VOLUME - 10, ISSUE - 05, MAY- 2021 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper | Obstetrics & Gynaecology

UNUSUAL CASE OF DIC AT 11 WEEKS GESTATION WITH MISSED ABORTION: A CASE REPORT

Dr. Shravani Mohite Junior Resident, MGM Women's and Children's Hospital, Navi Mumbai.

Dr. Arya Bodhe* Senior Resident, MGM Women's and Children's Hospital, Navi Mumbai.
*Corresponding Author

ABSTRACT Disseminated intravascular coagulation is defined as systemic activation of blood coagulation, which causes microvascular thrombi due to fibrin deposition causing multiple organ dysfunction. This coagulation cascade ultimately contributes to accelerated fibrinolysis causing severe bleeding. Hence, a patient with DIC can present with a simultaneously occurring thrombotic and bleeding problem. This is usually diagnosed with combination of clinical examination and thorough blood examinations. Treatment includes removal of the primary coagulopathic source, rapid coagulant replacement and vigorous blood transfusion in cases of massive bleeding types of DIC.

KEYWORDS : Disseminated intravascular coagulation, first trimester, missed abortion

INTRODUCTION

We report the case of a 22-year-old woman who presented to be in disseminated intravascular coagulation due to first trimester missed abortion following which dilatation and curettage was done with complete symptomatic recovery.

Abortion is defined as spontaneous or induced termination of pregnancy before 20 weeks gestation. Almost 80% spontaneous abortions occur within 12 weeks of gestation, of which, approximately half are euploid abortions and the other half occur as a result of aneuploidy/ chromosomal abnormality.

Missed abortion is described as non-viable products of conception that have been retained in utero for days or weeks. It usually causes vaginal bleeding, pain in lower abdomen and may be associated with passage of clots/tissue.

The incidence of DIC in pregnancy ranges from 0.03 to 0.35% and is more commonly associated with mid-trimester or late second trimester abortions. Clinically, it presents as defective haemostasis, persistent bleeding following any surgical procedure, generalized oozing from abdominal wall layers or retroperitoneal space or the incision sites for LSCS/ emergency hysterectomy. Prothrombin time (PT) and activated partial thromboplastin time (aPTT) are standard coagulation tests in addition to platelet and RBC count and analysis of blood smear. Also, plasma fibrinogen and fibrin levels can be informative. Bioassay is an excellent diagnostic tool for DIC.

CASE

A 22-year-old married female, came to casualty of MGM Hospital, with complaints of bleeding per vaginum and passage of clots since the past 1 day.

Patient was primigravida with 11.3 weeks gestation with respect to 10.2 weeks of scan. Patient did not have any known co-morbidities, no history of any addictions or did not gave any history of physical trauma/any history of intentional attempts for self-termination of pregnancy.

Patient was febrile, tachycardic and normotensive on evaluation. Abdomen was soft, non-tender. Per vaginal examination revealed bleeding with minimal clots with uterine size approximately 10 weeks with cervical os admitting tip of finger.

Following investigations were sent on admission:

Blood group: 'A' positive

CBC:	Hb - 9.5 g/dl	(12-16 g/dl)
	TLC - 6,100/mm^3	(4,000-11,000 /mm^3)
	Plt - 1.06X10^3/mm^3	(1.5-4.5 X10^3 /mm^3)
LFT:	T.Bili - 3.58 mg/dl	(upto 1.2 mg/dl)
	D.Bili - 2.90 mg/dl	(<0.3 mg/dl)
	SGOT - 335.3 units/l	(5-40 units/l)
	SGPT – 253.9 units/l	(7-56 units/l)
	ALP – 213.8 units/l	(45-145 units/l)
	T.Protein – 6.6 g/dl	(6-8.3 g/dl)
	Albumin – 3.6 g/dl	(3.4-5.4 g/dl)
	Globulin – 3.0 g/dl	(2-3.5 g/dl)
RFT:	Urea – 27.2 mg/dl	(7-20 mg/dl)
	Creatinine – 1.3 mg/dl	(0.6-1.2 mg/dl)
	Uric acid – 5.6 mg/dl	(2.7-7.3 mg/dl)
	BUN – 12.7mg/dl	(3.3-9.3 mg/dl)
Electrolytes:	Na^+ – 119 mEq/l	(135-145 mEq/l)
	K^+ - 3.1 mmol/l	(3.6-5.2 mmol/l)
	Ca^{+2} – 4.01 mmol/l	(8.5-10.5 mmol/l)
Coagulation profile:	PT – 30 seconds	(11-13.5 seconds)
	INR – 3.3	(<1.1)
CRP:	Reactive – 1:16	

Serology: HIV, HBsAg, VDRL – Negative

Ultrasonography was repeated:

Uterus:	Size – Gravid uterus with gestational sac seen. Echo – Poor choriodecidual reaction seen. Endo – Foetus noted with no evidence of cardiac activity. CRL – 45.7 mm ~ 11.3 weeks
Ovaries:	Both ovaries normal
Fluid:	Mild free fluid in interbowel pelvis

IMPRESSION: Missed Abortion

Medicine reference was given in view of deranged LFT, S.creatinine and PT/INR levels and to obtain opinion regarding fitness for taking up for dilatation and curettage.

Inj. Vitamin K 20mg IV was administered stat with continual of IV antibiotics. ECG was done and was reported to be normal. 2 units of FFP were issued.

After explaining high risks, delayed recovery and the need for MICU admission to the patient and relatives, patient was taken up for the said procedure under cover of FFP on flow under total intravenous anaesthesia.

Intraoperatively, patient's vitals as follows:

Pulse:	130/m
BP:	110/80mmHg;
SpO_2:	99-100% on O_2
CVS:	S_1S_2+
RS:	AEBE, clear

Postoperatively, patient was bleeding copiously despite making attempts to achieve haemostasis. Decision was transfusing 1 unit PRBC and 2 more units of FFP was made.

Patient was administered:
- Inj. Pitocin 20 IU IV
- Inj. Tranexamic acid 1 gm IV
- Inj. Ethamsylate 1 gm IV
- Inj. Prostodin 125 mcg deep IM
- Tab. Misoprostol 600 mcg per rectal
- Tab. Misoprostol 400 mcg sublingual
- Inj. D25 IV slowly
- Foley's catheterisation was done

Patient was transfused total 1-pint PRBC and 4 pints of FFP. Patient was monitored intensively in post-operative period for bleeding, urine output, abdominal circumference. Daily vaginal packing with roller gauze was done till bleeding almost subsided. Patient had one more fever spike and hence fever profile was sent which was reported to be negative. Higher antibiotics were started. CBC, LFT, electrolytes, S.creatinine, PT/INR and inflammatory markers were investigated. Medicine review reference was done in view of progressive deranging LFT and electrolytes.

Patient was administered:
- Inj. Monocef 1 gm IV 12 hrly
- Inj. Metronidazole 100cc IV 8 hrly
- Inj. Tranexamic acid 1 gm IV 8 hrly
- Inj. Ethamsylate 1 gm IV 8 hrly
- Inj. Vitamin K 10mg IV od X 3 days
- Inj. KCl 40 mEq in 500ml NS stat dose over 4-6 hrs
- Inj. Calcium gluconate 10 ml in 10ml NS 8 hrly
- Tab. Shelcal-HD P/O bd
- Syp. Kesol 30ml P/O tds

Medicine review was done daily and needed investigations were repeated as and when advised. Hence, Inj. KCl 60 mEq in 500ml NS stat dose was repeated. Inj. $MgSO_4$ 2gm in 100 ml NS od X 3days was added. On occurrence of one more fever spike, COVID RT-PCR was tested which was reported to be negative.

On day 4 of the procedure, patient had no bleeding/ spotting per vaginum and her deranged parameters of blood investigations also showed improving trend. All IV medications were shifted to oral.

Investigation Chart:

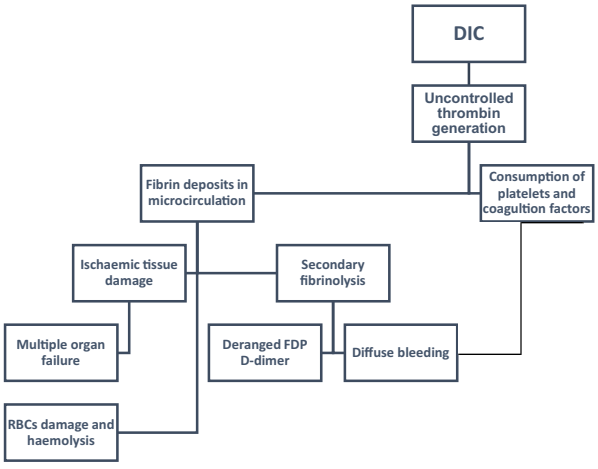
	Day 1 (pre OT)	Day 1 (post OT)	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Hb	9.5	7.3	8.8					10
TLC	6100	6500	5500					4400
Plt	1.06	1.80	1					2.46
T.Bili	3.58	3.78		3.25		3.25		2.88
D.Bili	2.90	2.15		2.56		2.15		1.93
SGOT	335.3	202.8		177.6		10.92		81
SGPT	253.9	135.7		124.1		116.1		93.1
ALP	213.8	170.8		151.1		263.3		270.4
T.Prot	6.6	5.0		6.0		5.9		5.5
Albumin	3.6	2.9		2.8		2.0		2.8
Globulin	3.0	2.1		3.2		3.1		2.7
Urea	27.2	17.3				15.2		
Creatinine	1.3	0.9		1.0		0.8		0.7
Uric acid	5.6	4.6				3.1		
BUN	12.7	8.0				7.1		
Na^+	119		126	127	128	129		130
K^+	3.1		2.3	2.6	3.80	4.6		3.8
Ca^{+2}	4.01		4.25	4.25	4.45	4.6		9.1
PT	30 sec	15	13					
INR	3.3	1	1					
aPTT	48.4							
CRP	1:16					1:4		
HIV	Negative							
HBsAg								
VDRL								
HCV		Negative						
HAV								
HEV								
LDH		805						
Ammonia		169.5						
PS for MP		Negative						
RMA								
Dengue								
WIDAL								
COVID-RTPCR			Negative					
URM: EC/PC				5-6/ 3-4				
UACR				83.84				
CK-MB				30				
Trop-T				13.34				
PS for cell type						Normal		
Retic count						2.9%		

Patient had no fresh complaints and complete S. electrolyte level correction was noted with markedly improvement in LFT levels. Patient was clinically and vitally stable. Hence, was discharged after reviewing it with medicine.

DISCUSSION

Disseminated intravascular coagulation (DIC) is a life threatening though reversible complication in pregnancy. It causes microvascular thrombosis which may reduce hemoperfusion to vital organs causing organ failure and uncontrolled bleeding. It may arise from variety of obstetrical conditions such as acute post-partum haemorrhage, eclampsia, pre-eclampsia, retained non-viable products of conception, septic abortion, placental abruption, amniotic fluid embolism, acute fatty liver of pregnancy.

Disseminated intravascular coagulation (DIC) is a pathologic disruption of the finely balanced process of haemostasis. It is characterized by systemic activation of blood coagulation, which results in generation and deposition of fibrin and formation of microvascular thrombi in the small blood vessels throughout the body (thrombosis) and activation of plasmin (fibrinolysis and haemorrhage), eventually leading to multiple organ dysfunction. Because widespread clotting depletes the platelets and clotting factors that are needed to control bleeding, excessive bleeding often occurs.



Normal pregnancy is a prothrombotic state. Pregnant females have a marked increase in some coagulation factors,

impaired endogenous anticoagulation, reduced fibrinolysis, and increased platelet reactivity. The shift in the balance between the haemostatic and fibrinolytic systems serves to prevent excessive haemorrhage. Amniotic fluid has been shown to activate coagulation in vitro leading to leakage of thromboplastin like material from the placental system which is responsible for occurrence of DIC.

Clinical manifestations of DIC are directly proportional to the magnitude of imbalance in haemostasis and to the underlying cause, or to both. The most common findings are defective haemostasis, persistent bleeding following any surgical procedure, generalized oozing from abdominal wall layers or retroperitoneal space or the incision sites for LSCS/ emergency hysterectomy. In acute DIC, haemodynamic complications and shock are usually common. The mortality ranges from 30-80% depending on the severity of DIC, the underlying cause and the age of the patient.

The investigations which include coagulation profile (PT, aPTT), FDP markers, platelet count and blood smear analysis should be assessed and repeated over a period of 6-8 hours since an initially mild abnormality may change dramatically in patients with severe DIC. A subsequent reduction of platelet count is sensitive to DIC. Prolongation of PT and aPTT is seen in 50-75% cases and 50-60% cases respectively. Blood smear shows presence of schistocytes (fragmented RBCs).

DIC is usually seen as a complication following mid-trimester/ late second trimester abortions or in puerperium. Disseminated intravascular coagulopathy (DIC) is a very rare complication in first trimester and can be due to undiagnosed hereditary thrombophilias, Von Willebrand disease, placenta percreta in first trimester, APLA. Inherited abnormalities of coagulation proteins that are essential for the pathophysiology of DIC, could contribute to the risk for DIC. Slowly evolving DIC produces mild thrombocytopenia, normal to minimally prolonged PT and PTT, normal or moderately reduced fibrinogen level, increased plasma D-dimer level. Rapidly evolving DIC results in more severe thrombocytopenia, more prolonged PT and PTT, rapidly declining plasma fibrinogen level, high plasma D-dimer level.

Management of DIC includes treatment of the underlying cause supportive therapy through blood and blood product transfusion and intravenous fluids. Immediate correction of the cause is the priority which includes broad-spectrum antibiotic, treatment of suspected sepsis, evacuation of the uterus in cases of RPOC/abruptio placentae). If treatment is effective, DIC usually subsides quickly. In cases of severe bleeding, or if there is an urgent need for surgery, then adjunctive replacement therapy is indicated which includes platelet concentrates to correct thrombocytopenia, cryoprecipitate to replace fibrinogen, fresh frozen plasma to increase clotting factors and anticoagulant levels. Heparin is useful in the treatment of slowly evolving DIC. Heparin usually is not indicated in rapidly evolving DIC with bleeding or bleeding risk. An exception is in women with a retained non-viable foetus and evolving DIC with a progressive decrease in platelets, fibrinogen, and clotting factors. In these patients, heparin is given for several days to control DIC, increase fibrinogen and platelet levels, and decrease excessive coagulation factor consumption. Heparin is then stopped and the uterus evacuated.

CONCLUSION

This case is an unusual manifestation of DIC in early pregnancy following a missed abortion, which was managed conservatively with blood and blood product transfusion and showed considerable improvement in clinical presentation as well as lab parameters. Bleeding was stopped completely and patient was stable vitally. On progressive investigations, coagulation profile of the patient appeared to be within normal limits with improvement in inflammatory markers.

Further evaluation in view of aetiology could not be done due to non-affordable condition of the patient. Patient was advised for pre conceptional counselling for her subsequent pregnancy.

REFERENCES

1. Disseminated intravascular coagulation in pregnancy: insights in pathophysiology, diagnosis and management; Erez O, Mastrolia SA, Thachil J.Am J Obstet Gynecol. 2015 Oct;213(4):452-63. doi: 10.1016/j.ajog.2015.03.054. Epub 2015 Mar 31.PMID: 25840271.
2. Disseminated intravascular coagulation (DIC) during pregnancy: Clinical findings, etiology, and diagnosis; Michael A Belfort, Charles J Lockwood, D L Hepner Dec 2020.
3. Placenta Percreta in First Trimester Leading to Disseminated Intravascular Coagulopathy (DIC): A Rare Case Report. Bansal CL, Gupta J, Asthana D, Kayal A.J Clin Diagn Res. 2015 Apr;9(4):QD03-4. doi: 10.7860/JCDR/2015/9338.5794. Epub 2015 Apr 1.PMID: 26023604.
4. Genetic Risk Factors for Disseminated Intravascular Coagulation; D. W. Sommeijer and P. H. Reitsma. Madame Curie Bioscience Database
5. Disseminated Intravascular Coagulation (DIC) By Joel L. Moake, MD, MSD Manual; Jan 2020