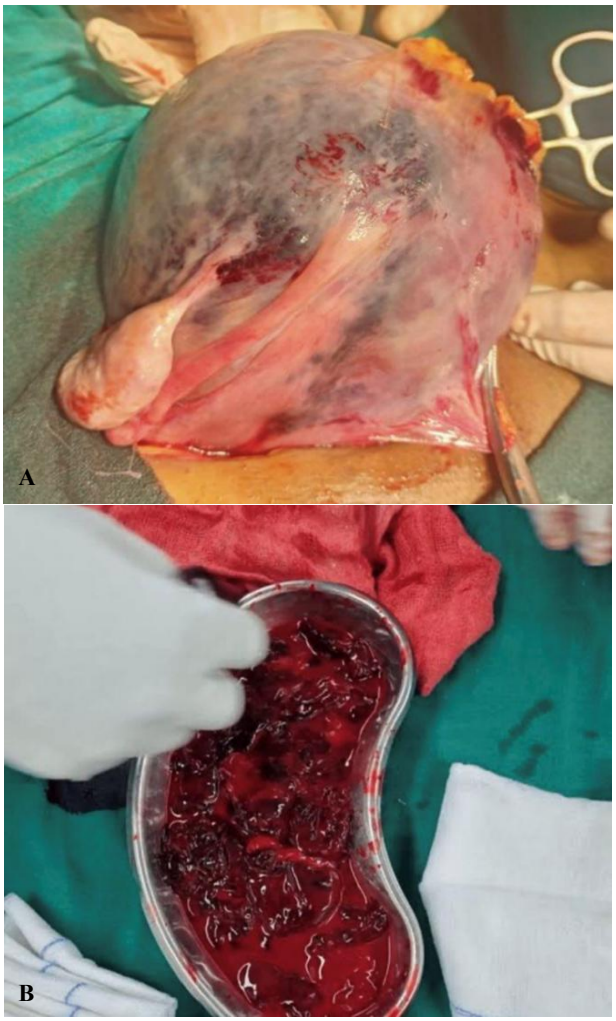


**Figure 1 (A and B): Intra-operative findings demonstrated a couvelaire uterus with a large retroplacental clot.**

Atonic PPH managed medically and surgically. Hemostasis was achieved with difficulty. Estimated blood loss exceeded 2 l with significant third-space losses. A drain was inserted with frank blood for 2 days. Intra/postoperative transfusion included 7 PRBC, 14 FFP, and 10 cryoprecipitates.

***Post-operative course***

The patient was shifted to ICU with invasive monitoring and mechanical ventilation. On Day 1, INR decreased to 2.7 after transfusion. She remained oliguric with generalized edema and deranged LFT/KFT. Drain output was blood-stained for 48 hours. POCUS showed no intra-abdominal collection or hematoma.

***Renal and hematological management***

*Diagnosis*

Abruptio placentae with DIC, AKI, and MODS. Dialysis was initiated on Day 2 and continued for 8 sessions during hospitalization. Cumulative transfusion: 7 PRBC, 14FFP, 10 cryoprecipitates. Gradual improvement was observed in urine output and renal function.

*Laboratory trend*

Day 1 Cr 2.28 mg/dl → Day 10 Cr 4.87 → Day 16 Cr 7.27 → Day 18 Cr 4.21 (Table 1). Edema decreased and hemodynamics stabilized.

***Outcome***

The patient was shifted from ICU to nephrology HDU on Day 18. She continued dialysis on an outpatient basis and was discharged in stable condition with Hb 8 g/dl, platelets $4.85\times10^5$/µl, Urea 40.7 mg/dl, and Creatinine 2.2 mg/dl.

## DISCUSSION

Massive placental abruption is a well-documented cause of obstetric disseminated intravascular coagulation (DIC) due to release of large quantities of tissue thromboplastin from the retroplacental clot into maternal circulation, leading to widespread microvascular fibrin deposition and consumption of clotting factors and platelets. This hypercoagulable-to-hypo-coagulable transition is the hallmark of obstetric DIC, often precipitating catastrophic haemorrhage and multi-organ dysfunction if not managed promptly.[4] In the index case, the patient presented in hypovolemic shock with severe DIC (INR 6.0) and early acute kidney injury (AKI), a triad associated with high maternal mortality. According to the WHO systematic review, abruptio placentae complicated by coagulopathy carries a maternal mortality rate of up to 20% in low-resource settings.[5] The survival in this case underscores the critical importance of early recognition, rapid blood component therapy, and multidisciplinary coordination. Massive transfusion protocols emphasising a balanced ratio of PRBC:FFP:platelets:cryoprecipitate have been shown to improve outcomes in obstetric haemorrhage.[3] Our patient received early and aggressive correction (7 PRBC, 14 FFP, and 10 cryoprecipitates), which restored hemostasis and reversed coagulopathy within 24 hours. This aligns with current RCOG and ACOG guidelines recommending immediate component therapy in DIC rather than awaiting lab correction before surgery.[2] Renal involvement in abruption-related DIC is typically multifactorial—ischemic injury from hypoperfusion, cortical necrosis from microthrombi, and tubular necrosis secondary to shock.[6] Early initiation of dialysis in this patient prevented progression to irreversible cortical

necrosis, a key factor in renal recovery, as also emphasised in previous studies.[7-9] Overall, this case reinforces key learning points: early and aggressive transfusion therapy, simultaneous surgical intervention without awaiting coagulation normalisation, and timely renal support are vital to survival in massive abruption with DIC and MODS. The patient's complete recovery demonstrates how structured multidisciplinary teamwork between obstetricians, anaesthesiologists, intensivists, and nephrologists can convert an almost fatal obstetric catastrophe into a success story.

## CONCLUSION

Even in catastrophic cases of abruption with DIC, AKI, and MODS, timely surgical intervention, massive transfusion, and early dialysis can be life saving. This case emphasizes the importance of individualized, multidisciplinary management.

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

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**Cite this article as:** Joshi S, Gupta M. Against all odds: successful maternal outcome after massive abruption with intrauterine fetal demise and severe coagulopathy. Int J Reprod Contracept Obstet Gynecol 2026;15:338-41.